Refiguring Old Age:
Shaping Scientific Research on Senescence, 1900-1960

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To Cassiel

Abstract

This dissertation traces the origin and the development of gerontology, the science of aging, in the United States and the United Kingdom. I argue that gerontology began to be formed as a multidisciplinary scientific field in the two countries from the 1900s to the 1950s. Unlike earlier scholars who had thought that the aging of the whole body was caused by the inevitable decline of an unknown critical factor, such as "vital heat," gerontologists of the twentieth century conceived aging as a contigent phenomenon whose rate and mode differed in distinct portions of the body. They also introduced systematic experimental approaches in their investigation which had seldom been employed in the study of aging before the twentieth century. Furthermore, with these new ideas and methodologies, gerontologists established their research field in which scholars from diverse disciplines could work in a cooperative manner, including biologists, physicians, psychologists, and social scientists. Amid the Great Depression, which threatened the very survival of the elderly, these multidisciplinary scholars formed professional societies and research institutes for more organized study of aging. But gerontology followed different paths of development in America and Britain due to their distinctive political and cultural conditions, academic traditions, and leading scholars' social and academic status. While British scientists of aging were struggling with various problems related to funding, professional recognition, and the recruitment of scholars interested in aging, American gerontologists came to have relatively ample and stable sources of financial support and an expanding network of national and local organizations. By analyzing this difference and tracing the beginnings of the new concepts and approaches, this dissertation aims at explaining the birth of a multidisciplinary scientific field within historical contexts.

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Abbreviations for Archival Sources

ABH Albert Baird Hastings Papers, National Library of Medicine, Bethesda, Maryland

AC Alexis Carrel Papers, Georgetown University Archive, Washington, D.C.

ACF Alexander Comfort Papers, University College London Archive, London, England

AEC Alfred E. Cohn Papers, RG 450C661, Rockefeller University Archive, Rockefeller Archive Center, Sleepy Hollow, New York

AF Alexander Fleming Papers, British Library, London, England

CMM Clive Maine McCay Papers, Cornell University Archive, Ithaca, New York

ECD Edward Charles Dodds Papers, Royal College of Physicians Library, London, England

EVC Edmund Vincent Cowdry Papers, Bernard Becker Medical Library, Washington University, St. Louis, Missouri

FB Frederic Bartlett Papers, University of Cambridge Archive, Cambridge, England

GBS George Bernard Shaw Papers, British Library, London, England

GS Gladys Sperling Papers, Cornell University Archive, Ithaca, New York

HHD Henry Hallett Dale Papers, Royal Society Archive, London, England

LBM Lafayette B. Mendel Papers, Yale University Archive, New Haven, Connecticut

LID Louis I. Dublin Papers, National Library of Medicine, Bethesda, Maryland

LKF Lawrence K. Frank Papers, National Library of Medicine, Bethesda, Maryland

MLN Manuscripts of Lord Nuffield, Nuffield College, University of Oxford, Oxford, England

NARA National Archives and Record Administration, College Park, Maryland

NAUK National Archives of the United Kingdom, London, England

NF Nuffield Foundation Archive, London, England

NWS Nathan W. Shock Papers, Bentley Historical Library, University of Michigan, Ann Arbor, Michigan

PBM Peter Brian Medawar Papers, Wellcome Library for the History and Understanding of Medicine, London, England

RAF Ronald A. Fisher Papers, University of Adelaide Archive, Adelaide, South Australia, Australia

RF Rockefeller Foundation Archive, Rockefeller Archive Center, Sleepy Hollow, New York

RP Raymond Pearl Papers, BP 312, American Philosophical Society Library, Philadelphia, Pennsylvania

RR Robert Robinson Papers, Royal Society Archive, London, England

UOA University of Oxford Archive, Oxford, England

WBC Walter B. Cannon Papers, HMS c40, Countway Library of Medicine, Boston, Massachusetts

WDM William deB. MacNider Papers, Manuscripts Department, Wilson Library, The University of North Carolina, Chapel Hill, North Carolina

Introduction

Science and Aging: The Making of a Research Field

On October 9, 1935, the renowned cytologist Edmund Vincent Cowdry (1888-1975) at Washington University wrote a letter to the Josiah Macy, Jr. Foundation with a deep concern about the current state of affairs surrounding old age and the elderly. The Great Depression since 1929 demolished many things which he had taken for granted for years: plenty of jobs for his students and friends, the prospects for a prosperous future, and the strong popular support of natural science. But what was the most urgent for him was the problem of old age. For Cowdry, the elderly, and even the middle-aged, were suffering from age discrimination in the job market and the loss of private pension plans that would support their livelihood. Admittedly, getting a job, or obtaining a new position after being laid-off, was difficult for the young as well as for the old at that time. However, the degree of difficulty was different for aged people who were increasingly feeling that younger people did not view them as productive members of society. While President Franklin D. Roosevelt signed the Social Security Act two months earlier to address this problem, the governmental measure was not enough for Cowdry who thought that it was necessary to do something fundamental for the elderly's active social participation rather than merely providing some money for them. He wrote to Ludwig Kast, president of the Macy Foundation, "The plea 'do make me of some use,' that we all hear [from the elderly], is tragic."[1] What was needed in this situation was not so much "throwing old people a few dollars" as a more systematic and organized approach to the problems of aging whose basis can be created by scientists.[2] "The problem should be of interest," Cowdry claimed, "to biologists, physicians, sociologists, and psychologists."[3]

[1] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.
[2] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.
[3] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.

Indeed, Cowdry noticed the growth of the new biological and biomedical sciences during the past years which, for him, would become a strong foundation for the new science of aging. At the Rockefeller Institute for Medical Research, he had been a junior colleague of Alexis Carrel, a renowned surgeon and Nobel laureate, who showed that senescence and natural death could be "contingent" at the cell level. Cytological research in which Cowdry himself had expertise also revealed that aging occurred at distinct rates in different portions of the body, and studies of protozoa showed that single-celled organisms did not die or age in certain environmental conditions. Furthermore, according to Charles Manning Child, one of the faculty members at the University of Chicago where Cowdry finished his Ph.D., many cells of invertebrates had an ability to "dedifferentiate" into embryonic cells which, consequently, could continue their life without senescence. To Cowdry, who learned the ideal of using biology for human welfare at Chicago, this new research would create the basis of what science could do for the welfare and happiness of the aged.

This dissertation discusses what Cowdry, along with many other scientists at that time, hoped and achieved—the creation of gerontology as a multidisciplinary scientific field studying aging. Whereas philosophical or medical ideas and practices on aging have existed for a long time, gerontology as a field of scientific inquiry is the product of a relatively recent development in Western history. Although it is true that many renowned scholars in the past—including Galen (129-ca. 200), Roger Bacon (1214-1294), Francis Bacon (1561-1626), and Marie François Xavier Bichat (1771-1802)—wrote about aging, few conducted systematic research on the subject. These scholars' mention of aging was usually limited to a small portion of their books devoted to other topics, such as general issues on health and disease as well as various philosophical and medical problems. Moreover, unlike astronomy or physiology which had a long tradition and many signficant research subjects established during and after the ancient Greek era, there were few issues of aging that consistently challenged scholars and led them to produce new solutions. The current dissertation aims at showing how this state gradually changed during the first half of the twentieth century. After the 1900s, gerontology began to be

established as a scientific field studying aging with new ideas, perspectives, and methodologies concerning the phenomenon of senescence.

This study historically traces the formation of gerontology as a scientific field. By "scientific field," I mean an academic arena devoted to a particular kind of scientific activity. Gerontology is a scientific field defined by its research subject, aging. However, a field cannot be created merely by determining its subject, which is a necessary but not sufficient condition for the formation of a field. In fact, while the Russian-French scientist Elie Metchnikoff coined the term, "gérontologie," in 1903 and wrote that it is the science dealing with aging, the field of gerontology was not formed until the 1930s.[4] Basically, the creation of a field needs a group of researchers who actively maintain it. These researchers should spend a significant portion of their time in pursuing a particular kind of study and think that they belong to a certain field. They also need to define what should be properly included in their research problems. Moreover, they have to have their own academic societies as well as professional journals devoted to the research whose boundaries they have settled. Societal endorsement and stable sources of funding are another important component of building and maintaining a scientific field. In this sense, I argue, gerontology emerged as a scientific field during the first half of the twentieth century. This dissertation will discuss how the scientists of aging developed new ideas and approaches on senescence and how they formed multidisciplinary academic organizations, such as the Gerontological Society and the British Society for Research on Ageing. This dissertation will also describe how gerontologists launched their journals devoted to the problems of aging, including the *Journal of Gerontology*, while, at the same time, having several funding agencies—the National Institutes of Health, the Rockefeller Foundation, and the Nuffield Foundation—support their research projects on a long-term basis for comprehensive solutions of the social and biomedical problems concerning senescence.

Interestingly, despite this success, gerontology has been considered marginal in Western society, and this marginality is often mentioned in relation to an essential

[4] Metchnikoff first mentioned "Gérontologie" in Elie Metchnioff, *Études sur la nature humaine: Essai de philosophie optimiste* (Paris: Masson, 1903), p. 386.

character of gerontology, its multidisciplinarity. As chapter five of this dissertation will show in more detail, gerontology has developed into a broad multidisciplinary scientific field consisting of many modern academic disciplines, including biology, medicine, psychology, and the social sciences. As sociologist Stephen Katz has pointed out, however, some people think that gerontology is a "nondiscipline" rather than a multidisciplinary field, because, they argue, the interaction among disciplines constituting gerontology is too weak and infrequent.[5] According to these critiques, there are hardly any conceptual tools or methodologies for producing knowledge shared across disciplinary boundaries, and for this reason some scholars regard gerontology as a "profession" for helping the aged or a mere "juxtaposition of academic specialties."[6] Historian of aging W. Andrew Achenbaum has also been quite cautious in designating the state of the science of aging. According to him, gerontology has remained "a field very much in the formative stages of emergence."[7] Its progress has been quite slow and the "theories of aging remain partial, tentative" despite the consistent efforts to understand aging in scientific terms.[8]

Strangely, the contemporary condition of the subject of gerontology in this seemingly sorry state is not very positive, either. Although the circumstances surrounding the elderly differ widely depending on their gender, race, class, and nationality, the

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[5] Stephen Katz, *Disciplining Old Age: The Formation of Gerontological Knowledge* (Charlottesville and London: University Press of Virginia, 1996), p. 105. On the nature and character of multi/inter/cross-disciplinarity, see Julie Thompson Klein, *Interdisciplinarity: History, Theory, and Practice* (Detroit: Wayne State University Press, 1990).

[6] Katz, *Discipling Old Age*, p. 106. See also David A. Peterson, *Career Paths in the Field of Aging* (Lexington, Mass.: Lexington Books, 1987); R. D. Bramwell, "Gerontology as a Discipline," *Educational Gerontology* 11 (1985), pp. 201-210; Ira S. Hirschfield and David A. Peterson, "The Professionalization of Gerontology," *The Gerontologist* 22 (1982), pp. 215-220.

[7] W. Andrew Achenbaum, *Crossing Frontiers: Gerontology Emerges as a Science* (Cambridge: Cambridge University Press, 1995), p. 13.

[8] Achenbaum, *Crossing Frontiers*, p. 13. Ironically, as several historians have mentioned, the beginning of a large-scale biomedical research on Alzheimer's disease supported by the National Institute on Aging (NIA) of the United States after the 1970s was partially due to the fact that scientists, including NIA director Robert Butler, dissociated the disease from aging about which not many things were known. Redefining Alzheimer's disease as a neurological disorder with specific causes was a better strategy for the survival of the research program. See Patrick Fox, "From Senility to Alzheimer's Disease: The Rise of the Alzheimer's Disease Movement," *The Milbank Quarterly* 67(1989), pp. 58-102; Martha Holstein, "Aging, Culture, and the Framing of Alzheimer Disease," in Peter J. Whitehouse, Konrad Maurer, and Jesse F. Ballenger (eds.), *Concepts of Alzheimer Disease: Biological, Clinical, and Cultural Perspectives* (Baltimore: Johns Hopkins University Press, 2000), pp. 158-180.

elderly in general are seldom preferred in industries, job markets, and other social gatherings despite affirmative action in many Western countries.[9] To explain this, social scientists studying aging during the mid-twentieth century proposed "modernization theory" which postulated that the elderly became increasingly marginalized during the process of modernization and industrialization.[10] Whereas the elderly were venerated and respected in traditional agrarian societies due to their scarcity, wealth as landowners, and symbolic status as sources of wisdom, the development of industrial capitalism substantially lowered their social and cultural status. The change in the mode of production from agriculture to industry, as well as the alteration of the major residential place from rural communities to urban areas, destroyed the elderly's traditional sources of wealth as well as their authority as bearers of accumulated expeience.

Although "modernization theory" has been criticized by many later historians and sociologists for being overly simplistic and ahistorical, similar ideas can still be heard, especially in relation to the role of the science and medicine of aging.[11] The most notable is the argument that "medicalization" of old age during the modern period defined the aged body as inherently pathological and unavoidably declining. According to the scholars who advocate this standpoint, modern medicine broadly defined—from late eighteenth century French clinical medicine to mid-twentieth century geriatrics—gave birth to the discourse of the elderly's inevitable physiological decline and pathological transformations. This discourse, engendered and supported by physicians' and medical researchers' study of aging cells and tissues, had a deep social and polical impact. For example, Katz has argued that modern medicine deprived the aged body of diverse symbolic meanings of former eras and replaced the hopeful prospect for prolongevity

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[9] For a thorough analysis of contemporary age discrimination, see John Macnicol, *Age Discrimination: An Historical and Contemporary Analysis* (Cambridge: Cambridge University Press, 2006).

[10] See, for example, Raymond Grew, "Modernization and Its Discontents," *American Behavioral Scientist* 21 (1977), pp. 298-312; Donald O. Cowgill and Lowell D. Holmes (eds.), *Aging and Modernization* (New York: Appleton-Century-Crofts, 1972).

[11] For criticisms on modernization theory, see Brian Gratton, "The New History of the Aged: A Critique," in David Van Tassel and Peter N. Stearns (eds.), *Old Age in a Bureaucratic Society: The Elderly, the Experts, and the State in American History* (Westport, Conn.: Greenwood, 1986); Katz, *Disciplining Old* Age, pp. 49-69.

with the pessimistic view on progressive degeneration.[12] According to him, this change accompanied broad alterations in social structure and discourse, including the transformation of almshouses, the growth of social surveys, and the introduction of pensions and mandatory retirement, all of which eventually contributed to "disciplining" old age. Laura Davidow Hirshbein has stressed the same point, although her subject is not gerontology but geriatrics, the medical specialty dealing with aged patients. According to her, early American geriatricians redefined the subjects it hoped to treat, the elderly, as a dependent, desexed, and disease-prone group of people.[13] This redefinition of old age by geriatricians, many of whom were gerontologists as well, illustrates how medicine and medical science of the twentieth century contributed to the negative view of old age. Historians Achenbaum, Carole Haber, and Thomas R. Cole have maintained similar standpoints. They have argued that modern biomedical research produced scientific bases that could be used for justifying the marginalizing place of the elderly in industrial capitalism.[14] When rising capitalistic society increasingly displaced the elderly from its sites of production, scientists of aging, by making the senile decline and pathogenesis an

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[12] Katz, *Disciplining Old Age*, pp. 27-48. For a related literature, see Hans-Joachim von Kondratowitz, "The Medicalization of Old Age: Continuity and Change in Germany from the Late Eighteenth Century to the Early Twentieth Century," in Margaret Pelling and Richard M. Smith (eds.), *Life, Death, and the Elderly: Historical Perspectives* (London and New York: Routledge, 1991), pp. 134-164; Daniel Schäfer, "'That Senescence Itself Is an Illness': A Transitional Medical Concept of Age and Ageing in the Eighteenth Century," *Medical History* 46 (2002), pp. 525-548.

[13] Laura Davidow Hirshbein, "'Normal' Old Age, Senility, and the American Geriatrics Society in the 1940s," *Journal of the History of Medicine and Allied Sciences* 55 (2000), pp. 337-362.

[14] W. Andrew Achenbaum, *Old Age in the New Land: The American Experience since 1790* (Baltimore: Johns Hopkins University Press, 1978), pp. 40-45, 110-113; Thomas R. Cole, *The Journey of Life: A Cultural History of Aging America* (Cambridge: Cambridge University Press, 1992), pp. 161-211; Carole Haber, *Beyond Sixty-Five: The Dilemmas of Old Age in America's Past* (Cambridge: Cambridge University Press, 1983), pp. 47-81. Also see Howard P. Chudacoff, *How Old Are You? Age Consciousness in American Culture* (Princeton: Princeton University Press, 1989), p. 59. But Richard Calhoun and Jesse Ballenger have argued that gerontologists did promote more positive outlook on the aged. See Richard B. Calhoun, *In Search of the New Old: Redefining Old Age in America, 1945-1970* (New York: Elsevier, 1978), pp. 69-72, 77; Jesse F. Ballenger, *Self, Senility, and Alzheimer's Disease in Modern America: A History* (Baltimore: Johns Hopkins University Press, 2006), pp. 56-75. Cole, in the conclusion of his book, also argues that gerontologists after the 1960s began to fight against ageism and tried to offer more positive viewpoint on senescence. See Cole, *The Journey of Life*, pp. 227-233. Yet Cole and Ballenger argue that even this "positive viewpoint" contributes to stigmatization of old age. According to them, this positive viewpoint was the other side of the same coin that defined old age according to the norms of industrial capitalism.

undeniable scientific fact, created the rationale for the marginalization of the elderly in society.

The picture that emerges from this brief historiographical survey is clear. The science of aging has contributed to the social isolation of the elderly and the stigmatization of old age amid the growth of modern industrial capitalism, even though its state as a scientific field has still been quite unstable and questionable. According to this view, both the science of aging and the elderly are marginal in modern society, and the aged people's marginalization was furthered by gerontology.

The current dissertation aims at providing a modified view on these issues. I admit that gerontology has not become a unified scientific discipline. I also think that gerontologists have contributed in some measure to the marginalization of the elderly. As a historian of science, however, I feel that the previous studies of gerontology have not based their view on a thorough historical analysis of the published and archival papers of early gerontologists in the United States and the United Kingdom. The scientists' correspondence, articles, and books reveal the changing character of their scientific programs, social ideals, research traditions, and interaction with their patrons and colleagues within cultural and political contexts. My study of these issues has led me to draw a historical picture which is quite different from that depicted by previous scholarship. I argue that the origin and development of gerontology can be traced in much more complex social and academic contexts than the mere "industrialization" of society and the "marginalization" of the elderly. This dissertation analyzes the impact of the development of biomedicine upon research on aging as well as the political and cultural conditions for the growth of gerontology. I also describe the challenges facing the scientists of aging as they built gerontology as a multidisciplinary field, along with their ideals for the welfare and active social life of the aged.

My arguments are fourfold. First, the birth of gerontology was indebted to the new research problems regarding senescence which were created during the development of the biomedical and biological sciences rather than through traditional research on senile decline and its inevitability. Second, the Great Depression that deepened cultural displacement of the aged motivated scientists to build gerontology to provide scientific

bases for the continuous social participation and welfare of the elderly rather than any rationale for disadvantaging them in industries and other social worlds. Third, while gerontology did not become a single discipline or a profession with unified standards and norms, it developed as a broad multidisciplinary scientific field which maintained a reasonable degree of cooperation among the disciplines constituting it. Fourth, the efforts at creating gerontology brought about different results in the United States and the United Kingdom, because of the two countries distinct conditions concerning scientific leadership, fundung, the number of interested scholars, and the social and political environments. The remaining part of this introduction will explain each of these arguments in more detail.

Biomedical and Biological Sciences of Aging

My first argument is that the growth of the biomedical and biological sciences provided new ideas, perspectives, and approaches, including the concepts of local distinctiveness and contingency of aging as well as new experimental methods. This was, I claim, at least a partial departure from the older view since the ancient Greek era which postulated that aging was an inevitable decline of the whole body due to a single critical factor such as “vital heat.” Although such an idea continued to be held in some measure by many scientists and physicians even during the twentieth century, the new concepts and approaches were gradually adopted by an increasing number of biological annd biomedical scholars. I also show how the scientists who created these new ideas and methods influenced later researchers who would build gerontology during the 1930s and the 1940s.

In terms of historiography, this part of my dissertation examines several historians’ assertion that gerontological knowledge contributed to the social displacement of the elderly by highlighting the inevitable decline of their physiological capacities. While this assertion is questionable in many respects, it is probably the most problematic in terms of the misleading impression it delivers about gerontologists’ research focus. The assertion leads us to think that the inevitability of senile decline was early gerontologists’ major study subject. But I claim that what fascinated the scientists of

aging were not such declines in the aged body but the new concepts and practices that emerged through the development of the biological and biomedical sciences, such as the contingency of aging, locally distinct modes of senescence, and experimentalism. I also point out that several major scientists of aging were not really interested in humans' senile changes which might be related to social problems of old age. In fact, there were many scientists who studied nonhuman species' senescence as a significant research subject by itself rather than as models of human aging. For instance, Herbert Spencer Jennings investigated protozoa's and invertebrates' aging, and William Crocker studied the plant's senescence.[15] I will show that these scientists' works contributed not so much to the social displacement of the aged as to the creation of new research problems.

The scientist of nonhuman aging who played the most important role in constructing gerontology was Clive McCay, a professor in the animal husbandry department at Cornell University. By focusing on his works and life, I will show how the development in animal husbandry and the science of nutrition brought about new research programs and experimental approaches in gerontology. McCay was genuinely interested in farm animals' senescence rather than that of humans, and his study of animal nutrition led him to discover the effect of restricted dietary calories on longevity and aging. The latter part of the chapter on McCay discusses how the domain of his research became substantially broadened beyond the field of animal husbandry and became a "boundary object" that connected various disciplines in gerontology including physiology, clinical medicine, pharmacology, psychology, and even dentistry. This broadening will be a subject of my case study of the making of gerontology as a multidisciplinary research field, using the concept of the "boundary object" postulated by historians and sociologists Ilana Löwy, Susan Leigh Star, and James Griesemer.[16]

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[15] Herbert Spencer Jennings was not a scientist who was uninterested in human problems. See, for example, Elazar Barkan, "Reevaluating Progressive Eugenics: Herbert Spencer Jennings and the 1924 Immigration Legislation," *Journal of the History of Biology* 24 (1991), pp. 91-112. When he studied aging, however, he was concerned only about experimental organisms. He even expressed this in his letter to E. V. Cowdry. See Jennings to Cowdry, 28 April 1938, Box 29, Folder 20, EVC. For Crocker's view on plants' aging, see William Crocker, "Ageing in Plants," in E. V. Cowdry (ed.), *Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1939), pp. 1-31.

[16] Ilana Löwy, *Between Bench and Bedside: Science, Healing, and Interleukin-2 in a Cancer Ward* (Cambridge, Mass.: Harvard University Press, 1996), pp. 247-253; Löwy, "The Strength of Loose

The Great Depression, the Elderly, and the Beginning of Gerontology

My second argument is that the Great Depression and the plight of the elderly at that time contributed to the construction of gerontology, which was expected to produce scientific knowledge for the elderly's continued social life and welfare. While the proportion of the elderly in the population had been consistently increasing in the Western world since the late nineteenth century due to improved hygiene, nutrition, and decreased birth rate, it was not until the 1930s that scientists felt that they needed to provide their expertise for the problems of aging as it became a serious social issue. The economic disaster during the 1930s was considered a serious blow to the elderly and even to the middle-aged, since it was thought to enhance age discrimination in job markets and industries and to destroy private pension systems. While the response of the United States federal government to this problem was to pass the Social Security Act in 1935 to make a national pension system,[17] that of scientists including cytologist Edmund Vincent Cowdry was to construct gerontology.[18] From the beginning, one of gerontology's primary aims was to help the aged live a productive and active social life, and this aim survived throughout the early years of the field.

Concepts—Boundary Concepts, Federative Experimental Strategies, and Disciplinary Growth: The Case of Immunology," *History of Science* 30 (1992), pp 371-396; Susan Leigh Star and James R. Griesemer, "Institutional Ecology, 'Translations,' and Boundary Objects: Amateurs and Professionals in Berkeley's Museum of Vertebrate Zoology, 1907-39," *Social Studies of Science*, 1989, 19: 387-420. Also see Peter L. Galison, *Image and Logic: A Material Culture of Microphysics* (Chicago: University of Chicago Press, 1997), pp. 781-844.

[17] Historians have not agreed upon the actual relationships between the elderly's problems and the establishment of the Social Security Act. But most scholars have agreed that the Act was the federal government's response to the problem of old age during the 1930s. See W. Andrew Achenbaum, *Social Security, Visions and Revisions: A Twentieth Century Fund Study* (Cambridge: Cambridge University Press, 1986), pp. 13-37; Achenbaum, *Old Age in the New Land*, pp. 127-141; William Graebner, *A History of Retirement: The Meaning and Function of an American Institution, 1885-1978* (New Haven: Yale University Press, 1980), pp. 181-214; Carole Haber and Brian Gratton, *Old Age and the Search for Security: An American Social History* (Bloomington: Indiana University Press, 1994), pp. 172-185; Macnicol, *Age Discrimination*, pp. 209-223.

[18] The relationship between the Great Depression and the making of gerontology was already briefly mentioned by several scholars. See Gerald J. Gruman, "Introduction," in Gerald J. Gruman (ed.), *Roots of Modern Gerontology and Geriatrics* (New York: Arno, 1979); Bramwell, "Gerontology as a Discipline," p. 208. My published article, which is a shortened version of this chapter, also deals with this issue. See Hyung Wook Park, "Edmund Vincent Cowdry and the Making of Gerontology as a Multidisciplinary Scientific Field in the United States." *Journal of the History of Biology* 41 (2008), pp. 544-553.

This part of my dissertation provides another argument challenging the traditional view expressed by many previous historians on the social place of gerontology. While many earlier historical studies have argued that gerontology highlighted the negative aspects of aging and even justified the elderly's social displacement through scientific means, I will show that early gerontologists had more constructive aims in mind on the place of the aged. In this sense, my dissertation joins recent works in the history of the life sciences which have emphasized more positive sides of biological research, especially in the United States. Whereas previous historians—who were skeptical of early twentieth century scientists' elite visions and agenda of scientific and social progress—tried to illuminate the negative aspects of the biological sciences by showing their close affiliation with conservative politics, social Darwinism, or mandatory eugenic sterilization, Philip Pauly, Gregg Mitman, and other historians of science have recently argued that the relationship between the life scientists and politics was more complex and multilayered. In fact, they have extensively studied how biology in early twentieth century America promoted and was guided by the ideals of democracy, cooperation, and social integration.[19] During my research, I have found that this recent historical view of the place of biological science agrees with the state of early gerontology, which was mostly constituted by life scientists at that time. My dissertation investigates how gerontologists tried to offer the scientific bases on the possibility of the elderly's prolonged health, social participation, and active life and work.

Development of Gerontology as a Multidisciplinary Scientific Field

My third argument is that gerontology grew as a multidisciplinary scientific field in America and Britain despite many challenges and problems that it had to face. While I partially admit several scholars' evaluation of the weak status of gerontology as a

[19] See Philip J. Pauly, *Biologists and the Promise of American Life: From Meriwether Lewis to Alfred Kinsey* (Princeton: Princeton University Press, 2000); Gregg Mitman, *The State of Nature: Ecology, Community, and American Social Thought, 1900-1950* (Chicago: University of Chicago Press, 1992); Sharon Kingsland, "Toward a Natural History of Human Psyche: Charles Manning Child, Charles Judson Herrick, and the Dynamic View of the Individual at the University of Chicago," *The Expansion of American Biology*, pp. 195-230; Kathy J. Cooke, "Duty or Dream? Edwin G. Conklin's Critique of Eugenics and Support for American Individualism," *Journal of the History of Biology* 35 (2002), pp. 365-384.

scientific field, I still claim that it survived as a viable multidisciplinary research field through various strategies and initiatives. Early gerontologists, especially those in the United States, succeeded in encouraging cooperation across various disciplines including biology, medicine, psychology, and the social sciences, although the main core in such cooperation was biological and medical professionals. I argue that the first step in making this multidisciplinary structure of gerontology was taken when Cowdry edited *Problems of Ageing* (1939). He already had much experience in editing several biology textbooks and handbooks for which he invited a number of eminent authors from diverse subfields within biology and medicine. This experience led him to ask many prestigious scientists from a variety of disciplines to write the chapters of *Problems of Ageing*. I will show how these multidisciplinary contributors, with the encouragement of the Russian-British scientist Vladimir Korenchevsky, formed the "American Branch" of the Club for Research on Ageing in 1940, which, in turn, developed into the Gerontological Society in 1945. In Britain, Korenchevsky organized the British Club for Research on Ageing in 1939, which was transformed into the British Society for Research on Ageing (BSRA) in 1947. After the formation of these societies, gerontologists adopted various strategies—especially using their journals and the societies' organizations—to enhance the field's multidisciplinary cooperation, professional maturation, and the general public's support. Even though there were several failed attempts, especially in terms of funding, their struggles to survive as a field came to fruition.

It should be stressed that this analysis of the making of a multidisciplinary field does not include a study of the "professionalization" of gerontology. While some commentators say that gerontology is now a "profession," in the period dealt with in this dissertation gerontologists made little progress toward forming a profession.[20] During the first half of the twentieth century, the science of aging did not meet all the criteria of a profession, including a set of specialized training following a determined curriculum, the recognition of authority in an area by other professions and the general public, and the

[20] Hirschfield and Peterson, "The Professionalization of Gerontology," pp. 215-220.

autonomous decision-making and quality control by peers.[21] But there were some efforts and certain aspects in gerontology that would lead to its professionalization. Bascially, the biomedical scientists of aging shared new ideas and methodologies that were developed during the early twentieth century as well as broader social visions on the place of the elderly in modern society. It is also noticeable that the *Journal of Gerontology*, the first academic periodical in the science of aging, chose articles for publication based on a peer-review system, which was used to impose high academic standards upon the papers in the *Journal*. Yet the kinds of expertise and training constituting gerontology was too diverse and heterogeneous for gerontology to become a profession, and gerontologists' authority as the experts on the problems of aging was not easily recognized by philanthropies, governments, and the general public.

But the scientists of aging who dealt with such initial problems still identified themselves as belonging to a single field, gerontology. In analyzing how this identity arose, this dissertation focuses on the role of biological and medical scientists and physicians. I emphasize the works of biomedical researchers and practitioners because most early gerontologists had academic backgrounds in biological and medical fields. Indeed, previous historical works on gerontology written by Achenbaum and Katz did not study this peculiar composition of early gerontology in detail, and thereby did not reveal the process of the formation of multidisciplinarity in gerontology in historical contexts. I point out that the ideal of multidisciplinary interaction was born among biological and medical researchers who, due to the nature of their specialties, could communicate with one another relatively easily. They could have been satisfied with interaction only among themselves and could have contructed gerontology as a field for biomedical research on aging. This is actually what happened in the birth and development of many biomedical (sub)disciplines which are primarily defined by their subjects, such as immunology and

[21] On professionalization, see, for example, Nathan Reingold, "Definitions and Speculations: The Professionalization of Science in America in the Nineteenth Century," in *Science: American Style* (New Brunswick: Rutgers University Press, 1991), pp. 24-53; George H. Daniels, "The Process of Professionalization in American Science: The Emergent Period, 1820-1860," *Isis* 58 (1967), pp. 151-166; M. P. Crosland, "Development of a Professional Career in Science in France," *Minerva* 13 (1975), pp. 38-57; Dorinda Outram, "Politics and Vocation: French Science, 1793-1830," *The British Journal for the History of Science* 13 (1980), pp. 27-43; R. Steven Turner, "The Growth of Professional Research in Prussia, 1818-1848," *Historical Studies in the Physical Sciences* 3 (1971), pp. 137-182.

virology. Yet gerontologists' shared concerns about the elderly's place and role in society led them, especially those in America, to discuss the problems of old age in broader social contexts during their meetings, and eventually prompted them to invite social scientists, psychologists, and anthropologists interested in aging—who had already been growing in number independently of biomedical scientists of senescence—in their group. This further extended the character of multidisciplinarity in gerontology in the United States, which substantially departed from the more restricted biomedical defintion of "gérontologie" coined by Metchnikoff.[22]

Gerontology in the United States and the United Kingdom

But the situation in British gerontology was different. Although gerontology in Great Britain was also multidisciplinary in some sense, the BSRA was composed mostly of biological and medical scientists. Their multidisciplinarity was defined only *within* biomedicine. This character of the BSRA partially originated from the intention of its founder, Korenchevsky, who did not try to include social scientists in his new organization. In Britain, social scientists of aging formed their own separate professional society, the British Society of Gerontology (BSG), in 1971. The BSRA and BSG maintained a relatively peaceful relationship, perhaps because there were not many substantial academic contacts between them.

There is another important difference between American and British gerontology. Unlike their American counterparts, the British scientists of aging failed to create an active academic field until the late-1960s for at least three reasons. First, British gerontology did not have a strong leadership. Although Korenchevsky founded gerontology in Britain, his identity as an émigré scientist, marginal academic status, and financial difficulties in his own old age led him to make constant troubles with his patrons and potential collaborators, hampering his organizational efforts. Second, there were not many scientists who could become leaders of the field instead of him or build a strong research tradition in the country. Indeed, most leading scientists of aging before

[22] Metchnioff, *Études sur la nature humaine*, p. 386.

the 1940s were Americans or other nationals who worked at American institutions. Third, there were not many strong supporters of gerontology in Britain. The Nuffield Foundation was the only source of research money for gerontology and there was little funding coming from the British government until the late 1950s.

But I do not argue that aging itself was not an significant issue in Britain. Rather, the lack of support for gerontology implies that the social condition in Britain was different from that in the United States. Indeed, aging had been an important problem in Britain since the 1900s when the Old Age Pensions Act was passed. Britons thereafter had tried to construct a welfare state supporing their elderly citizens in terms of both healthcare and pension. I claim that the troubles of British gerontology implies that these ways of caring for the elderly were considered much more impotant than supporting the science of aging which was considered too far from the immediate necessities of senior citizens.

In contrast, the United States had relatively weaker welfare systems supporting the health and livelihood for the elderly, and this weakness, ironically, became a background factor for the progress of gerontology. In fact, in 1948 when Britain established the National Health Service for free healthcare of its citizens including the elderly, the United States federal government substantially expanded the National Institutes of Health (NIH) for better biomedical research. It is important to notice that the growth of gerontology in America was heavily indebted to this expansion of the NIH, which supported both intramural and extramural gerontological research including the NIH Unit on Gerontology and the Gerontology Study Section. As Stephen Strickland has put it, the public support of biomedical research was the only politically acceptable way for the federal government to assist the citizens' health when national health insurance could not be introduced. I argue that the expansion of gerontology in America, especially the rapid growth of its research funds, reflects this political situation.[23] I also point to the other factors that were favorable to gerontology in America, such as a large number of

[23] Stephen P. Strickland, *Politics, Science, and Dread Disease: A Short History of United States Medical Research Policy* (Cambridge, Mass.: Harvard University Press, 1972), pp. 154-156, 213. Also see Victoria A. Harden, *Inventing the NIH: Federal Biomedical Research Policy, 1887-1937* (Baltimore: Johns Hopkins University Press, 1986), p. 182.

researchers interested in aging and the strong leadership of seveal major scientists, such as Cowdry, McCay, and Nathan Shock.

Finally, I have to say that my comparative analysis is limited to America and Britain, because gerontologists in the two countries played the most active role in building gerontology as an international research field. It was Korenchevsky who visited many countries in Europe and North America in person to encourage scientists there to organize the Club for Research on Ageing. Many of them also became members of the International Association of Gerontological Societies in 1950. Among them, American gerontologists were the most enthusiastic in responding to Korenchevsky's call. They developed gerontology more extensively than any other countries in the world, as can be seen in their construction of the National Institute on Aging in 1974 which has grown into the largest funding agency and the reserch intitute on aging in the world.

Other Concerns: Gender, Class, and Race

Gerontologists mentioned in this dissertation were primarily interested in the aging of the middle-class white male population, if they studied human aging. Their major concerns in terms of social issues were the elderly's mandatory retirement, age discrimination, and social isolation, which were most frequently found among white middle-class aged men during the early twentieth century.[24] The case was not different when gerontologists studied physiological and clincal problems of aging, since their research subject was usually chosen among Caucasian men.[25] Admittedly, some gerontologists, such as cardiologist Alfred Cohn, studied how different racial groups

[24] People of color were hardly mentioned in early volumes of gerontology journals. The only paper I found before the late 1950s was Frederic D. Zeman, "The Needs of the Negro Aged," *Journal of Gerontology* 3 (1948), p. 234.

[25] Although some people of color were sometimes used as research subjects by the NIH's intramural gerontology program during the 1940s and the 1950s, this posed a problem. Since these people were poor inmates of the Baltimore City Hospitals, gerontologists thought that their poverty and the accompanying poor health certainly influenced their course of aging and made it deviate from the "normal" pattern. This was a reason why the gerontologists of the NIH began the Baltimore Longitudinal Study of Aging from 1958 and used volunteers, most of whom were middle-class white males, as the volunteers. While "negro male volunteers," whose "unusual characteristics" would be "very much worth studying," the research project began without using them. See William Wesley Peter to Nathan Shock, 3 September 1958, Box 21, Folder Longitudinal Studies W. W. Peter July-Dec. 1958, NWS.

showed distinct patterns of age changes.[26] Cowdry also briefly mentioned that both women's and men's aging had to be considered in gerontologists' prospective works.[27] Yet there were not many scientists like Cohn and Cowdry at that time, and their research on that problem was far from being extensive or systematic. Not surprisingly, this reflects the fact that the race, class, and gender composition of early gerontologists was quite homogeneous: Almost all the members of the Club for Research on Ageing in the United States and the United Kingdom were white men with middle-class backgrounds, apart from a few exceptional cases, such as the renowned British geriatrician Marjorie Warren. Given these conditions, it was taken for granted that a core subject of gerontology should be Caucasian males.

This situation began to change after the 1970s. Most notably, the Baltimore Longitudinal Study of Aging, which had traced age changes of many volunteers' various physiological parameters after 1958, began to recruit female volunteers in 1978. After that, an increasingly large number of gerontologists in the United States started investigating senescence in women, people of color, and those from the woking class. In a larger perspective, this change is a part of the broader movement in medical research in general. During the latter half of the twentieth century, medical scientists became more aware of the gender, class, and racial factors than before as significant variables influencing the results of clinical trials.[28] With this awareness, gerontology has also become more cautious about different aging experiences and physiological changes among distinct groups divided by gender, class, and race.

Whereas this new medical research trend is certainly an important topic for historical research, this dissertation, whose scope is limited to the first half of the twentieth century, does not thoroughly deal with it. Instead, this dissertation focuses on the rise of the criterion of age, which, along with gender and race, has functioned as an

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[26] Wilhelm Ehrich, Clarence de la Chapelle, and Alfred E. Cohn, "Anatomical Ontogeny. B. Man. I. A Study of the Coronary Arteries," *The American Journal of Anatomy* 49 (1931), p. 251.

[27] Cowdry to Kast, 19 November 1935, Box 31, Folder 9, EVC.

[28] For a review of the reasons for the under-representation of ethnic minorities in medical research, see, for example, Mahvash Hussain-Gambles, Karl Atkin, and Brenda Leese, "Why Ethnic Minority Groups Are Under-Represented in Clinical Trials: A Review of the Literature," *Health and Social Care in the Community* 12 (2004), pp. 382-388.

important basis of classifying and using humans in political, cultural, and scientific arenas. Through gerontologists' works, the significance of age has become more widely discussed and disseminated among both professional researchers and the general public.

Chapter Organization

This dissertation has six chapters. Chapter one analyzes how biological and biomedical scientists adopted the new ideas, perspectives, and methodologies that could be used by later scientists who would construct gerontology. I argue that biomedical and biological research of the early twentieth century introduced new experimental methodologies as well as the notion that aging was a contingent and localizd phenomenon. Chapter two extends the argument in chapter one by showing how research in animal husbandry produced a novel scientific program and an experimental tool that became a focal point of interaction across disciplinary boundaries. By studying the life and work of animal nutritionist Clive McCay, this chapter claims that research on farm animals' longevity led to the discovery of the relationship between caloric intake and lifespan, initiating a viable multidisciplinary research program in gerontology. Chapter three discusses another biological research project conducted by the renowned British scientist and Nobel laureate Peter Medawar. I argue that his evolutionary theory of aging stemmed from his interest in mathematical and theoretical approaches to age changes in living organisms, and his new theory prompted other researchers to study evolutionary problems of aging further. Yet I also point out that Medawar himself did not pursue more advanced research following his initial theorization and did not actively participate in the growth of gerontology in Britain. An important reason for Medawar's meager participation in gerontology is suggested in chapter four. This chapter argues that gerontology did not become a strong research field in Britain as it did in America because of Korenchevsky's lack of leadership, the small number of researchers interested in aging, and the social environment that favored direct assistance for the elderly through free healthcare and pension rather than science. In contrast, the establishment of gerontology in the United States proceeded in a better condition, and chapters five and six discuss the reasons why the case was different on the other side of the Atlantic. Chapter five analyzes

how gerontology was born as a multidisciplinary scientific field in America by tracing Cowdry's career as a biologist and textbook editor. I argue that his broad vision on the social role of biology and his editorial experience contributed to his endeavors to organize gerontology as a multidisciplinary field, especially when the whole country was severely agitated by the Great Depression. Chapter six traces the later development of the multidisciplinary field of gerontology in America. I claim that the growth of the science of aging in the United States during the 1940s and 1950s was made possible by gerontologists's constant efforts at securing research funds, interacting with the general public, and promoting cross-disciplinary cooperation and professional maturation. Chapter six also analyzes the larger social and political factors that allowed these efforts to bring about meaningful outcomes, which included, most notably, the support from the NIH which was considerably expanded after World War II. My historical analysis ends at the late 1950s when gerontologists completed the first major phase in shaping a new research field that would further contribute to refiguring our notion and practice concerning old age.

Chapter 1

"Senility and Death of Tissues Are Not a Necessary Phenomenon"—Locality, Contingency, and Experimentalism in the Science of Aging

"I don't want to die," wrote Paul de Kruif (1890-1971), a renowned American biomedical scientist and writer.[1] In his popular article published in *Ladies' Home Journal*, he expressed deep worries about his old age and death that would come in the future. Even though he was only thirty-nine years old in 1930 when the article came out, he knew that he was undergoing senescence which was bringing him ever closer to death. While he, along with the seventy percent of his cohorts born in 1890, was fortunate to survive in 1930, vital statistics at that time told him that he would live only for twenty-five more years on the average. But he thought that "there are too many birds left that I don't know the songs of, and it's going to take a long, long time to learn them." It was also "too much fun to fight Lake Michigan's strong blue water." He wanted to "go on and and cut down more thousands of saplings, and make barricades of sandbags and saplings, to keep Lake Michigan from taking our house." Unfortunately, de Kruif thought, he could not do everything he liked because he would become old and probably die in two or three decades.

But de Kruif certainly knew that he was living in an era when average longevity had substantially increased. Whereas "babies born in 1850 could expect, on the average, to live only thirty-five years," he wrote, "babies born today have a gambling chance to live to be fifty-five."[2] Yet he was also aware that the expectation of life of the people who arrived at their middle life had not changed much since 1850. According to him, the extension of average lifespan in the Western world was mostly due to the decrease of

[1] Paul de Kruif, "How Long Can We Live?" *Ladies' Home Journal* (February, 1930), p. 3.
[2] De Kruif, "How Long Can We Live," pp. 3-4.

death rate among the young. This was a consequence of the medical advancement which brought about the control of many infectious diseases. In contrast, middle aged or elderly people's expectation of life underwent little change. The modern biomedical sciences, to which he himself contributed as a microbiologist, were not yet able to deal effectively with chronic diseases and their underlying cause, aging.

Nevertheless, de Kruif thought that new ideas and research on senescence were emerging since the 1900s. In fact, as a renowned popular writer and scientist, he was well aware of the development of scientific and medical research on aging. In the article, he wrote about Raymond Pearl's study of longevity and heredity, Alexis Carrel's immortal tissue culture, and Jacques Loeb and John Northrop's study of the relationship between temperature and lifespan. De Kruif also knew of Louis Dublin's statistical investigations of the death rate as well as recently developed new medications such as digitalis and insulin that were effective upon chronic illness. Even though what he could immediately recommend to his readers for long and healthy life was only "the clean air of outdoors and the sun," biomedical scientists studying senescence and chronic diseases, he thought, were producing new knowledge on aging process and its potential applications.[3] This scientific progress would eventually change humans' longevity and their health in old age.

[3] De Kruif, "How Long Can We Live," p. 197.

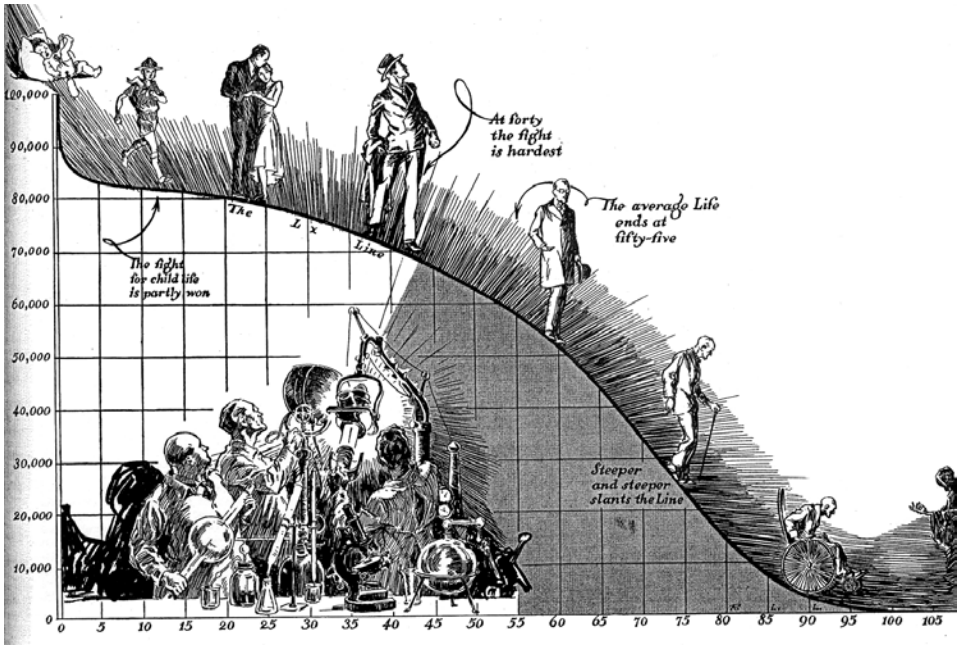

Figure 1.1. *Scientists and the Ages of Life. An Illustration by R. L. Lambdin in Paul de Kruif, "How Long Can We Live?" Ladies' Home Journal (February, 1930), p. 3. The Picture Was Modified by the Author.*

For de Kruif, the new research on aging was imperfect yet had an immense potential for further progress, as the first illustration in the article indicated. (See Figure 1.1.) The three scientists, with a microscope, flasks, and other scientific apparatus, were illuminating the process of aging, although the man whose life-course was being observed was still sliding into his final destination. Admittedly, there were not many prescriptions that scientific professionals could offer to this aging person. But what was important was that scientists had begun to investigate senescence in a much more systematic and organized way than before. Just as biomedicine had been immensely successful in helping humans deal with infectious disease, as described in his famous monograph, *Microbe Hunters* (1926), the science of aging was also expected to tackle aging and chronic illness. While the effective control of infectious disease in childhood or early adulthood increased longevity, the science of aging would further extend the lifespan and bring about happier and healthier life.

This chapter examines the basis of this optimism that de Kruif's article sought to convey to readers. I argue that aging became a subject of modern biomedical research

during the early twentieth century, and in this sense, I claim that de Kruif's article reflected—at least partially—an important change that was occurring at that time. Indeed, while many philosophers, physicians, and theologians had written about aging, the discourse on senescence as a natural phenomenon was not dominant in Western culture before the late nineteenth century. Aging had been a subject related more to religion, morality, and social responsibility than to biological and medical theories and practices. If anybody had ever discussed aging without mentioning its relation to religion or morality, that discussion was usually limited to a short paragraph or a brief chapter in scholarly or popular treatises on health and longevity. However, the changes in biomedicine during the late nineteenth and early twentieth centuries altered the state of biological and medical approach to aging and formed a critical basis upon which the new science of senescence could grow. At that time, many new research subjects, study fields, and institutions were created and developed. New medicine based on germ theories of disease, along with experimental approaches in general, transformed medical practice and its possibilities. Biological science was also changing rapidly through the growth of genetics, experimental embryology, and other new subfields in the life sciences. Simultaneously, the institutional niche for these changes was created by newly reorganized hospitals, well-funded biomedical institutes, and the biology departments in research universities. The scientists of aging whose works will be discussed in this chapter were direct heirs of this change. They were trained in biology departments or medical schools, worked as professional researchers and teachers, and wrote articles in peer-reviewed journals. Moreover, they were much more experienced in experimental methodologies than researchers of earlier generations, and were connected with one another through their national or international professional networks. The current chapter traces how these researchers created the novel perspectives and research methodologies on senescence. Their primary way of approaching aging came from their research programs created amid the intellectual and institutional transformation within the biological and biomedical sciences.

I will argue that these scientists introduced three novel ways of approaching senescence. First, they focused on how each locality within the body showed a distinct

rate and mode of aging. This was a departure from the traditional notion prevalent in Western culture which insisted that aging was caused by the decrease or decay of one critical entity or principle such as the "vital power" that caused aging of the whole body. Second, the scientists began to recognize that aging was a contingent phenomenon at least at the cell level. Although few could deny that senile changes were unavoidable in humans and other multicellular species at the level of the whole individual, unicellular organisms, plants, and some somatic animal cells were thought to have the capacity to continue their life without undergoing senile changes. Third, scientists began to use experimental methods in studying aging. This was a fundamentally new way of investigating senescence which made possible a substantial departure from the traditional mode of approaching aging.

These three factors are interrelated. The notion that aging was a contingent phenomenon provides a conceptual basis for experimental manipulation of the aging process. Since aging was thought to be contingent at the cell level, it was easier to manipulate its rate and progress by controlling environmental condition or manipulating genetic constitution. Moreover, the idea of each locality's distinct rate and mode of aging could support the contingency concept and encourage experimental manipulation of aging. As aging was no longer explained by the decay or decrease of some central entity, scientists began to study senescence in different parts of the body using new experimental techniques such as tissue culture, which showed that certain somatic cells did not age and die in particular local environmental states. This result provided further support for the idea of contingency of aging and promoted the necessity of broader and more intensive experimental studies of senescence in each part of the body.

The current chapter examines previous historical works on the science of aging. In particular, this chapter asks whether the implications of the arguments of several historians of aging—including Carole Haber, W. Andrew Achenbaum, and Thomas R. Cole—were reliable regarding the relationship between society and the major research subjects in the science of senescence. They have claimed that the science of aging since the late nineteenth century mostly functioned as a means of justifying the social place of

the elderly in capitalist culture and economy.[4] As industrial capitalism marginalized the elderly in its culture and employment structure, these historians have asserted, the science of aging provided—regardless of scientists' intention—the rationale for justifying this changing place of older people by making the inevitable bodily and mental decline of the aged as a scientific fact or equating it with a pathological alteration. As some of these historians have admitted, however, it is important to notice that biological and medical theories on the declining health and vigor of the elderly were not suddenly born in the era of industrial capitalism. The origins of the theories could be traced at least as far back as to the writings of Hippocrates, Galen, and numerous doctors and natural philosophers after the ancient Greek and Roman era.[5] If that was the case, how could the modern scientific discourse on senile decline become the basis of marginalizing older people in industrializing society? While I partially admit that scientists of aging played some roles in marginalizing the elderly, I think that historians' emphasis on such a role has been exaggerated. Moreover, their argument can mislead readers on the actual research activity that fascinated many scientists of senescence during the early twentieth century. I claim that even if it was true that the scientists of senescence contributed to the declining social status of the aged their major subject of academic interest was not so much the inevitable decline of the aging person as the novel aspects of aging revealed through new methodologies. I agree that these scientists were working within, and perhaps were influenced by, their social environment that marginalized the elderly. But it is also true that their research was structured by the tradition within which they were trained, the nature of their research environment, and the professional relationship with their

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[4] W. Andrew Achenbaum, *Old Age in the New Land: The American Experience since 1790* (Baltimore: Johns Hopkins University Press, 1978), pp. 40-45, 110-113; Thomas R. Cole, *The Journey of Life: A Cultural History of Aging America* (Cambridge: Cambridge University Press, 1992), pp. 161-211; Carole Haber, *Beyond Sixty-Five: The Dilemmas of Old Age in Amerca's Past* (Cambridge: Cambridge University Press, 1983), pp. 47-81. Also see Howard P. Chudacoff, *How Old Are You? Age Consciousness in American Culture* (Princeton: Princeton University Press, 1989), p. 59.

[5] Thomas R. Cole and Mary G. Winkler, "Aging in Western Medicine and Iconography: History and the Ages of Man," *Medical Heritage* 1 (1985), pp. 336-347; M. D. Grmek, "On Ageing and Old Age: Basic Problems and Historic Aspects of Gerontology and Geriatrics," in F. S. Bodenheimer and W. W. Weisbach (eds.), *Monographiae Biologicae* vol. 5 no. 2 (Den Haag: Junk, 1958), pp. 8-22; Richard L. Grant, "Concepts of Aging: An Historical Review," in Gerald J. Gruman (ed.), *Roots of Modern Gerontology and Geriatrics* (New York: Arno, 1979), pp. 443-478.

colleagues. I think that these factors led the scientists to find the contingency and local distinctiveness of aging which could not have been observed if their only aim had been to justify the received social order.

But this chapter does not argue that the changes in the science of aging constitute a "scientific revolution" in the sense of Thomas S. Kuhn.[6] While the optimism described in de Kruif's article might indicate that a sudden scientific change would completely alter the scientific understanding of aging, what the scientists of aging during the early twentieth century did was more multilayered.[7] Indeed, unlike the classical Kuhnian examples like the chemical and Copernican revolutions, no single unified theory of aging was constructed at that time. As a contemporary commentator has pointed out, a number of divergent theories on the causes of aging still existed even in the mid-twentieth century, and many of them were very similar to older theories.[8] Even the ideas of some scientists mentioned in this chapter maintained the traditional theoretical standpoint. In some sense, Raymond Pearl's notion of the "vital capital" or Charles Minot's theory of "cytomorphosis" sounds like newer versions of the tradition theories which postulated that the decrease or decay of some central element brought about aging.

I argue that what really emerged during the early twentieth century was not a coherent theory on the cause of senescence but a cluster of new ideas, perspectives, and research methods on aging. Perhaps Kuhn's controversial and multipurpose notion of "paradigm" might include this cluster. Unlike Kuhn, however, I do not claim that any formal theory appeared or was established in the early twentieth century. Instead, I show that scientists of aging, while disagreeing on the cause of aging, shared a set of concepts and methodologies that were based on or stemmed from modern biomedical research. For example, tissue culture of animal and plant cells led Alexis Carrel and William Crocker to argue that some animal somatic cells and most plant cells had an ability to live indefinitely without undergoing senescence. Although many researchers, including Pearl,

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[6] Thomas S. Kuhn, *The Structure of Scientific Revolutions* (Chicago: University of Chicago Press, 1962).
[7] Indeed, de Kruif never mentioned any theories of aging proposed before the twentieth century. See de Kruif, "How Long Can We Live?" pp. 3-5, 190, 192, 195, 197.
[8] See Alfred H. Lawton, "The Historical Developments in the Biological Aspects of Aging and the Aged," *Roots of Modern Gerontology and Geriatrics*, p. 30.

still advocated theories similar to traditional ones, the new tissue culture brought forth the idea that life was inherently continuous, at least at the cell level, and that aging could be experimentally manipulated. It is also important that the scope of research subjects was substantially broadened during the early twentieth century through the development of cytology, botany, embryology, and pathology. This broadening of the scope, I will argue, enabled researchers to focus on more detailed studies of aging in each body part of humans, animals, plants, and even microbes. Moreover, experimental approaches imported from genetics, protozoology, nutrition research, and other scientific fields created new problems on the nature of aging which had not been suggested before that time.[9] I do not argue that the introduction of experimental methods made the researchers of aging more "rigorous" or even more "scientific." Rather, I claim that experimental research provided scientists with new means of manipulating and intervening into the process of aging, enabling them to reveal hitherto unexplored dimensions of senescence.[10]

This chapter also points out that with the introduction of experimental methodologies the science of aging became a part of the growing biomedical research enterprise pursued by professional researchers. Unlike many pre-twentieth century scholars and theorists of aging, the researchers who employed experimental methods were professional scientists who worked in biology departments, medical schools, or research institutes. They also belonged to professional societies and interacted with one another through direct encounters, exchange of letters, and peer-reviewed articles and books. In this chapter, I will trace the network of this communication among researchers

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[9] Indeed, historians and sociologists of science have analyzed how the use of experiments has transformed researchers' approaches and brought forth new epistemic challenges. Many research articles and monographs have been published on this issue. See, for example, David Gooding, Trevor Pinch, and Simon Schaffer (eds.), *The Uses of Experiment: Studies in the Natural Sciences* (Cambridge: Cambridge University Press, 1993); Steven Shapin and Simon Schaffer, *Leviathan and Air-Pump: Hobbes, Boyle, and the Experimental Life* (Princeton: Princeton University Press, 1985); Hans-Jörg Rheinberger, *Toward a History of Epistemic Things: Synthesizing Proteins in the Test Tube* (Stanford: Stanford University Press, 1997); Peter Galison, *Image and Logic: A Material Culture of Microphysics* (Chicago: University of Chicago Press, 1997).

[10] Peter Galison has succinctly summarized this problem in his recent historiographical review. If Einsteinium and Fermium can be observed only through experimental means, then it becomes difficult to say whether they are nature or artifact. See Peter Galison, "Ten Problems in History and Philosophy of Science," *Isis* 99 (2008), pp. 111-124.

through their correspondence and citations. This will help draw, in preparation for a further discussion on the issue in the following chapters, a preliminary picture on how the professional network of gerontology was constructed.

Before moving onto the main body, I have to mention one important issue, namely, that most scientists mentioned in this chapter were Americans or Europeans who worked at American institutions. This does not mean that no other countries made a contribution to the development of the science of aging, and the later chapters do discuss the works of the scientists who stayed in the United Kingdom. Moreover, there were certainly many Europeans, especially Germans, who were deeply involved in the biomedical studies related to aging. Indeed, some American scientists mentioned in this dissertation, including Charles Minot and Alfred Cohn, were trained in Germany. The renowned German biologist August Weismann's influence upon American scientists of aging was also important in the growth of research on aging. Nevertheless, this dissertation deals only with researchers in America and Britain. The investigations of aging done in other European countries than Britain are outside of the scope of my historical study. Even though an analysis of their research tradition will certainly enrich the historiography, it will make the current dissertation an overly ambitious piece of work. What conclusion, then, can be drawn from this relatively circumscribed study of the science of aging focused on America and Britain? I will show that the role of investigators who worked in the United States was more significant than those in the United Kingdom. This is not because Americans gave a special attention to aging as a scientific subject. Rather, the biological and medical sciences in general developed rapidly in the United States during the early twentieth century, and the science of aging also grew along with other sciences. The new ideas and approaches related to aging discussed in this chapter were indebted to the expansion of the biological and medical studies, particularly in the United States.

Aging Revised: Local Distinctiveness of Senile Changes and Their Occurrence during Early Phases of Life

Most scholars and doctors since the ancient Greek era who wrote about aging attributed it to the inevitable decline or decay of one critical factor.[11] For example, the Hippocratic doctors in ancient Greece wrote that aging was the process of losing the "innate heat," while Galen (129-ca. 216) claimed that aging proceeded through "drying" of the body.[12] Similarly, Roger Bacon (1214-1294), an English philosopher of the thirteenth century, asserted that the "natural heat" was diminished with aging in "two ways: by the Decay of Natural Moisture, and By the Increase of Extraneous Moisture."[13] Francis Bacon (1561-1626), an influential philosopher of early modern England, also argued that the "native spirit," which was a normal constituent in every living body, eventually caused senile changes by drying and destroying the structure of the body.[14] Christopher Hufeland (1762-1836), a renowned German physician, also argued that the enfeeblement of the "vital power, the grand cause of all life" caused aging and natural death.[15] Marie François Xavier Bichat (1771-1802), a leading French physician and pathologist during the heydays of French clinical medicine, had a similar idea. He asserted that aging was a process of the weakening of the "internal principle" which resisted the actions of the external forces that brought about death.[16] Even in the early twentieth century, Aldred Scott Warthin (1866-1931), a pathologist at the University of Michigan, explained aging as a consequence of gradual exhaustion of "energy" whose amount in each body was fixed.[17] Ignatz L. Nascher (1863-1944), an Austria-born American physician who coined the term "geriatrics," also argued that aging was caused

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[11] But it was often not clear whether this factor was the cause of aging or its consequence. Since there are already several survey articles on theories of senescence, this chapter does not aim at exhaustive historical survey. See Cole and Winkler, "Aging in Western Medicine," pp. 336-347; Grmek, "On Ageing and Old Age," pp. 8-22; Grant, "Concepts of Aging," pp. 443-478.

[12] Hippocrates, "Aphorisms," in Francis Adams (tr.), *The Genuine Works of Hippocrates* (Baltimore: Williams and Wilkins, 1939), p. 294; Galen, "The Nature and Sources of Growth and of Disease," in Robert Montraville Green (tr.), *A Translation of Galen's Hygiene* (Springfield, Illinois: Charles C. Thomas, 1951), pp. 6-8.

[13] Roger Bacon, *The Cure of Old Age and Preservation of Youth* (London: Tho. Flesher and Edward Evets, 1683), p. 2.

[14] Francis Bacon, "The Preface," in *History Natural and Experimental of Life and Death or the Prolongation of Life* (London: William Lee and Humphrey Moseley, 1658).

[15] Christopher William Hufeland, *The Art of Prolonging Life* (London: J. Bell, 1797), pp. 35, 63-64.

[16] Marie François Xavier Bichat, *Physiological Researches upon Life and Death*, Tobias Watkins (tr.) (Philadelphia: Smith and Maxwell, 1809), pp. 1-2.

[17] Aldred Scott Warthin, *Old Age: The Major Involution: The Physiology and Pathology of the Aging Process* (New York: Hoeber, 1929), pp. 11-16.

by the inevitable "tissue-cell evolution"—the decreasing adaptibility of cells as they proliferated during a human's life.[18]

While some scholars introduced variations by arguing that aging was caused by the accumulation of waste products or by the progressive wearing-out of the body's components, the essential point did not differ. Aging was an unavoidable phenomenon brought about by one critical cause. For example, an American physician Homer Bostwick claimed in 1851 that senile changes were caused by "the gradual accumulation of solid, earthy elements."[19] Although he was very confident that there was "no other cause" of aging, there is little evidence that his idea influenced any later scientists or physicians.[20] Even the renowned Russian-French scientist Elie Metchnikoff (1845-1916), who coined the term "gerontology," had an idea that was essentially similar. Aging was a result of accumulated toxic wastes in the large intestine, which was often accompanied by the activities of overly stimulated macrophages that eventually destroyed the body.[21] Although Metchnikoff's idea led many lay people to drink yoghurt regularly as he did to stimulate the beneficial intestinal bacteria, there were not many scientists who considered his theory of aging seriously. Despite the fact that his argument for the creation of gerontology was eventually realized, his theory of aging was just one of many ideas that did not have sufficient explanatory power.[22]

But the development of embryology and human physiology introduced an important change, as can be seen in the case of Aldred Warthin. While insisting on a tradition notion that aging was caused by the exhaustion of "energy," Warthin's book shows an interesting and significant twist. In his book, *Old Age: The Major Involution* (1929), he argued that there were two kinds of aging process, the major and minor

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[18] Ignatz Leo Nascher, *Geriatrics: The Diseases of Old Age and Their Treatment* (Philadelphia: Blakiston, 1916), pp. 43, 47-48. Although he thought that aging could have multiple causes, he still argued that there was "a determining factor." All other causes were merely "contributing factors."

[19] Homer Bostwick, *An Inquiry into the Cause of Natural Death or Death from Old Age* (New York: Stringer and Townsend, 1851), p. 7.

[20] Bostwick, *An Inquiry into the Cause of Natural Death*, p. 7.

[21] Elie Metchnikoff, "Old Age," in *Annual Report of the Board of Regents of the Smithsonian Institution* (Washington: Government Printing Office, 1905), pp. 536-550.

[22] W. Andrew Achenbaum, *Crossing Frontiers: Gerontology Emerges as a Science* (Cambridge: Cambridge University Press, 1995), pp. 30-33, 40.

involutions. While the former was the senile change occurring in old age, the latter, he argued, was the degeneration during the developmental stages which was necessary for the growth of the embryo. For example, the tail of spermatozoa and the polar bodies of the ovum disappeared after fertilization. The gill slits and the notochord were also degenerated during embryogenesis and the umbilical cord underwent rapid senescence right after the birth of the baby. It was also important that even during puberty and early adulthood some minor involutions occurred, such as the atrophy of the thymus and tonsils. According to Warthin, although these minor involutions played an important role in the maturation of the organism, they were "prophetic of the future fate of the organism as a whole."[23] In other words, "growth and retrogression [went] hand in hand in the broad economy of the organism."[24]

Whereas Warthin maintained the distinction between the major and minor involutions, other researchers viewed the phenomenon differently. Most notably, the Canadian-American cytologist Edmund Vincent Cowdry (1888-1975), who played a significant role of building gerontology as a scientific field in America, did not make a distinction between major and minor involutions. (See Chapter 5.) Each cell and tissue underwent aging at a distinct time and rate. For example, there was no essential difference except the time of onset between the aging of arteries and veins within the umbilical cord and the senescence of other blood vessels commonly found in middle-aged or elderly arteriosclerosis patients.[25] It is notable that similar ideas were suggested by other scientists of aging, such as Raymond Pearl, Alexis Carrel, and Alfred Cohn. Chapters two and six will also deal with the same notion advocated by two other significant researchers of aging, Clive McCay and Nathan Shock.[26] Through these

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[23] Warthin, *Old Age*, p. 62.
[24] Warthin, *Old Age*, p. 69.
[25] E. V. Cowdry, "The Structure and Physiology of Blood Vessels," *Arteriosclerosis: A Survey of the Problem* (New York: Macmillan, 1933), p. 63.
[26] Alfred E. Cohn and Henry A. Murray, Jr., "Physiological Ontogeny I. The Present Status of the Problem," *Quarterly Review of Biology* 2 (1927), pp. 482, 490; Raymond Pearl, *The Biology of Death* (Philadelphia: Lippincott, 1922), pp. 138-149, 225; Alexis Carrel, "Tissue Culture and Cell Physiology," *Physiological Reviews* 4 (1924), pp. 1-20; Carrel, "The New Cytology," *Science* 73 (1931), pp. 297-303; E. V. Cowdry, "Ageing of Tissue Fluids," in E. V. Cowdry (ed.), *Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1939), pp. 643, 685, 689. Also see Nathan W. Shock, "Ageing of Homeostatic Mechanism," in Albert I. Lansing (ed.), *Cowdry's Problems of Ageing*, 3rd edition

scientists' works, aging became a phenomenon localized at a specific part of the body which should be studied by more focused analysis of cells and tissues.

Another important scientist who supported the notion of the local nature of aging was the American anatomist and embryologist Charles S. Minot (1852-1914). While arguing that aging was caused by "cytomorphosis," namely, the gradual increase of the size of the cell's cytoplasm over that of the nucleus during aging, he stated that this phenomenon occurred at a distinct rate in distinct portions of the body.[27] The senile change of the whole body was thus not a single and synchronous phenomenon but a composite of many different aging processes which occurred at their own unique rates. Since this statement was published in 1908, he was probably one of the first scientists who promulgated the idea of local distinctiveness of aging. But Minot's contribution to the science of aging was not limited to this idea. He proposed an interpretation of aging and development which introduced a completely changed order of aging in a rather surprising way.

Born in 1852, Minot belonged to the first generation of American scientists who imported the new lines of biological and medical research from Germany. After studying at the Massachusetts Institute of Technology and Harvard University, he went to the University of Leipzig, to work with the renowned German physiologist Carl Ludwig. There he learned the new physico-chemical and experimental approaches, which were vigorously advocated by contemporary German leaders of the life sciences. Indeed, like his teachers and colleagues in Germany, Minot refused to employ the concept of "vital force," and advocated rigorous experimental and statistical methods in biology, especially for his specialty, embryology.[28] Yet he used older biological ideas and methodologies as well in his research pursued at Harvard Medical School as a professor of embryology. In particular, he accepted the biogenetic law, which postulated that embryogenesis repeated a species' evolutionary history. Indeed, he proposed in 1897 a new phylogenetic tree on

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(Baltimore: Williams and Wilkins, 1952), pp. 421, 429-31, 436, 438; Clive M. McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), p. 574.

[27] Charles S. Minot, *The Problem of Age, Growth, and Death: A Study of Cytomorphosis* (New York: Putnam, 1908), p. 216.

[28] Minot, *The Problem of Age, Growth, and Death*, pp. xv-xxii.

the evolution of vertebrates through his study of the morphological changes of *Amphioxus* during its developmental stages.[29] His research stood at the juncture between the new and old biological traditions.

But Minot's research on aging was more closely linked to the new trends in German biology than older ideas and methodologies. As early as in 1886, he fully accepted the view that the nucleus and chromosomes were the "physical basis" of heredity.[30] Minot was also favorable to the German biologist August Weismann's theory of germ plasm, which helped guide Minot's interest toward the problem of aging. He was well aware of Weismann's statement that senescence and natural death were not inevitable for unicellular organisms or the germ plasm of multicellular species, even though other portions of the multicellular species, which Weismann called "soma," did become senile and eventually perish.[31] Minot reinterpreted this statement in his own way basesd on his and other scientists' microscopic observation and measurement. Every cell in multicellular organisms underwent a process that he called "cytomorphosis" from the earliest stage of its life cycle, during which the proportion of its cytoplasm gradually increased over that of the nucleus, ultimately resulting in the cell's death.[32] Since the nucleus was the core material of heredity and function, the decrease of its proportion implied that the cell was losing its vitality and becoming senile. But this process was reversed during the earliest phase of life right after fertilization, when the nuclear division without increasing the total embryonic volume resulted in the dramatic reduction of the proportion of the cytoplasm in each cell. This phenomenon, which Minot called "rejuvenation," was followed by another round of cytomorphosis during the

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[29] Charles S. Minot, "Cephalic Homologies: A Contribution to the Determination of the Ancestry of Vertebrates." *American Naturalist* 31 (1897), pp. 927-943. For the historical context of this paper, see Peter J. Bowler, *Life's Splendid Drama: Evolutionary Biology and the Reconstruction of Life's Ancestry, 1860-1940* (Chicago: University of Chicago Press, 1996), pp. 157-171.

[30] Charles S. Minot, "The Physical Basis of Heredity," *Science* 8 (1886), pp. 125-130.

[31] Charles S. Minot, "On Heredity and Rejuvenation," *American Naturalist* 30 (1896), pp. 6-9. See August Weismann, *Essays upon Heredity and Kindred Biological Problems*, Edward B. Poulton, Selmar Schönland, and Arthur E. Shipley (eds.) (Oxford: Clarendon, 1889), pp. 111, 158-159.

[32] Minot's first mention of "cytomorphosis" appears in 1901. See Charles S. Minot, "The Embryological Basis of Pathology," *Science* 13 (1901), p. 494. For a more developed theory, see Minot, *The Problem of Age, Growth, and Death*, pp. 38-85, 131-168, esp. p. 78, 157.

developmental periods, adulthood, and old age.[33] The unicellular organism, which did not age, did not need to undergo such a cycle of cytomorphosis and rejuvenation.

While this view was not based upon any new experiments, it certainly contained a novel conceptual element. For Minot, aging was a process that proceeded from the early stages of life after fertilization. By emphasizing their essential continuity, this idea contributed to the breakdown of the traditional periodization of human life, namely, infancy, childhood, adolescence, adulthood, middle age, and old age.[34] Admittedly, Galen had held a similar notion in his argument that humans, who were in the wettest and softest state during infancy, became increasingly dry and hard as they aged and arrived at the driest and hardest condition in their old age.[35] Yet Minot was different from Galen, since he was more explicit in his view that the process of senescence progressed even during embryogenesis and continued in the youngest organisms' body. In contrast, Galen had never written that young organisms were necessarily becoming senile. To Galen, old age was still a distinct phase of life, even though the gradual drying process occurred throughout the whole life.

But a really novel aspect of Minot's work on aging can be found in his own observational research on the rate of growth that completely reversed the prevalent notion on the aging process. As I have explained in the previous section, it had been generally accepted that aging was a period of decline, which followed the phases of embryogenesis, growth, and maturity. Based on his measurement of the rate of growth of guinea pigs and other animals, however, Minot arrived at the conclusion that the "power of growth," which he thought was revealed through the rate of growth, declined at the highest rate during the earliest phases of life and increasingly slowly in later periods.[36] For example, the following graph clearly showed that the "daily percentage increments in weight" of

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[33] Charles S. Minot, "Senescence and Rejuvenation," *Journal of Physiology* 12 (1891), p. 98; Minot, *The Problem of Age, Growth, and Death*, p. 166.

[34] This periodization of human life is deeply imbedded in the Western culture. See Cole and Winkler, "Aging in Western Medicine," pp. 336-347; Cole, *Journey of Life*, pp. 3-31. Many modern scholars still had the essentially same concept. See G. Stanley Hall, *Senescence: The Last Half of Life* (New York: Appleton, 1922), p. vii; Warthin, *Old Age*, pp. 17-52.

[35] Galen, "The Nature and Sources of Growth and Disease," pp. 6-8.

[36] Minot, "Senescence and Rejuvenation," p. 147; *The Problem of Age, Growth, and Death*, pp. 86-130, esp. pp. 116, 126.

guinea pigs diminished most rapidly during their earliest life while the rate of decline gradually decreased as they aged. (See Figure 1.2.)

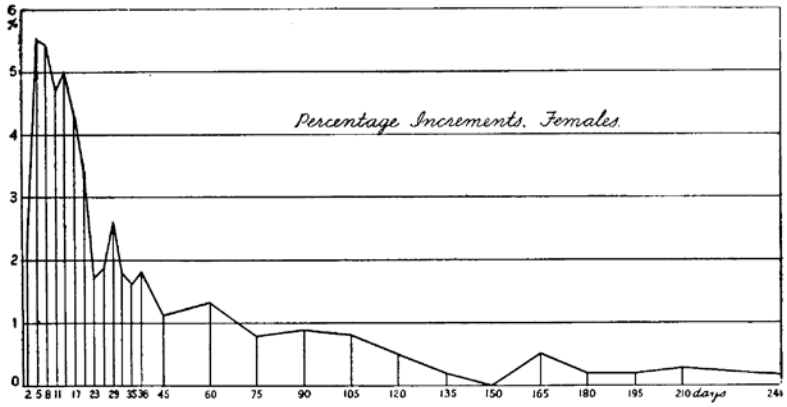

Figure 1.2. *A "Curve Showing the Daily Percentage Increments in Weight of Female Guinea-Pigs." Charles S. Minot, The Problem of Age, Growth, and Death (New York: Putnam, 1908), p. 96.*

He thus argued,

> ….we commonly think of old as those who have lost most, who have passed beyond the maximum of development and are now upon the path of decline, going down ever more rapidly. One of the chief objects at which I shall aim….will be to explain to you that that notion is erroneous, and that the period of old age, so far from being the chief period of decline, is in reality essentially the period in which the actual decline going on in each of us will be the least. Old age is the period of slowest decline—a strange, paradoxical statement, but one which I hope to justify fully….[37]

Minot supported this argument through other examples as well. He wrote that cell death, which was necessary for morphogenesis, took place at the highest rate during embryogenesis and occurred at increasingly lower rates during the later parts of life.[38]

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[37] Minot, *The Problem of Age, Growth, and Death*, p. 5.
[38] Minot, *The Problem of Age, Growth, and Death*, pp. 38-85.

This was actually what Warthin would point out in 1929. Minot also noted that the rate of decline of humans' "learning power" was the highest during their early life and became increasingly lower in later periods.[39]

This theory, which reversed the traditional view on the rate of aging during the span of life, was highly influential among later researchers of aging. Minot's idea was cited by a number of authors on senescence. Admittedly, some of them were quite critical. For instance, protozoologist Jennings claimed that "the decline in growth and the later decline in effectiveness…are evidently not identical."[40] Nevertheless, there were far more scientists who were favorable toward Minot's claim. In particular, as I will show in chapter three, the British biomedical scientist Peter Medawar's evolutionary theory of aging was heavily indebted to Minot's concept of senescence. Vladimir Korenchevsky, who organized the British Club for Research on Ageing, as well as Alexis Carrel, a founder of tissue culture technique, also fully embraced Minot's argument.[41] But the American researchers who created the field of gerontology in the United States were probably the most enthusiastic about Minot's views. Indeed, Alfred Cohn, Edward Stieglitz, and Lawrence Frank cited Minot's work and discussed its meanings.[42] For them, Minot's argument implied that aging was not so much a phenomenon limited to the latter half of an organism's life as a process that occurred throughout its whole lifespan. This indicated that the scope of gerontology was much wider than what had been thought.

Minot's research brought forth other novel ideas on aging. First of all, following Weismann, Minot emphasized that "natural death" through aging was a contingent

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[39] Minot, *The Problem of Age, Growth, and Death*, p. 243.

[40] Herbert Spencer Jennings, "The Problem of Age, Growth, and Death. A Study of Cytomorphosis. Charles S. Minot. New York and London, 1908. Pp. 280," *Psychological Bulletin* 6 (1909), p. 142.

[41] Vladimir Korenchevsky, Report of Dr. Korenchevsky to the Medical Research Council for 1941-1942, 3 Nov 1942, p. 4, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK; Alexis Carrel, "Physiological Time," *Science* 74 (1931), pp. 618-621; "The Mechanism of Senescence," *Bulletin of the New York Academy of Medicine* 4 (1928), p. 1146.

[42] Lawrence K. Frank, "Gerontology," *Journal of Gerontology* 1 (1946), p. 5; Cohn and Murray, "Physiological Ontogeny I.," p. 473; "The Aging of the Heart Muscle Regarded from a General Biologic Point of View," Address Delivered at Annual Graduate Fortnight—The Problems of Aging and of Old Age, New York Academy of Medicine, 3 October 1928, p. 622; Edward J. Stieglitz, "Aging as an Industrial Health Problem," *Journal of the American Medical Association* 116 (1941), pp. 1385. Herbert Spencer Jennings, who contributed to Cowdry's *Problems of Ageing* and participated in the first conference on aging at Woods Hole, also read Minot's book.

phenomenon that "has been acquired during the process of evolution of living organisms."[43] Since unicellular organisms did not naturally age or die without accidents, aging was not a cosmic phenomenon or destiny that had been determined for all living organisms but a contingent product of evolution from protozoa to metazoa. Minot thought that even these metazoan organisms were not without a capacity for eternal life, since some of their cells were still able to undergo the process of "rejuvenation" during the earliest developmental phase. It is important that this idea, which reintroduced the controversial notion of rejuvenation into scientific discourse by equating it with early embryogenesis, was followed by similar or more strengthened claims by other scientists. For instance, the renowned American embryologist Edwin Conklin argued that rejuvenation was possible in other cases than those of early phase of development. Conklin asserted that polyzoa, tunicates, and gland cells could sometimes rejuvenate themselves by eliminating some portions of their overly expanded cytoplasm that contained metabolic waste products.[44] As I will show in the next section, Alexis Carrel also asserted that animals' somatic cells could be rejuvenated outside of their body in a certain environmental condition which was made possible through tissue culture. Although Minot's idea that gradual cytomorphosis led to the cell's aging and death was similar to traditional theories which postulated the decline of some imaginary entities as the cause of aging, his works contained an element that could be extended to a completely different direction.

However, Minot did not have the viewpoint that was widely shared among the founders of American gerontology. Whereas most gerontologists during the 1930s and 1940s tried to fight against the prevalent notion that old age was a hopeless final phase of human life, Minot, despite his very novel viewpoint, used his research for defending, rather than challenging, the idea that the later scientists vehemently opposed. For Minot, old age came much earlier than usually thought, since his own research indicated that senescence occurred even during the very early phases of human life. It was thus

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[43] Minot, *The Problem of Age, Growth, and Death*, p. 215.
[44] Edwin G. Conklin, "The Size of Organisms and of Their Constituent Parts in Relation to Longevity, Senescence and Rejuvenescence," *The Harvey Lectures*, Series 8 (Philadelphia: Lippincott, 1913), pp. 252-279.

necessary to begin education and vocational life as early as possible, before senile changes put an end to a human's learning capacity. This idea brought Minot closer to the ageist viewpoint. Citing Osler's notorious statement on the "fixed period," Minot even argued that "Dr. Osler probably took a far too amiable view of mankind, and that in reality the period when the learning power is nearly obliterated is reached in most individuals very much earlier."[45] While Osler said that forty years of age was the upper age limit that a human being could still be useful, Minot asserted that "few men after twenty-five are able to learn much."[46] If Osler's "fixed period" was the life after forty, it was the period after twenty-five for Minot. Although later gerontologists were well aware of Minot's works, they did not accept this extremely ageist viewpoint.

Aging as a Contingent Phenomenon and a Subject for Experimental Manipulation

The contingency of aging was another important concept that appeared during this period along with the local distinctiveness of aging which Minot and others promoted. In many organisms, biological and biomedical scientists since the late nineteenth century found that aging and death were not unavoidable. Whereas few scholars seriously doubted the inevitability of humans' senile decline, aging itself began to be conceptualized as a contingent phenomenon rather than an inevitable destiny or cosmic process. As the German biologist August Weismann had already stated, aging and natural death was "not a primary necessity" in the germ plasms, unicellular organisms, plant cells, and the "lower Metazoa" with a regenerative capacity.[47] Observing these and other statements, anthropologist and historian Hannah Landecker has correctly pointed out that "immortality was not an unusual topic in the biology of this period."[48]

This new direction in aging research accompanied the beginning and development of experimental approach to senescence. If senescence and death could be evaded in certain conditions, it seemed possible to manipulate aging process experimentally to

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[45] Minot, *The Problem of Age, Growth, and Death*, p. 245.
[46] Minot, *The Problem of Age, Growth, and Death*, p. 246.
[47] Weismann, *Essays upon Heredity*, pp. 24, 27, 32.
[48] Hannah Landecker, "On Beginning and Ending with Apoptosis: Cell Death and Biomedicine," in Sarah Franklin and Margaret Lock (eds), *Remaking Life and Death: Towards an Anthropology of the Biosciences* (Santa Fe, New Mexico: School of American Research Press, 2003), p. 43.

investigate its nature. In fact, this brought about a significant departure from the tradition mode of understanding senescence. If previous scholars took senile decline for granted and tried to explain it with various theories—some of which were based on their clinical observation—the scientists discussed in this section actively and more systematically sought for ways to intervene into the seemingly usual course of life by changing its environmental or internal conditions.

Botanists were among these scientists who contributed to the idea of contingency through their experimental study of plant life. Indeed, many plant species' capacity to regenerate the whole organism if cut into pieces had been known since Aristotle, and some trees' exceptionally long lifespan had fascinated both botanists and lay people.[49] But it was during the late nineteenth century that botanists began to study this property of plants more systematically. They found that some differentiated mature plant cells could be de-differentiated and become meristemic cells that could reinitiate the entire developmental process.[50] Moreover, after Weismann's proposal of the germ plasm theory, it was discovered that plants did not have such a strict distinction between somatic and germ cells. In fact, many plant somatic cells could be induced to become reproductive cells and to proliferate without any limits of longevity. Through plant tissue culture techniques, botanists also revealed that it was possible to culture plants without making them senile, by transferring them to a fresh medium at a regular interval. Of course, temporary organs such as leaves or fruits did undergo senescence with the change of season. Moreover, it was evident that many short-lived plants such as annuals and biennials had a definite duration of life. However, plant parts other than leaves or fruits tended to remain alive for a much longer term, and even the short-lived plant life could be lengthened, sometimes indefinitely, in carefully controlled environmental

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[49] Aristotle, "On Youth, Old Age, Life and Death, and Respiration," in Jonathan Barnes (eds.), *The Complete Works of Aristotle* (Princeton: Princeton University Press, 1984), p. 746.

[50] Friedrich Hildegrand, "Die Lebensdauer und Vegetationsweise der Pflanzen, ihre Ursache and ihre Entwickelung," *Botanische Jahrbücher für Systematik, Pflanzengeschichte und Pflanzengeographie* 1 (1881), pp. 51-135. This article was cited in Weismann, *Essays upon Heredity*, p. 32. Also see Charles Manning Child, *Senescence and Rejuvenescence* (Chicago: University of Chicago Press, 1915), p. 246; Weismann, *Essays upon Heredity*, p. 32.

conditions.[51] For these reasons, the renowned American botanist William Crocker argued that "certain plants are theoretically immortal."[52]

These properties of plants began to be known to other scientists of aging, especially after 1937 when Crocker attended the first conference on aging at Woods Hole and contributed to Cowdry's *Problems of Ageing*. Crocker also participated in the Club for Research on Ageing as a founding member and discussed plant aging with other gerontologists. During their discussion, senescence and death of plants were considered an adequate and proper research subject in experimental gerontology.[53] In fact, the case of plants was highly interesting, because many kinds of plant cells, unlike most somatic animal cells, were potentially immortal.

Herbert Spencer Jennings (1868-1947) and other biologists studying protozoa were also an important group of scholars who promoted the notion of the contingency of aging through experimental study. A number of protozoologists—including Lorande Woodruff and G. N. Calkin—observed that many species of protozoa could be cultured in a vegetative manner without any decline of activity. In certain environmental conditions, however, some species did show decline in vigor and eventually die, and some other species, if cultured in a vegetative way, could not avoid death in any case. Nevertheless, many of them could undergo "rejuvenescence" through conjugation—sexual reproduction—which led to the replacement of worn-out macronucleus by micronucleus that remained dormant during most of the protozoa's life.[54] Newly activated micronucleus could then continue the life of the protozoa which would die otherwise. For this reason, Jennings argued that "senescence and death of the cell are not inevitable accompaniments or results of living."[55]

As a professor of zoology at the Johns Hopkins University, Jennings studied aging of both multicellular and unicellular organisms. During the late 1920s, he studied

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[51] William Crocker, "Ageing in Plants," in E. V. Cowdry (ed.), *Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1939), p. 27.
[52] Crocker, "Ageing in Plants," p. 1.
[53] Minutes of the Meetings of the Club for Research on Ageing, p. 1, 11-12 January 1940, Box 41, Folder 2, EVC
[54] Herbert Spencer Jennings, "Genetics of the Protozoa," *Bibliographia Genetica* 5 (1929), pp. 213-226.
[55] Hebert Spencer Jennings, "Senescence and Death in Protozoa and Invertebrates," *Problems of Ageing*, p. 40.

the aging of rotifers, especially the relation of their age to their siblings' egg size, mortality, and fecundity.[56] Senescence of unicellular organisms was another significant research subject, because this study was related to one of the most critical issues in biology, namely, the mechanism of evolution. The protozoa's "aging" under an inadequate environment could be regarded as evidence for supporting Lamarckian evolution since it was a case showing that environmental influences could cause physiological changes inherited by later generations.[57] Unlike the aging of multicellular organisms, the senescence of protozoa had an evolutionary implication beyond the domain of cells, tissues, and their interactions.

With this research experience, Jennings participated in the development of gerontology when Cowdry edited *Problems of Ageing* during the 1930s. He wrote a chapter on protozoa's and invertebrates' aging in Cowdry's volume and participated in the first scientific conference on aging held at Woods Hole. After retiring from his chair at Johns Hopkins in 1938, however, he moved to the University of California as a research associate and did not attend any following gerontology meetings which were mostly held in the East Coast.[58] Although he kept publishing research articles on senescence of paramecia in his later years, he did not participate in any later activities of gerontologists, probably because he, unlike his immortal unicellular organisms, was too infirm for long-distance travel.[59]

Charles Manning Child (1869-1954), a zoologist at the University of Chicago, also contributed to the notion of contingency of aging. In his book, *Senescence and*

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[56] Herbert Spencer Jennings and Ruth Stocking Lynch, "Age, Mortality, Fertility, and Individual Diversities in the Rotifer Proales Sordida Gosse. I. Effect of Age of the Parent on Characteristics of the Offspring," *Journal of Experimental Zoology* 50 (1928), pp. 345-407; "Age, Mortality, Fertility, and Individual Diversities in the Rotifer Proales Sordida Gosse. II. Life-History in Relation to Mortality and Fecundity," *Journal of Experimental Zoology* 51 (1928), pp. 339-381.

[57] Jennings and Lynch, "Age, Mortality, Fertility, and Individual Diversities. I.," p. 346. In general, however, Jennings thought that both environmental and genetic factors were involved in the changes of protozoa through generations. See Jennings, "Senescence and Death in Protozoa and Invertebrates," p. 48. See also, Jan Sapp, *Beyond the Gene: Cytoplasmic Inheritance and the Struggle for Authority in Genetics* (Oxford: Oxford University Press, 1987), p. 91. His interest in both heredity and environment was a part of his larger and philosophical interest in the nature of life. See Sharon Kingsland, "A Man Out of Place: Hebert Spencer Jennings at Johns Hopkins, 1906-1938," *American Zoologist* 27 (1987), pp. 807-817.

[58] See Jennings to Cowdry, 5 October, 1944, Box 48, Folder 70, EVC.

[59] Hebert Spencer Jennings, "Paramecium bursaria: Life History. I. Immaturity, Maturity and Age," *Biological Bulletin* 86 (1944), pp. 131-145.

Rejuvenescence (1915), he proposed a comprehensive theory on the mechanism of aging and rejuvenation which was based on his own research on coelenterates and flatworms as well as others' work on plants and different animal species. In fact, he had conducted experimental research on regeneration and asexual reproduction of coelenterates and flatworms since the 1900s, which revealed that cells in these organisms could de-differentiate into a type found in earlier stages of life, which could then be used for reproduction and regeneration of damaged parts. This observation showed that aging was not inevitable in some cells of invertebrate multicellular animals, since they could undergo "rejuvenescence" in certain conditions. Since plant cells had the same capacity, and even some vertebrate animal cells seemed to undergo regular rejuvenation in a limited degree, Child argued that "the idea that life proceeds only in one direction from youth to age and death must be abandoned."[60] According to him, "rejuvenescence," which was associated with de-differentiation and regeneration, was "as essential a feature of life as senescence."[61]

Child exerted a certain influence upon later generations of researchers on aging, although some of them were rather critical toward his works. For example, Peter Medawar thought that Child's approach and methodology was too old-fashioned and obsolete to be meaningful for his own work on the evolution of senescence.[62] (See Chapter 3.) But it is important that Korenchevsky mentioned Child's *Senescence and Rejuvenescence* in his "reading list" for the early members of the Club for Research on Ageing.[63] (See Chapter 4.) Moreover, Child influenced Cowdry, a leader in the birth of gerontology in the United States. Child was a biology professor at the University of Chicago, where young Cowdry finished his Ph.D. work. Cowdry was well aware of Child's work and cited it in his writings.[64] (See chapter 5.)

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[60] Child, *Senescence and Rejuvenescence*, p. 186.
[61] Child, *Senescence and Rejuvenescence*, pp. 58-59, 186.
[62] Medawar, "Demography: notes," pp. 3, 9, Box 17, Folder C.26, PBM.
[63] Vladimir Korenchevsky, "Some books and papers on senility," 28 August 1939, Box 41, Folder 12, EVC.
[64] E. V. Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 4 (3)-5(4), undated, Box 142, Folder 1, EVC.

The life and work of the renowned French surgeon Alexis Carrel (1873-1944) should also be discussed, since he established tissue culture as a most significant tool for biomedical research, including biogerontology. His "immortal" chicken heart cells brought a new hope for experimental manipulation of senile process to the scholars who would establish gerontology as a scientific field after the 1930s.

Carrel was born in Lyons, France in 1873, and finished his scientific and medical training at the University of Lyons. Failing to gain a hospital appointment there, he went to Canada in 1904 with a new hope for professional career. Fortunately, his presentation on the new blood vessel surgery techniques that he had been developing impressed many attendees at a medical meeting held at Montreal. Consequently, he was invited to the University of Chicago where he further refined his blood vessel surgery techniques and began to practice tissue and organ transplantation as well. He then went to the Rockefeller Institute for Medical Research through the invitation of Simon Flexner, and in 1912, was appointed a full member of the Institute which was equivalent to a tenured professor position in regular universities. The year of 1912 was important for him for another reason. In that year, he was awarded the Nobel Prize in Physiology and Medicine for his innovation in blood vessel suture techniques and transplantation research.[65]

As Hannah Landecker has pointed out, Carrel's tissue culture originated from this medical and surgical research that he had begun in Lyons and continued at Chicago and the Rockefeller Institute.[66] For Carrel, tissue culture, which had been first carried out by the American embryologist Ross Harrison, was a means to study the most fundamental process of healing at the cell level. While Harrison was interested in observing cells survive and differentiate outside of the body, Carrel wanted to make cells not only survive but also proliferate *in vitro*. This was expected to illuminate how the healing process of reconnected blood vessels and transplanted organs after surgery could proceed within the body. In pursuing this research, Carrel maintained the procedures and practices

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[65] On Carrel's early career, blood vessel surgery, and winning of the Nobel Prize, see Shelly McKellar, "Innovation in Modern Surgery: Alexis Carrel and Blood Vessel Repair," in Darwin H. Stapleton (ed.), *Creating a Tradition of Biomedical Research: Contributions to the History of the Rockefeller University* (New York: Rockefeller University Press, 2004), pp. 135-150.

[66] Hannah Landecker, "Building 'a New Type of Body in Which to Grow a Cell': Tissue Culture at the Rockefeller Institute, 1910-1914," *Creating a Tradition of Biomedical Research*, pp. 151-174.

that he had used for his former surgical operations, such as strict asepsis, artificial circulation of body fluids, and the careful regulation of temperature and humidity.

Tissue culture techniques came to be used for basic biological as well as medical investigations, which included research on the mechanisms of senescence. Indeed, the significance of Carrel's tissue culture for the aging process was observed at the very moment when the technique was devised. In 1911, in one of his earliest papers on tissue culture, he wrote that "it may easily be supposed that senility and death of tissues are not a necessary phenomenon and that they result merely from accidental causes, such as accumulation of catabolic substances and exhaustion of the medium."[67] In fact, his success in continuous culture of chicken's connective tissue outside of the body was dependent upon his prevention of such "accidental causes." When the symptoms of "senility" began to appear among the cells, namely, when "the rate of growth decreased or when large granulations appeared in the cytoplasm of the cells," he extirpated "with a cataract knife the fragment of coagulated plasma containing the original piece of tissue and the surrounding new cells, which [were] washed for several minutes in normal or slightly hypotonic Ringer's solution."[68] The washed cells were then replanted into a fresh and nutritious medium that could bring about "rejuvenation" of the cultured cells. Since this process could be repeated indefinitely, he thought that senescence, at least at the cell level, could be postponed permanently using his intricate tissue culture technique. Indeed, he and his colleagues argued that they kept culturing the cells derived from a chicken's heart for more than thirty years, which was much longer than most chickens' maximum lifespan of approximately ten years.

But this "immortal" tissue of Carrel, whose reality was hardly doubted till the 1960s, became a significant problem thereafter. In fact, based on their own tissue culture experiments, Leonard Hayflick and Paul Moorhead argued in 1961 that normal human diploid fibroblasts could not proliferate indefinitely.[69] Since this seemed to be true of

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[67] Alexis Carrel, "Rejuvenation of Cultures of Tissues," *Journal of the American Medical Association* 57 (1911), p. 1611.
[68] Carrel, "Rejuvenation of Cultures of Tissues," p. 1611.
[69] Leonard Hayflick and Paul S. Moorhead, "The Serial Cultivation of Human Diploid Cell Strains," *Experimental Cell Research* 25 (1961), pp. 585-621.

other types of cells, it was claimed that normal somatic animal cells except for tumor tissues should die after a certain number of divisions, which was later called the "Hayflick limit." With this idea in mind, J. A. Witkowski has claimed that Carrel's culture of "immortal" cells must be a fraud, especially considering Carrel's secretive attitude toward his own work.[70] In a recent monograph, however, Landecker has tried to offer a more historical interpretation on the problem. She has pointed out that understanding the historical reason why Carrel's immortality argument was not seriously challenged till the 1960s is more important than trying to explain, with meager evidence available, why his chicken heart cells did not seem to die for more than thirty years.[71] In quite a different context, biologists have recently begun to question the existence of the Hayflick limit, since not every non-tumor cell should die after a certain number of divisions. It has recently been found that human embryonic stem cells may be cultured without any limit outside of the body.[72]

The current chapter does not attempt to suggest any new interpretation on this problematic issue. Rather, following Landecker, I try to put Carrel and his research in a historical context. Considering this chapter's topic, it is more important to ask what the meaning of his works was with respect to the new conception of aging that arose during the early twentieth century and how influential his research was upon the scientists who constructed gerontology as a scientific field. The answers to these questions should begin with what experiments Carrel actually conducted to understand senescence at the cell level.

With tissue culture techniques, Carrel and his assistants tackled various issues related to aging. Initially, he found that blood plasma was not an effective medium for proliferation of cultured cells, and its meager usefulness further decreased with aging of

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[70] J. A. Witkowski, "Dr. Carrel's Immortal Cells," *Medical History* 24 (1980), pp. 129-142.
[71] Hannah Landecker, *Culturing Life: How Cells Became Technologies* (Cambridge, Mass.: Harvard University Press, 2007), p. 91.
[72] For example, see James Thomson, Joseph Itskovitz-Eldor, Sander Shapiro, Michelle Waknitz, Jennifer Swiergiel, Vivienne Marshall, and Jeffrey Jones, "Embryonic Stem Cell Lines Derived from Human Blastocysts," *Science* 282 (1998), pp. 1145-1147. This article was found from Melinda Cooper, "Resuscitations: Stem Cells and the Crisis of Old Age," *Body and Society* 12 (2006), pp. 1-23.

the individual providing the plasma.[73] While it seemed that each type of cell had its own most effective medium for growth, blood plasma was not optimal for any kind of cell, and its capacity to stimulate growth substantially declined with senescence. What, then, was the most effective medium for tissue culture in general? Carrel found such a medium in a material he got from the youngest body, the ground embryonic tissue extracts which he called the "embryonic juices."[74] The embryonic juices, whose precise biochemical composition was not known, were highly effective in stimulating the growth of cultured tissues when mixed with normal blood plasma.

During the late 1910s and the 1920s, Carrel further studied the factors involved in aging at the level of both the whole organism and the cell. While working at a hospital in France during World War I, he examined with his mathematically trained colleague Pierre LeComte du Noüy the decline of the wound healing rate in accordance with the age of patients.[75] With this experience, Carrel began to study senescence at the cell level in a more quantitative manner after he returned to the Rockefeller Institute after the War. Through his series of experiments using pure culture of fibroblasts, Carrel found that "the rate of cell multiplication [varied] in inverse ratio to the age" of the organism that offered blood plasma as culture media.[76] Since the relationship between age and cell division rate seemed to be quite precise, he even argued that the rate could be used in measuring the degree of changes occurring in blood with senescence. He also studied the character of the factors that were primarily responsible for age-related alterations in blood. Through his experiments on the changes in the proliferating capacity of blood serum after heating and CO_2 precipitation, he argued that there were both "growth-activating" and "growth-inhibiting" substances in serum, and that the former's function decreased while

[73] Alexis Carrel and Montrose T. Burrows, "On the Physicochemical Regulation of the Growth of Tissues: The Effects of the Dilution of the Medium on the Growth of the Spleen," *Journal of Experimental Medicine* 13 (1911), pp. 562-570; Alexis Carrel, "Contributions to the Study of the Mechanism of the Growth of Connective Tissue," *Journal of Experimental Medicine* 18 (1913), p. 289.
[74] Carrel, "Contributions to the Study of the Mechanism," p. 289.
[75] Landecker, *Culturing Life*, p. 81.
[76] Alexis Carrel and Albert H. Ebeling, "Age and Multiplication of Fibroblasts," *Journal of Experimental Medicine* 34 (1921), p. 623.

that of the latter increased with aging.[77] This explained why blood plasma from very old animals could not support the growth of cultured cells any more.

Carrel pursued little research on aging after 1930, probably because of the limitation of his research techniques and his public activities. Although it may be difficult to know all the reasons why he virtually ceased his study of senescence after 1930, it is meaningful to notice that his major methodologies, most of which came from his surgical training, were not adequate for any further elucidation of the subjects he was interested in, including the biochemical or molecular properties of the factors involved in the aging of the cell in relation to its media. Admittedly, he did publish a paper in which he argued that the serum lipoid, lecithin, cholesterol, and some sorts of proteins were growth-inhibiting factors whose concentration in blood increased with senescence.[78] However, he did not have expertise in biochemistry to make a further analysis of the properties and functions of these molecules with respect to aging. In the 1930s, rather than conducting any advanced research on this subject, he spent his time and energy in publishing popular or philosophical works and delivering public lectures.

But Carrel's nonprofessional works were another important part of his career, since they played an important role in creating new discourses on aging among both lay people and professional scientists. In the Progressive Era which highly valued the advancement of human life through scientific innovations, Carrel's research on tissue culture and organ transplantation was enthusiastically welcomed by the general public. For instance, a popular article in 1912 praised the potential usefulness of Carrel's research for rehabilitation medicine. He was described as offering "new organs for old ones."[79] In a language analogous to the modern discourse on stem cells, the reporter wrote, "If the heart is not performing its proper function, what is easier than to throw it away and secure a new one from cold storage"?[80] Carrel's research would eventually make this possible, perhaps in the future. His tissue culture research also became the

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[77] Alexis Carrel and Albert H. Ebeling, "Antagonistic Growth Principles of Serum and Their Relation to Old Age," *Journal of Experimental Medicine* 38 (1923), p. 425.
[78] Lillian E. Baker and Alexis Carrel, "Effect of Age on Serum Lipoids and Proteins," *Journal of Experimental Medicine* 45 (1927), pp. 305-318.
[79] "New Organs for Old Ones," *The Wilkes-Barre Record* (8 June 1912).
[80] "New Organs for Old Ones."

source of imagination and enthusiasm about the immortal life without old age and death. Indeed, a popular article in the *Examiner* claimed that "A GREAT scientist, Dr. Alexis Carrel has shown…that permanent life is not impossible."[81] Another writer in the same year even argued that "a revolt against old age and death" was proceeding primarily due to Carrel's research.[82] His public lecture on his tissue culture delivered at the third "Race Betterment Conference" held at Battle Creek, Michigan in 1928 intensified this popular enthusiasm and hope. An article in *New York Sun* summarized this talk by writing that "given proper environment, proper feeding and care, unicellular organisms….can be maintained in a stage of eternal youth."[83] A reporter of *Brooklyn Eagle* also described Carrel's study of potential immortality at the cell level in a detail, while admitting that the whole organism had to die eventually.[84] Through these popular writings, Carrel's research promoted discourse on the possibility of immortality and the deferment of aging by means of science.

Carrel also advocated the establishment of an institute for aging research. With an invitation by the Association of Life Insurance Presidents, he delivered a lecture on human lifespan at their annual meeting in 1937, and during this lecture he claimed that a research institute for the scientific study of aging should be built.[85] According to Carrel, the lengthening of the human's lifespan would not necessarily be beneficial since "the importance of human life depends on its quality, not on its length."[86] Therefore, it was important to "find the means of improving its quality," and "there should be somewhere in the civilized world an institute dedicated to the study of the process of aging."[87] The scientists in this institute would study "the chemical, physical, and physiological changes

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[81] "Why We Ought to Live 100 Years," *The Examiner* (23 June 1912).
[82] "May We Live 200 Years?" *The Kansas City Star* (30 June 1912).
[83] "Eternal Youth a Dream," *New York Sun* (5 January 1928).
[84] Zoe Beckley, "There is No Eternal Youth, But We Can Curb Old Age, Woman Researcher Says," *Brooklyn Eagle* (19 January 1928).
[85] Vincent P. Whitsitt to Carrel, 30 October 1937, Box 65, Folder 23, AC.
[86] Alexis Carrel, "The Problem of the Prolongation of Life," p. 5, 3, December 1937, Box 23, Folder 15, AC.
[87] Alexis Carrel, "The Problem of the Prolongation of Life," p. 5, 3, December 1937, Box 23, Folder 15, AC.

that manifest the progress of age in tissues" using dogs, monkeys, mice, and rats.[88] This research was necessary, Carrel argued, because "the problem of the prolongation of life has extended beyond the frontiers of hygiene and medicine into an uncharted country," the exploration of which required a new research institute studying novel problems.[89] This institute would certainly improve humans' life, since "there is no example of a scientific search for truth which has not been rewarded."[90]

Carrel's advocacy for an institute for aging research highly impressed both journalists and scientists. Most notably, *New York Times* described his lecture in detail in an article titled, "CARREL URGES FUND FOR STUDY OF AGING,"[91] and *New Health* also summarized Carrel's argument for building an institute for the study of aging.[92] His lecture elicited responses from scientists as well, including Clive McCay at Cornell University who had just found that restricted dietary caloric intake increased longevity. (See Chapter 2.) In his letter to Carrel, McCay wrote that Carrel had recently "[deplored] the neglect of problems of old age by research laboratories."[93] However, McCay added, "you are stimulating interest and the future will be brighter."[94] While McCay then explained his own research on aging and dietary caloric intake, Carrel already knew it very well.

In fact, Carrel interacted with and influenced many scientists who would build gerontology after the 1930s. He was a colleague of Cowdry and Alfred Cohn in the Rockefeller Institute and knew well Ludwig Kast and Lawrence Frank of the Josiah Macy, Jr. Foundation.[95] Carrel also corresponded with Edward Stieglitz, the first head of the Gerontology Unit at the National Institute of Health, as well as William MacNider,

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[88] Alexis Carrel, "The Problem of the Prolongation of Life," pp. 5-6, December 1937, Box 23, Folder 15, AC.
[89] Alexis Carrel, "The Problem of the Prolongation of Life," p. 6, December 1937, Box 23, Folder 15, AC.
[90] Alexis Carrel, "The Problem of the Prolongation of Life," p. 6, December 1937, Box 23, Folder 15, AC.
[91] "Carrel Urges Fund for Study of Aging," *New York Times* (4 December 1937).
[92] "Scientific Study of Senescence," *New Health* (June 1938).
[93] McCay to Carrel, 6 December 1937, Box 64, Folder 9, AC.
[94] McCay to Carrel, 6 December 1937, Box 64, Folder 9, AC.
[95] See, for example, Kast to Carrel, 18 June 1928, Box 42, Folder 12, AC; Frank to Carrel, 14 May 1938, Box 66, Folder 3, AC; Kast to Carrel, 18 June 1928, Box 42, Folder 12, AC. Cohn discussed the problem of life expectancy with Carrel. See Cohn to Carrel, 5 December 1940, Box 41, Folder 22, AC.

the first president of the Gerontological Society.[96] These people deeply respected Carrel and his research. A letter from T. Wingate Todd—a contributor to Cowdry's *Problems of Ageing*—written after visiting Carrel's laboratory exemplifies the feeling of the American scientists of aging toward his contributions.

> Having now returned home to Cleveland, I have had time to get into perspective the thoughts and hopes which flow from my visit to you last Wednesday. The opportunity to chat with you and to look over those living tissues, especially the conversation, was not merely invigorating, but full of imaginative lighting for my thought and I do not wish to let this immediate period of enthusiasm, while the conversation is still vivid in my mind, pass without again thanking you for this privilege.[97]

In fact, the members of the Club for Research on Ageing, which was primarily constituted by the contributors to *Problems of Ageing*, discussed the various issues related to tissue culture and immortality.[98] (See Chapter 5.) Carrel's research and his immortal tissue came to exert a considerable influence upon the thoughts and experimental practices of many gerontologists.

In terms of more technical aspects, the impact of Carrel upon research of early gerontologists can be summarized as follows. First of all, he introduced tissue culture as an experimental tool for studying and manipulating aging at the cell level. As will be discussed in chapter three, Medawar began his career as an experimental scientist with tissue culture, which revealed that even embryonic cells underwent senile changes. Henry Simms, an early member of the Gerontological Society, also used tissue culture to challenge Carrel's argument that blood plasma from older animals inhibited the growth of

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[96] See Stieglitz to Carrel, 26 June 1940, Box 69, Folder 27, AC; MacNider to Carrel, 7 April 1931, Box 58, Folder 88, AC.
[97] Todd to Carrel, 4 April 1938, Box 67, Folder 39, AC.
[98] The Club for Research on Ageing Minutes of Meeting Part I, pp. 1-5, 13-14 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS.

tissue.[99] It is important that Peter Leslie Krohn, a recipient of the Nuffield Gerontological Research Fellowship, also studied aging and immortality of cells using tissue culture. (See chapter 4.) In fact, while scientists in the 1960s such as Hayflick and Moorhead criticized Carrel's immortality argument, it should be noticed that even they used tissue culture as a major research tool. By the 1960s, Carrel's technique of growing cells outside of the body was firmly established as a means of measuring and controlling aging process at the cell level. The second contribution of Carrel is that his finding of each type of cell's unique need for culture medium became a basis of Cowdry's belief that a cell's aging was dependent upon its local fluid environment which determined its rate of senescence as well as growth. (See Chapter 5.) Since different kinds of cells needed different kinds of media, the rate of proliferation and aging of each type of cells should also be distinct from one another. Carrel, along with Minot, contributed to a departure from the older notion that aging in the whole body was caused by a single universal factor.

Despite this influence, Carrel himself did not join the new field of gerontology. Carrel did not accept Cowdry's invitation to contribute to *Problems of Ageing*, which would initiate the process of establishing gerontology in the United States. (See Chapter 5.) While "this would interest me very much," Carrel replied to Cowdry, "unfortunately, the nature of my work this winter makes it materially impossible for me to assume any outside activities."[100] Carrel did not participate in any other early projects and meetings concerning gerontology in America, and died in France in 1944 during his efforts to help his war-torn home country with his scientific expertise.

The Beginnings of Experimental Study of Longevity

As Carrel opened ways to manipulate the aging process at the cell level through experimental means, other scientists devised new methods to alter the longevity of laboratory animals by various methods. While many of these scientists were influenced

[99] Henry S. Simms, Abstract of Presentation at the Meeting of the Club for Research on Aging, 13 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS.
[100] Carrel to Cowdry, 23 October 1936, Box 41, Folder 36, AC.

by Carrel, they conducted research using their own techniques which they learned in their disciplines. Through these researchers' efforts, longevity began to become a subject of systematic experimental approaches.

The works of Jacques Loeb (1859-1924) and John Northrop (1891-1987) at the Rockefeller Institute were probably the first experimental studies of longevity in the early twentieth century. As early as in 1902, after successfully inducing a sea urchin's artificial parthenogenesis, namely, the development without fertilization, Loeb stated that death was not so much a mere negative phenomenon as a specific chemical process that was actively controlled by biochemical machinery.[101] Indeed, he observed that an unfertilized egg's longevity could be extended and its death postponed through the manipulation of the chemical composition of environment. Inducing artificial parthenogenesis was certainly one such way to do so, because unfertilized eggs through this process could avoid death and continue to live. But it was also possible to extend an unfertilized egg's lifespan without provoking parthenogenesis with diluted potassium cyanide, which prevented the unfertilized egg's further development as well as its necrosis. This observation prompted them to think that it was possible to control and to extend the longevity of other organisms using comparable measures that controlled physico-chemical machinery of the cell. In fact, during the 1910s, Loeb and Northrop found that fruit flies' longevity could be extended by lowering their environmental temperature.[102] Since the rate of chemical reactions was known to have temperature coefficients, this finding indicated that the duration of life was also related to some kinds of chemical reactions occurring within the body.[103] This series of experimets illuminates what historian of biology Philip Pauly has called, the "engineering ideal" in biology—the ideal

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[101] Jacques Loeb and Warren H. Lewis, "On the Prolongation of the Life of the Unfertilized Eggs of Sea-Urchins by Potassium Cyanide," *American Journal of Physiology* 6 (1902), pp. 305, 317.

[102] Jacques Loeb and J. H. Northrop, "Is There a Temperature Coefficient for the Duration of Life?" *Proceedings of the National Academy of Sciences of the United States of America* 2 (1916), pp. 456-457; "What Determines the Duration of Life in Metazoa?" *Proceedings of the National Academy of Sciences of the United States of America* 3 (1917), pp. 382-386.

[103] Although the precise identity of these reactions was not known, Loeb and Northrop further studied more details of the phenomenon, especially the length of each life stage of the fruit fly under different temperature conditions. See Jacques Loeb and J. H. Northrop, "On the Influence of Food and Temperature upon the Duration of Life," *Journal of Biological Chemistry* 32 (1917), pp. 103-121.

and enthusiasm for manipulating rather than merely observing the organismic process—of which Loeb was an early advocate.[104]

Loeb and Northrop's experimental manipulation of longevity was deeply related to the discourse on immortality at that time. They were well aware of scientific research on immortal life which was promoted by Weismann, Carrel, and other researchers.[105] If life was inherently immortal, Loeb and Northrop asked, why, then, should many living organisms undergo senescence and death? They thought that there must be some physico-chemical basis of this phenomenon which could be controlled by experimental means. Indeed, their research on the temperature coefficient of fruit flies' longevity was a case study indicating that this physico-chemical basis actually existed.

Loeb and Northrop influenced later developments in experimental longevity research. While coauthoring an article, "On the Influence of Food and Temperature upon the Duration of Life" with Loeb, Northrop also pursued independent research on the relationship between the amount of food and longevity.[106] As I will show in the next chapter, this research, along with other studies by J. R. Slonaker at Stanford and H. Louise Campbell at Columbia, constituted the expanding literature on nutrition and lifespan during the 1910s and 1920s. These investigations formed a basis upon which Clive McCay discovered the relation of dietary caloric restriction to lifespan. It is also important that Loeb's and Northrop's works on the relation of temperature to longevity were known to Raymond Pearl (1879-1940) who conducted his own research on the duration of the fly's life.[107] As I will write later in this section, Pearl regarded Loeb's and Northrop's conclusion as a fact which supported his theory of the "rate of living."

Pearl was born in New Hampshire in 1879 and finished his Ph.D. in 1902 under Herbert Spencer Jennings at the University of Michigan. After teaching zoology for three years at the same university, Pearl went to University College London in 1905 to study

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[104] Philip J. Pauly, *Controlling Life: Jacques Loeb and the Engineering Ideal in Biology* (Oxford: Oxford University Press, 1987), esp. pp. 93-117.

[105] Loeb and Northrop, "What Determines the Duration of Life," pp. 382-383; Jacques Loeb, "Natural Death and the Duration of Life," *The Scientific Monthly* 9 (1919), pp. 578-585;

[106] Loeb and Northrop, "On the Influence of Food and Temperature upon the Duration of Life," pp. 103-121; John H. Northrop, "The Effect of Prolongation of the Period of Growth on the Total Duration of Life," *Journal of Biological Chemistry* 32 (1917), pp. 123-126.

[107] Raymond Pearl, *The Biology of Death* (Philadelphia: J. B. Lippincott Company, 1922), p. 209.

with the renowned biometrician and eugenicist Karl Pearson. After being trained in statistics under Pearson, Pearl returned to America in 1906 and worked at the University of Pennsylvania, the University of Maine, and the Statistical Division of the United States Food Administration. In 1918, he accepted an offer of professorship in vital statistics and biometry at the Johns Hopkins University, where he spent the rest of his academic life and conducted a series of experimental studies of longevity.

Pearl's interest in longevity can be traced back to the British statistical tradition in which he was trained. In fact, the age of death of a human being, along with its causes, had been a subject of various statistical surveys conducted in Britain since the early nineteenth century. During these surveys, the relationship between heredity and longevity was observed by several scientists. First of all, the renowned eugenicist and biometrician Francis Galton briefly mentioned the inheritance of longevity in his book, *Inquiries into Human Faculty and Its Development*.[108] Karl Pearson, a biographer of Galton and the holder of the Galton Chair of Eugenics at University College London, also studied how longevity was influenced by hereditary factors by examining the age of death of family members.[109]

After returning to America, Pearl, as a student of Pearson, used his statistical expertise in studying longevity and mortality of fruit flies as well as humans.[110] First of all, he and his assistant Sylvia Parker depicted the survival curves of their fruit flies which became the basis of devising formulas revealing the mathematical relationship between time and the number of surviving flies in a cohort. These curves and formulas indicated, within a certain margin of error, that the mortality curve of the fly followed a certain pattern which was highly similar to that of humans despite their completely different time scale.[111] (See Figure 1.3.) Another important finding was the fact that

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[108] Francis Galton, *Inquiries into Human Faculty and Its Development* (London: Dent: 1907), p. 212.

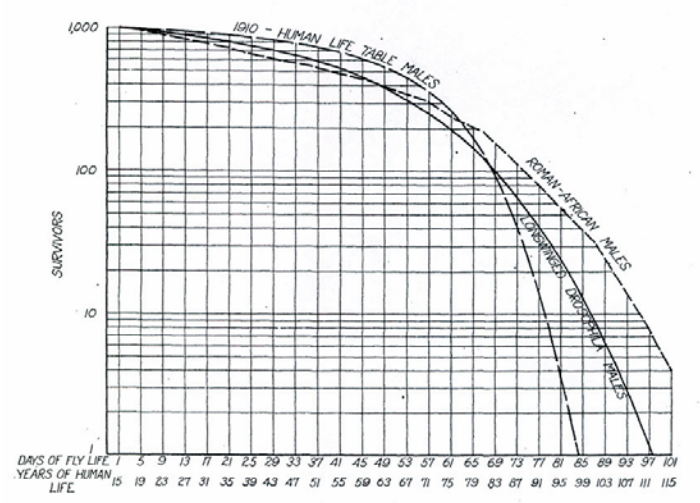
[109] See, for example, M. Beeton and Karl Pearson, "On the Inheritance of the Duration of Life, and on the Intensity of Natural Selection in Man," *Biometrika* 1 (1901), pp. 50-89.

[110] Pearl began to use fruit flies at the suggestion of Jacques Loeb and Thomas Hunt Morgan. See Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. I. Introductory Discussion of the Duration of Life in Drosophila," *American Naturalist* 55 (1921), p. 482.

[111] Pearl and Parker, "Experimental Studies on the Duration of Life. I.," pp. 490-504. He superimposed the human's mortality curves on that of the flies by equating fly's nine days with the human's twenty-three years. The result showed that the people in the 1910s showed a mortality curve which was different from

genetically different strains of flies with distinct outward appearance—such as "longwinged" and "shortwinged"—showed quite different mortality and average longevity, which proved the importance of hereditary factors in lifespan. (See Figure 1.4.) While formal research on the relation of heredity to longevity had been initiated by his predecessors like Pearson, Pearl made it a subject of experimental investigations.[112]

In his later studies, Pearl obtained more evidence supporting his earlier finding using seven distinct lines of flies, each of which showed a different mortality curve as well as the length of lifespan. Interestingly, these fly lines maintained very similar average longevity and mortality even after more than six months' of brother-sister matings.[113] These experiments supported his argument that genetic factors, in a statistical sense, played an important role in determining how long a fly could live.[114]



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that of the fly and the ancient Romans and Africans due the effect of the improved modern hygiene and environment.

[112] The "predecessors" may include the renowned American physician Benjamin Rush (1745-1813), who mentioned that longevity could probably be inherited. See Benjamin Rush, *Medical Inquiries and Observations* vol. 1 (Philadelphia: Griggs and Dickinsons, 1815), p. 235.

[113] Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. II. Hereditary Differences in Duration of Life in Line-Bred Strains of Drosophila," *American Naturalist* 56 (1922), pp. 174-187.

[114] Pearl and Parker's research published in 1924, which used genetically more homogeneous fly lines, revealed the hereditary influence upon longevity more sharply. See Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. IX. New Life Tables for Drosophila," *American Naturalist* 58 (1924), pp. 71-82.

Figure 1.3. *A Comparison of Humans' and Flies' Mortality Curve. Raymond Pearl and Sylvia Louise Parker, "Experimental Studies on the Duration of Life," American Naturalist 55 (1921), p. 502.*

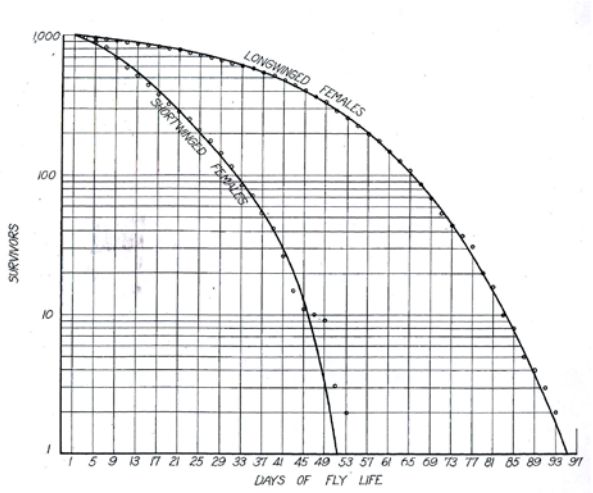

Figure 1.4. *A Comparison of "Longwinged" and "Shortwinged" Flies' Mortality Curve. Raymond Pearl and Sylvia Louise Parker, "Experimental Studies on the Duration of Life," American Naturalist 55 (1921), p. 498.*

Pearl and Parker studied the relation of longevity to factors other than heredity. For example, they investigated whether etherization which researchers often used for counting the number of flies affected the fly's lifespan. Pearl and Parker concluded that "no sensible error will be introduced into duration of life experiments on *Drosophila* as a result of completely anesthetizing the flies with ether."[115] Pearl also studied whether and how ventilation influenced longevity, and claimed that approximately ten percent increase of lifespan was observed after providing adequate ventilation to the bottle containing flies.[116] In a different set of experiments, he found that flies living in a congested environment tended to have a shorter longevity, confirming the British

[115] Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. III. The Effect of Successive Etherizations on the Duration of Life of Drosophila," *American Naturalist* 56 (1922), p. 280.
[116] Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. V. On the Influence of Certain Environmental Factors on Duration of Life in Drosophila," *American Naturalist* 56 (1922), pp. 385-405.

biostatistician William Farr's observation on humans.[117] Certainly, environmental factors should not be neglected in determining the duration of life.

But Pearl argued that heredity played a more important and fundamental role in determining longevity. Citing a statistical study by his British mentor, Karl Pearson, Pearl wrote that family members tended to die at comparable ages, even though they had lived in distinct environments for a long time.[118] He also discovered that fruit flies in the F_2 generation, which were obtained from the brother-sister mating of the F_1 flies that were born between a long-lived and a short-lived strain, were segregated into two kinds of flies whose lifespan closely resembled that of their two grandparents. Pearl and his colleagues also claimed that the ratio of the number of these two fly groups was close to 3:1, and the genes related to longevity were located in specific sites within the chromosome.[119] These results implied that the genes determining lifespan were inherited and segregated through generations following the Mendelian Laws. Another important observation in this regard was made by comparing the lifespan of his and Loeb and Northrops' flies. Pearl found that the lifespan of Loeb and Northrop's fruit flies, which had been grown in aseptic conditions, was shorter than his own flies that were kept in a normal, germ-laden, environment. Since Pearl assumed that the major, or perhaps the only, role that the germ played in longevity was to shorten it by causing diseases, the longer life duration of his own flies meant that they had hereditary factors whose influence upon longevity was stronger than germs which were considered an environmental factor.[120]

While conducting this research during the 1920s, Pearl tried to understand how aging was involved in longevity. He was certainly aware that "the death rates….generally

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[117] Raymond Pearl and Sylvia Louis Parker, "Experimental Studies on the Duration of Life. IV. Data on the Influence of Density of Population on Duration of Life in Drosophila," *American Naturalist* 56 (1922), pp. 312-321.

[118] Pearl, *The Biology of Death*, pp. 170-171.

[119] Usually, the genes related longevity had other phenotypic characters that could be easily observed and had been studied by othere geneticists such as the renowned geneticist Thomas H. Morgan. See Raymond Pearl, Sylvia Parker, and Bienvenido Maria Gonzalez, "Experimental Studies on the Duration of Life. VII. The Mendelian Inheritance of Duration of Life in Crosses of Wild Type and Quintuple Stocks of Drosophila melanogaster," *American Naturalist* 57 (1923), pp. 153-192; Bienvenido Maria Gonzalez, "Experimental Studies on the Duration of Life. VIII. The Influence upon Duration of Life of Certain Mutant Genes of Drosophila melanogaster," *American Naturalist* 57 (1923), pp. 289-325. Also see Pearl, *The Biology of Death*, p. 197.

[120] Pearl, *The Biology of Death*, pp. 201-202.

increase steadily with advancing age."[121] But what was the mechanism through which constitutional changes in the body occurring with senescence affected lifespan? By what degree did aging contribute to longevity? These questions could not be easily answered, because longevity was determined by a complex set of factors. Yet Pearl tried to obtain at least a partial answer to these questions by devising an experiment. Inspired by Alexis Carrel, he investigated whether the embryonic juices that Carrel used for his immortal chicken tissue could change the mortality or longevity of flies in any degree. He fed his flies the embryonic juice Carrel used when their mortality curve showed the signs of senescence. If the embryonic juice could modify the process of aging in any degree, Pearl could measure that degree by observing the change in the flies' mortality curve. Unfortunately, the result of this experiment showed that the embryonic juice hardly altered the lifespan of fruit flies in any meaningful measure.[122]

To Pearl, however, this result did not indicate that Carrel's experiments were not valid. Since Pearl deeply respected Carrel's work and even visited his laboratory to learn more about his immortal cells,[123] Pearl was convinced that Carrel's embryonic juice was certainly effective in prolonging the lifespan of cultured cells, despite the fact that its influence upon the longevity and aging process of the entire organism was not remarkable. Furthermore, in his semi-popular monograph, *The Biology of Death* (1922), he regarded the immortality at the cell level advocated by Carrel as an established conceptual basis upon which he explained other phenomena. What needed explanation was why the living organism, especially multicellular species, had to undergo senescence and death rather than why life at the cell level could be prolonged indefinitely.

Pearl thought that there were other examples besides Carrel's tissue culture experiment that supported this possibility for indefinite prolongation of life. Weismann had already argued that the life of the germ plasm could be continued indefinitely through generations, although soma had to age and perish. The experiments using unicellular organisms, including Jenning's research on protozoa, also indicated that life at the cell

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[121] Pearl and Parker, "Experimental Studies on the Duration of Life. I.," p. 500.
[122] Pearl and Parker, "Experimental Studies on the Duration of Life. V.," pp. 392-397.
[123] Pearl to Carrel, 22 October 1920; Carrel to Pearl, 10 December 1920, Folder Carrel, Alexis, RP.

level could avoid natural death. The same conclusion could be arrived at by observing the properties of tumor tissues investigated by Leo Loeb, as well as the character of plants propagated through a series of grafts. All these cases showed that "life itself is inherently continuous."[124]

Pearl offered a theory on why multicellular animals' lifespan was limited despite this inherently continuous nature of life. According to him, somatic cells in multicellular organisms eventually had to undergo natural death because they were organized in a way that could be disturbed during an organism's lifespan. If a certain part of the organism failed to function normally, other portions in the organized body could also be broken down because the stability of each portion of the body was organically dependent upon that of other parts. Hence, after the initial breakdown occurred, the gradual process leading to the whole organism's death would begin.[125] By proposing this theory, Pearl agreed with Weismann's and Minot's idea that senescence and natural death were the price that the higher forms of life in the evolutionary scale had to pay because of their more advanced and organized structure.

To Pearl, the most important factor determining when this breakdown occurred was heredity rather than environment. Since the gene determined "the way the organism is put together,"[126] it might be assumed that those individuals with a better-organized body inherited from their parents could live longer. Strangely, instead of providing any further explanation on how this occurred, Pearl offered in the same book another account which was very similar to the traditional theory of senile decline. He argued that "heredity determines the amount of capital placed in the vital bank upon which we draw to continue life," while environment only influenced the rate at which the capital was spent.[127] An organism which had inherently a longer span of life could thus be

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[124] Pearl, *The Biology of Death*, p. 48.
[125] Pearl, *The Biology of Death*, p. 147. He thought that this series of breakdown was the primary way aging disrupted the body of multicellular organisms, although he also thought that it was related to the susceptibility of the body parts to disease. Pearl argued that tissues originated from endoderm during embryogenesis were particularly susceptible to breakdowns which would invite diseases, because endoderm was "a very old-fashioned and out-out-date ancestral relic, which causes…an infinity of troubles." See Pearl, *The Biology of Death*, p. 142.
[126] Pearl, *The Biology of Death*, p. 225.
[127] Pearl, *The Biology of Death*, p. 226.

considered having the genes that led to a larger amount of "vital capital" which could be used for a longer period. Admittedly, even this organism might not live long if it was placed in an environmental condition that made it spend its vital capital rapidly. Loeb's and Slonaker's experiments were relevant to this issue, because they showed that animals in a warmer environment or in a condition in which they had to do more physical exercise brought about a higher "rate of living"—which was equivalent to increased metabolic rate—that led to more rapid exhaustion of their vital capital and earlier death.[128] Even in these cases, however, the primary factor determining longevity was heredity, because environment itself could not modify the absolute amount of the vital capital.

To study the problem of longevity more systematically, Pearl established the Institute for Biological Research within the Johns Hopkins University in 1925. The Institute aimed at studying various problems in the life sciences and medicine including hypertension and cancer, although its main research focus was longevity, mortality, and senescence of humans and animals.[129] The Institute was intensively funded by the Rockefeller Foundation which enabled Pearl and his colleagues to investigate various factors that could influence the fruit flies' and other animals' duration of life, such as light, food, and heredity.[130] Humans' longevity, population growth, and health were also investigated with surveys and statistical investigations.

But Pearl was not successful in continuing and expanding this research program in the Institute after 1930. As historian Sharon Kingsland has documented, Pearl had significant career trouble from 1929 to 1930, primarily due to the attack of Edwin Bidwell Wilson, a statistician at Harvard University.[131] Wilson's criticism on Pearl's

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[128] Pearl, *The Biology of Death*, pp. 208-217. Pearl further extended this idea in his later book. See Raymond Pearl, *The Rate of Living: Being an Account of Some Experimental Studies on the Biology of Life Duration* (New York: Alfred A. Knopf, 1928).

[129] See A Report of Progress from July 1, 1925 to February 1, 1926, Folder Institute for Biological Research 1926-1929; Pearl to Edwin R. Embree, 21 December 1925, Folder Embree, Edwin #1 1924-25; "Plans for Research on the Biology of Life Duration and Extension," Folder Plans for Research on the Biology of Life Duration and Extension, RP.

[130] On the Rockefeller Foundation's "Human Biology" Program, see Robert E. Kohler, *Partners in Science: Foundations and Natural Scientists, 1900-1945* (Chicago: University of Chicago Press, 1991), pp. 125-128. Embree's Program was also related to publication of *Human Biology and Racial Welfare* (1930) edited by E. V. Cowdry. See chapter five of this dissertation.

[131] Sharon Kingsland, "Raymond Pearl: On the Frontier in the 1920s," *Human Biology* 56 (1984), pp. 1-18.

competence as a statistician inflicted severe damage upon his reputation as a scientist and frustrated his attempt to move to Harvard to take the chair occupied by the retiring entomologist William Morton Wheeler. After this failure, the Rockefeller Foundation also became more critical of Pearl's research programs, particularly when Max Mason began to direct the Foundation's Natural Science Division.[132] The Foundation ultimately decided to discontinue its funding for the Institute, which thus had to be disbanded.

In retrospect, Wilson's attack upon Pearl reflected a larger movement within the field of population science. Pearl's authority as a population scientist began to be challenged with the growth of social scientific approaches to the population problems.[133] From the 1930s, social scientists of population criticized the alleged biological determinism of Pearl and attributed the stigma of eugenic ideology to him and other biologically oriented population researchers. To the social scientists, who increasingly became a dominant group in the field of population research, Pearl was a misguided biological fatalist whose methodology as well as ideology was not only wrong but also dangerous.

Although Pearl spent the last ten years of his life in frustration and bitter controversy, his contribution to later developments in science was not small. Historian Sharon Kingsland has already pointed out that Pearl's works were "the start of the first systematic effort to apply demographic techniques to animal populations, an approach that would later, after Pearl's time, become important in a different field, population ecology."[134] Another important contribution of Pearl was his pioneering study of genetic factors involved in longevity and his experimental approach. As I will discuss in chapter two, the importance of genetic factors in determining lifespan was not properly

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[132] Kingsland, "Raymond Pearl," p. 15. Also see Joseph S. Ames to Pearl, 13 December 1929, Folder Johns Hopkins University-Correspondence with Joseph S Ames, RP.

[133] Edmund Ramsden, "Carving up Population Science: Eugenics, Demography, and the Controversy over the 'Biological Law' of Population Growth," *Social Studies of Science* 32 (2002), pp. 857-899. In this article, Ramsden analyzes the controversy between social scientists of population and Pearl and other biological scientists as a kind of "boundary work" defined by Thomas Gieryn. For Gieryn's work, see Thomas F. Gieryn, *Cultural Boundaries of Science: Credibility on the Line* (Chicago: University of Chicago Press, 1999).

[134] Kingsland, "Raymond Pearl," p. 8. Also see Sharon Kingsland, *Modeling Nature: Episodes in the History of Population Ecology* (Chicago: University of Chicago Press, 1995), 56-97.

understood by McCay and other early scientists who studied the relationship between life's duration and dietary caloric intake. Yet later researchers of longevity gradually realized the significance of genes and therefore began to use genetically homogenized animals in their experimental works on lifespan. Pearl and his colleagues' works were also read and cited by Medawar who constructed an evolutionary theory of aging. While Medawar thought that Pearl's hypothesis did not explain why any part of the body should break down in the first place before it brought about the unsettling of the whole body's balance, Medawar accepted Pearl's conclusion that "an animal's span of life was governed by inherited factors and was within certain limits subject to experimental modification."[135] In fact, Medawar's evolutionary theory was based on Pearl's conviction that the genes were the primary causal agent in the symptoms of senescence, and this conviction was shared by Medawar and later scientists of aging. For instance, George Williams, who proposed the "antagonistic pleiotropy theory" of the evolution of aging after Medawar, also used in his work a paper published by Bienvenido Gonzalez, a colleague of Pearl in the Department of Biometry and Vital Statistics at Johns Hopkins.[136] (See Chapter 3.)

In retrospect, Pearl and Medawar shared an important scientific methodology, statistics, which would constitute a part of gerontology's multidisciplinary structure. It is significant that Pearl was trained in Britain, where Medawar also lived and studied. As historian Theodore Porter has pointed out, Britain was the country where modern mathematical statistics was created.[137] There, Medawar, by interacting with the leading statistical scholars in the country like R. A. Fisher, and Pearl, by studying under an early champion of mathematical statistics, Pearson, came to introduce statistical approach into the science of aging. As chapter five will also show, Louis I. Dublin, a contributor to

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[135] Peter B. Medawar, "Old Age and Natural Death," *The Modern Quarterly* 2 (1946), pp. 38, 40. The same kind of criticism can be found in Thomas Hunt Morgan's letter to Pearl. See Morgan to Pearl, 8 October 1921, Folder Morgan, Thomas Hunt #1, RP.

[136] George C. Williams, "Pleiotropy, Natural Selection, and the Evolution of Senescence," *Evolution* 11 (1957), p. 400; Gonzalez, "Experimental Studies on the Duration of Life VIII," pp. 289-325. For much later works which still cites Pearl, see, for example, James Curtsinger, "Density and Age-specific Mortality," *Genetica* 96 (1995), pp. 179-182.

[137] Theodore M. Porter, *The Rise of Statistical Thinking, 1820-1900* (Princeton: Princeton University Press, 1986), p. 255.

Cowdry's *Problems of Ageing*, was also a pioneer of statistical approach in American gerontology, and another important statistician Alfred Lotka gave Medawar an important theoretical insight. While statistics, as Porter has shown, had developed during the nineteenth century as a "truly interdisciplinary" research tool shared by a number of research fields—such as physics, astronomy, psychology, biology, and the social sciences—during the early twentieth century it became incorporated into gerontology as a part of its multidisciplinary structure.[138]

Pearl's place in the history of early gerontology was important in terms of human relationship as well. The fact that Dublin and Lotka were Pearl's close professional colleagues shows how significant statistics' contribution to gerontology was at that time.[139] It is also important that Pearl was a graduate student of Jennings during his doctoral training at the University of Michigan and later became his professional colleague at Johns Hopkins. Moreover, Pearl knew well and interacted with F. A. E. Crew, a British geneticist and an early member of the British Society for Research on Ageing, and regularly met Cowdry at Woods Hole, Massachusetts where the Marine Biological Laboratory was located.[140] (See Chapter 5.) Pearl contributed to *Human Biology and Racial Welfare* (1930) edited by Cowdry.[141]

Pearl's research represented the three new standpoints concerning senescence that arose during the first half of the twentieth century.[142] First, his works reflected the new ideal that aging could be manipulated through experimental means. By appropriate selection, crossing, and environmental alterations, he showed that it was possible to make an organism that could live longer or shorter. Second, his monograph, *The Biology of Death* (1922), provided further support for Carrel, Jennings, and others' argument that

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[138] Porter, *The Rise of Statistical Thinking*, p. 8.

[139] For the relation of Pearl to Lotka and Dublin, see, for example, Lotka to Pearl, 28 June 1921, Folder Lotka, Alfred J Apr-June 1921, RP; Dublin to Pearl, 29 April 1930, Folder Dublin Louis I, RP.

[140] Crew was a British member of the International Union for the Scientific Investigation of Population Problems in which Pearl played a leading role. See F. A. E. Crew and Eldon Moore, "Outline of Problems to Be Investigated by Commission II on Differential Fertility, Fecundity and Sterility," Folder Crew F A E #1, RP.

[141] Cowdry to Pearl, 21 July 1926; Pearl to Cowdry, 24 July 1926, Box 158, Folder 12, EVC.

[142] It is quite noticeable that Pearl's works, containing these new perspectives, were cited by many contributors to Cowdry's *Problems of Ageing*, including Jennings, Todd, Dublin, MacNider, Edward Krumbhaar, and Earl T. Engle.

aging was a contingent phenomenon, at least at the cell level. This book, which was the first synthetic account on the state of aging research, summarized for both professional and general readers what the scientists of aging were doing. Third, his theory of aging supported the idea that aging was a local phenomenon. According to him, senile changes leading to death began with the breakdown of a certain part of the body which led other portions to be broken down as well. In this process, each body part's aging began in a distinct phase and proceeded at a unique rate.

But Pearl himself did not join the new field of gerontology when it began to be constructed by the efforts of Cowdry and others during the 1930s. A possible reason can be found in the fact that there were substantial troubles between Cowdry and Pearl during the editorial process for *Human Biology*.[143] Since Pearl refused to follow many of Cowdry's editorial comments on his chapter, Cowdry probably did not want to invite Pearl for his later projects leading to the establishment of gerontology.

Yet another, perhaps more important, reason is related to Pearl's view on aging itself. As historian Garland Allen has pointed out, Pearl was an active advocate of hereditarianism throughout his whole life, even though he became more critical of eugenics in his later career.[144] Pearl argued that those who carried favorable genetic traits had to be encouraged to reproduce while those who did not should not have children. As far as the records show, however, Pearl did not clearly assert the eugenic implication of longevity, even though he was confident that longer life was an inherited trait. Why didn't he do so? While the genes for longer life might make their carriers live a healthier life, it was uncertain from the perspective of population whether it was really beneficial to encourage those who had such genes to reproduce. To Pearl, the reason was simple. Longer life of these people would eventually contribute to the aging of the population and thereby increase the social cost. He thought that the expansion of the elderly population opened a way toward an inefficient, unhealthy, and senile society which would be never

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[143] See, Pearl to Cowdry, 23 January 1928, Folder Cowdry E V, RP. But it was certain that Cowdry was influenced by Pearl's study of longevity. (See Chapter Five.)

[144] Garland E. Allen, "Old Wine in New Bottles: From Eugenics to Population Control in the Work of Raymond Pearl," in Keith R. Benson, Jane Maienschein, and Ronald Rainger, *The Expansion of American Biology* (New Brunswick: Rutgers University Press, 1991), pp. 231-261.

good for the progress of the American civilization. With this viewpoint on the elderly, he even made the following remark during his lecture for the centennial celebration of the American Statistical Association in 1940,

> The wisdom of the founding fathers led them to the view that youngsters under 21 years of age were, on the whole, too foolish to be entrusted with the power of the vote. But…it apparently never occurred to them that there might conceivably be an age *beyond* which people would also be too foolish to be allowed to vote.[145]

This statement, which was reported through several newspapers and magazines, shocked many elderly people and prompted them to send angry letters to Pearl.[146] In fact, the scientists who constructed gerontology at that time had a very different outlook. Few of them denied the elderly's social place and political right like Pearl, and a major aim of gerontology was to promote a meaningful social and vocational life for the aged. In this respect, Pearl was not a scholar who was expected to join gerontology, and there is no record that any of the members of the Club for Research on Ageing encouraged him to join them. But even if they had invited Pearl, he would not have been able to accept the offer; he suddenly died in November, 1940, perhaps due to heart attack, when he was sixty-one years old.

Conclusion

This chapter traced the birth and development of new ideas and approaches in aging research in the early twentieth century. I have argued that the concepts of contingency and local distinctiveness, along with experimental approaches, were introduced into aging research through the development of the modern biological and biomedical sciences of the early twentieth century. While the traditional idea on aging

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[145] Raymond Pearl, "The Aging of Population," *Journal of the American Statistical Association* 35: 209: Pt 2 (1940), p. 294.

[146] See the papers in Folder Pearl Raymond "No Ballot for Aged" (Speech-replies), RP. See, in particular, "No Ballot for Aged," *La Grande Evening Observer* (6 January 1940); J. R. McKeevan McAllen to Pearl, undated; Al Mechem to Pearl, 8 January 1940, Folder Pearl Raymond "No Ballot for Aged" (Speech-replies), RP.

postulating the inevitable decline due to one critical factor continued, the new perspectives and methodologies became a basis of creating the novel field of gerontology. These developments showed that while Paul de Kruif's wish to remain a young and playful boy without aging and death could not be realized, the new science of aging he enthusiastically described was preparing its way for maturing as a scientific field.

But a number of scientists—most notably, Carrel and Pearl—whose works were discussed in this chapter did not participate in organizing the field of gerontology. Indeed, some of them had already died or became too infirm when the efforts for creating the field began during the late 1930s. Others were explicitly unsympathetic and even hostile toward the elderly, and scientists like Carrel were probably too busy in pursuing other affairs or different research projects to join the field. In fact, few of these researchers thought that they could become professional scientists whose major job was to study aging.

By analyzing the major participants in early gerontology, the following chapters will make a detailed analysis of what changed this situation. The concepts and approaches discussed in this chapter were not enough to lead scientists to build gerontology that became a multidisciplinary research field in which scholars with various backgrounds and methodologies could participate. This field could be created, I will argue in chapter five, when the Western society met a difficult condition, the Great Depression. The next chapter will discuss another important factor driving the change, the development of new research programs that could be used as focal point of multidisciplinary interaction among the scientists of aging. The life and work of the renowned nutrition scientist Clive McCay can be regarded as an exemplary case in the formation of these research programs.

Chapter 2

"The Thin Rats Bury the Fat Rats" Clive Maine McCay and the Beginning of Research on Caloric Restriction, Longevity and Aging

Clive Maine McCay (1898-1967) created a research program on caloric restriction and longevity which held a significant place in the early history of gerontology.[1] Although several people, including, most notably, the Italian nobleman Luigi Cornaro (1468-1566), had claimed that small amounts of food could bring about a longer life, McCay and his colleagues transformed these claims, derived from personal experience and episodic observation, into a formal research project based on systematic experiments on the relation of longevity, aging, and caloric intake. Through his series of experiments during the 1920s and 1930s, he and his colleagues at Cornell University found that a restriction in dietary caloric intake substantially retarded aging and increased the longevity of experimental animals. Since then, his research has been established as the basis of a highly successful research program on the relation of diet to lifespan and senescence. Currently, many prestigious biomedical researchers are actively studying how and why restricted caloric intakes can make a positive contribution to the length of life. While still controversial, the application of McCay's discovery to humans has also been seriously considered and discussed by both scientists and the general public.

By examining two historical issues, this chapter discusses how McCay and his colleagues created such a highly successful scientific program. First, I will analyze the

[1] Following McCay's own words, I use the term "caloric restriction" rather than "calorie restriction" or "dietary restricton." Clive M. McCay and his works have barely been historically studied. For secondary literature, see Patricia B. Swan, "To Live Longer, Eat Less! (McCay, 1934-1939)," *Journal of Nutrition* 127 (1997), pp. 1039S-1941S; J. K. Loosli, "Clive Maine McCay (1898-1967)—A Biographical Sketch," *Journal of Nutrition* 103 (1973), pp. 3-10; Jeanette B. McCay, *Clive McCay, Nutrition Pioneer: Biographical Memoirs by His Wife* (Charlotte Harbor, Florida: Tabby House, 1994); "Clive M. McCay," in W. Andrew Achenbaum and Daniel M. Albert (eds.), *Profiles in Gerontology: A Biographical Dictionary* (Westport, Conn.: Greenwood, 1995), pp. 232-233.

institutional and intellectual factors that made McCay's initial discovery possible. Particularly, I will discuss how his training in the science of nutrition at Berkeley and Yale and the research priorities at the animal husbandry department of the New York State College of Agriculture at Cornell shaped the direction of his early investigation of longevity and aging. Second, I will investigate how the scope and the meaning of this research, which was a small project in animal husbandry and agriculture, became considerably expanded from the mid-1930s, when American science underwent major changes in its funding priorities and institutional structures. I will analyze how these changes brought about McCay's interactions with scholars in various fields such as physiology, dentistry, pharmacology, physics, and psychology, who would employ McCay's method in their diverse research projects. This made McCay's investigation a channel of multidisciplinary communication which was critical for the development of gerontology at that time. Moreover, I will also show that McCay's research became deeply assimilated to American culture as a way to live long and healthy life, even though its direct application to humans was still thought to be questionable.

These discussions on McCay offer an opportunity to reexamine an important issue on the multidisciplinarity in gerontology. While several historians have discussed how gerontology became a multidisciplinary scientific field during the 1930s and 1940s through cooperation of biologists, physicians, sociologists, and psychologists, most historians described gerontologists' research on aging as if all of them had been ultimately concerned with human aging.[2] Even when they discuss the studies of the aging of nonhuman beings such as rats, mice, or cultured tissues, they still implied that these studies had been done as models of human senescence. Yet this view does not reflect the wide scope of gerontology as revealed, for example, in the list of authors in E.

[2] See, for example, W. Andrew Achenbaum, *Crossing Frontiers: Gerontology Emerges as a Science* (Cambridge: Cambridge University Press, 1995); Stephen Katz, *Disciplining Old Age: The Formation of Gerontological Knowledge* (Charlottesville: University Press of Virginia, 1996). Also see W. Andrew Achenbaum, *Old Age in the New Land: The American Experience since 1790* (Baltimore: Johns Hopkins University Press, 1978); Thomas R. Cole, *The Journey of Life: A Cultural History of Aging in America* (Cambridge: Cambridge University Press, 1992); Jesse F. Ballenger, *Self, Senility, and Alzheimer's Disease in Modern America: A History* (Baltimore: Johns Hopkins University Press, 2006); Gerald J. Gruman (ed.), *Roots of Modern Gerontology and Geriatrics* (New York: Arno, 1979); Carole Haber, *Beyond Sixty-Five The Dilemma of Old Age in America's Past* (Cambridge: Cambridge University Press, 1983).

V. Cowdry's *Problems of Ageing* (1939), which included a protozoologist, a botanist and an entomologist as well as a scientist of animal husbandry. As experts in their own field, many of them were interested in the aging of the organisms they studied for its own sake. Among them, McCay was a professor of the department of animal husbandry in the New York State College of Agriculture at Cornell University, and one of his professional duties was to find ways to lengthen the productive lifespan of livestock and other domesticated animals, such as cows, dogs, goats, and trout. Even when he experimented with rats, he always had this job in mind and emphasized that the end result of his research would be useful for breeders, farmers, and commercial food producers. By illuminating this neglected aspect of gerontology, I will show that the spectrum of multidisciplinarity in gerontology was much broader than has often been described.

The latter part of this chapter deals with the actual construction process of this multidisciplinarity by examining how McCay's research which was initiated for a particular purpose—animal husbandry and agriculture—transcended its origin and became a project relevant for many disciplines and fields. I will show that this began in 1936 with a research grant from the Natural Science Division of the Rockefeller Foundation under Warren Weaver's directorship. As a recipient of a substantial grant from the Division, McCay was encouraged to develop multiple lines of attack on the problem, in order to follow the Foundation's policy which aimed at building cross-disciplinary projects combining two or more scientific expertise and disciplines. Interestingly, the science of gerontology was also developing during the same period, and by joining this new multidisciplinary field McCay gained another opportunity to extend the scope of the research initiated by him. His research was welcomed by gerontologists from various backgrounds who regarded it as an important way of manipulating aging processes through experimental means. By participating in these scholars' professional associations such as the Club for Research on Ageing and the Gerontological Society, McCay could make his research a legitimate part of the multidisciplinary science and could attract gerontologists who were willing to do further research on the topic with their expertise.

Fishery, Game, and Nutrition: The Early Years, 1925-1930

Clive Maine McCay was born in Winamac, Indiana in 1898. (See Figure 2.1.) He finished his undergraduate education in chemistry at the University of Illinois in 1920 and his master's degree in biochemistry at Iowa State College in 1923. He then went to the University of California, Berkeley to study biochemistry and nutrition under C. L. A. Schmidt and took two courses from the renowned nutrition scientist E. V. McCollum, who was then temporarily at Berkeley.[3] After earning his Ph.D. degree in 1925, he was awarded a National Research Council Fellowship that enabled him to go to Yale University to study nutrition with Lafayette B. Mendel.

Figure 2.1. *Clive Maine McCay Weighing One of His Rats. 8 Jan. 1962. Folder McCay, Clive Photos, Cornell University Faculty File, Cornell University Archive.*

McCay was very fortunate, because the science of nutrition was growing rapidly at that time, especially at Yale under the strong leadership of Mendel and Thomas B. Osborne. They had trained E. V. McCollum as well as a large number of eminent biochemists and nutrition researchers, many of whom were women.[4] McCollum had established the rat as a standard experimental animal in nutrition research and discovered

[3] "Programme of the Final Public Examination for the Degree of Doctor of Philosophy of Clive Maine McCay," 18 April 1925, The University of California Graduate Division. Folder Montana, Box 47, CMM; McCay to MacNider, 12 March 1942, Folder McCay, Clive [comments listed], Box 6, NWS.

[4] Margaret W. Rossiter, "Mendel the Mentor: Yale Women Doctorates in Biochemistry, 1898-1937," *Journal of Chemical Education* 71 (1994), pp. 215-219. Also see Joseph S. Fruton, "Thomas Burr Osborne and Chemistry," *Bulletin for the History of Chemistry* no. 17/18 (1995), pp. 1-8.

essential dietary elements such as vitamin A, whose absence in rats' diets retarded growth.[5] Mendel and Osborne, too, were involved in vitamin research and also studied how the absence of specific amino acids within certain protein diets fed to rats caused the failure of their growth and health, and the addition of these amino acids to the diet brought back their normal pattern of development and life.[6] It is during this research program that Mendel and Osborn observed an interesting fact which would become the basis of McCay's later research on longevity. In 1917, after a series of experiments on how long the rats on a defective food could remain in the state of retarded growth before resuming development on a normal diet, they discovered that these underfed rats lived longer than the control groups that had been fed a normal diet.[7] But this experiment was prematurely terminated because of the death of the two underfed rats due to a lung disease. In the same year, John H. Northrop at the Rockefeller Institute for Medical Research also found that the lengthening of the larval period of fruit flies by feeding them inadequate diets increased their total lifespan, although he was interested not so much in aging and longevity as in confirming that "the relative duration of each [stage in life] is independent of that of the other two stages."[8]

McCay's opportunity to continue similar research came when he joined Mendel's Yale research group and studied the brook trout's nutrition at the Connecticut State Hatchery with the support of fishing and game enthusiasts under the authority of the State Board of Fisheries and Game.[9] As the name of the sponsor suggests, McCay's work was

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[5] E. V. McCollum and Marguerite Davis, "The Necessity of Certain Lipins in the Diet during Growth," *Journal of Biological Chemistry* 15 (1913), pp. 167-175. On McCollum's early career, see Harry G. Day, "The Fruitful Role of E. V. McCollum in Herbert Hoover's U. S. Food Administration during World War I," *Perspectives in Biology and Medicine* 40 (1996), pp. 7-17.

[6] Thomas B. Osborne and Lafayette B. Mendel, "Amino-Acids in Nutrition and Growth," *Journal of Biological Chemistry* 17 (1914), pp. 325-349.

[7] Thomas B. Osborne, Lafayette B. Mendel, and Edna L. Ferry, "The Effects of Retardation of Growth upon the Breeding Period and Duration of Life of Rats," *Science* 45 (1917), pp. 294-295. For the origin of Osborne and Mendel's research on growth retardation and other scientists' influence on them, see Thomas B. Osborne and Lafayette B. Mendel, "The Suppression of Growth and the Capacity to Grow," *Journal of Biological Chemistry* 18 (1914), pp. 95-108; "The Resumption of Growth after Long Continued Failure to Grow," *Journal of Biological Chemistry* 23 (1915), pp. 439-454.

[8] John H. Northrop, "The Effect of Prolongation of the Period of Growth on the Total Duration of Life," *Journal of Biological Chemistry* 32 (1917), p. 126.

[9] Indeed, before coming to Yale, McCay began to work on the impact of water pollution upon local fish population for the Fish and Game Commission of California. See Jeanette McCay, *Clive McCay*, p. 273.

supported by hunters and anglers who initiated the first conservation movements out of concerns over the decrease of their game animals with pollution and commercial hunting. These movements were important for the development of science as well, because it became a basis of systematic research on the conservation of nature.[10] Since McCay was a scientist involved in this research, he studied nutrition to devise a better diet for brook trout which were reared in hatcheries and released into local rivers to supplement the decreasing number of their wild counterpart. In his lecture delivered to local fish culturalists, McCay argued that his research was a "cooperative effort of two different types of experimenters, first the man who knows how to hatch and rear fish and has a good plant at his disposal and second the man who has devoted his entire efforts to chemistry, physiology, and nutrition."[11]

McCay and his team tried to discover an ideal food and its chemical composition for brook trout at the Burlington State Fish Hatchery. According to him and his colleagues, this project was initiated to rear trout with an inexpensive artificial diet which nevertheless contained all essential nutrients at a time when the increasing price of fish food had become a burden for many hatcheries.[12] McCay was particularly interested in understanding the identity of factor "H" in raw meat whose absence resulted in retarded growth even when all the other essential nutrients were provided.[13] But growth was not the only problem he was concerned about. Since the brook trout was a kind of game fish for anglers, the food also had to fulfill another requirement—"turning out a

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Also see retyped letter of McCay to Beyer, January 14, 1925, Folder CMM Letters Berkeley-New Haven 1925, Box 50, CMM.

[10] John F. Reiger, *American Sportsmen and the Origins of Conservation* (New York: Winchester, 1975), esp. pp. 114-151; Daniel J. Philippon, *Conserving Words: How American Nature Writers Shaped the Environmental Movement* (Athens: Univ. Georgia Press, 2004), esp. pp. 159-218; Thomas R. Dunlap, *Saving America's Wildlife: Ecology and the American Mind, 1850-1990* (Princeton, N.J.: Princeton University Press, 1988), pp. 5-17, 65-97.

[11] According to his wife, Jeanette B. McCay, this lecture was published as Clive M. McCay, "Goals in Nutrition," *Transactions of the American Fisheries Society* 57 (1927), p. 265. See Jeanette McCay, *Clive McCay*, p. 276.

[12] McCay, "Goals in Nutrition," p. 261; Clive M. McCay, F. C. Bing, and W. S. Dilley, "The Effect of Variations in Vitamins, Protein, Fat and Mineral Matter in the Diet upon the Growth and Mortality of Eastern Brook Trout," *Transactions of the American Fisheries Society* 57 (1927), pp. 240-241; Franklin Bing, "A Progress Report upon Feeding Experiments with Brook Trout Fingerlings at the Connecticut State Fish Hatchery," *Transactions of the American Fisheries Society* 57 (1927), p. 277.

[13] Clive M. McCay and W. E. Dilley, "Factor H in the Nutrition of Trout," *Transactions of the American Fisheries Society* 57 (1927), pp. 250-260.

product[trout]," which "is sufficiently hardy to readily adjust itself after it is freed in the stream" so that "the most atrocious tale of a broken line must come true after the virtuous fisherman comes in contact with a virulent trout."[14] A successful diet thus had to support adult fish's characteristics such as the longevity, reproductive power, and the ability to maintain its health in their environment as well as normal growth.[15]

McCay's team investigated the effect of various dietary components—such as protein, vitamins, and minerals—upon these characteristics of brook trout. In particular, to understand the nutritive value of different amounts and kinds of protein, they fed the trout synthetic diets consisting of low, medium, and high protein supplied in the form of casein, while giving other groups of trout dried skimmed milk as the control food. In terms of growth, the trout with dried skimmed milk grew considerably, although their size was still smaller than that a trout was supposed to attain if it was fed raw meat as the protein source.[16] The fish with medium and high protein diets made of casein showed only a moderate level of growth but were larger than those with the low protein diet. Interestingly, however, many of the last group of trout survived when others were dying after the eleventh week. Even in the twentieth week, seventeen trout among the fifty that had received the low protein diet were still alive, whereas those with mid- or high protein food completely perished and less than ten trout remained in the group fed with dried skimmed milk.[17] At that point, McCay divided the trout on the low protein diet into two, and gave one group raw liver as the source of protein while keeping the other group on the low protein diet. While the latter group died soon, the former resumed their growth and remained active even when almost all the other trout had disappeared. Indeed, this result could be seen as a demonstration that what Mendel and Osborne had already shown with their rats was also true for the trout.[18] Why, then, did the low protein diet bring about such an effect? Since complete fasting led to the weight decrease, the low level of protein, he thought, seemed to be used for weight maintenance if not its increase. As it

[14] McCay, "Goals in Nutrition," pp. 261, 263.
[15] McCay, "Goals in Nutrition," p. 263.
[16] McCay, Bing, and Dilley, "The Effect of Variations," p. 244.
[17] McCay, Bing, and Dilley, "The Effect of Variations," p. 248.
[18] One of Mendel and Osborne's papers was cited. But it was not their 1917 paper which explicitly mentioned longevity of rats. McCay, Bing, and Dilley, "The Effect of Variations," p. 247.

was known that the level of dietary caloric intake was related to weight, he concluded that the protein in this diet was used as a calorie source.[19]

Yet confirming the effect of underfeeding on longevity was only a small part of their project at that time. In their papers published in 1927 and 1928, McCay and his team gave much less emphasis to their discovery on trout's longevity than to the effect of vitamins, fat, and minerals upon their growth and the identity of factor H in raw liver that did not seem to exist in the synthetic diets.[20] In their 1929 article on an advanced study of the same topic, however, McCay, W. E. Dilley, and M. F. Crowell clearly stated that "trout stunted upon low protein diet live twice as long as those that are allowed to grow upon similar synthetic rations with a higher protein level."[21] "This seems to indicate," the authors thought, "that their bodies contain a store of some substance that is essential for life but is consumed in growth."[22] While this article, which was written after McCay moved to Cornell, still did not deal with longevity as a major point of interest and never mentioned aging as a research subject, he began to study these issues as his main topic after 1930.

Rats, Calories, and Aging in the Department of Animal Husbandry at Cornell

While at Yale, McCay's research highly impressed Leonard Maynard, a professor of the department of animal husbandry within the New York State College of Agriculture at Cornell University.[23] Maynard thus offered McCay an assistant professorship at

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[19] John W. Titcomb, Eben W. Cobb, Mary F. Crowell, and C. M. McCay, "The Nutritional Requirements and Growth Rates of Brook Trout," *Transactions of the American Fisheries Society* 58 (1928), p. 218. The relation of caloric intake to weight was widely discussed among both the lay public and professional scientists during the 1920s. See Margaret A. Lowe, "From Robust Appetites to Calorie Counting: The Emergence of Dieting among Smith College Students in the 1920s," *Journal of Women's History* 7 (1995), pp. 37-61.

[20] McCay, Bing, and Dilley, "The Effect of Variation," pp. 246-247; Clive M. McCay, F. C. Bing, and W. E. Dilley, "Factor H in the Nutrition of Trout," *Science* 67 (1928), pp. 249-250.

[21] Clive M. McCay, W. E. Dilley, and M. F. Crowell, "Growth Rates of Brook Trout Reared upon Purified Rations, upon Dry Skim Milk Diets, and upon Feed Combinations of Cereal Grains," *Journal of Nutrition* 1 (1929), p. 245.

[22] McCay, Dilley, and Crowell, "Growth Rates of Brook Trout," p. 245.

[23] According to a biography of Maynard, one of Maynard's major achievements was the recognition of "the genius of those around him" such as McCay. See Daphne A. Roe, "Leonard Amby Maynard," *Biographical Memoirs of the National Academy of Sciences*, vol. 62 (Washington D.C.: Government Printing Office, 1993), p. 304.

Cornell's animal husbandry department, which McCay accepted in 1927. Thereafter, McCay's research came to be influenced by the research environment of the animal husbandry department and the larger goals of the State College of Agriculture. (See Figure 2.2.)

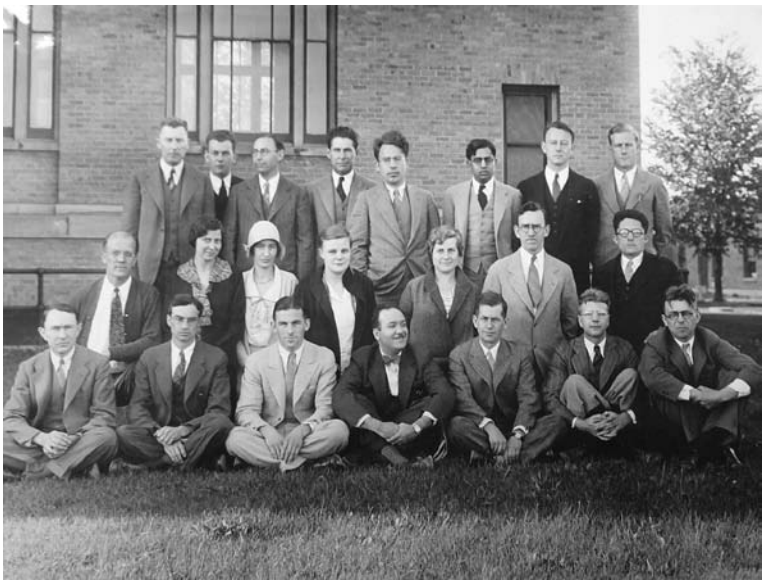

Figure 2.2. *Nutrition Seminar in the Animal Husbandry Department, 1930-1931. McCay is the first person from the right in the second row. Mary Crowell, his graduate student and coauthor of the famous 1934 paper, is the fourth from the right in the second row. Leonard Maynard is the third from the right, in the front row. Box 30, The Clive Maine McCay Papers, Cornell University Archive.*

Cornell's Agricultural College was established by New York State in 1904 to "improve the agricultural methods of the State; to develop the agricultural resources of the State in the production of crops of all kinds, in the rearing and breeding of live stock, in the manufacturing of dairy and other products, in determining better methods of handling and marketing such products, and in other ways; and to increase intelligence and elevate the standards of living in the rural districts."[24] Scientific investigation for solving practical problems of farmers and breeders of New York State was thus one of the most important jobs of the College professors, whose salary and the majority of their research

[24] *Cornell University Official Publication: The Register for 1927-1928* (Ithaca, New York: Cornell University Press, 1928), p. 79.

expenses were coming from the State budget and New York's portion of the federal funds appropriated for encouraging systematic agricultural research.[25] Indeed, as Charles Rosenberg, Diane Paul, Barbara Kimmelman, and Jonathan Harwood have pointed out, agricultural colleges and state experimental stations attached to them were the institutions where such agricultural research was pursued, which provided a basis of many important developments in the basic as well as practical life sciences during the early twentieth century, such as the discovery of vitamins and the introduction of Mendel's law of heredity into the United States.[26] Cornell's Agricultural College was one of these institutions that had provided its faculty and graduate students a strong support for both basic and agricultural investigations.

McCay's previous training and research experience fit well within this new academic niche. At Berkeley, McCay had taken courses in nutritional chemistry with E. V. McCollum, who discovered vitamin A at the University of Wisconsin where he had been hired to solve nutrition problems of farm animals. At Yale, McCay worked with Mendel, who, along with his colleague Osborne, investigated nutrition at the Connecticut Agricultural Experimental Station. McCay himself had studied fishery as a part of animal husbandry at the Burlington State Hatchery, where he observed an important

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[25] *The Register for 1927-1928*, p. 80. About the political contexts of the introduction of these laws, see Charles E. Rosenberg, "The Adams Act: Politics and the Cause of Scientific Research," in *No Other Gods: On Science and American Social Thought* (Baltimore: Johns Hopkins University Press, 1976), pp. 173-184; "Unintended Consequences: The Ideological Shaping of American Agricultural Research, 1875-1914," in *No Other Gods*, pp. 185-199.

[26] Charles E. Rosenberg, "Science Pure and Science Applied: Two Studies in the Social Origin of Scientific Research," in *No Other Gods*, pp. 200-210; "Science, Technology, and Economic Growth: The Case of the Agricultural Experimental Station Scientist, 1875-1914," in *No Other Gods*, pp. 153-172; Diane B. Paul and Barbara A. Kimmelman, "Mendel in America: Theory and Practice, 1900-1919," in Ronald Rainger, Keith R. Benson, and Jane Maienschein (eds.), *The American Development of Biology* (Philadelphia: University of Pennsylvania Press, 1988), pp. 281-310. The articles in a recent volume of the *Journal of the History of Biology* are particularly important in this regard. See, first of all, Jonathan Harwood, "Introduction to the Special Issue on Biology and Agriculture," *Journal of the History of Biology* 39 (2006), pp. 237-239. Also see the historical works on the relationship between biological research and agriculture in the American context, such as Sharon E. Kingsland, "The Battling Botanist: Daniel Trembly MacDougal, Mutation Theory, and the Rise of Experimental Evolutionary Biology in America, 1900-1912," *Isis* 82 (1991), pp. 479-509; Paolo Palladino, "Wizards and Devotees: On the Mendelian Theory of Inheritance and the Professionalization of Agricultural Science in Great Britain and the United States, 1880-1930," *History of Science* 32 (1994), pp. 409-444; Deborah Fitzgerald, *The Business of Breeding: Hybrid Corn in Illinois, 1890-1940* (Ithaca: Cornell University Press, 1990); Kathy J. Cooke, "From Science to Practice, or Practice to Science? Chickens and Eggs in Raymond Pearl's Agricultural Breeding Research, 1907-1916," *Isis* 88 (1997), pp. 62-86.

phenomenon regarding trout's longevity and nutrition. This background fit well within the research priorities of the Cornell Agricultural College, which had been renowned for its investigation of longevity of poultry since the early 1920s.[27] His move to Cornell was thus a very natural course of events, especially as a student of Mendel who had trained a large number of scholars who went to agricultural experimental stations.[28]

During his early years at Cornell, McCay's major research subject was the metabolism and nutrition of various farm animals such as cows, goats, and sheep as well as rats. He also continued his trout feeding experiments at the Cortland Hatchery near Ithaca and studied the relation of fish blood to water pollution at Buffalo, with the support from the New York State Conservation Department.[29] While the scope of these studies was quite broad, many of them had a bearing upon the practical problems of farmers and breeders. For example, McCay, Maynard, and E. S. Harrison studied how a low-fat diet affected the level of lipid in cows' milk and blood and whether the cows' ability to synthesize lipid could compensate for the low level of lipid in their diets.[30] Along with Maynard and L. L. Madsen, McCay also investigated the problem of synthetic diets, particularly the adequacy of cod liver oil in the artificial feed for farm herbivores, such as sheep and goats. They argued that "cod liver oil should not be fed to farm Herbivora for extended periods at any but the lowest levels," since its toxic effect was confirmed during their experiments.[31] McCay by himself also studied the effect of a high level of "roughage," a diet with high cellulose content, upon the growth of the trout

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[27] In 1920, Alexis Carrel's colleague A. E. Ebeling sought for an expert on the longevity of the chicken, whose tissue was being cultured at the Rockefeller Institute. H. D. Goodale at Massachusetts Agricultural College recommended contacting Cornell's Agricultural College, which had been keeping very old chickens for research purpose. See Goodale to Ebeling, November 30, 1920; O. B. Kent to Ebeling, 7 December 1920; Ebeling to Kent, 14 December 1920, Box 48, Folder 6, AC.

[28] Rossiter, "Mendel the Mentor," pp. 215-219.

[29] Clive M. McCay, "Studies upon Fish Blood and Its Relation to Water Pollution: A Biological Survey of the Champlain Watershed," *Supplement to the Nineteenth Annual Report the New York State Conservation Department* (New York, 1929). Also see Jeanette B. McCay, *Clive McCay*, pp. 273-304.

[30] L. A. Maynard and C. M. McCay, "The Influence of a Low-Fat Diet upon Fat Metabolism during Lactation," *Journal of Nutrition* 2 (1929), pp. 67-81; C. M. McCay and L. A. Maynard, "The Interrelationship between the Dietary Fat and the Phosphorus Distribution in the Blood of Lactating Cows," *Journal of Biological Chemistry* 92 (1931), pp. 273-280.

[31] L. L. Madsen, C. M. McCay, and L. A. Maynard, "Synthetic Diets for Herbivora, with Special Reference to the Toxicity of Cod-Liver Oil," *Cornell University Agricultural Experimental Station Memoir* (Ithaca, New York: Cornell University, 1935).

and rats. He concluded that the trout showed an optimal rate of growth in spite of the high level of roughage in its food, while the rat's development was remarkably retarded with such diets.[32]

In 1933, McCay published in *Science* a short paper on longevity and optimum growth, which showed how this research environment at the animal husbandry department influenced his work. In the first paragraph of the paper, he criticized the "philosophy" of modern nutrition that "a diet which produces optimum growth in the young animal is the ideal."[33] According to him, this philosophy led to the assumption that "optimum growth means optimum health" and even to the idea that optimum health obtained from fast growth eventually brought about longer duration of life.[34] As an expert of animal husbandry, McCay also noticed that this idea had been accepted as a norm by many breeders who raised animals which were "slaughtered for meat shortly after they matured."[35] Unfortunately, however, "the same philosophy dominates the practices of rearing dairy calves and horses to maturity as rapidly as possible although it is desirable that they have a long productive life span," during which they could serve humans by providing milk and labor.[36]

McCay argued that some scientists' support of this wrong idea furthered the misunderstanding of the relation between longevity and growth rate. For example, H. Louise Campbell at Columbia University argued in her Ph.D. thesis of 1928 that more milk in the diet increased both the longevity and rate of growth of the rat.[37] However, McCay questioned the validity of Campbell's experiments by pointing out that her long-lived rats died at the age of 664 days, while the median lifespan of J. R. Slonaker's three

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[32] C. M. McCay, "The Effect of Roughage upon Growth," *Proceedings of the Society for Experimental Biology and Medicine* 27 (1929), pp. 209-211.
[33] Clive M. McCay, "Is Longevity Compatible with Optimum Growth?" *Science* 77 (1933), p. 410.
[34] McCay, "Is Longevity Compatible with Optimum Growth?" pp. 410-411.
[35] McCay, "Is Longevity Compatible with Optimum Growth?" p. 411.
[36] McCay, "Is Longevity Compatible with Optimum Growth?" p. 411.
[37] H. Louise Campbell, "Growth, Reproduction and Longevity of Experimental Animals as Research Criteria in the Chemistry of Nutrition" (Ph.D. Thesis, Columbia University, 1928).

rats at Stanford University was 1,222 days.[38] Campbell's rats had died too early to be regarded as showing longer lifespan.

Indeed, McCay found an interesting point in Slonaker's and others' experiments, although he thought that Slonaker's main argument itself—that rats fed a strictly vegetarian diet died earlier than those with mixed diets—was not very meaningful.[39] McCay stated that a careful reading of Slonaker's data indicated that the slow growth rate of the rats had some relation with their long lifespan of 1,222 days. This was also supported by the Polish scientist Jan Żabiński, who also argued in 1929 that insects whose growth was inhibited sporadically with deficient diets lived longer than others individuals.[40] Since McCay's previous experiments on brook trout indicated the same relation, he concluded that "no one has ever found it possible….to have both rapid growth with early attainment of maturity, and longevity. It is possible that longevity and rapid growth are incompatible."[41]

In the next year, McCay published a paper in the *Journal of Nutrition* that directly tackled the issue of the vegetarian diet and growth studied by Slonaker. While he asserted that a vegetarian diet made a rat grow slowly and become senile quickly, McCay concluded through his research that the reverse should be true. In an experiment following his 1929 study of the effect of "roughage" on growth, he found that rats with high cellulose diets lived longer than those that had been fed a stock food which contained less cellulose.[42] But he thought that the "longer life span in the present case of high cellulose diets probably has no relation to the cellulose, but is the result of the slow growth."[43] If other nutritional factors, such as vitamins and the number of calories, were

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[38] James Rollin Slonaker, *The Effect of a Strictly Vegetable Diet on the Spontaneous Activity, the Rate of Growth, and the Longevity of the Albino Rat* (Stanford, California: Stanford University Press, 1912).

[39] McCay wrote that he had "disregarded Slonaker's data upon exercised rat, because there is nothing in the literature for comparison" and had "not cited his data upon the vegetarian groups because they died relatively young and need not be considered in a discussion of longevity." See McCay, "Is Longevity Compatible with Optimum Growth?" p. 411.

[40] Jan Żabiński, "The Growth of Blackbeetles and of Cockroaches on Artificial and on Incomplete Diets. Part I," *Journal of Experimental Biology* 6 (1929), p. 384.

[41] McCay, "Is Longevity Compatible with Optimum Growth?" p. 411.

[42] C. M. McCay, "Cellulose in the Diet of Rats and Mice," *Journal of Nutrition* 8 (1934), p. 445.

[43] McCay, "Cellulose in the Diet," p. 436.

adequately provided, the rats with high cellulose diets grew as quickly as those fed with a normal food.[44]

The caloric content in diets was becoming important again in this and other research published during the late 1920s and the early 1930s. While he initially assumed that the rats with a low rate of growth might have a problem in ingesting an adequate number of calories due to a large amount of cellulose in their diets, he found that the actual cause of the problem was the source of vitamins rather than the presence of cellulose.[45] If a different source of vitamin B was provided with a sufficiently large number of calories, the rat showed a normal rate of growth. In other research done with the cooperation of Maynard, he also paid a close attention to the number of calories. To examine the role of dietary fats as a raw material for milk produced by cows rather than as an energy source, McCay and Maynard maintained with starch a constant calorie level in the foods used for his research on low-fat diets.[46] The cows with these low-fat diets secreted lower levels of milk and fat and had less phospholipid and total phosphorous in the blood.

In the same year, McCay and his student Mary Crowell announced the remarkable result of another series of experiments on the relation of caloric ingestion to growth, longevity, and aging. After dividing one hundred and six weaned rats roughly into three groups, the first of them were allowed to eat as they wanted while the second received the diet with insufficient calories from the beginning. The rats belonging to the third group were given food with an adequate number of calories only for the first two weeks and then were fed low-calorie diets. As expected, the second and third group of rats showed remarkably retarded rates of growth. Strikingly, however, the mean lifespan of the male rats in the second and third group was 792 and 883 days respectively, while that of rats in the first group was only 509.[47] What was more surprising was that the mean durations of life of the latter two groups were increasing when the paper was written because many of

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[44] McCay, "Cellulose in the Diet," pp. 438, 446.
[45] McCay, "Cellulose in the Diet," pp. 437-438.
[46] McCay and Maynard, "The Interrelationship between the Dietary Fat and the Phosphorus Distribution," p. 273.
[47] C. M. McCay and Mary F. Crowell, "Prolonging the Life Span," *The Scientific Monthly* 39 (1934), p. 412.

them were still alive. The only exception to this general trend was that the average lifespan of the female rats in the second group (755 days) was slightly shorter than that of the first (801 days). Nevertheless, this was merely because "some of the females in the retarded growth groups were lost during the hot summer of 1931."[48] Indeed, the median, rather than average, lifespan of the female rats belonging to the second and third groups was 904 and 894 days each, whereas that of the first group was only 820. After 1,200 days, no rats in the first group remained while thirteen survived in the second and third groups.

This experiment was more systematic and extensive than the previous works by Mendel, Osborne, Northrop, and Żabiński. Unlike Northrop who simply removed the yeast in the fly food to make it deficient,[49] McCay, as far as he could, tried to determine the accurate composition of the food given to his white rats, which contained all the essential nutrients and vitamins but was low in calories. McCay also used far more rats than Mendel and Osborn and succeeded in keeping thirteen among them alive after the 1,200th day, while all four rats of his teachers that were alive after two years died before becoming 1,000 days old.[50] While Żabiński's paper was quite comparable to McCay's in terms of accurate determination of the composition of diets, the former's research did not deal with longevity as the main topic and never discussed aging, while McCay made the two the central concerns for his investigation.

This result seemed to contradict some previous studies on longevity, particularly those of Campbell, T. B. Robertson, and L. A. Ray, who argued that a rapid growth accompanied a long life. Yet McCay had already pointed out problems in Campbell's research in 1933, and critically examined the meaning of Robertson and Ray's work in another paper published in 1935.[51] Strictly speaking, the relation of nutrition to longevity was not their study subject, but McCay had to explain their result in his terms since it could be used to challenge his conclusion. For McCay, it was possible that Robertson and

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[48] McCay and Crowell, "Prolonging the Life Span," p. 413.
[49] Northrop, "The Effect of Prolongation of the Period of Growth," p. 124
[50] Osborne, Mendel, and Ferry, "The Effect of Retardation of Growth," p. 295.
[51] T. Brailsford Robertson and L. A. Ray, "Experimental Studies on Growth. XV. On the Growth of Relatively Long Lived Compared with That of Relatively Short Lived Animals," *Journal of Biological Chemistry* 42 (1920), pp. 71-107.

Ray's rapidly growing mice lived longer because they already had better constitutions than those that grew slowly and died early due to their weak body. Therefore, it was necessary to divide the rats "into groups at the time of weaning or shortly thereafter" in a random manner and to made them "grow at different rates" with distinct diets.[52] The mean lifespan measured in this state would reflect the effect of the diets, rather than the bodily constitution, upon growth and longevity.

But what was more important in McCay's research, which was absent in any of these previous investigations, was that it dealt with how caloric restriction influenced the process of *aging*. The rats whose growth was retarded due to insufficient caloric intake looked younger than the normally grown ones. He wrote, "In studies with animals it is customary to observe the hair, since its condition frequently reveals changes that are taking place within the animal body." On this point, it was very striking that "the hair of the animals retarded in growth remained fine and silky for many months after that of the rapidly growing animals had become coarse." He supported this statement with three pictures showing the two rats in one camera frame. One of these pictures had a legend, "THESE TYPICAL RATS ARE BOTH 900 DAYS OLD." (See Figure 2.3.) The normally grown rat on the left looked more senile than the retarded one in the right.

Figure 2.3. *"THESE TYPICAL RATS ARE BOTH 900 DAYS OLD." C. M. McCay and Mary F. Crowell, "Prolonging the Life Span," The Scientific Monthly, 1934, 39, p. 407. The original picture came from Box 11, The Clive Maine McCay Papers, Cornell University Archive.*

[52] C. M. McCay, Mary F. Crowell, and L. A. Maynard, "The Effect of Retarded Growth upon the Length of Life Span and upon the Ultimate Body Size," *Journal of Nutrition* 10 (1935), pp. 64-65.

However, it may be asked whether the rat on the right was actually less senile than that on the left, or it just *looked* so. At that time, McCay could not answer this question, even though he was convinced that he needed to know more about what was going on within his underfed rats' body rather than being satisfied with observing the difference of their outward appearance.

Yet the rats' outward appearance was important for many reasons. Indeed, while the insects Northrop, Żabiňski, and others used might be ideal for measuring longevity due to their short lifespan, these organisms did not lead anybody to study *aging*. Their neglect of aging was probably due to the fact that the senile change of insects, unlike rats, was not quite evident in their gross morphology.[53] In this sense, rats were in a much better position, since their morphological changes accompanying aging were more clearly visible through their gross morphological alterations. Indeed, rats' outward appearance was important to McCay's teachers and colleagues who studied the consequence of differential nutrition. McCollum often contrasted in his publications two rats, one of which always showed the deleterious consequence of the lack of a particular dietary factor such as vitamin A.[54] Mendel and Osborne also contrasted two rats in one picture to demonstrate the effect of restricted diet upon the rat's growth.[55] The change in the rats' outward appearance caused by differential diets was important in convincing both professional scientists and the general public of the importance of specific dietary factors. McCay, who realized the importance of aging by looking at the gross difference of the rats made through caloric restriction, also used the picture of these rats to persuade other researchers and lay people of the significance of his work. Of course, as I will show in this section, such pictures did not yet show the complex problems that were invisible in the rats' outward appearance.

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[53] Admittedly, few scientists at that time were interested in insects' aging. See Leland Ossian Howard, "Ageing of Insects," in E. V. Cowdry (ed.), *Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1939), pp. 53-70. If compared with other chapters in Cowdry's book, Howard's chapter reveals that much less original research was done on insects' aging than that of other organisms.

[54] E. V. McCollum, *The Newer Knowledge of Nutrition: The Use of Food for the Preservation of Vitality and Health* (New York: MacMillan, 1919). In particular, see Figure 5.

[55] Osborne and Mendel, "The Resumption of Growth," p. 447.

There was another important feature of McCay's rats which could also be found in his teachers' research with rats—its connection with animal husbandry and agriculture. While theoretical issues were also important, farmers' and breeders' practical problems were significant as well for Mendel, McCollum, and McCay. Analyzing the reasons why aging had been neglected, McCay asserted,

> The healthy adult is a matter of little interest, even to himself, and the sick one usually rates as a pest. This philosophy belongs properly to the butcher. Every producer of meat animals wants to rear them rapidly because it is economical. These animals are killed as soon as they mature. What agricultural expert can tell the effect of the feeding during the growth period upon the milk-producing capacity of a cow during her entire life? What chicken specialist can tell the effect of the rate of growth of the chicken upon the egg production of the laying hen?[56]

Although it was usual and perhaps more economical for meat producers to kill their animals before they aged, the breeders who raised cows and hens not for meat but for milk and egg could certainly benefit by extending the lifespan of their animals to old age. While few scientists of animal husbandry had studied how to lengthen the longevity of farm animals, McCay's experiment showed an effective means to do so, and demonstrated that "the potential life span of an animal species is unknown and greater than we have believed," even though his experiments were limited to rats.[57]

During the May and June of 1934, McCay further emphasized the relevance of his research to animal husbandry by giving a talk during the "Agricultural College Hour" program of Cornell University Radio Station. To local farmers and breeders as well as scholars and students in the Ithaca area, he announced his discovery and how it could be potentially used to benefit their job. According to him, his work did not simply aim at extending the life of animals. It was intended to extend the *productive* portion of animals' as well as humans' life by retarding their aging.

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[56] McCay and Crowell, "Prolonging the Life Span," p. 405.
[57] McCay and Crowell, "Prolonging the Life Span," p. 414.

> In attempting to prolong the life span there is no doubt that we will agree that nothing is gained by extending the period of dotage, the period of pain, disease and failing senses in either animals or men. No more are we interested in maintaining a cow after she has ceased to be profitable in producing milk. Whatever the pessimists may say, most people agree that the extension of the active, productive part of the life of either man or his domestic animals is worthwhile. It is good business to attempt to rear calves that can maintain milk production after they mature, for a long period of years.[58]

Indeed, since 1933, McCay had tried to contact actual farmers through E. S. Savage, a professor in his department, who had known some farmers interested in the relation of growth retardation to longevity.[59] For example, J. E. Ellsworth at Folly Farm in East Weatogue, Connecticut, wrote that he had tried not to "over-stimulate" his young Jersey heifers during their growth after watching the cattle from the Island of Jersey, which "even at five years old appeared to be younger and in better bloom than those which had been bred and raised in this country as a rule."[60] These animals were "finer in their bones" and "kept on developing after they had reached an age at which American bred and raised Jerseys seemed to have reached their maximum."[61] Hugh W. Bonnell at Cranberry Run Farm in Ohio also stated that "excess feed which is usually given in this country promotes excess growth in all parts of the body" and "placed fat etc. around the ovaries and uterus that made for non breeding and homely bodies."[62]

After publishing his results, McCay kept in touch with farmers and breeders, who wrote that his method of producing longevity was very useful. In 1939, O. B. Kent of the Poultry Service Department of the Quaker Oats Company wrote that he had been

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[58] C. M. McCay, "Some Old Beliefs about Long Life," p. 8, 26 May 1934, Agricultural College Hour Radio Talk Manuscript, Box 6, Folder McCay [comments listed], Clive, NWS.
[59] Savage to Ellsworth, 19 April 1933, Box 6, Folder McCay [comments listed], Clive, NWS. It seems that with McCay's request Savage wrote similar letters to other farmers, including Hugh W. Bonnell.
[60] Ellsworth to Savage, 25 April 1933, Box 6, Folder McCay, Clive [comments listed], NWS.
[61] Ellsworth to Savage, 25 April 1933, Box 6, Folder McCay, Clive [comments listed], NWS.
[62] Bonnel to Savage, 22 April 1933, Box 6, Folder McCay, Clive [comments listed], NWS.

interested in McCay's research for several years and had "been recommending restricted feeding, low protein in rearing chicks in order to delay the egg production or maturity."[63] This method, according to Kent, was highly effective in saving feed and reducing mortality of his fowls in their old age. Kent later visited McCay's lab and disseminated the caloric restriction method to farmers and breeders outside of his company. According to Kent, these people testified to the effectiveness of McCay's method, and Kent's own hens also showed "higher egg production, better livability, very much better hatchability, and a very sharp reduction in the amount of thin shelled and poor shelled eggs" as well as longer lifespan.[64] Walter F. Wood, Jr., manager of the experimental farm of the Park and Pollard Dairy and Poultry Feeds at New York, also thought that "an intake of food greater than the bird's actual needs….has caused both an excessive growth….and an extra amount of wear and tear in the internal organs of the bird."[65] Wood then asked McCay for more information and some reprints of his papers on this issue. McCay recommended his chapter in *Problems of Ageing* (1939), edited by Edmund Vincent Cowdry at Washington University.[66] McCay wrote to Wood, "There are a number of people who are finding this very profitable in the Poultry Industry."[67]

Yet caution may be needed in reading this statement of McCay. Indeed, it is doubtful that what these professional breeders did was the same as what the professional scientists—McCay and his colleagues—did. Moreover, McCay never used the poultry, the subject of the above breeders' practice, and his research produced animals with too many structural and functional problems to be used in actual animal husbandry. Indeed, in his 1934 radio talk he said that "these experiments offer no suggestions for practical diets for either men or animals at the present time."[68] Although he did not detail these problems to the listeners of his talk, he and his colleagues did mention them in their article published in 1935 in the *Journal of Nutrition*, which targeted more professional

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[63] Kent to McCay, 6 January 1939, Box 6, Folder McCay, Clive [comments listed], NWS.
[64] Kent to McCay, 28 October 1942, Box 6, Folder McCay, Clive [comments listed], NWS.
[65] Wood to McCay, 4 June 1941, Box 6, Folder McCay, Clive [comments listed], NWS.
[66] Clive M. McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), pp. 572-623.
[67] McCay to Wood, 7 June 1941, Box 6, Folder McCay, Clive [comments listed], NWS.
[68] McCay, "Longevity—The Life Span as a Biological Project," p. 3, 16 June 1934; "Longevity—Extending the Life Span Experimentally," p. 7, 2 June 1934, Aricultural College Hour Radio Talk Manuscript, Box 6, Folder McCay [comments listed], Clive, NWS.

audience. First of all, the underfed rats failed to reach the "normal size" that a rat was supposed to attain if sufficient calories were given.[69] Perhaps this was a crucial problem that made it virtually impossible to use McCay's method for the actual raising of some farm animals, because size was important if they were being reared for human food. Moreover, "some of the femurs of the retarded groups proved to be very fragile," and in many instances their bones "crumbled in the course of dissection."[70] In general, the bones of the long-lived rats were less dense than those of normally grown individuals, and some of the former had numerous pores. Another issue was the structure and function of underfed animals' reproductive organs. The underfed female rats showed delayed opening of the vagina and no estrus cycle until normal diets were given, and even after the normal diets were restored, their estrus cycle was irregular.[71] Likewise, underfed male rats had penis deformity which might cause sterility or diminished fertility.

But McCay also reported that the rats raised with reduced calories in the diets were "better" than normal ones in many respects. Besides looking much younger in terms of silky hair and the general outward appearance, the underfed rats had better teeth, developed fewer tumors, and had fewer middle ear infections and lung diseases, which had often caused death of many old rats.[72] Furthermore, these retarded animals were "alert and active even after long periods of retardation."[73] In this sense, he hoped to give a positive answer to "a question commonly asked concerning the happiness of retarded animals."[74]

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[69] McCay, Crowell, and Maynard, "The Effect of Retarded Growth," pp. 67-69.
[70] McCay, Crowell, and Maynard, "The Effect of Retarded Growth," p. 75.
[71] "The Influence of Diet upon the Physiological and Biochemical Changes Which Accompany Aging in the Animal Body: General Statement of Project: Supplementary Study," p. 1, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF. This document was written probably in 1935. See Livingston Farrand to Alan Gregg, 30 Nov. 1935, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF. Also see "Conference on Nutritional Requirements for the Ageing Population," pp. 1, 6, 1-2 Nov. 1941, Box 1, Folder Nutritional Requirements Conference 1941, GS.
[72] Clive M. McCay, "Nutrition, Ageing, and Longevity," *Transactions and Studies of the College of Physicians of Philadelphia* 10 (1942), pp. 7, 9; "Conference on Nutritional Requirements for the Ageing Population," pp. 12-13, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[73] McCay, "Nutrition, Ageing, and Longevity," p. 9.
[74] McCay, "Nutrition, Ageing, and Longevity," p. 9.

Yet these issues surrounding McCay's rats were more complex. In fact, besides having little direct applicability to farming or animal breeding, McCay's research had significant theoretical problems as well. One of them was related to the question of maintaining a homogeneous genetic background. When McCay was developing his rat experiments, many biologists and biomedical scientists, influenced by the pioneering works of Thomas Hunt Morgan, Clarence C. Little, and others, were already using experimental animals with standardized genetic constitutions.[75] Although not in such a large scale as Morgan's flies or Little's mice, rats were also being genetically standardized at the Wistar Institute of Anatomy and Biology in Philadelphia.[76] These genetically homogenized animals were thought to produce more reliable experimental results, since the effect of an experimental manipulation could be clearly detected in contrast to the controls, which had the same genetic constitution. However, most contemporary nutrition researchers including McCollum, Mendel, Osborn, and Henry Sherman did not use genetically homogeneous rats, and McCay was no exception.[77] But Raymond Pearl and his colleagues had already shown through their fruit fly experiments the importance of genetic factors in longevity, and it was possible that the long life of McCay's underfed rats also was due to their peculiar genetic constitutions rather than the effect of reduced calories in diets.[78] In fact, Warren Weaver, head of the Natural Science Division of the Rockefeller Foundation, inquired in 1936 whether McCay's team used genetically homogeneous rats. Weaver heard that "there has been no program of inbreeding" of the rats, and most of the rats had originated from Mendel and Osborn's previous rat colony at Yale which was random-bred and had uncertain origins.[79]

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[75] Robert E. Kohler, *Lords of the Fly: Drosophila Genetics and the Experimental Life* (Chicago: University of Chicago Press, 1994); Karen A. Rader, *Making Mice: Standardizing Animals for American Biomedical Research, 1900-1955* (Princeton: Princeton University Press, 2004).

[76] Bonnie Tocher Clause, "The Wistar Rat as a Right Choice: Establishing Mammalian Standards and the Ideal of a Standardized Mammal," *Journal of the History of Biology* 26 (1993), pp. 329-349.

[77] See J. Russell Lindsey and Henry J. Baker, "Historical Foundations," in Mark A. Suckow, Steven H. Weisbroth, and Craig L. Franklin (eds.), *The Laboratory Rat*, 2nd edition (Amsterdam: Elsevier, 2006), pp. 15-20.

[78] Raymond Pearl, *The Biology of Death* (Philadelphia: Lippincott, 1922), p. 197.

[79] Warren Weaver Diary, 29 July 1936, Series 200D, Record Group 1.1, RF. See Lindsey and Baker, "Historical Foundations," p. 19.

Another significant problem was pointed out by Irving Fisher of Yale University, a renowned economist and eugenicist as well as chairman of Hygiene Reference Board of the Life Extension Institute, of which McCay's advisor Lafayette Mendel had also been a member.[80] In April 18, 1934, Fisher asked, "If allowing the rats to eat all they want while they are in captivity does them harm healthwise, their shortness of life may be explained on that score."[81] He continued, "The query in essence is whether there is any optimum health which is also an optimum longevity and whether possibly your low fed rats may be nearer that optimum than your high fed ones in a study of activity."[82] This implied that McCay's underfed rats might actually be normal individuals rather than the product of a new feeding method, and his "normal" rats could be no more than mere experimental artifacts produced through overfeeding. Indeed, this issue had already been raised in 1933 by a short comment in the *Journal of the American Medical Association*, whose author was probably none other than McCay's Yale advisor, Mendel. While praising McCay's experiments, the author suddenly pointed out,

> Twenty years ago, rats fed on the crude mixed diets of that period were reported to be of slow growth, many of them not reaching full adult size till the end of one year. Certain recorded groups of that time, maturing slowly, showed an average life span of more than three and one-half years. [McCay]'s rats of today, fed modern diets for accelerating nutrition, reach full adult size before the end of the first six months and live on an average for less than eighteen months.[83]

If previous experimental rats fed crude mixed diets had lived longer than modern ones fed well-balanced diets, then McCay's rats produced through restricted caloric intake

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[80] On Mendel's membership in the Institute, see Irving Fisher and Haven Emerson to Mendel, 25 November 1925, Box 1, Folder 14, LBM. This letter suggests that one of the major activities of the Institute was to fund "research and educational work in connection with the prevention of disease and the prolongation of human life."

[81] Fisher to McCay, 18 April 1934, Box 6, Folder McCay [comments listed], Clive, NWS.

[82] Fisher to McCay, 18 April 1934, Box 6, Folder McCay [comments listed], Clive, NWS.

[83] "Growth versus Longevity," *Journal of the American Medical Association* 100 (1933), p. 2015. McCay says that this comment was written by Mendel. See McCay to unknown correspondent, February 1934, Box 48, Folder Reprints, CMM. The content of the comment also indicates that it was written by Mendel.

could hardly be considered a new discovery. Jan Żabiński, who was cited by McCay, pointed to a similar problem.[84] If somebody wanted to claim that the underfeeding could really extend the longevity of an animal, it was necessary to know how long it could live in its "normal" condition, which could be used as a point of reference. The problem in McCay's experiment was that it was not known how long a rat could usually live in its "normal" environment and nutritional state.

But what is this "normal" condition? While McCay claimed that the extension of lifespan through caloric restriction was "so great that it exceeded the bounds of any normally fed group of animals," he did not define what the "normal" was.[85] Fisher wrote that such a "normal" state of humans in terms of feeding could be found in wartime when people were forced to underfeed themselves due to the scarcity of food.[86] Following this definition, the natural counterpart of this state would be the condition of the wild. Indeed, the wild animals could not eat as much as experimental animals, and probably due to this natural underfeeding, the organisms in the wild might live longer than those in the lab. But even if the actual number of calories wild rats consumed were to be known, it was still not possible to measure the effect of such caloric intake upon the longevity, because few "normal" animals in the wild could live as long as laboratory organisms due to predation, disease, or other accidents.

This problem continued to haunt later researchers and has been dealt with only recently.[87] It has been suggested that it is feasible to estimate how many calories a wild animal consumes by measuring the amount of its caloric *expenditure* and comparing it with that of experimentally underfed organisms, since caloric expenditure of an animal can be thought to almost equal to its consumption.[88] If the wild rat's caloric expenditure differs substantially with that of underfed rats, an old problem in the science of aging and longevity may find an answer. And this comparative research on wild and laboratory

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[84] Żabiński, "The Growth of Blackbeetles and of Cockroaches," p. 384.
[85] McCay, "Nutrition, Ageing, and Longevity," p. 6.
[86] Fisher to McCay, 18 April 1934, Box 6, Folder McCay [comments listed], Clive, NWS.
[87] For a general summary on the problem, see Leonard Hayflick, *How and Why We Age* (New York: Ballantine Books, 1994), pp. 277-295.
[88] Steven N. Austad, "Does Caloric Restriction in the Laboratory Simply Prevent Overfeeding and Return House Mice to Their Natural Level of Food Intake?" *Science of Aging Knowledge Environment* vol. 2001 issue 6, pe3.

animals may give another benefit to the scientists of aging, since it could lead them to combine their expertise with that of researchers working in the field.

Such a combination of the science of aging with new specialties was already actively going on during the 1930s and 1940s when McCay tried to build a cooperative research program around his investigation of caloric restriction and longevity. Rather than leading to the abandonment of the investigation, many of the theoretical and practical problems in McCay's research led to cooperative research involving diverse expertise in various branches in science as well as in agriculture and animal husbandry. Originally a small local project for a state's farmers and animal breeders, it expanded beyond its initial domain into medicine, physiology, psychology, dentistry, and even into the American popular culture. Interestingly, some of the above problems *enabled*, rather than hindered, this expansion, which in turn produced further problems that would attract more researchers from various fields. The next section will discuss how this expansion occurred despite, or in some sense, because of, the above problems.

Reception of McCay's Research: The Rockefeller Foundation, the New Science of Aging, and the Making of a Research Project

The first step in the expansion of McCay's research program was taken when Alan Gregg, director of the Medical Science Division of the Rockefeller Foundation, visited McCay's laboratory on May 29, 1935. While it is not known how Gregg initially came to know about McCay's research, he was very impressed by it and reported to the Foundation that "L. A. Maynard (former IEB fellow) and Assistant Professor C. M. McCay were doing some interesting work on longevity in rats."[89] At that time, most of the potential problems in McCay's research were not evident either to Gregg or to McCay himself. It was a very promising project, although it had been "much neglected" due to its difficulties of long-term maintenance of large number of old animals.[90] Stable and

[89] "Cornell University—Nutrition," 31 April 1941, Box 137, Folder 1689, Series 200D, Record Group 1.1, RF; Gregg to Maynard, 3 June 1935, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.
[90] "The Influence of Diet upon the Physiological and Biochemical Changes Which Accompany Aging in the Animal Body: General Statement of Project," p. 4, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF. This document seems to be written by McCay or co-authored by McCay and Maynard, although

extensive funding from a large foundation was thus urgently needed if the project could produce more important results in the future. Since Gregg thought that the Natural Science Division would be more appropriate for supporting this program, he handed over the project to the officers of the Division, Warren Weaver and Frank Blair Hanson.[91]

The Natural Science Division of the Rockefeller Foundation underwent a major transformation under Weaver's directorship during the 1930s. According to historian Robert Kohler, the Division at that time became focused on supporting cross-disciplinary cooperative research project rather than established disciplines in the natural sciences.[92] In particular, Weaver's program concentrated on the research projects in the biological sciences rather than natural science in general, when the Great Depression agitated the whole country and substantially cut the budget of the Foundation. The decrease in funds made the Foundation abolish many of its prospective programs except those in the life sciences, to which the Foundation had traditionally been deeply committed.[93] Yet Weaver wanted the life sciences he supported to be interdisciplinary investigations using the tools borrowed from physics and chemistry, rather than remaining as traditional "pure" biology. What was important was innovative investigations, which, Weaver thought, were possible only through the cooperation across disciplinary borders. Among these investigations, nutrition research was an important subcategory for support.[94]

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the correspondence from Livingston Farrand, Cornell President, to Gregg states that it was prepared by L. A. Maynard, the head of animal husbandry department. See Farrand to Gregg, 30 November 1935, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.

[91] Gregg to Farrand, 3 December 1935, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.

[92] Robert E. Kohler, *Partners in Science: Foundations and Natural Scientists, 1900-1945* (Chicago: University of Chicago Press, 1991), pp. 303-329.

[93] Kohler, *Partners in Science*, p. 281. Other scholars have offered different interpretations. Daniel Kevles has argued that biological science received more attention since it could be an alternative to the physical sciences, which were thought to be the basis of new technology that was considered responsible for the economic crisis, at least partially. On the other hand, Lily Kay has argued that biological science was expected to reveal the basis of human behavior and might contribute to the proper solution of contemporary social problems in labor, heredity, and immigration. See Daniel J. Kevles, *The Physicists: The History of a Scientific Community in Modern America* (Cambridge, Mass.: Harvard University Press, 1995), pp. 247-251; Lily E. Kay, *The Molecular Vision of Life: Caltech, The Rockefeller Foundation, and the Rise of the New Biology* (Oxford: Oxford University Press, 1993), pp. 22-57. Also see A. Hunter Dupree, *Science in the Federal Government: A History of Policies and Activities* (Baltimore: Johns Hopkins University Press, 1986), pp. 344-368.

[94] "Committee Paper No. I," p. 3, Box 2, Folder 9, Record Group 3.1 Series 915, RF.

This funding policy of Weaver's Division fit well with the new direction of research proposed by McCay and Maynard. Most importantly, they claimed that their research belonged to the science of "nutrition," which was a major area of support by Weaver's Division.[95] Admittedly, McCay could have chosen another important word related to his research, *aging*, to describe his project. If he had done so, the foundation might not have decided to support him, since aging was not included in the list of the topics Weaver had created for his program.[96] Another aspect of the research that made it attractive to Weaver was that it would be cross-disciplinary, borrowing various approaches used in biochemistry, biophysics, physiology, pathology, and psychology. McCay and Maynard proposed to study the changes of biochemical, pathological, and physical properties in the aged animal body after long-term feeding of specific diets.[97] Mental changes accompanying aging in rats on a particular form of diets were another topic that would be studied with the help of experimental psychologists.[98] Moreover, the research could be extended to an investigation of diet's effect upon sex and reproduction, which was also supported by the Rockefeller Foundation at that time.[99] Indeed, underfed rats showed "delay in the opening of the vagina" and "a penis deformity" which might cause problems in reproduction.[100] Further research could thus reveal the complex relations among age, diet, and reproduction that had broader implications, including those for human life and society.

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[95] Minutes of Meeting of the Executive Committee of the Rockefeller Foundation, 26 March 1936, Record Group 16, RF.

[96] For the new program of the Natural Science Division adopted in 1933, see "Committee Paper No. I," p. 3, Box 2, Folder 9, Series 915, Record Group 3.1, RF.

[97] "The Influence of Diet upon the Physiological and Biochemical Changes Which Accompany Aging in the Animal Body: General Statement of Project," pp. 4-5, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.

[98] "The Influence of Diet upon the Physiological and Biochemical Changes Which Accompany Aging in the Animal Body: General Statement of Project," p. 6, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.

[99] "Committee Paper No. I," p. 3, Box 2, Folder 9, Record Group 3.1 Series 915, RF. On the NRC Committee on the Problems of Sex, supported by the Rockefeller Foundation's program on sex and reproduction, see Adele E. Clarke, *Disciplining Reproduction: Modernity, American Life Sciences, and the Problems of Sex* (Berkeley: University of California Press, 1998), pp. 90-120.

[100] "The Influence of Diet upon the Physiological and Biochemical Changes Which Accompany Aging in the Animal Body: General Statement of Project: Supplementary Study," p. 1, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.

Yet the funding decision depended upon other factors as well. It needed the evaluation by the applicants' scientific colleagues of the strength and value of their work and their past achievements. Therefore, Frank Blair Hanson asked other scientists to evaluate McCay and Maynard. McCay's doctoral thesis advisor, C. L. A. Schmidt of the University of California, described McCay "as a strong and promising man in nutrition."[101] But Henry Sherman of Columbia University, another expert on nutrition and longevity and a teacher of H. Louise Campbell, was not very positive in evaluating McCay. According to Hanson, Sherman stated that McCay "is out for publicity, his enthusiasm runs away with him, and some of his work has not been sound."[102] Although it is not certain why Sherman made such a critical statement, it probably reflects the fact that McCay produced a result that contradicted the research of Campbell and Sherman who argued that diets leading to rapid growth delayed senility as well.[103] But this statement of Sherman on McCay was not taken into a serious consideration, because Sherman also said that "Maynard is head of the project and is a thoroughly sound man."[104] The latter statement was indeed important, since Maynard was considered the Principal Investigator of the project as the chairman of the department, although it was McCay, still a junior faculty member, who actually directed and conducted the research. Other people on the Cornell campus, including R. A. Gortner (visiting lecturer in chemistry) and Carl E. Ladd (dean of the College of Agriculture), highly recommended the Foundation's support of McCay's research.

Another significant issue, which was related to the direction of McCay's research, was how the Rockefeller money might be supplemented by other sources of funding, including the state and federal budgets appropriated for the state college' agricultural research. Indeed, the Foundation's position was that the responsibility of supporting a scientific program should be shared with other patrons, usually the institution itself, up to

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[101] Frank Blair Hanson Diary, 17 and 18 December 1935, Record Group 12.1, RF.

[102] Frank Blair Hanson Diary, 16 December 1935, Record Group 12.1, RF. Some of this statement is not completely unfair, since McCay tried to give publicity to his work to the general public using various newspapers and magazines. This will be discussed in the last part of this chapter.

[103] See, for example, H. C. Sherman and H. L. Campbell, "Growth and Reproduction upon Simplified Food Supply," *Journal of Biological Chemistry* 60 (1924), pp. 5-15. Campbell's Ph.D. thesis arrived at the same conclusion and was criticized by McCay.

[104] Frank Blair Hanson Diary, 16 December 1935, Record Group 12.1, RF.

the amount the latter could pay. To resolve this issue and to discuss other problems, Hanson visited Ithaca in December, 1935, and suggested that Cornell Agricultural College could fund McCay and Maynard from its annual budgets, particularly those appropriated according to the Bankhead-Jones Act. Yet McCay and Maynard asserted that Cornell would receive only $15,000 for that year from the Act, and there was some "[difficulties] of getting State or Federal money for work with rats" that were thought to be appropriate for basic, rather than agricultural, investigations.[105] Hanson wrote that McCay and Maynard claimed that they had to "continue on the small funds available or get outside help," if they were "to do pure research."[106] Perhaps such a statement on their financial difficulty might be an exaggeration, because McCay's study using rats had been moderately, though not sufficiently, supported by the Agricultural College, whose research funds came from the state budget and New York's portion of federal grants intended to help agricultural research there.[107] A major part of the research fund of the University of Wisconsin's Agricultural College, where McCay's teacher McCollum studied vitamins using rats, also came from the budget of Wisconsin state.[108] Then, does this mean that McCay and Maynard exaggerated their funding problems simply to get Rockefeller money? To be awarded a grant from a particular source, however, means that the awardees would follow the policy and expectation of the funding agency. As will be seen in the remaining part of the chapter, the above assertion showed that he was trying to become a new type of researcher studying more fundamental problems in nutrition, longevity, and aging, rather than remaining as an agricultural investigator for farmers and breeders.

Nevertheless, McCay did not abandon the hope that his research would contribute to agriculture. According to Hanson, McCay and Maynard stated that "at present farmers are interested in how early the hens begin to lay [eggs] and how early the heifers give

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[105] Frank Blair Hanson Diary, 17 and 18 December 1935, Record Group 12.1, RF.
[106] Frank Blair Hanson Diary, 17 and 18 December 1935, Record Group 12.1, RF.
[107] The Agricultural College provided $180,000 from 1927 to 1950 for McCay's research. See "Federal Security Agency, Application for Grant-in-aid," 17 November 1949, p. 3, Box 21, Folder USPH 584 Prints 1952, CMM.
[108] Rosenberg, "Science, Technology, and Economic Growth: The Case of the Agricultural Experimental Station Scientist, 1875-1914," in *No Other Gods*, pp. 160-162.

milk, but with no account taken as to the length of time the hens lay [eggs] or the cows give milk."[109] Hence, "it might be a better economics to have eggs and milk given over a longer period," and thus to eliminate the "present wasteful method" to "get all the milk and eggs possible from the young animals and then discard."[110] Indeed, Carl E. Ladd, dean of the Agricultural College, also wrote that "the projects….are of great interest to all of us" and suggested to Hanson that the College could provide not more than $3,500 per year from the Bankhead-Jones Act to supplement the Rockefeller Foundation's support of McCay and Mayard's program.[111]

Yet Ladd was not completely confident about the usefulness of McCay's research for New York farmers' immediate problems. On March 27, 1936, he wrote, "We are under very great pressure from practical farmers to undertake certain investigations that mean much to them financially and that would undoubtedly have been supported by the State before this if we had not had the depression."[112] He suggested that he would "immediately relocate these funds in such a way as to start work on some of the more pressing problems with the beginning of the crop year this spring," if the Rockefeller Foundation would not decide to support McCay.[113] For Ladd, who was more deeply concerned about the farmers within the state during the difficult times of the Great Depression, McCay's program was too basic to be supported by the state college's public funds.

However, Ladd did not need to worry about this issue, because the Executive Committee of the Rockefeller Foundation already decided on March 26th to award $42,500 to McCay and Maynard for five and a half years beginning in 1936.[114] As promised, Cornell University also offered $1,200 for 1936 and $3,500 per annum for five years thereafter to supplement Rockefeller funding. This was quite a large amount as a grant awarded to a particular scientist's project, since only nine out of fifty-two

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[109] Frank Blair Hanson Diary, 17 and 18 December 1935, Record Group 12.1, RF.
[110] Frank Blair Hanson Diary, 17 and 18 December 1935, Record Group 12.1, RF.
[111] Ladd to Hanson, 2 January 1936, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.
[112] Ladd to Hanson, 27 March 1936, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.
[113] Ladd to Hanson, 27 March 1936, Box 136, Folder 1687, Series 200D, Record Group 1.1, RF.
[114] Minutes of the Executive Committee of the Rockefeller Foundation, 26 March 1936, Record Group 16, RF.

individual scientists funded by the Natural Science Division from 1933 to 1936 received more money than McCay.[115]

The Rockefeller grant gave McCay and his coworkers material and social support necessary for the expansion of the program. They increased the number of rats in the rat colony from three hundred to one thousand, and bought such new equipment as X-ray apparatus, microtomes, galvanometers, electrocardiographs, and an air conditioner.[116] Moreover, the fact that McCay and Maynard were awarded a grant by a major philanthropy such as the Rockefeller Foundation demonstrated to their contemporaries that their research was an important scientific work worthy of long-term support. Soon after the decision of the Rockefeller Foundation was made, *Time* enthusiastically reported it with the comment that McCay and his colleagues were "learned, industrious biochemists," who "hope to validate the theory that the characteristics of youth can be retained….by special diets."[117]

The grant also enabled McCay to hire more scientific personnel who could pursue novel lines of research, including histologist Katherine Hummel, chemist Gordon Ellis, and lab technician Gladys Sperling, who had a master's degree in bacteriology. The Rockefeller grant also paid the salary of Leroy Barnes, a physics Ph.D., although he had already been working at McCay's lab before 1935. He and Ellis studied the age changes of the physical and chemical properties of the rat's bones and other organs, such as their degree of calcification, recalcifying ability, breaking strength, moisture, and density.[118] They also used X-ray apparatus to examine the growth and senescence of rats' bones under the influence of exercise and varying mineral contents in the diets without killing

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[115] "Appropriations Made in New Program, 1933, 1934, 1935, 1936," Box 1, Folder 2, Series 915, Record Group 3.1, RF. Of course, a comparison of absolute amounts of grants might not reveal how well funded a scientist was, because other factors, such as the length of the term of funding, should also be considered.

[116] "Diet and Longevity: Excerpt from Trustee's Confidential Monthly Report," June 1937, Box 136, Folder 1688, Series 200D, Record Group 1.1, RF; "Rockefeller Foundation Research in Longevity, Outstanding Orders, December 31, 1936, Revised Statement March 19, 1937," Box 136, Folder 1688, Series 200D, Record Group 1.1, RF; "Expenditure from the Rockefeller Foundation Grant for Research in Longevity: January 1, 1937 to January 1, 1938," Box 137, Folder 1689, Series 200D, Record Group 1.1, RF. Air conditioning was important to keep the rats at a constant temperature. Indeed, many of McCay's rats in his previous experiments died due to the summer heat.

[117] "Diet for Age," *Time* (20 April 1936).

[118] Leroy Barnes and Gordon Ellis, "Studies of the Skeletons of Animals: A Progress Report by Leroy Barnes and Gordon Ellis," 1 May 1940, Box 137, Folder 1689, Series 200D, Record Group 1.1, RF.

them. Furthermore, they investigated the effects of low calcium diets upon the bones' decalcification and fragility by adding in some rats' food sodium metaphosphate, which could make calcium ions in the food unavailable to the animal by holding them chemically.[119]

A number of scientists in other parts of the country also became involved in the research on nutrition and longevity using McCay's rats, because the increased number of rats in his colony enabled him to offer some of his animals to the scientists from other disciplines and institutions.[120] For example, Clifton A. H. Smith, a dental researcher in New York City, received McCay's rats to investigate their teeth and jaws.[121] A. Baird Hastings at Harvard Medical School and J. R. Murlin at the University of Rochester also studied metabolism and muscle changes of McCay's animals, and pharmacologist William deB. MacNider at the University of North Carolina used the tissue of McCay's rats in his own investigation of aging.[122] The Josiah Macy, Jr. Foundation also contributed to this expanding network of cross-disciplinary research by funding the pathological research on McCay's underfed rats by John Saxton at Cornell Medical School in New York City.[123]

McCay also offered his rats to scientists who wanted to use them for their research subjects. For example, John Nelson of the Rockefeller Institute requested some of McCay's aged rats to obtain a bacterium that he thought could be found in the aged animal's body.[124] Ernst Gellhorn of the University of Minnesota hoped to investigate the

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[119] Leroy Barnes and Gordon Ellis, "Studies of the Skeletons of Animals: A Progress Report by Leroy Barnes and Gordon Ellis," 1 May 1940, Box 137, Folder 1689, Series 200D, Record Group 1.1, RF.

[120] Frank Blair Hanson Diary, 17 January 1939, Record Group 12.1, RF.

[121] On Smith's publication using McCay's rats, see C. A. H. Smith, R. F. Light, and C. M. McCay, "Advanced Age in Relation to Dental Caries in White Rats," *Journal of the American Dental Association* 26 (1939), pp. 1700-1703; C. A. H. Smith and C. M. McCay, "Advanced Age in Relation to Dental Caries in White Rats. II," *Journal of the American Dental Association* 34 (1947), pp. 340-344.

[122] See, for example, MacNider to McCay, 13 May 1941, Box 1, Folder Correspondence 1940-1941, GS; Frank Blair Hanson Diary, 21 October 1940, Record Group 12.1, RF. Also see O. H. Lowry, C. M. McCay, A. B. Hastings, A. N. Brown, "Histochemical Changes Associated with Aging. III. The Effects of Retardation of Growth on Skeletal Muscle," *Journal of Biological Chemistry* 143 (1942), pp. 281-284; Hastings to McCay, 31 July 1940, Box 30, Folder Club for Aging Misc. II, NWS.

[123] Lawrence Frank to McCay, 14 October 1937, Box 6, Folder Clive, McCay [comments listed], NWS; McCay to Frank, 15 October 1937, Box 6, Folder Clive, McCay [comments listed], NWS; Maynard to Weaver, 2 December 1937, Box 136, Folder 1688, Series 200D, Record Group 1.1, RF.

[124] Sperling to Nelson, 25 March 1941, Box 1, Folder Correspondence 1940-1941, GS.

reaction time of autonomic nerve system using McCay's very old rats.[125] Herbert Evans, a specialist in epiphysis at the University of California, also asked McCay to send him his old rats to do research on epiphysis with them, while William F. Windle at the University of Pennsylvania proposed using McCay's rats to study the senile changes of the central nervous system.[126] As McCay became known among other biological and biomedical scientists as a chief source of old animals,[127] his rats crossed disciplinary boundaries and were disseminated to other scientists and labs as experimental organisms useful for diverse research projects, which increasingly considered age and aging as important variables that needed to be controlled and studied.

Such an expansion of the academic network through the sharing of the rat resembled the cases of Thomas Hunt Morgan's fruit flies and Clarence C. Little's mice, which have been extensively studied by Robert Kohler, Karen Rader, and other historians of biology. By tracing the uses of flies and mice in biological labs, Kohler and Rader studied how scientists made the networks of exchanging and spreading experimental animals, which transformed, strengthened, and enlarged their scientific communities, the hierarchy within them, and the scope and character of the scientific knowledge produced by them.[128] Admittedly, as I have pointed out in the previous section, McCay's rats were not genetically standardized as were *Drosophila melanogaster* and *Mus musculus*. Moreover, reproduction was hardly possible for the rats, since they were already aged when shipped to other laboratories or had defects in the reproductive capacity due to restricted feeding. Nevertheless, McCay's rats gradually occupied an important place in the development of aging research just as Morgan's flies and Little's mice had done for genetics and cancer research. The rats began to be used in many laboratories as a research material embodying the idea and methodology employed by McCay and his predecessors, which could be combined with other types of expertise available in different labs. Furthermore, the rats added a new dimension—age of the organism—to biological and

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[125] See Gellhorn to McCay, 22 January 1945, Box 1, Folder Correspondence 1944-1945, GS.
[126] Evans to McCay, 21 February 1944, Box 1, Folder Correspondence 1944-1945, GS; Windle to Gladys Sperling, 26 April 1949, Box 1, Folder Correspondence and Christmas and Easter Newsletter 1949, GS.
[127] See, for example, Willard D. Roth to McCay, 10 January 1956, Box 1, Folder Correspondence 1956-1957, GS; V. W. Steward to McCay, 19 May 1958, Box 1, Folder Correspondence 1958-1959, GS.
[128] Kohler, *Lords of the Fly*, pp. 133-170; Rader, *Making Mice*.

medical research, which had seldom been considered in the previous investigations using mice or flies.

Other researchers who did not use McCay's rats read his articles and studied longevity and aging in relation to dietary factors. Anton J. Carlson and Frederick Hoelzel at the University of Chicago examined how the lifespan of the rats obtained from the Wistar Institute was influenced by intermittent fasting and omnivorous/vegetarian diets.[129] W. H. Riesen, E. J. Herbst, Catherine Walliker, and C. A. Elvehjem at the University of Wisconsin also studied the relation of dietary calories to longevity, cancer formation, and respiratory infection with Sprague-Dawley rats and arrived at a conclusion similar to McCay's.[130] Scientists using other organisms also became interested in McCay's research, and investigated the relation of diets to lifespan, senescence, and other features of life and disease. For example, a group of researchers at Brown University studied how *Daphnia longispina* was influenced by limiting the amount of food.[131] While they found complex relations of the quantity of diets to the daphnia's longevity, growth, and heartbeat, it was evident that insufficient food tended to lead to longer lifespan. Even a protozoan species, *Tokophrya infusionum*, was used for research on longevity and diets by Maria Rudzinska at New York University.[132]

The relation of caloric intake to tumor formation was another important subject that began to be studied extensively after McCay's research. While the connection between underfeeding and tumor formation had already been noticed during the 1900s, cancer researchers came to focus more on calories rather than underfeeding in general

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[129] Anton J. Carlson and Frederick Hoelzel, "Apparent Prolongation of the Life Span of Rats by Intermittent Fasting," *Journal of Nutrition* 31 (1946), pp. 363-375; "Growth and Longevity of Rats Fed Omnivorous and Vegetarian Diets," *Journal of Nutrition* 34 (1947), pp. 81-96.

[130] W. H. Riesen, E. J. Herbst, Catherine Walliker, and C. A. Elvehjem, "The Effect of Restricted Caloric Intake on the Longevity of Rats," *American Journal of Physiology* 148 (1947), pp. 614-617.

[131] Lester Ingle, "Effects of Environmental Conditions on Longevity," *Science* 78 (1933), pp. 511-513; Lester Ingle, Thelma R. Wood, and A. M. Banta, "A Study of Longevity, Growth, Reproduction and Heart Rate in Daphnia Longispina as Influenced by Limitations in Quantity of Food," *Journal of Experimental Zoology* 76 (1937), pp. 325-352; H. Howard Dunham, "Abundant Feeding Followed by Restricted Feeding and Longevity in Daphnia," *Physiological Zoology* 11 (1938), pp. 399-407.

[132] Maria Rudzinska, "Overfeeding and Life Span in Tokophrya Infusionum," *Journal of Gerontology* 7 (1952), pp. 544-548.

after McCay's experiments were published in the 1930s.[133] For instance, H. P. Rusch, B. E. Kline, and C. A. Baumann at the Medical School and the Agricultural College of the University of Wisconsin cooperated in their study of how diets with varying amounts of fats with high caloric content influenced tumor formation of mice exposed to ultraviolet rays.[134] Zelda Ball, Richard Barnes, and Maurice Visscher also used mice to investigate how caloric restriction influenced sexual maturity, lifespan, and the occurrence of cancer,[135] and Albert Tannenbaum at Chicago investigated the relation of caloric intake and overweight to tumor incidence.[136]

During this period, the problem of experimental organisms' genetic background that Warren Weaver addressed to McCay began to be dealt with. While rat researchers were slower in adopting inbred animals, scientists using other organisms quickly noticed the importance of this issue. As early as in 1938, H. Howard Dunham at Brown used clones of a single daphnia in his research on longevity and dietary restriction.[137] The scientists using mice also began to adopt inbred strains, particularly those supplied by the Jackson Memorial Laboratory. For example, Ball, Barnes, and Visscher at Minnesota employed C_3H mouse strain for the investigation of caloric restriction and cancer incidence, and Martin and Ruth Silberberg at Washington University used an inbred C57 strain in their study of high fat diet and joints of aging mice.[138] In the case of McCay himself, the introduction of inbred animals was not an issue that could be resolved in his lifetime. Even in his final experiments published in 1960, he did not seem to use inbred

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[133] As far as the published records are concerned, the first research paper on cancer and underfeeding is C. Moreschi, "Beziehungen zwischen Ernärung und Tumorwachstum," *Zeitschrift für Immunitatsforschung und Experimentelle Therapie* 2 (1909), pp. 651-675.

[134] H. P. Rusch, B. E. Kline, and C. A. Baumann, "The Influence of Caloric Restriction and of Dietary Fat on Tumor Formation with Ultraviolet Radiation," *Cancer Research* 5 (1945), pp. 431-435.

[135] Zelda B. Ball, Richard H. Barnes, and Maurice B. Visscher, "The Effect of Dietary Caloric Restriction on Maturity and Senescence, with Particular Reference to Fertility and Longevity," *American Journal of Physiology* 150 (1947), pp. 511-519; Maurice B. Visscher, Zelda B. Ball, Richard H. Barnes, and Ivar Sivertsen, "The Influence of Caloric Restriction upon the Incidence of Spontaneous Mammary Carcinoma in Mice," *Surgery* 11 (1942), pp. 48-55.

[136] Albert Tannenbaum, "The Genesis and Growth of Tumors. II. Effects of Caloric Restriction per se," *Cancer Research* 2 (1942), pp. 460-467; "Relationship of Body Weight to Cancer Incidence," *Archives of Pathology* 30 (1940), pp. 509-517.

[137] H. Howard Dunahm, "Abundant Feeding," pp. 399-407.

[138] Visscher, Ball, Barnes, and Sivertsen, "The Influence of Caloric Restriction," pp. 48-55; Martin Silberberg and Ruth Silberberg, "Effects of a High Fat Diet on the Joints of Aging Mice," *Archives of Pathology* 50 (1950), pp. 828-846.

rats.[139] Yet he did conduct an experiment in 1946 to examine the problem of hereditary factors and longevity. This experiment showed that a rat's litter of origin was closely related to its lifespan, indicating that genetically close individuals had similar duration of life. Indeed, he had been concerned about this problem and tried to minimize hereditary influence upon longevity "by the assignment of individuals from the same litter to different groups."[140] In fact, this procedure had been a critical element in his experiments and formed a basis from which he criticized Robertson and Ray's claim that rapidly growing animals lived longer. When inbred rats were not available, this was a relatively reasonable means of minimizing genetic influences upon the results of the experiments, and showed that McCay did not completely neglect the hereditary factors in longevity.[141] He even claimed in 1948 that "inheritance plays a vital part in predetermining the life span" and thus genetics approaches should be used for research on longevity along with pathological, psychological and nutritional methods.[142] Although he himself was still not confident on conducting experiments on heredity, genetics had to be employed in the multidisciplinary efforts to investigate the influence of caloric restriction on longevity and aging.[143]

Indeed, a significant factor responsible for the extension of McCay's Rockefeller grant in 1941 was that he encouraged multiple lines of research on longevity and aging in other laboratories. When Frank Hanson requested an evaluation of McCay's research, Eugene L. Opie of Cornell Medical School emphasized that McCay's works contributed to the medical understanding of tumors, which emerged as a major disease of the twentieth century. Opie wrote that "in animals whose development has been retarded by a

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[139] C. E. Brown, L. L. Barnes, G. Sperling, and C. M. McCay, "Radioactive Calcium Osteosarcomas and Squamous Carcinomas: Influence of Dietary Restriction and Retarded Growth on Incidence," *Cancer Research*, 1960, 20: 329-337, on p. 330.

[140] Gladys Sperling, J. K. Loosli, L. L. Barnes, and C. M. McCay, "The Effect of Coffee, Human Diets, and Inheritance upon the Life Span of Rats," *Journal of Gerontology* 1 (1946), p. 431.

[141] Most rat researchers did not use inbred animals until the 1960s. While the Wistar Institute maintained a small number of inbred rats, most of rats they sold were random-bred till the 1960s. See Lindsey and Baker, "Historical Foundations," p. 10; Clause, "The Wistar Rat," p. 332.

[142] C. M. McCay, "Gerontology and Nutrition," in *Biological Foundations of Health Education: Proceedings of the Eastern States Health Education Conference, April 1-2, 1948* (New York: Columbia University Press, 1950), p. 84.

[143] For McCay's feeling about genetics, see McCay to E. V. Cowdry, 28 October 1946, Box 41, Folder 10, EVC.

diet deficient in energy requirements we have found conspicuous diminution of disease, with special reference to the occurrence of tumors, as compared with normal controls."[144] William S. Ladd, dean of Cornell Medical School, also pointed out that McCay's rats could suggest an important fact concerning public health and nutrition. According to Ladd, "This work stimulates such thoughts as whether it is a wise thing to feed one's children so that they grow rapidly to large stature and are kept well nourished all along through childhood and adolescence, or whether it might possibly be wiser for the sake of health and longevity to not so force them."[145] It was not only to the faculty of Cornell Medical College that Hanson's evaluation request was sent. Robert E. Coker, ex-chairman of Division of Biology and Agriculture of the National Research Council, commented that "McCay's animals could serve not only for him and his staff but also be sources of authentic materials for research by others in different institutions."[146] He felt that "something is going to be done….to stimulate broader and more effective research on aging" through McCay and his aged animals.[147] Anton J. Carlson, a professor of physiology at the University of Chicago, also commented that "he is willing to distribute valuable materials to other workers in this field, such as MacNider of the University of North Carolina, who are working on other phases of aging."[148] MacNider himself was highly enthusiastic on the value of McCay's research. "So far as I know," he claimed, "this work on ageing which Dr. McCay is doing through his studies on these animals is the only instance in which age factor has been controlled."[149] MacNider was "certain that [McCay's] work….has been of the greatest value in obtaining information concerning the influence of fundamental factors on the process of ageing."[150] Such statements brought about a highly positive comment on McCay's project during the

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[144] Hanson to Opie, 31 January 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF; Opie to Hanson, 1 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF. Hanson sent the same letter to R. E. Coker, H. C. Sherman, William MacNider, A. J. Carlson, C. L. A. Schmidt, and W. S. Ladd.
[145] Ladd to Hanson, 6 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.
[146] Coker to Hanson, 5 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.
[147] Coker to Hanson, 5 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.
[148] Carlson to Hanson, 5 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.
[149] MacNider to Hanson, 4 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.
[150] MacNider to Hanson, 4 February 1941, Box 137, Folder 1690, Series 200D, Record Group 1.1, RF.

Executive Meeting of the Rockefeller Foundation, held on April 2, 1941. It was emphasized,

> A valuable by-product of these researches is co-operation with a number of investigators in other institutions by supplying them with materials from the aged rats: the teeth are sent to a dental investigator in New York City; the brains go to Dr. Warren Sperry of Columbia University College of Physicians and Surgeons; certain organs are being sent to Dean William MacNider of the University of North Carolina Medical School and to Dr. O. H. Lowry of the Harvard Biochemistry Department. In addition, the Department of Pathology of Cornell Medical College has been associated with the work of Dr. McCay in studying pathological changes associated with aging.[151]

The Executive Committee decided to award $60,000 to McCay's research on "aging and longevity" from January 1, 1942.

It is important to notice that Maynard's name disappeared in the project description, and McCay's research was designated as a study of "aging and longevity" rather than "nutrition."[152] This change, which is very significant for McCay and his colleagues' later career, is related to the emergence of "this field" mentioned by Anton Carlson in the above comment, to which McCay, MacNider, Coker, and Carlson himself belonged. What, then, is "this field"?

This question can be answered by appreciating a larger movement in the American academia, which gave McCay another opportunity for the expansion of his research. Its first step was taken when Edmund Vincent Cowdry, a renowned cytologist at Washington University, asked McCay to contribute a chapter to *Problems of Ageing*

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[151] Minutes of Meeting of the Executive Committee of the Rockefeller Foundation, 2 April 1941, Record Group 16, RF. But the fact that McCay supplied the whole rats as well as their organs to other labs was not mentioned in this statement.

[152] Indeed, Maynard had not been so closely involved in research on aging and nutrition as McCay, and he was further dissociated from the actual research after being appointed the chairman of a federal nutrition laboratory at Cornell. Meanwhile, McCay was promoted to a full professor in 1936 and became the official and actual, director of the research. See Frank Blair Hanson Diary, 21 October 1940, Series 200D, Record Group 1.1, RF.

(1939), the first multiauthored handbook on senescence. As will be discussed later, Cowdry tried to make *Problems of Ageing* a comprehensive and cooperative survey on the current state of the science of aging by a group of researchers drawn from various academic disciplines. On November 25, 1936, Cowdry asked McCay to tell him his thoughts on this "cooperative attempt" that should "present and interpret what is known concerning the basic phenomenon of aging."[153] McCay replied that while age had barely been considered a meaningful category in scientific research, he and his colleagues had "appreciated the importance of the problem in both human life and in animal husbandry" and had investigated since the 1920s the phenomenon that restricted feeding increased longevity and retarded aging.[154] According to him, an important reason why aging had not been extensively studied was that it usually took a very long time to examine senile changes, which were "extremely slow and difficult to measure" at least at the level of the whole body of large animals.[155] This difficulty also made it hard to find adequate sources of funding committed to supporting long-term projects. Yet age was an important variable in living organisms that should not be neglected in biological studies. Moreover, as Cowdry emphasized, aging research had to be a cooperative work by scientists in various fields with different backgrounds and approaches. McCay wrote that "the most hopeful fields of attack" were biochemistry, physiology, pathology, histology, physics, and psychology should also be included to get a broader understanding of the phenomenon of senescence.[156] Indeed, such a multidisciplinary approach was what McCay had adopted in his laboratory to study the relation between aging and nutrition. Cowdry was very pleased with this reply and asked McCay to join Cowdry's multidisciplinary book project whose scope was even larger than McCay's—it included anthropology, statistics, pharmacology, cardiology, botany, and protozoology as well as the fields mentioned by McCay.[157] McCay accepted this request by agreeing to write a chapter on "chemical aspects of aging," which reviewed contemporary biochemical

[153] Cowdry to McCay, 25 November 1936, Box 32, Folder 21, EVC.
[154] McCay to Cowdry, 30 November 1936, Box 32, Folder 21, EVC.
[155] McCay to Cowdry, 30 November 1936, Box 32, Folder 21, EVC.
[156] McCay to Cowdry, 30 November 1936, Box 32, Folder 21, EVC.
[157] Cowdry to McCay, 12 December 1936, Box 32, Folder 21, EVC.

studies of senesence, including, of course, his own research on caloric restriction and longevity.[158]

McCay's participation in Cowdry's handbook project led to his deep involvement in the early development of the multidisciplinary science of aging, gerontology. As will be discussed in chapter five, Cowdry, while editing the book, promoted the contributors' interaction with one another. He distributed the summary of each contributor's chapter to other authors, who were encouraged to comment on it. Furthermore, with the support of the Josiah Macy, Jr. Foundation, he planned a conference on aging at Woods Hole, Massachusetts to facilitate cross-disciplinary discussion among the contributors and other participants. On June 25 and 26, 1937, McCay attended this conference, which was indeed the first scientific meeting of researchers on aging in the United States. There he enjoyed meeting with the other contributors to *Problems of Ageing*, many of whom would later become the founding members of the Club for Research on Ageing and the Gerontological Society. Some of them—particularly William MacNider, Baird Hastings, and Anton Carlson—studied longevity and caloric restriction using their expertise. Henry Simms at Columbia University was also attracted to the study of caloric restriction and longevity after 1950.[159] Furthermore, MacNider, Anton Carlson, and Robert Coker wrote strong letters of recommendation for McCay when he applied for the extension of his Rockefeller grant in 1940. As discussed above, their letters were important factors for the Foundation's decision to continue the funding.

Along with these people, McCay actively contributed to the development of the early professional organizations. In 1937, he was appointed a member of the Committee on Cellular Physiology within the National Research Council, which later changed its name to the Committee on the Biological Processes of Ageing.[160] Although the Committee was not very successful in its main job—obtaining funds for gerontological research—it provided a discussion forum for the early researchers on aging in the United

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[158] McCay to Cowdry, 22 December 1936, Box 32, Folder 21, EVC; McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), pp. 572-623. The reason McCay took charge of this chapter was that he had a Ph.D. degree in nutrition and biochemistry.

[159] Benjamin N. Berg and Henry S. Simms, "Nutrition and Longevity in the Rat," *Journal of Nutrition* 71 (1960), pp. 255-263.

[160] Robert E. Coker to Edmund Vincent Cowdry, 8 June 1937, Box 25, Folder 31, EVC.

States. From 1940, he also actively participated in the Club for Research on Ageing, which became the basis for the establishment of the Gerontological Society in 1945. McCay was a core member of both organizations and was elected the fifth president of the Gerontological Society in 1949. Moreover, he worked as a panel member in the Gerontology Study Section within the National Institutes of Health from 1946 to 1949. Although the Section was short-lived, it was the first NIH extramural grant committee specializing in gerontology that offered fellowships to researchers on aging through peer-review process. According to McCay, the Section also functioned as a place of discussion for professional researchers on aging—it gave "an opportunity to compare efforts and exchange ideas with other workers in the field."[161] Indeed, along with his gerontologist colleagues, he reviewed and selected applications, particularly those aimed to study aging in relation to nutrition. If necessary, he visited the applicant's laboratory before giving his advice on the desirability of funding.[162] In reviewing McCay's own grant applications, other members of the Gerontology Study Section stated that "his research in the same field has been so extensive and has been carried on so long that there was little question as to the merit of this request."[163] His grant for 1947-1948 was $29,109, and the NIH kept supporting his research thereafter.[164]

Such a successful integration of McCay and his work into the new science of gerontology was closely related to the fact that his views on senescence were in harmony with the novel ideas on aging promoted and shared by other scientists in the field. First, his research showed that aging could be manipulated through experiments, as Alexis

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[161] McCay to David E. Price, 13 July 1949, Box 21, Folder USPH 584 Prints 1952, CMM.

[162] Gerontology Study Section Minutes of Meeting, 20 November 1946, p. 5; 21 February 1947, p. 4, Box 1, Folder Minutes of Meetings, Record Group 443 Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, NARA.

[163] Gerontology Study Section Minutes of Meeting, 21 February 1947, p. 5, Box 1, Folder Minutes of Meetings, Record Group 443 Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, NARA.

[164] Ernest M. Allen to McCay, 7 July 1947; David Price to McCay, 30 June 1949; Price to McCay, 1 March 1950; Allen to McCay, 24 February 1951; Box 21, Folder USPH 584 Prints 1952, CMM. After the Gerontology Study Section was disbanded, the National Heart Institute, to which Nathan Shock's Gerontology Research Center belonged, supported his research. See Brown, Barnes, Sperling, and McCay, "Radioactive Calcium Osteosarcomas and Squamous Carcinomas," p. 329; Frank Pope, Wanda Lunsford, and Clive McCay, "Experimental Prolongation of the Life Span," *Journal of Chronic Diseases* 4 (1956), p. 153; "NIH Research and Training Grants Active on January 31, 1958," p. 17, Box 12, Folder Annual Reports 1957, NWS.

Carrel, Edwin Conklin, and C. M. Child had argued. The figure of two rats of the same chronological age in his chapter in *Problems of Ageing*, which McCay himself thought had "always [interested] people," was a striking example of how the rate of aging could be artificially altered.[165] (See Figure 2.4.) The rat on the left, which looked quite senile and was definitely dying, was a normally fed one, while the "young" rat on the right, which seemed to be healthy, growing, and even smiling, was given diets with reduced calories. As McCay intended, this picture fascinated both professional scientists and the lay public, although the complex problems emerged through caloric restriction could not be seen in it. Among McCay's professional colleagues, Alexis Carrel, whom McCay had respected as the discoverer of the "immortality" of cultured tissues, discussed in his lecture the meaning of the two rats, and wrote to McCay that they looked "quite striking" to him.[166]

Figure 2.4. *Two Rats of the Same Chronological Age. Clive Maine McCay, "Chemical Aspects of Ageing," in Problems of Ageing: Biological and Medical Aspects, ed. E. V. Cowdry (Baltimore: Williams and Wilkins, 1939), p. 578. The original picture from Box 15, Folder Food-Nutrition Photo, The Clive Maine McCay Papers, Cornell University Archive.*

Second, his experiments supported the concept of aging as an accident or contingency, which was advocated by Alexis Carrel, Raymond Pearl, Edwin Conklin, C.

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[165] McCay to Cowdry, 12 February 1937, Box 32, Folder 21, EVC.

[166] "Carrel Sees Lives Extended for Ages," *New York Times* (13 December 1935); Carrel to McCay, 21 December 1937, Box 64, Folder 9, AC. Actually, McCay sent Carrel his picture of the two rats along with a reprint of his article, which Carrel already had read. See McCay to Carrel, 18 December 1937, Box 64, Folder 9, AC. McCay's respect toward Carrel can be found in McCay's letter to his fiancée. See Retyped letter of McCay to Beyer, 27 April 1924, Box 50, Folder CMM Letters 1923-1927, CMM.

M. Child, Herbert Spencer Jennings, and Peter Medawar.[167] For McCay, the idea that senile changes were merely an "accident" "affords a suitable working hypothesis to discover means of preventing them."[168] Although the complete prevention of senility was not possible, he thought that his experiments showed a way to slow down aging substantially. According to McCay, this proved that "the potential life span is known for no animal species."[169]

Third, McCay's rats supported the widely held idea that aging was not a single process but consisted of many localized processes which were not synchronized. As mentioned in the previous section, the above picture alone did not reveal whether these underfed rats were actually young or merely young-looking, and it was thus necessary to study the animals' body parts more thoroughly and systematically in cooperation with experts in many fields. These studies revealed that aging proceeded with different rates in distinct portions of the body, as advocated by many other scientists of aging, such as Cowdry, Carrel, Pearl, Alfred Cohn, and Charles Minot.[170] First of all, McCay and others' research revealed that while the hair of the underfed rats looked quite young, the bones, which were highly decalcified, showed a sign of advanced senescence.[171] McCay and his colleagues including physicist Leroy Barnes also studied the levels of

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[167] Alexis Carrel, "Tissue Culture and Cell Physiology," *Physiological Reviews* 4 (1924), pp. 1-20; Alexis Carrel, "The New Cytology," *Science* 73 (1931), pp. 297-303; Edwin Conklin, "The Size of Organisms and the Their Constituent Parts in Relation to Longevity, Senescence and Rejuvenescence," *Harvey Lectures*, Series 8, (Philadelphia: Lippincott, 1913), pp. 252-279; C. M. Child, *Senescence and Rejuvenation* (Chicago: University of Chicago Press, 1915); Pearl, *The Biology of Death*, pp. 2-50; H. S. Jennings, "Senescence and Death in Protozoa and Invertebrates," *Problems of Ageing* (1939), pp. 32-52; Peter B. Medawar, "Old Age and Natural Death," *The Modern Quarterly* 2 (1946), pp. 30-49.

[168] McCay, "Nutrition, Ageing, and Longevity," pp. 1-2.

[169] McCay to W. Kaempffert, 15 October 1934, Box 6, Folder Clive, McCay [comments listed], NWS; McCay, "Prolonging the Life Span," p. 414.

[170] Charles S. Minot, *The Problem of Age, Growth, and Death: A Study of Cytomorphosis* (New York: Putnam, 1908), pp. 214-216; Alfred E. Cohn and Henry J. Murray, Jr., "Physiological Ontogeny I. The Present Status of the Problem," *Quarterly Review of Biology* 2 (1927), pp. 482, 490; Pearl, *The Biology of Death*, pp. 138-149, 225; Carrel, "Tissue Culture and Cell Physiology," pp. 1-20; Carrel, "The New Cytology," pp. 297-303; E. V. Cowdry, "Ageing of Tissue Fluids," *Problems of Ageing* (1939), pp. 643, 685, 689. Also see Nathan W. Shock, "Ageing of Homeostatic Mechanism," in Albert I. Lansing (ed.), *Cowdry's Problems of Ageing*, third edition (Baltimore: Williams and Wilkins, 1952), pp. 421, 429-31, 436, 438; A. J. Carlson to Cowdry, 28 June 1937, Box 10, Folder 397, WDM.

[171] C. M. McCay, "Chemical Aspects of Ageing," in *Problems of Ageing*, p. 574; "The Conservative Attack upon Problems of Aging," *Geriatrics* 13 (1958), p. 712; Frank Pope, Wanda Lunsford, and C. M. McCay, "Experimental Prolongation of the Life Span," *Journal of Chronic Disease*, 1956, 4:153-158, on p. 156.

calcification and decalcification in other portions of the body such as the costal cartilages, the aorta, and the kidneys, which, the researchers thought, would indicate the rates of senescence in different parts of the body. They found less calcified costal cartilages in underfed rats, which implied that their aging was retarded, while the aorta and the kidneys of the same rats were more extensively calcified.[172] Probably based on the result of this research, McCay wrote in 1939 that "this may also be an illustration of the tendency of different organs to age at different rates."[173] Later pathological and dental research conducted by Saxton, Smith, and others led them to a similar conclusion. When the underfed rats' teeth did not demonstrate much difference from those of control rats, the frequency of several age-associated chronic diseases, such as lymphosarcoma, leukemia, and chronic nephrosis, was considerably decreased by caloric restriction.[174] Likewise, histochemical examination by Hastings and his colleagues at Harvard Medical School revealed that the chemical composition of the underfed rats' skeletal muscle was closer to that of younger rats.[175]

McCay's research was favorably received by other gerontologists, probably because of the above factors—the local distinctiveness and contingency of aging as well as the possibility of experimental manipulation of senile changes—in which many other gerontologists were interested as well. Indeed, McCay's research was regarded as a project in need of special attention, demanding more systematic investigation and brainstorming whose first step could be taken by scientific conferences.[176] Hence, gerontologists convened at Ithaca on November 1 and 2, 1941 for the "Conference on

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[172] C. M. McCay, G. H. Ellis, LeRoy L. Barnes, C. A. H. Smith, and Gladys Sperling, "Chemical and Pathological Changes in Aging and After Retarded Growth," *Journal of Nutrition* 18 (1939), pp 15-25.
[173] McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), p. 574. Also see C. M. McCay, "The Conservative Attack upon Problems of Aging," p. 712.
[174] Saxton and others' pathological research till 1945 was summarized in John Saxton, "Nutrition and Growth and Their Influence on Longevity in Rats," in Jaques Cattell, (ed.), *Biological Symposia*, vol. 11 (Lancaster, Pennsylvania: The Jaques Cattell Press, 1945), pp. 177-196. See also Smith, Light, and McCay, "Advanced Age in Relation to Dental Caries in White Rats," pp. 1700-1703; Smith and McCay, "Advanced Age in Relation to Dental Caries in White Rats. II," pp. 340-344.
[175] Lowry, McCay, Hastings, and Brown, "Histochemical Changes," pp. 281-284.
[176] For the reception of McCay's work among professional gerontologists, see E. J. Stieglitz to Walter B. Cannon, 14 April 1941, Box 82, Folder 1114, WBC; V. Korenchevsky to W. MacNider, 24 April 1941, Box 82, Folder 1113, WBC; The Club for Research on Ageing: Report of the Committee upon the Importance of Future Research into the Biology of Senescence, 15 September 1941, Box 82, Folder 1113, WBC.

Nutritional Requirements for the Ageing Population." Many of the founding members of the Club for Research on Ageing and the scientific staff of Cornell's animal husbandry department attended the conference to talk about the future directions of the research, its meaning for gerontology in general, and the implications for the social problems of the ageing population in the United States. The list of these participants—including A. J. Carlson (physiology), John Saxton (pathology), Nathan Shock (psychological physiology), Leroy Barnes (physics), Lawrence Frank (B.A. in economics, Executive Secretary of the Macy Foundation), and McCay and his colleagues at the animal husbandry department—showed that the research on nutrition and longevity transcended its origin in animal husbandry and became a focus of gerontology's multidisciplinary investigation.

Interestingly, many of the problems found in McCay's underfed rats, which made them less useful for animal husbandry or agricultural applications, greatly interested these multidisciplinary participants and motivated them to suggest further research, in addition to those that had already been done by McCay's team. For example, his team's research found the underfed rats' severely decalcified bones and heavily calcified aorta and kidneys, although these phenomena became less intense during the second experiments.[177] Frank Fremont-Smith of the Josiah Macy Foundation pointed out that to get a more reliable result on this issue McCay should control an important variable—the degree of rats' exercise—which influenced the deposition of calcium in the bone.[178] A. Baird Hastings of Harvard Medical School pointed to a more basic issue concerning this problem, namely, the underfed rats' calcium metabolism. He argued that retarded and control rats' ability to use and maintain calcium in the blood should be tested using calcium citrate or radioactive strontium, if the reasons why underfed rats' bones were decalcified were to be known.[179] Ephraim Shorr of Cornell Medical College agreed with

[177] "Conference on Nutritional Requirements for the Ageing Population," p. 7, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[178] "Conference on Nutritional Requirements for the Ageing Population," p. 7, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[179] "Conference on Nutritional Requirements for the Ageing Population," p. 8, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.

Hastings and proposed that the effect of introducing factors influencing decalcification, such as vitamin D, citrates, and estrogen, needed to be understood as well.[180]

The reproductive problem of underfed rats also became a focus of an intense discussion. McCay reported that retarded male rats might show diminished fertility since their sperm's motility was low.[181] Yet Earl T. Engle of Columbia University immediately pointed out that the retarded male rats' actual breeding ability should be directly examined, because the "motility of sperm is not an adequate or sure criterion of fertility."[182] While McCay admitted that none of his rats had yet been allowed to breed, it was certain that future research should include tests of their reproductive capacity, particularly under the influence of various dietary factors such as vitamins. The absence of estrus cycle of female underfed rats and its recovery after returning to normal diets was another topic of discussion. Shorr stated that he had noticed a similar case through his clinical experience. Just as the amenorrhea of underfed rats was "treated" through sufficient calories in the diets, the amenorrhea of human females could also be treated with estrogen therapy.[183] This statement was followed by further comments from Carlson and Hastings, who emphasized the need for studying endocrinal functions of retarded animals.[184] It was also suggested that a further study could be conducted about the influence of adding extra hormones upon longevity and aging.[185]

Interestingly, these physiological problems caused by caloric restriction did not hinder gerontologists from thinking about the possibility of applying McCay's research to the contemporary social problems of aging. Indeed, as many historians have pointed out, the United States during the 1930s and 1940s underwent major changes in the status of its

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[180] "Conference on Nutritional Requirements for the Ageing Population," p. 8, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[181] "Conference on Nutritional Requirements for the Ageing Population," p. 1, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[182] "Conference on Nutritional Requirements for the Ageing Population," p. 2, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[183] "Conference on Nutritional Requirements for the Ageing Population," p. 6, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[184] "Conference on Nutritional Requirements for the Ageing Population," p. 7, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[185] "Conference on Nutritional Requirements for the Ageing Population," p. 7, 1 and 2 November, 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.

elderly citizens.[186] Particularly, the severe economic recession during the Great Depression was thought to eliminate the elderly workers' jobs and other means of support. This concern eventually contributed to establishing the Social Security Act, which institutionalized mass retirement of those over sixty-five years of age. This series of events led many people to ask whether it was natural for workers to retire at sixty-five and whether there were any other ways for them to keep working and to adapt themselves better in the job market and workplaces. One of these ways could be found in reducing body weight through caloric restriction. A. V. Moore of Cornell University pointed out that during his clinical practice he found "few obese persons above the age of seventy."[187] Agreeing with Moore, L. S. Cottrell suggested that actual investigation into the relation of mortality and obesity should be undertaken. At this suggestion, Lawrence Frank, Shorr, and Carlson pointed out that such an investigation should be done using the population with more than average social status such as retired farmers, since the use of poor elderly people, especially those in almhouses and mental hospitals might exaggerate the aged human's inferiority.[188] At the same time, Frank asked whether it was possible to conduct an experiment to test the effect of better diets plus proper exercise upon an aged person's body and mind "with the idea that retirement would not be demanded at sixty-five if significant improvement in physical and mental conditions occurred" through such means.[189] In agreement with Frank, Carlson pointed out that "one of the chief aspects of ageing is the psychological effect of being cut off from the "stream of life" by

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[186] Achenbaum, *Old Age in the New Land*, pp. 127-141; *Social Security: Visions and Revisions, A Twentieth Century Fund Study* (Cambridge: Cambridge University Press, 1986), pp. 13-37; Carole Haber and Brian Gratton, *Old Age and the Search for Security: An American Social History* (Bloomington: Indiana University Press, 1994), pp. 139-142; 181-185; John Macnicol, *Age Discrimination: An Historical and Contemporary Analysis* (Cambridge: Cambridge University Press, 2006), pp. 211-223; William Graebner, *A History of Retirement: The Meaning and Function of an American Institution, 1885-1978* (New Haven: Yale University Press, 1980), pp. 181-214.

[187] "Conference on Nutritional Requirements for the Ageing Population," p. 10, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS. Indeed, the Metropolitan Life Insurance Company's report arrived at the conclusion that underweight was statistically related to the longer life among the elderly. See "Further Facts on Body-Weight and Longevity," *Statistical Bulletin, Metropolitan Life Insurance Company* 4:3 (1923), pp. 2-4.

[188] "Conference on Nutritional Requirements for the Ageing Population," p. 10, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.

[189] "Conference on Nutritional Requirements for the Ageing Population," p. 10, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.

retirement."[190] Therefore, "absolute retirement" should be avoided by all means.[191] On this issue, Frank further commented that the "new physiological criteria of ageing" should be developed, which could substantially differ from chronological age.[192] Other participants, such as Nathan Shock of the National Institutes of Health, discussed the physiological parameters that should be measured to determine physiological age, such as reaction time, renal function, responses to lowered oxygen, muscles' mechanical efficiency, basal pulse rate, and so on. Many of these parameters would actually be measured by Shock in his longitudinal study of aging at the NIH that began in 1958. (See Chapter 6.) Fremont-Smith suggested that these tests could benefit large manufacturing corporations like General Motors which might hope to "adjust jobs to capacities of aged."[193]

Psychological aspects concerning nutrition and longevity were also mentioned, since, according to Frank, "nutrition is not purely a biological problem."[194] Carlson commented that "food habits in man may have a psychological basis" because "eating is often an escape mechanism in the human."[195] But this "escape" might lead a person to eat food with too many calories, such as candy, cake, and ice cream, which eventually could bring about obesity and early death. While these issues were not discussed further during the conference, McCay later succeeded in beginning a study of the influence of caloric restriction upon mental capacities. In the early 1940s, he studied underfed dogs' behavior in cooperation with W. T. James at the University of Colorado.[196] Indeed, McCay had already recognized the importance of psychological approaches when he had

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[190] "Conference on Nutritional Requirements for the Ageing Population," p. 10, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[191] "Conference on Nutritional Requirements for the Ageing Population," p. 10, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[192] "Conference on Nutritional Requirements for the Ageing Population," p. 11, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[193] "Conference on Nutritional Requirements for the Ageing Population," p. 11, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[194] "Conference on Nutritional Requirements for the Ageing Population," p. 9, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[195] "Conference on Nutritional Requirements for the Ageing Population," p. 9, 1 and 2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.
[196] W. T. James and C. M. McCay, "An Analysis of the Effect of Retarded Growth on Behavior in Dogs," *Journal of Comparative Psychology* 37 (1944), pp 173-185.

applied for a grant from the Rockefeller Foundation in 1935. Although the Foundation refused to award a grant for employing a psychologist,[197] McCay was able to cooperate during the 1950s with the department of psychology at Cornell to study the learning capacity of retarded and normal rats with the support from the NIH.[198]

In retrospect, this 1941 conference and the subsequent developments in gerontology show that McCay's research program and his rats functioned as a "boundary object" or "trading zone" among disciplines. Peter Galison has studied how people trained in distinct traditions could collaborate by making a "trading zone" where they could partially share theories and practices.[199] In a similar vein, Ilana Löwy, Susan Leigh Star, and James Griesemer have argued that the "boundary objects," which might be either a concept or an artifact, could be created as a channel of communication between clinicians and experimenters or amateurs and professionals, while the boundaries themselves remained intact.[200] As the discussions during the conference showed, McCay's rats and his project began to be used as such a channel of communication among scholars from various fields, including physiologists, clinicians, psychologists, pathologists, and foundation officers with social science background. While all these scholars remained as specialists in their own field, they could talk to one another through their common object—McCay's underfed rats and their complex problems concerning aging and longevity. The multidisciplinary science of gerontology offered McCay an

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[197] Warren Weaver to Madison Bentley, 27 April 1937, Box 136, Folder 1688, Record Group 1.1, Series 200D, RF. Madison Bentley was a professor of psychology at Cornell, and proposed to study the psychological aspects of aging with cooperation of McCay.

[198] "Progress from Research upon Aging during the Year 1951"; "Progress from Research upon Aging during the Year 1953," Box 21, Folder USPH 584 Prints 1952, CMM. Unfortunately, however, this cooperative project did not produce a publishable result. See C. M. McCay, Frank Pope, and Wanda Lunsford, "Experimental Prolongation of the Life Span," *Bulletin of the New York Academy of Medicine* 32 (1956) pp. 91-101.

[199] Peter L. Galison, *Image and Logic: A Material Culture of Microphysics* (Chicago: University of Chicago Press, 1997), pp. 781-844.

[200] Ilana Löwy, *Between Bench and Bedside: Science, Healing, and Interleukin-2 in a Cancer Ward* (Cambridge, Mass.: Harvard University Press, 1996), pp. 247-253; "The Strength of Loose Concepts—Boundary Concepts, Federative Experimental Strategies, and Disciplinary Growth: The Case of Immunology," *History of Science* 30 (1992), pp. 371-396; Susan Leigh Star and James R. Greisemer, "Institutional Ecology, 'Translations,' and Boundary Objects: Amateurs and Professionals in Berkeley's Museum of Vertebrate Zoology, 1907-39," *Social Studies of Science* 19 (1989), pp. 387-420.

opportunity to make his project relevant for many different research programs and interests without disturbing their disciplinary borders.

Yet the investigation on caloric restriction and longevity could be a boundary object only in so far as the researchers did not attempt an actual human application. Indeed, the expansion of McCay's research brought about the involvement of the fields concerning humans, such as psychology, medicine, human nutrition, and the social sciences. If the scientists in these fields, particularly those with more practical bent, wanted to make actual use of McCay's work, they would face several significant problems incurred by caloric restriction, such as decreased body size and the problems in sexual organs. As McCay's colleage Gladys Sperling wrote to the elderly lay people who inquired about "longevity diets," McCay's method was a "too drastic treatment to be used on humans."[201]

Admittedly, McCay and his colleagues did conduct an experiment in 1941 with a more practical implication for humans, namely, the study of the effect of caloric restriction applied after the rat passed their middle age. Unlike his previous experiments which supplied drastically reduced number of calories to the rats right after weaning, this 1941 study allowed rats to eat as they wanted before their 485th day, when the caloric restriction began. This experiment showed that the rats underfed after their middle life lived longer than the controls. Yet the degree of life extension was not great in this case, and the rats' health state was not studied in any detail.[202] Moreover, McCay never conducted any further experiments on the effect of underfeeding after middle life, and his earlier work on caloric restriction right after weaning was considered his major achievement.

Regardless of what these early experiments showed about the state of the underfed rats' body, the implications of his discovery for human life fascinated many people. For physicians, it suggested a way to reduce tumors in old age, although its direct medical application was hardly practicable. To Lawrence Frank and others who were

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[201] Sperling to Forest J. Sur, 6 December 1944, Box 1, Folder Correspondence 1943-1944, GS.
[202] C. M. McCay, L. A. Maynard, G. Sperling, and Harlow S. Osgood, "Nutritional Requirements during the Latter Half of Life," *Journal of Nutrition* 21 (1941), pp 45-60.

concerned about the elderly's employment problems, McCay's research might reveal a way to enhance old laborers' fitness in their work places. For McCay himself, the relevance of his research to humans was a way to get recognition and support from both professional scientists and general public. Thus he often implied how his work was related to human longevity by mentioning in his research articles historical figures such as Luigi Cornaro, a sixteenth century Italian nobleman who had attributed his long life to restricted diets.[203] He also tried to show his readers the meaning of his rats' longevity from the standpoint of human life by emphasizing that the rat's ten days were equivalent to the human's one year.[204] According to this scheme, many humans had a chance to pass their 120th birthday, just as many underfed rats could survive after their 1,200 days.

The general public enthusiastically received this message. From the 1930s, he and his rats frequently appeared in newspapers and magazines and attracted readers' attention. Sometimes, journalists contacted McCay to hear more about his work,[205] and he and his colleagues publicly displayed their rats to disseminate the meaning of their research among doctors and lay people.[206] Indeed, gerontology as a scientific field was established as responses to human's social problems in the 1930s and 1940s, and gerontologists tried to communicate with the general public as far as they could. (See Chapter 6.) The enthusiastic public reception of McCay's research and its implication for human longevity can be understood as a result of this effort on his part, which Henry Sherman, in response to the Rockefeller Foundation's query, critically described as an unprofessional attempt of going "out for publicity." Despite such a critical comment by

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[203] McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), p. 581. Indeed, history was important for McCay, who frequently mentioned previous scholars of nutrition in his articles and even opened a course on the history of nutrition at the College of Agriculture. About McCay's interest in the history of nutrition, see Franklin C. Bing, "Clive McCay and Our Job of Work in Recording the History of Nutrition: A Paper Presented at a Meeting of the American Institute of Nutrition in Ithaca, New York," August 16, 1973, Cornell University Library; E. N. Todhunter, "Clive M. McCay: Nutrition Historian, Teacher, and Bibliophile," Box 53, Folder Todhunter Nutrition History, CMM. McCay's lectures on the history of nutrition were published after his death. See Clive M. McCay, *Notes on the History of Nutrition Research*, edited by F. Verzár (Berne: Huber, 1973).

[204] McCay, "Nutrition, Ageing, and Longevity," p. 2.

[205] McCay to W. Kaempffert, 15 October 1934, Box 6, Folder Clive, McCay [comments listed], NWS.

[206] "Notes on A.M.A. Meetings, June 6-12, 1942," Box 1, Folder Notes on AMA meeting 1942, GS. At the American Medical Association's annual meeting of 1942, held at Atlantic City, McCay's team made a booth to display their rats. Physicians, medical scientists, and laymen came to see their booth. For a picture of the booth, see Box 1, Folder Atlantic City, GS.

one of his colleagues, however, McCay's public activities contributed to a more important change that occurred during this period, that is, the expansion of his research from the domain of agriculture and animal husbandry to other broader arenas. At the same time that his work became a boundary object for cross-disciplinary research in gerontology, it gained a meaning for Americans' healthy and active old age. The next section will explain how McCay's attempt of popularizing his work was received by the general public in the American culture.

Calories, Simple Diets, and the Civilized Old Age in American Culture

In the history of the United States, precursors to McCay's low-calorie food can be traced back to the simple vegetarian diets advocated by Sylvester Graham (1794-1851). According to him, most forms of debility were caused by the foods that overly stimulated the body, such as meat, alcohol, and coffee, especially when they were consumed in a large amount. These kinds of foods also encouraged excessive sexual desire and led to the indulgence in masturbation and sexual intercourse, which further worsened the debility and moral deterioration. To maintain healthy and ethical life, therefore, he recommended temperance, sexual continence, vegetarianism, and the consumption of small amount of food, including the bread he himself developed.[207]

Historian Stephen Nissenbaum and others have argued that some contemporaries enthusiastically accepted and practiced Graham's ideas, and certain medico-religious groups—such as the advocates of hydrotherapy, Ellen G. White (1827-1915) and the Seventh Day Adventists, and John Harvey Kellogg (1852-1943) at his Battle Creek Sanitarium—continued his program into the twentieth century.[208] Interestingly, in his

[207] Sylvester Graham, *Lectures of the Science on Human Life*, vol. II (Boston: Marsh, Capen, Lyon and Webb, 1839), pp. 361-399, 523-610; *A Lecture to Young Men on Chastity, Intended Also for the Serious Consideration of Parents and Guardians* (Boston: Light, 1839); *Treatise on Bread and Bread Making* (Boston: Light and Stearns, 1837).

[208] Stephen Nissenbaum, *Sex, Diet, and Debility in Jacksonian America: Sylvester Graham and Health Reform* (Westport, Conn.: Greenwood, 1980), pp. 140-157. Also see John P. Coleman, "Casting Bread on Troubled Water: Grahamism and the West," *Journal of American Culture* 9 (1986), pp. 1-8; Jayme A. Sokolow, *Eros and Modernization: Sylvester Graham, Health Reform, and the Origins of Victorian Sexuality in America* (London: Associated University Press, 1983).

later years, McCay himself considered Graham one of the "pioneers of nutrition."[209] McCay also felt a deep sympathy toward the teaching of White and the Seventh Day Adventists, and wrote articles on how his research could "confirm" their claims on simple diets.[210] It is thus not surprising that McCay even exchanged correspondence with the White family.[211]

Indeed, the science of nutrition of the early twentieth century supported, at least partially, Graham, White, and Kellogg's emphasis on simple diets. Historians Margaret A. Lowe and Harvey Levenstein have pointed out that this science contributed to a considerable change in Americans' thoughts and practice about diets during the early twentieth century.[212] While food had been prepared and chosen according to its taste and appearance, new nutrition scientists—including McCay's teachers and colleagues E. V. McCollum, Henry Sherman, and Lafayette Mendel—during these decades made it a norm for American laymen to check nutritive value and, more importantly, the number of calories in diets before consumption. The food with high calorie content and low essential nutrients such as vitamins and minerals began to be condemned, and obesity, which was thought to be caused by such food, was abhorred in both medical and esthetic point of view.

An article published in *Popular Science* in 1941 with the statement, "Hungry people, Dr. McCay has found, live longest," shows how McCay's works were received in America where Grahamite tradition left a strong mark and new scientists of nutrition further transformed its dietary ideas and practices.[213] This article summarized the result of McCay's works on longevity and nutrition with a picture of McCay and his two famous rats, along with a legend saying that "RAT LIFE IS A SPEED-UP OF HUMAN

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[209] C. M. McCay, "Four Pioneers in the Science of Nutrition—Lind, Rumford, Chadwick, and Graham," *Journal of the American Dietetic Association* 23 (1947), pp. 397-402.
[210] C. M. McCay, "Science Confirms Adventist Health Teachings," *Review and Herald* (19 February 1959), pp. 5-6.
[211] See, for example, Herbert C. White to McCay, 21 January 1959, McCay to White, 26 January 1959, Box 16, Folder Mrs White Aug 1959, CMM.
[212] Lowe, "From Robust Appetites to Calorie Counting," pp. 37-61; Harvey A. Levenstein, *Revolution at the Table: The Transformation of the American Diet* (New York: Oxford University Press, 1988), pp. 147-160.
[213] Edwin Teale, "Stay Hungry and Live Longer," *Popular Science* (October 1941), p. 97.

LIFE."[214] But this article never mentioned that nobody had ever tried to use for human infants the method McCay had devised for his rats. What was more important, however, was the general message rather than a specific scientific method and the result obtained by it: Too many calories in the food were harmful for health and longevity. This article also emphasized that what McCay advocated was not malnutrition but less calorie and full nutritive content in the diet. It asked, "Why are not the Chinese, who have been underfed for generations, the longest-lived race on earth?"[215] The reporter said that Chinese had not lived long because they simply had been starved, while McCay's rats were fed enough essential nutrients, although their dietary calories were reduced.

Major newspapers such as *New York Times* also enthusiastically reported the findings of McCay. An article of March 31, 1938 called his rats "the Methuselahs of the Animal World" and stated that the birth of such a "Methuselah" by a scientific method might mean that "parents will have it within their power to control the prospective life-span of their children and even make it possible for them to 'begin life at 100.'"[216] Yet another article of April 3, 1938 approached the issue more critically. It pointed out that "apart from the fact that physiological and chemical actions are not necessarily the same in rats and men and that we may not therefore conclude that we humans may become centenarians by starving ourselves in early life, we wonder what a century means in these experiments."[217] This article then asked, "Would life be worth living," if a person succeeded in attaining a greatly increased longevity by applying McCay's method to his or her own body?[218] With this question, the article raised a question on the *quality* of life in old age, which should be "more than a watery eye, a leathery, wrinkled skin, a loss of interest in the affairs of the world."[219] It would be better for a human to "become like the butterfly, which, according to Anatole France, presents an example to man by dying at the height of its beauty and its joy in life."[220]

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[214] Teale, "Stay Hungry and Live Longer," p. 97.
[215] Teale, "Stay Hungry and Live Longer," p. 99.
[216] "Longer Life-Span in Biologists' Aim," *New York Times* (31 March 1938).
[217] "Rats, Men, Longevity," *New York Times* (3 April 1938).
[218] "Rats, Men, Longevity."
[219] "Rats, Men, Longevity."
[220] "Rats, Men, Longevity."

Historian Thomas Cole has argued that such an attitude was the basis of the ideal of "civilized old age" which had existed in America since the mid-nineteenth century.[221] According to Cole, this ideal was born among middle-class gentlemen, who cherished diligence, hard-working, and rational design of life which were thought to prevent or considerably delay decrepitude, dependence and chronic diseases in old age. According to Cole, this ideal began to be formed in American culture along with the rise of industrial capitalism, which encouraged self-discipline and rational planning of life. An ideal elderly gentleman was thus the person who maintained health and vigor even in his extreme old age through his industrious and productive life style which he had kept since his younger years. As he came to the end of his lifespan determined by god, this elderly gentleman was expected to die a "natural death," without suffering from chronic diseases and decrease of biological and social activity.

While the author of the above article in *New York Times* implied that McCay's method merely increased lifespan and could not lead to the "civilized old age," other journalists thought differently. J. D. Ratcliff in *Collier's* magazine asked, "Can man's useful, productive life be prolonged through what are now his declining years?"[222] Although this question could not be easily answered, "a most promising start has already been made" through McCay's research on aging and caloric restriction.[223] After summarizing McCay's work briefly, Ratcliff described him as the person who "practices what he preaches."[224] Thinking that "overeating is criminal," McCay was holding "weight rigidly at 140 pounds" by being careful about the amount of energy and nutrients in his food.[225] Perhaps for this reason, he had maintained himself as a highly productive

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[221] Cole, *Journey of Life*, pp. 139-158. Cole has also pointed to the negative aspects of this ideal in relation to biomedical scientists' works. According to Cole, even when these scientists argued for the possibility of longer and healthier life—and even rejuvenation and immortality—which could be realized through new scientific methods, they ultimately highlighted the tragedy of aging. Ironically, their argument for a better and healthy old age defined usual phenomena of growing old, including decreasing vigor and occurrences of disease, as an undesirable state, because these phenomena did not meet the criteria of ideal old age that could be reached through science. See Cole, *The Journey of Life*, pp. 209-211, 227-230.

[222] J. D. Ratcliff, "Let's Live a Little Longer," *Collier's* (13 March 1943), p. 11.

[223] Ratcliff, "Let's Live a Little Longer," p. 11.

[224] Ratcliff, "Let's Live a Little Longer," p. 72.

[225] Ratcliff, "Let's Live a Little Longer," p. 72.

scientist, who had "enough energy for two or three average men."[226] Another article in *Lifetime Living* highlighted this aspect of McCay further. He was depicted as a lean and self-disciplined middle aged man, who practiced the best way of life he himself advocated. He "works smoothly, continuously and without hurry, whether he is watching his stock, nursing a sick dog, preparing lecture notes for his full-time teaching schedule at the College of Agriculture and in the Cornell School of Nutrition, planning and directing a dozen current research projects or [persuasively] returning the fire of a hostile interrogator at a Congressional hearing."[227] Indeed, "nothing interrupts the schedule, including the numerous visitors, who are simply swept into the steady current of the McCay activities."[228] Probably, an important factor making this busy and orderly life possible was the food he had eaten with his family. The article did not fail to emphasize that "they eat well" the "nutritionally sound and tasty" food prepared at "a functional, well-equipped kitchen" that was "Mrs. McCay's laboratory."[229] The article detailed her recipes in a separate page.

Another article in *Harper's Magazine*, titled "The Thin Rats Bury The Fat Rats," approached the issue of civilized old age as that of national significance. James Rorty, the author of the article, argued that "because we eat too much we are too fat and because we are too fat we victimize ourselves and burden our relatives and the state by becoming the premature victims of the degenerative diseases."[230] He then recommended the diet which was "cheap, easy to prepare, and shy on the high caloric sugars, starches, and fats," such as "coffee with milk but no sugar or cream….One egg. Citrus and other fresh fruit in quantity sufficient to satisfy hunger. Whole grain muffin or bread. One part of butter."[231] Although this diet was easy to prescribe, it was less easy even for "intelligent and reasonably well disciplined laymen" to practice it since the whole American food industry was structured around producing and selling the food with high-calories and low

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[226] Ratcliff, "Let's Live a Little Longer," p. 72.
[227] "Nutrition….And You," *Lifetime Living* (April 1953), pp. 23-24.
[228] "Nutrition….And You," p. 24.
[229] "Nutrition….And You," p. 24.
[230] James Rorty, "The Thin Rats Bury the Fat Rats," *Harper's Magazine* (May 1949), p. 31.
[231] James Rorty, "The Thin Rats Bury the Fat Rats," p. 32.

nutritive value.[232] However, it was extremely important to find a way to live with the diets with low caloric value and high nutritive content, since an "overfed and overweight America is aging rapidly."[233] As the size of elderly population was continuously increasing, widespread adoption of better but simple diets would substantially reduce the cost of the care of the aged and make the whole population healthier and more active. In this sense, McCay's research was highly valuable.

McCay himself wrote popular articles, stressing the importance of food with low calories and adequate nutritive content.[234] After reading one such article in *New York Times Magazine*, W. Ross McKnight at Stanfordville, New York, who was then sixty-five years old, wrote to McCay how he became a slender and healthy person, desired by his employer, doctor, and insurance company.[235] McKnight said that when he had been thirty-five years old, he had gone on an extended trip for eleven months, eating only very bad food that had made him "a walking copy of Rameses II, no good to [himself] or to [his] employer."[236] He was "dog-tired of being "treated" for symptoms by [his doctor]," and life insurance companies refused to offer him any coverage.[237] However, he gradually realized what the real problem was about him. "Based on vitally important personal experience as well as on extended observation," he knew that "the number one curse paid on humanity is just plain gluttony."[238] He then "went on a simple diet of antitoxic foods—no animal products at all save a little butter, cheese, and milk."[239] Through overcoming gluttony in this way, he "rebuilt [his] body….[and] walked into the head medical offices of the life insurance companies and got the insurance [he] wanted

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[232] James Rorty, "The Thin Rats Bury the Fat Rats," p. 32.
[233] James Rorty, "The Thin Rats Bury the Fat Rats," p. 34.
[234] See, for example, C. M. McCay, "Better Food for the Later Years," in T. Lynn Smith (ed.), *Living in the Later Years* (Gainesville, University of Florida Press, 1952), pp. 107-122.
[235] C. M. McCay, "America Is Learning What to Eat," *New York Times Magazine* (28 March 1943), pp. 10, 27. Although McCay did not directly mention his work on caloric restriction and longevity using rats, he did write, "Thousands of Americans have gone to premature graves from overeating and keeping their bodies too fat."
[236] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS.
[237] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS.
[238] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS. The line under "gluttony" was drawn by McKnight himself.
[239] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS.

without any strings to it."[240] After that, he had not "had an illness that would cause [him] to miss time from work….and [hadn't] had a 'cold' or a twinge of rheumatism, for longer than [he] can remember—certainly not within the past 20 years."[241] By taking care of his food and restraining gluttony, he could be a healthy and active old man who had seldom missed his work due to illness. In Thomas Cole's words, this was his "civilized old age," which he wanted to show as a good example of what meaning McCay's work had for the general public.

Conclusion

McCay did not stop working for animal husbandry and agriculture, even while the meanings of his research for human life were widely discussed among the general public. In 1943, he published *Nutrition of the Dog*, which would later become a classic monograph summarizing the current knowledge on feeding dogs.[242] In 1947 he also constructed a dog farm near the Cornell campus to pursue a more systematic research in dog raising and nutrition. For these works, he won "the National Dog Week Award" in 1948.[243] He also kept thinking about how his work on caloric restriction could be used for agriculture and animal husbandry, and argued in the early 1950s that farm animals' growth could be retarded by feeding low calorie diets when their food was expensive and its amount insufficient.[244] He claimed that the animals could be returned to a normal diet when the food price fell and its amount increased. This diet would immediately make the animals resume their normal growth and might lead to a longer and healthier life.

McCay worked on the general problems in human nutrition as well. During the Second World War, he advocated the consumption of inexpensive yet nutritive foodstuff such as the sprouted soybean, which, he argued, contained sufficient amounts of proteins,

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[240] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS.
[241] McKnight to McCay, 8 May 1943, Box 6, Folder McCay, Clive [comments listed], NWS.
[242] C. M. McCay, *Nutrition of the Dog* (Ithaca, N.Y.: Comstock, 1943).
[243] C. M. Olson to McCay, 7 June 1948, Box 11, CMM.
[244] "Studies in the Longevity of Farm Animals," Box 53, Folder Warnerton Farm, CMM. This manuscript by McCay cites C. M. McCay, "A Simplified Nutrition Program for the Latter Half of Life," *Journal of the American Geriatrics Society* 2 (1954), pp. 417-421. Also see C. M. McCay, "Effect of Restricted Feeding upon Aging and Chronic Diseases in Rats and Dogs," *American Journal of Public Health*, 37 (1947), p. 522.

vitamins, and essential minerals.[245] During the same period, he served the Navy as a lieutenant commander and the head of the "Mobile Nutrition Unit" of the National Naval Medical Research Institute, in order to investigate the nutritional adequacy of the military rations.[246] After the war, McCay also studied the effect of soft drinks upon teeth,[247] and participated in the study of the nutritive value of irradiated food, which was sponsored by the U. S. Army, when irradiation was considered a method of long-term food storage as well as a way for peaceful uses of atomic energy.[248]

Whereas McCay did include aging as a topic in this series of human nutrition studies, he was interested in only moderate ways of maintaining health in old age rather than drastic measures such as caloric restriction during early life.[249] With the cooperation of New York State Department of Mental Hygiene and the American Dry Milk Institute, this interest and research led him to invent the "Cornell Bread," just as Graham had made his bread.[250] McCay argued that his bread, enriched with dry skim milk, wheat germ, and soy flour, was particularly good for elderly people due to its high calcium and protein content.[251] Since the aged tended to lose more calcium than the young,[252] the Cornell

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[245] C. M. McCay, "Sprouted Soybeans," Box 1, Folder Bread Analysis Formula Discussion, CMM; Leonard A. Maynard, *Early Years of the Graduate School of Nutrition at Cornell, 1941-1956*, p. 29, Box 56, CMM.

[246] See, for example, "Food Experts Here for Study," *Norfolk Seabag: U. S. Naval Training Station* (13 May 1944). Other papers and files on McCay's works at the Navy can be found in Box 2, Folder Navy Clippings, CMM. He served the navy from July 13, 1943 to July 15, 1946. See "World War II Military Service Certificate of the New York State: Employees' Retirement System," Box 11, CMM.

[247] J. S. Restarski, R. A. Gortner, Jr., and C. M. McCay, "Effect of Acid Beverage Containing Fluorides upon the Teeth of Rats and Puppies," *Journal of the American Dental Association* 32 (1945), pp. 668-675; C. M. McCay and Lois Will, "Erosion of Molar Teeth by Acid Beverages," *Journal of Nutrition* 39 (1949), pp. 313-324.

[248] U. S. Army Natick Laboratories, "Fact Sheet: The Army Food Irradiation Program and the Radiation Laboratory," Box 5, Folder Third, CMM; C. M. McCay and G. L. Rumsey, "Final Report—Part I: Effect of Ionized Radiation on the Nutritive Value of Food (Beef) as Determined by Growth, Reproduction, and Lactation Studies with Dogs," 15 March 1960, Box 8, CMM.

[249] See, for example, C. M. McCay, "Ten Rules for Good Nutrition After Forty," 6 June 1950, Box 1, Folder Age, CMM; C. M. McCay, "Better Food for the Later Years," in T. Lynn Smith (ed.), *Living in the Later Years* (Gainesville: University of Florida Press, 1952), pp. 107-122; C. M. McCay, "Nutritional Factors in the Maintenance of Bones and Teeth," *New York State Dental Journal* 17 (1951), pp. 429-435.

[250] C. M. McCay, "What Consumers Should Know about Bread," *Journal of Home Economics* 41 (1949), pp. 179-181.

[251] C. M. McCay, "Bread, Potatoes, and Milk….For Long, Healthy Life," *Farm Research* (June 1958), p. 8; Leonard A. Maynard, *Early Years of the Graduate School of Nutrition at Cornell, 1941-1956*, p. 29, Box 56, CMM.

Bread could restore the lost calcium as well as supply diverse other essential amino acids and minerals.

While trying to increase the sale and consumption of his new bread, McCay kept conducting the research that made him famous. Using several new techniques such as "parabiosis" and new materials including radioactive calcium, he studied caloric restriction's impact on aging and longevity until the late 1950s.[253] Indeed, this continued research left a strong and enduring legacy that survived beyond the walls of the New York State College of Agriculture even after he retired in 1962. For lay people, McCay's research remained a warning against overeating and intemperate life, which could make their later years unhappy.[254] But it was to gerontologists, whose community steadily expanded after the mid-twentieth century, that McCay's research was the most meaningful. In many animal species, including mice, dogs, hamsters, fish, and even yeasts, gerontologists observed the phenomenon that diets with reduced calories increased longevity.[255] In 1988, Laura Beil in *Science News* wrote that "after more than 50 years it has endured as an unusual research tool," which, according to gerontologist Edward Masoro, "gives you a handle for looking at the mechanisms of aging."[256] Whereas other factors such as the amount of exercise and hormones were examined for their influence upon aging and longevity, none of them was as effective as caloric restriction. But Masoro and other gerontologists admitted that the reason why restricted caloric intake increased longevity and retarded aging was not yet clearly understood. A large number of factors seemed to be involved in aging, and dietary caloric restriction

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[252] Girija G. Kane and C. M. McCay, "Calcium Requirements of Old and Young Hamsters and Rats," *Journal of Gerontology* 2 (1947), pp. 244-248.

[253] C. M. McCay, Frank Pope, Wanda Lunsford, Gladys Sperling, and P. Sambhavaphol, "Parabiosis between Old and Young Rats," *Gerontologia* 1 (1957), pp. 7-17; Emily M. Horrington, Frank Pope, Wanda Lunsford, and C. M. McCay, "Age Changes in the Bones, Blood Pressure, and Diseases of Rats in Parabiosis," *Gerontologia* 4 (1960), pp. 21-31; Brown, Barnes, Sperling, and McCay, "Radioactive Calcium Osteosarcomas and Squamous Carcinomas," pp. 329-334.

[254] For example, see "Eating Less Extends Lifespan, Research on Animals Shows," *Sarasota Herald-Tribune* (17 April 1990); Paul Mcglothin and Meredith Averill, *The CR Way: Using the Secrets of Calorie Restriction for a Longer, Healthier Life* (New York: Harper-Collins, 2008).

[255] Edward Masoro, "Overview of Caloric Restriction and Ageing," *Mechanisms of Ageing and Development* 126 (2005), p. 913.

[256] Laura Beil, "Lean Living," *Science News* 134 (1988), p. 143.

was known to be related to many of them in multiple ways.[257] However, this did not mean that the research should be discouraged. Rather, according to a recent report, it meant that dietary caloric restriction should be studied through the cooperation of diverse experts in many fields, such as "epidemiology, clinical trials, nutrition, metabolism, endocrinology, neuroendocrinology, genetics, pharmacology, and behavioral medicine."[258] McCay's research thus contributed to, as well as benefited from, the gathering of diverse expertise, which constituted the multidisciplinary character of gerontology.

The next chapter will deal with another important product of research that contributed to expanding the scope of gerontology—the evolutionary theory of aging proposed by the British biologist Peter Brian Medawar. By illuminating the academic and social contexts in which he was located, Medawar's conception of the theory will be discussed in detail. The next chapter also examines the reason why his work, unlike McCay's, failed to encourage his contemporaries to become involved in the related lines of research.

[257] Beil, "Lean Living," p. 142.
[258] Evan C. Hadley, Chhanda Dutta, Judith Finkelstein, Tamara B. Harris, Mark A. Lane, George S. Roth, Sherry S. Sherman and Pamela E. Starke-Reed, "Human Implications of Caloric Restriction's Effects on Aging in Laboratory Animals: An Overview of Opportunities for Research," *Journals of Gerontology: Series A* 56A(Special Issue) (2001), p. 5.

Chapter 3

"The Shape of the Human Being as a Function of Time" Peter Brian Medawar and the Evolutionary Conception of Senescence

While the previous chapter has dealt with a research program created by an American scientist, this chapter discusses a British scientist's investigation and its influence. The main topic is Peter Brian Medawar's (1915-1987) construction of his evolutionary theory of aging, its subsequent development, and the roles he played in the creation of gerontology in his home country, Great Britain.[1] According to contemporary gerontologists, the evolutionary theory of aging is "central to biogerontology" and "the only one known to us which is still a plausible candidate for an exceptionless, general theory of aging."[2] Medawar's formulation of his theory in the 1940s played a key role in the subsequent development of this evolutionary theory of senescence which has been extended to the domain of experimental research as well.

Admittedly, Medawar was not the first scientist who tried to provide an understanding of aging basesd on evolutionary viewpoints. The renowned German biologist August Weismann (1834-1914) had already proposed a theory of the evolution of senescence in 1881.[3] Medawar employed Weismann's idea that aging was a

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[1] For a review on Medawar's and others' evolutionary theory of aging, see Brian Charlesworth, "Fisher, Medawar, Hamilton, and the Evolution of Aging," *Genetics* 156 (2000), pp. 927-931; Leonid A. Gavrilov and Natalia S. Gavrilova, "Evolutionary Theories of Aging and Longevity," *The Scientific World Journal* 2 (2002), 339-356; Robin Holliday, "The Evolution of Human Longevity," *Perspectives in Biology and Medicine* 40 (1996), pp. 100-107.

[2] Michael R. Rose and Joseph L. Graves, Jr., "What Evolutionary Biology Can Do for Gerontology," *Journal of Gerontology: Biological Sciences* 44 (1989), p. B27; George M. Martin, "How Is the Evolutionary Biological Theory of Aging Holding Up against Mounting Attacks?" *American Aging Association Newsletter* (April 2005), p. 2.

[3] In 1881, Weismann delivered a lecture on "Über die Dauer des Lebens" at the meeting of the Association of German Naturalists held at Salzburg. This lecture was published as a part of a large book. See August Weismann, *Essays upon Heredity and Kindred Biological Problems*, Edward B. Poulton, Selmar Schönland, and Arthur E. Shipley (eds.) (Oxford: Clarendon, 1889), esp., pp. 8-35.

contingent phenomenon that emerged through natural selection rather than an inherent character of every living creature. Yet Medawar was different from Weismann in many respects. In particular, while Weismann based his idea on the concept of group selection which would be questioned by later scientists—especially the biologists during the Modern Synthesis of the 1930s—Medawar's theory was constructed upon the new evolutionary ideas after the Synthesis, which regarded the individual as the unit of natural selection.

But Medawar has been known more as an immunologist than as a gerontologist, and his spendid succeess in the former field has contributed to his image as a quintessential *experimental* biologist. His winning of the Nobel Prize in 1960 together with the Australian scientist Frank Macfarlane Burnet solidified this image, because the award was for his experimental demonstration of the theory of immunological tolerance proposed by Burnet in 1949.[4] The fact that Medawar's research on immune tolerance was related to his medical investigation into skin homograft for burned patients during World War II further strengthened his image as a practically oriented experimenter, particularly in contrast to Burnet, who has been known as a great theoretician and biological thinker.[5]

The initial goal of this chapter is to show that Medawar was an ingenious theoretician as well as a careful experimentalist, and that his evolutionary theory of aging was a result of this theoretical research. I will reveal that Medawar used mathematics extensively in order to describe and analyze the changes of the living organism over time, including its growth, evolution, immunity development, and senescence. An intellectual basis of doing this research came from his reading of the works of several British mathematical biologists and an American statistician—in particular, Ronald A. Fisher,

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[4] Alfred I. Tauber and Scott H. Podolsky, "Frank Macfarlane Burnet and the Immune Self," *Journal of the History of Biology* 27 (1994), p. 564; Arthur M. Silverstein, *A History of Immunology* (San Diego: Academic Press, Inc., 1989), pp.285-291; Hyung Wook Park, "Germs, Hosts, and the Origin of Frank Macfarlane Burnet's Concept of 'Self' and 'Tolerance,' 1936-1949," *Journal of the History of Medicine and Allied Sciences* 61 (2006), pp. 532-533.

[5] Silverstein, *A History of Immunology*, pp. 72-74, 175-179; Eileen Crist and Alfred I. Tauber, "Selfhood, Immunity, and the Biological Imagination: The Thought of Frank Macfarlane Burnet," *Biology and Philosophy* 15 (2000), pp. 509-533; Frank Fenner, *Sir Macfarlane Burnet: Scientist and Thinker* (St. Lucia: University of Queensland Press, 1988).

D'Arcy Thompson, J. B. S. Haldane, Julian Huxley, and Alfred J. Lotka. But Medwar's research was very peculiar, because he was deeply obsessed with approaching the nature of life through its *changes* over time. Indeed, he was very interested in the living organism's growth, aging, and evolution, and tried to explain them in a single theoretical framework. Even in his immunological research, he focused on biological changes over time, unlike chemical immunologists whose static account of immunity formation was accepted as the orthodoxy at that time.[6] This obsession, I claim, guided his theoretical as well as experimental research, from which his evolutionary theory of aging was conceived.

But the main argument of this chapter is about what conceptual contribution he made to the science of aging through this obsession rather than describing the obsession in detail. I will first show that he absorbed the new evolutionary reasoning that emerged after the Modern Synthesis through the efforts of Fisher, Haldane, Huxley, Theodosius Dobzhansky, E. B. Ford, and others. With these scholars' theoretical resources, I argue, he conceived his new evolutionary concept which transformed aging from a cosmic process to a *contingent* phenomenon dependent upon random mutation, natural selection, and the place where the organism happened to live, which, as a whole, began to be regarded as the factors making evolution haphazard and directionless process. Medawar thereby strengthened one of the three new conceptual and practical elements in aging research—contingency, locality, and experimentalism—which was introduced during the early twentieth century. (See Chapter 1.)

This chapter analyzes Medawar's contribution to the institutionalization of gerontology as well. In the final section of this chapter, I will show that while his theory did not immediately lead to a research program on the evolution of aging and he was not deeply involved in the birth of gerontology in Britain, he did make contributions to the development of the science of aging in at least three respects. First, he participated in the British Society for Research on Ageing and the Advisory Committee of the Nuffield

[6] The chemical orientation in immunology during the first half of the twentieth century has been extensively discussed by many historians. See, for example, Silverstein, *A History of Immunology*, pp. 49-56, 64-75.

Gerontological Research Fellowship. Second, he supported and mentored Alex Comfort, who later became a leading British gerontologist. Third, his theory prompted other scientists to pursue the study of the evolution of aging, although his influence was not immediately visible. In particular, Medawar led the American biologist George Williams to propose another evolutionary theory of aging which opened a new possibility of experimental manipulation of aging process. Williams and other later scholars inspired by Medawar contributed to making the evolutionary theory of aging become a genuine part of the multidisciplinary science of gerontology.

The first section will trace how this new theory of aging was constructed by tracing Medawar's research after 1937. The following section will discuss how he contributed to the development of gerontology in the United Kingdom and how this contribution influenced the multidisciplinary field of gerontology and the scientific understanding of aging.

Mathematics and the Modern Synthesis in Peter Brian Medawar's Evolutionary Theory of Aging

Peter Medawar was born in Rio de Janeiro, Brazil in 1915 and was educated in Magdalen College at Oxford from 1932 to 1936. (See Figure 3.1.) There he finished his undergraduate education with a "First" in zoology and worked as a demonstrator and research fellow before being appointed Mason Professor of Zoology at the University of Birmingham in 1947. Four years later, Medawar moved to University College London as Jodrell Professor of Zoology.

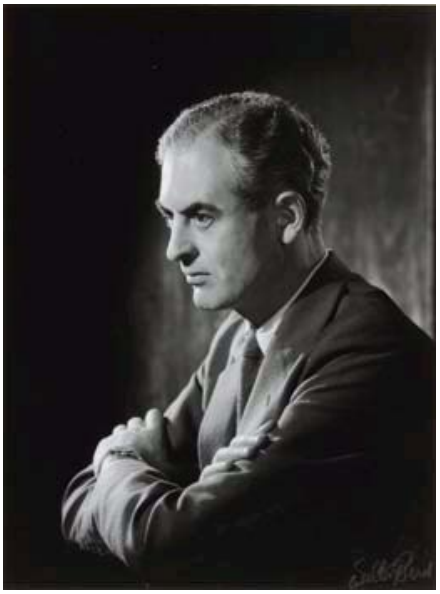

Figure 3.1. *Peter Brian Medawar. Undated. P. B. Medawar, IM/GA/WRS/8627 GB 117, Royal Society Archive.*

During this career, he developed his expertise in theoretical as well as experimental research. In his autobiography, he wrote that he read Alfred North Whitehead and Bertrand Russell's *Principia Mathematica* while staying at Oxford, and joined the "Theoretical Biology Club," in which Karl Popper, Joseph Needham, and C. H. Waddington also participated.[7] In particular, he trained himself as a mathematical theoretician by thoroughly reading the works of several contemporary British mathematical biologists and some American scholars. Medawar enthusiastically studied the British biologist D'Arcy W. Thompson's *Growth and Form* (1917), which described the growth of organisms and interspecies relations through mathematical formulas. Interestingly, Thompson also read Medawar's papers and gave his comments to help Medawar revise his paper for publication.[8] Medawar, deeply appreciating this help and the insights he gained from Thompson's work, wrote a chapter in *Essays on Growth and Form Presented to D'Arcy Wentworth Thompson* (1945), edited by Le Gros Clark and Medawar himself. Another major influence on Medawar's mathematical thinking was

[7] P. B. Medawar, *Memoir of a Thinking Radish: An Autobiography* (Oxford: Oxford University Press, 1986), pp. 65, 85.
[8] Thompson to Medawar, 21 February 1942, Box 2, Folder A.24, PBM.

The Genetical Theory of Natural Selection (1930) and its author, Ronald A. Fisher. Medawar closely read Fisher's book in which he found several important mathematical formulas that he would use in his own work. In turn, Fisher helped Medawar by reading and commenting on his papers, one of which Fisher transmitted to *The Proceedings of the Royal Society*.[9] Medawar also read Julian Huxley's *Problems of Relative Growth* (1932), which was written in the spirit of Thompson, to whom the book was dedicated. Indeed, as a promising student and brilliant fellow at Oxford, Huxley influenced many zoologists within the University, including John Z. Young who was Medawar's tutor at Magdalen College.[10] Another important mathematical biologist, J. B. S. Haldane, who also taught at Oxford, influenced Medawar through his publications and his written feedback on Medawar's article on the evolution of aging.[11] Among the scholars on the other side of the Atlantic, the American statistician Alfred J. Lotka, the author of *Elements of Physical Biology* (1925), was also very important as a source of theoretical inspiration for Medawar's mathematical ideas.

Medawar's first published paper, which was based on his early research at Howard Florey's physiology lab at Oxford, shows how Medawar began to use mathematical approaches he learned from these scholars to analyze a biological phenomenon—the "ageing" of tissues explanted from embryonic animals. Although his first paper, which was basically his D. Phil degree thesis, did not contain any mathematical formulas, he nevertheless tried to account for his experimental results in quantitative terms.[12] In this paper, he studied biological properties of a factor in malt extracts which had been known to inhibit the proliferation of cells. He observed that the

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[9] Fisher to Medawar, 25 March 1943, RAF; Medawar to Fisher, 9 September 1943, RAF. This article Fisher transmitted to the Royal Society is P. B. Medawar, "The Shape of the Human Being as a Function of Time," *Proceedings of the Royal Society of London: Series B. Biological Sciences* 132 (1944), pp. 133-141.

[10] Jack Morrell, *Science at Oxford, 1914-1939: Transforming an Arts University* (Oxford: Oxford University Press, 1997), pp. 273-286. Also see Medawar, *Memoir*, p. 51.

[11] Haldane to Medawar, undated, Box 17, Folder C. 23, PBM. This letter contains Haldane's comments on Medawar's evolutionary theory of aging.

[12] A copy of Medawar's unsubmitted thesis can be found in the Robert Robins Papers at the Royal Society Archive. See P. B. Medawar, "A Factor Inhibiting the Growth of Mesenchyme," B313, RR. According to Medawar, he did not submit this thesis to the University even after its completion, because "the degree served no useful purpose and cost….as much as it cost in those days to have an appendectomy." See Medawar, *Memoir*, p. 71. For a historical account of Oxford's D. Phil degree, see Morrell, *Science at Oxford*, pp. 14-16, 26-29.

susceptibility of explanted tissue to the inhibitory effects of the factor increased with the tissue's age. This observation led him to think that the "growth energy" of tissues could be represented as the capacity to grow under the influence of the inhibitory factor.[13] Younger tissues with more growth energy tended to grow at a higher rate than older tissues in the presence of the same amount of the inhibitory factor.

In his second article published in 1940, he studied this phenomenon further with carefully designed experiments and mathematical analysis. He wrote that the growth energy of a tissue increased in proportion to the concentration of the inhibitory factor that was "*just sufficiently high* to inhibit all [outgrowths] from a series of explants of differing embryonic ages."[14] He then actually measured these concentrations using the explanted embryonic chicken heart aged from 6 to 18 days and found that the growth energy (*GE*) had the following relation to the age of the tissue (*t*).

$$\log GE = \log A - kt$$

It is unlikely that the establishment of this equation was very difficult. If the way to measure the growth energy was determined appropriately, it was possible to calculate its logarithmic value. Medawar then found that this value was inversely proportional to the age of the tissues. The constants in the above equation, *A* and *k*, could be calculated from the observed relationship between *t* and *GE*. From this equation, Medawar also deduced the following one.

$$GE = Ae^{-kt}$$

This showed that "the growth energy is found to decline exponentially over the range 6-18 days."[15] Another important issue Medawar wanted to resolve was the "specific

[13] P. B. Medawar, "A Factor Inhibiting the Growth of Mesenchyme," *Quarterly Journal of Experimental Physiology* 27 (1937), pp. 156-158.
[14] P. B. Medawar, "The Growth, Growth Energy, and Ageing of the Chicken's Heart," *Proceedings of the Royal Society of London: Series B. Biological Sciences* 129 (1940), p. 337.
[15] Medawar, "The Growth," p. 354.

growth rate," which was defined as the rate of change of size (or mass, *W*) divided by the current size (or mass). The specific growth rate ø(*W*) was the actual growth rate *per unit size* (or mass), which was expressed through the following equation.

$$\text{ø}(W) = \frac{1}{W}\frac{dW}{dt} = \frac{d\log W}{dt}$$

He then assumed that this specific growth rate was in direct proportion to the growth energy. $GE = K\text{ø}(W)$. Therefore, since *GE* equaled Ae^{-kt},

$$\frac{d\log W}{dt} = \frac{A}{K}\ e^{-kt}$$

If this equation were to be correct, log *W* should be in proportion to growth energy, Ae^{-kt}. Proving this relation by other means, he deduced from the above equation $W = W_i\, e^{-ae^{-kt}}$, which meant that "the heart of the chicken grows *at a rate of continuous compound interest which itself declines by continuous compound interest*."[16] That is, while the growth of the tissue occurred exponentially through the duplication of existing cells, the rate of this duplication decreased exponentially. For Medawar, this was a peculiar character of *senescence*, which occurred even during embryo development.

Interestingly, some of these equations could be found in other scholars' books Medawar read, although they employed the equations to describe a different phenomenon statistically. In 1930, to determine the specific *death* rate of a population, Fisher used the equation Medawar adopted in describing the specific growth rate.[17] Lotka employed the same equation in his *Elements of Physical Biology*, and wrote that the formula designated "the force of mortality."[18] Medawar would use Lotka's term "the force of mortality" in his evolutionary theory of aging.

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[16] Medawar, "The Growth," p. 344. The italic characters were written by Medawar himself.
[17] Ronald A. Fisher, *Genetical Theory of Natural Selection* (Oxford: Clarendon, 1930), p. 23.
[18] Alfred J. Lotka, *Elements of Physical Biology* (Baltimore: Williams and Wilkins, 1925), p. 102.

This shows that Medawar's use of mathematics in aging research was influenced by mathematical statistics, which, according to historian Theodore Porter, had developed into a mature form in Britain during the late nineteenth century.[19] Indeed, Medawar deeply respected Fisher, a senior British scientist who pioneered in using statistical methods in population genetics. From Fisher and others, Medawar imported statistical methodology and used it for his own research problem. The way Medawar did so also agrees with a well-known pattern in which mathematical statistics developed. Like previous scientists who adopted analogical reasoning to analyze various different phenomena, Medawar proposed an analogy between the growth of cultured tissues and the changes of population while introducing mathematical statistics into his research.[20]

In the following year, Medawar published another theoretical paper about growth and aging, which dealt with an important problem suggested by two renowned biomedical scientists, Charles S. Minot at Harvard Medical School and Alexis Carrel at the Rockefeller Institute. Medawar stated that since the rate of growth declines with aging by continuous compound interest, the specific acceleration of growth was always negative. This could be expressed in the following formula, which, as he wrote in his 1940 article, designated an important character of senescence.

$$\frac{d(\frac{1}{W}\frac{dW}{dt})}{dt} < 0$$

Medawar noticed that the specific acceleration of growth had another interesting feature, which Minot had explained as a paradox in *The Problem of Age, Growth, and Death* (1908): "organisms age fastest when they are young."[21] This seemingly paradoxical statement reflected Minot's observation that the rate of the decline of growth rate was highest in the early phase of an organism's life and gradually slowed down in its later

[19] Theodore M. Porter, *The Rise of Statistical Thinking, 1820-1900* (Princeton: Princeton University Press, 1986), p. 255.
[20] Porter, *The Rise of Statistical Thinking*, p. 319.
[21] P. B. Medawar, "The 'Laws' of Biological Growth," *Nature* 148 (1941), p. 773; Charles S. Minot, *The Problem of Age, Growth, and Death: A Study of Cytomorphosis* (New York: Putnam, 1908), p. 5.

course. Medawar translated this observation in his own terms: The specific acceleration of growth, which was expressed in the above formula, "while always negative, rises progressively to zero during the course of life."[22] Yet Medawar did not fully accept another part of Minot's main arguments—that the decline of the growth rate was an inevitable feature of life destined by "cytomorphosis," the gradual increase of the proportion of the cytoplasm in the cell over that of the nucleus with aging.[23] Medawar knew that Carrel had already shown that this might not be true in certain conditions. In artificial environments such as media for tissue culture, cells could survive and divide indefintely without undergoing aging or death. Under such conditions, Medawar wrote, "*growth proceeds with uniform specific velocity*" that did not decline over time.[24]

But Medawar addressed this issue in a more skeptical way at a discussion forum held by the Royal Society of Medicine on March 4th, 1942. While discussing the unlimited growth of cancer cells, he stated that such growth might not be restricted to them, since Carrel and other researchers had shown that normal somatic cells could also be induced to grow indefinitely in a special tissue culture environment such as the "embryonic juices" Carrel used. If that was the case, the limited lifespan and decline of the growth rate of the normal somatic cells could simply be due to some kind of the "inhibitory factors" contained in the culture media rather than to the cells' inherent limitations. However, Medawar was a cautious person who did not always accept other scientists' arguments without deeply thinking about their possible pitfalls. In fact, while not completely denying the validity of Carrel's idea and experiments, Medawar thought that Carrel's argument might not be supportable. This skeptical attitude came from Lotka's *Elements of Physical Biology* (1925), through which Medawar conceived an analogy with the second law of thermodynamics.

> The rate at which heat is lost from a cooling body is initially high, and falls off as its temperature approaches that of the environment. The rate at which the

[22] Medawar, "The 'Laws,'" p. 773.
[23] Minot, *The Problem of Age, Growth, and Death*, pp. 38-85.
[24] Medawar, "The 'Laws,'" p. 773.

> distribution of molecules in a closed diffusion system tends towards uniformity is likewise rapid at first, and slower and slower thereafter. In these cases, and in others similar to them, we are dealing with rates which fall off "of their own accord."[25]

These non-biological examples showed that the slowing down of the growth rate—which proceeded quickly at first and became gradually slower—belonged to a widespread phenomenon in nature. If the decrease of the cells' specific growth rate with aging, like the heat loss process mentioned above, occurred "of their own accord" rather than through some sorts of inhibitory environmental substances, then "the belief that cells stop growing *because* something stops them, is not self-evident, even if it is true."[26]

There seems to be nothing new about the idea, because it had been generally agreed that aging was unavoidable despite the fame and influence of Carrel's work. Nevertheless, Medawar incorporated a highly counterintuitive concept into his notion that had originally been proposed by Minot. Aging was a form of change that occurred even in the earliest phases of life, and growth itself always accompanied aging. This idea was completely different from older notion that divided human life into four or five distinct periods, such as infancy, childhood, adulthood, middle age, and old age.[27] While traditionally it had been thought that humans, or the living organism in general, went through the periods of "growing up" and "growing old," Medawar's research on the aging of embryonic cells, along with Minot's earlier view, implied that no such distinction was meaningful in scientific understanding of aging, since senile changes occurred continuously from the developmental phases to later periods of life.

Another paper published in 1943, titled "The Shape of the Human Being as a Function of Time," shows how Medawar tried to describe this age change—whether it

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[25] "Discussion on Growth and New Growth," *Proceedings of the Royal Society of Medicine* 35 (1942), p. 593. After this statement, Medawar mentioned Lotka's own statement on the same phenomenon contained in *Elements of Physical Biology*.

[26] "Discussion on Growth and New Growth," p. 593.

[27] Thomas R. Cole, *The Journey of Life: A Cultural History of Aging in America* (Cambridge: Cambridge University Press, 1992), pp. 24-31; Thomas R. Cole and Mary G. Winkler, "Aging in Western Medicine and Iconography: History and the Ages of Man," *Medical Heritage* 1 (1985), pp. 336-347.

meant growth or senescence—in mathematical terms. While his previous works concerned only the cells' growth and aging, this article dealt with those of the whole human being, particularly the change of the relative proportion of its parts with age. Citing the books of Thompson and Julian Huxley whose methodology Medawar used, he first showed a picture of a growing male human whose height was fixed so that the change of the relative proportion of each body part could be easily compared. (See Figure 3.2.)

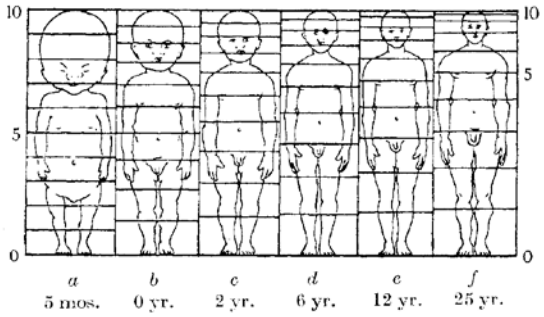

Figure 3.2. *The Growth of a Human. Peter Brian Medawar, "The Shape of the Human Being as a Function of Time," Proceedings of the Royal Society of London: Series B. Biological Sciences 132 (1944), p. 136.*

Since two parameters, the base-line and the height did not alter, the changing distances of four portions of the body (the fork, navel, nipples, and chin) from the base-line could be traced as follows. (See Figure 3.3.)

TABLE 1. MEASUREMENTS, IN ARBITRARY UNITS ALONG THE AXIS OF SYMMETRY $\bar{y}$, OF VARIOUS LANDMARKS ILLUSTRATED BY FIGURE 1

	time in years from conception					
	0·42	0·75	2·75	6·75	12·75	25·75
base-line	0·00	0·00	0·00	0·00	0·00	0·00
fork	1·00	1·59	1·80	1·94	2·09	2·23
navel	1·81	2·29	2·50	2·67	2·77	2·90
nipples	2·52	3·00	3·20	3·38	3·51	3·64
chin	3·20	3·62	3·83	4·03	4·13	4·22
total height	4·85	4·85	4·85	4·85	4·85	4·85

Figure 3.3. *Changing Proportions. Peter Brian Medawar, "The Shape of the Human Being as a Function of Time," Proceedings of the Royal Society of London: Series B. Biological Sciences 132 (1944), p. 136.*

Based on the numerical data given in this table, Medawar constructed an equation that enabled him to calculate the distance (t^x) of a certain portion of the body from the base-line if the age (t) and the initial distance (x when t = 0.42) were given. The variable that changed according to age was,

$$F(t) = 1.4176 + 0.4127\log (t - 0.406) + \frac{0.147}{t}$$

This variable was a part of the following equation.

$$t^x = F(t)x + \frac{1-F(t)}{4.85}x^2$$

This showed that the growth pattern of each body part was not independent. It followed a general scheme that could be expressed as a mathematical formula like the above one. For Medawar, his equation described each part's growth "as a single process of continuous deformation in time."[28]

For Medawar, however, making these equations was not the activity that took the most energy and time. In a letter to Fisher, Medawar wrote that he obtained the above equations "in [his] spare time from medical research."[29] According to Medawar's recollection, this medical research was initiated at the beginning of the Second World War which led him to study the restoration of severed peripheral nerves.[30] While pursuing this research, he also investigated other related issues, such as the effects and toxicity of sulfonamide drugs and the proper way of using fixatives for the treatment of burned skin.[31] But the most important job for him at that time was the research on the "homograft problem" which he pursued with Leonard Colebrook and Thomas Gibson at

[28] Medawar, "The Shape of the Human," p. 133.
[29] Medawar to Fisher, 23 March 1943, RAF.
[30] P. B. Medawar, "Tolerance and Tissue Transplantation," undated, Box 18, Folder C.34, PBM.
[31] For example, see P. B. Medawar, "The Rate of Penetration of Fixatives," *Journal of the Royal Microscopical Society* 60 (1941), pp. 46-57; F. Jacoby, P. B. Medawar, and E. N. Willmer, "The Toxicity of Sulphonamide Drugs to Cells in Vitro," *British Medical Journal* 2 (1941), pp. 149-153.

the Burns Unit of the Glasgow Royal Infirmary.[32] Indeed, the use of skin homograft—a piece of the skin transplanted from a different individual—for burned patients engendered a severe problem at many hospitals at that time. Although it was highly urgent to treat the patients severely burned in warfare, many of them did not have enough of their own skin that could be used to cover their damaged surface. Yet it was not possible to use the skin from a different person other than a monozygotic twin brother or sister, since such skin would be invariably rejected by the patient's body. To investigate the basic causes of this phenomenon, Medawar began his own research using rabbits and mice at Oxford with a small grant awarded by the Medical Research Council.[33] He studied whether homograft rejection was caused by an immune reaction or by local cellular response, and, if the former was true, whether it occurred through an acquired immunity—which was normally used to protect the organism against microbial infection—or a "natural immunity," which was thought to be innate but magnified by blood transfusion.[34] He also investigated the genetic mechanism underlying homograft rejection through the inbred mice he acquired from Fisher's laboratory.[35]

Although Medawar's primary approach in this war-related research was experimental, mathematics was also important. He extensively used mathematics in designing the immunological experiments. One of them was conducted to determine the number of antigens responsible for homograft rejection. He first inferred that there should be $2^n - 1$ kinds of antigen combinations provided that there were n distinct antigens involved in responding to homograft.[36] He then thought that the number of elements in

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[32] Medawar, *Memoir*, pp. 80-82. Medawar and Gibson's research was published. See T. Gibson and P. B. Medawar, "The Fate of Skin Homografts in Man," *Journal of Anatomy* 77 (1943), pp. 299-310.

[33] In 1943, Medawar received £250 per annum. See F. J. C. Herald to Medawar, 19 January 1943, FD 1/6959, NAUK. Indeed, the nature of homograft response had been investigated by several researchers before Medawar. See Silverstein, *A History of Immunology*, pp. 278-283.

[34] P. B. Medawar, "Notes on the Problem of Skin Homografts," *Bulletin of War Medicine* 4 (1943), pp. 1-4. Medawar made a distinction between "acquired" and "natural" immunity, because it was not known at the time whether the immune response against microbes was the same as the reaction of the body against cells or tissues from a different individual.

[35] Medawar to Fisher, 19 November 1943, RAF. But Medawar was not very successful in doing genetic experiments using Fisher's mice, since they often died due to weakness, illness, or other problems. For his research on immune tolerance that gave him the Nobel Prize, Medawar used different inbred mice strains, which probably came from the Jackson Memorial Laboratory in America.

[36] In a set that has n elements, the number of subsets except the null set is $2^n - 1$.

the "principal subgroup," a group of antigens with a certain type of combination with the largest number of its members, was ${}_nC_r$.[37] For example, if there were four types of antigens, *A*, *B*, *C*, *D*, they then could be combined in 15 ($2^4 - 1 = 15$) different ways.

A, B, C, D; AB, AC, AD, BC, BD, CD; ABC, ABD, ACD, BCD; ABCD.

Among them, the principal subgroup is the one consisting of *AB*, *AC*, *AD*, *BC*, *BD*, *CD*, and the number of its members was ${}_4C_2 = 6$. If this was the case, Medawar concluded, it was possible to choose six animals randomly and to transplant each individual's tissue to all others to examine whether there were at least four different antigens. If all these transplantations failed, it could be concluded that at least four antigens existed that could be combined in the six different ways shown above. Of course, some cases of successful homograft transplantation did *not* mean that there were fewer than four antigens, because it was possible that these six animals happened to include individuals which were genetically closely related and perhaps sharing the same genes for homograft rejection. In that case, further study would be necessary. Yet Medawar decided to initiate a series of tissue transplantation experiments based on this scheme, because it could be the first step toward identifying the number of antigens. He used twenty-five rabbits to test whether there were at least seven antigens. Indeed, the number of combinations in the principal subgroup was thirty-five rather than twenty-five, if there were seven antigens. But he used twenty-one plus four more as "insurance" since twenty-one was "one more than the number of combinations in the principal subgroup" for six antigens, "which are *just* sufficient to account for the failure of a cross-grafting test between twenty animals."[38] Medawar wrote, "If the test fails with twenty-one animals, then the existence of one antigen more must be invoked."[39] The result of the transplantation experiments indicated that this was indeed the case. Since every rabbit, except the two that died prematurely and the one excluded due to illness, rejected all the tissues transplanted from other rabbits, he

[37] P. B. Medawar, "A Second Study of the Behavior and Fate of Skin Homografts in Rabbits," *Journal of Anatomy* 79 (1945), p. 166. Medawar added that "r = ½n when n is even and (n/2)±½ when n is odd."
[38] Medawar, "A Second Study," p. 166.
[39] Medawar, "A Second Study," p. 166.

concluded that there were at least seven antigens in the rabbit responsible for homograft rejection.

He also used mathematical method to infer the "tempo" of the breakdown of homografts from the graph on the changing number of surviving skin patch over time. (See Figure 3.4.)

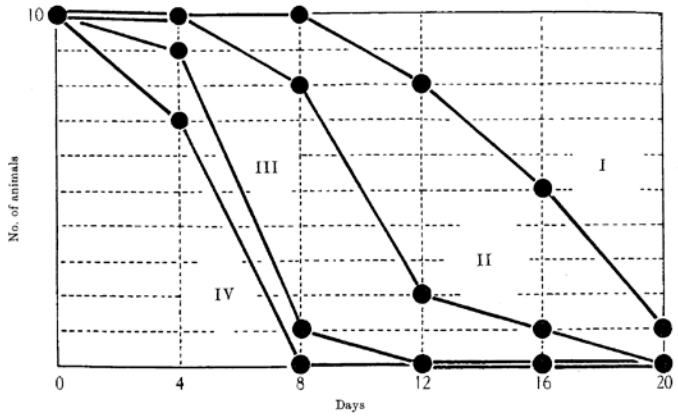

Figure 3.4. *Changing Number of Surviving Grafts over Time. P. B. Medawar, "The Behavior and Fate of Skin Autografts and Skin Homografts in Rabbits," Journal of Anatomy 78 (1944), p. 186.*

The vertical axis designated the number of surviving skin transplants, and the horizontal axis was the days after the surgery. In this graph, "I" was the case of the "low dosage" homograft and "II" was that of the "high dosage," while "III" was that of the second homograft which was transplanted into the same individual's skin but at a part different from that where the first skin patch was transplanted.[40] "IV" was the case of the homograft which was attached to the very place where the first one from the same donor was rejected by the recipient body. He arrived at two conclusions from this graph. First, the cause of the homograft rejection was the actively acquired immunity, since the second-set homograft was rejected more rapidly, as could be seen in the difference

[40] The "high dosage" homograft meant that the transplanted tissue which weighed from 0.045 to 0.055g, and the "low dosage" meant the skin patch weighing 0.006g. See Medawar, "The Behavior and Fate," p. 186.

between the cases I and III.[41] Second, the amount of grafted tissue had *something* to do with the pattern of rejection, as clearly seen in the difference between I and II in the above graph. But what was the precise nature of this difference? To answer this question, Medawar used C. I. Bliss' statistical method of expressing "the percentages of graft mortality as areas of the normal curve of error in terms of the normal deviate."[42] Medawar converted the above graph into a different one using the same data. (See Figure 3.5.) This new graph revealed the difference between I and II more clearly.

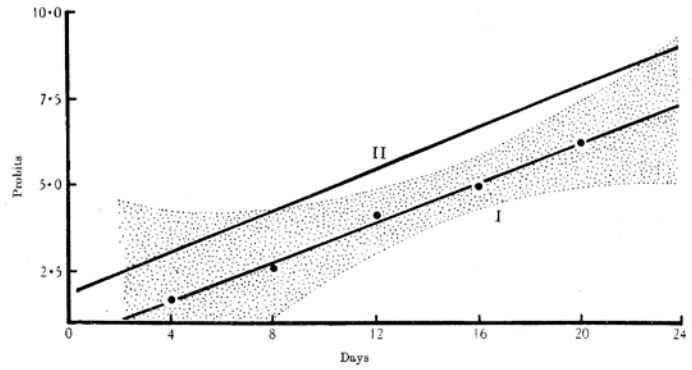

Figure 3.5. *Probit Mortality of Two Homografts. P. B. Medawar, "The Behavior and Fate of Skin Autografts and Skin Homografts in Rabbits," Journal of Anatomy 78 (1944), p. 188.*

The vertical axis was the "probit mortality," which, to put it simply, meant the probability that a randomly chosen skin homograft would be dead by the time designated by the horizontal axis. Since the slope of the two lines in the new graph was almost the same, Medawar concluded, "the *tempo* at which breakdown proceeds, once the process has started, is the same for both: the difference between them lies in the length of the latent period which must pass before the homograft reaction becomes effective."[43]

Medawar also studied another phenomenon related to organismic changes over time—how *age* of an organism influenced the regeneration of its peripheral nerves and the result of the tissue transplantation—although he did not use any mathematics in

[41] P. B. Medawar, "The Behavior and Fate of Skin Autografts and Skin Homografts in Rabbits," *Journal of Anatomy* 78 (1944), p. 194.
[42] Medawar, "The Behavior and Fate," p. 188.
[43] Medawar, "The Behavior and Fate," p. 189.

investigating this issue. He and his colleagues at Oxford Zoology Department studied the rate of regeneration of rabbits' severed peripheral nerves under varying conditions as a wartime research project, and found that young rabbits of one month old did not differ from adult rabbits in their rate of the advancement of an axon tip of a severed nerve cell, whereas the time for functional completion and the "scar delays," the time for a severed nerve fiber to retrogress before growing forward, were shorter in younger rabbits.[44] He also studied how the age of skin donors and recipients influenced the outcome of tissue transplantation, and found that young rabbits aged between 2½ and 4½ weeks old did not show any difference from adult rabbits in homograft rejection.[45] This result did not mean, however, that age was irrelevant in tissue transplantation. It simply implied that "the power of resistance to skin homografts is fully developed in rabbits ranging between 2½ and 4½ weeks in age."[46]

When, then, was the critical period in an organism's life, during which "the power of resistance to skin homografts" was formed? Indeed, after the experiment of James Murphy at the Rockefeller Institute in 1913, several researchers found that the embryonic organism did not develop resistance against extrinsic agents, although it did seem to react against them after the end of its developmental periods.[47] By examining these findings, Medawar came to suspect that embryogenesis was the time during which the resistance capacity to skin homografts developed. He thus felt that it was necessary to study the developmental phase further to understand the nature of changes occurring in embryogenesis and the factors that made the embryo accept agents of extrinsic origins without resistance. To Medawar, this research was important for another reason. Study of embryo development was a way to appreciate the repair process after injury, since the two processes—embryogenesis and tissue regeneration—resembled each other very closely. He expected that by studying embryo development he could know more about

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[44] E. Gutmann, L. Guttmann, P. B. Medawar, and J. Z. Young, "The Rate of Regeneration of Nerve," *Journal of Experimental Biology* 19 (1942), pp. 37, 43.
[45] Medawar, "A Second Study," pp. 162-163. It was also found that the skin patch from younger donors was not different from that from older donors in eliciting rejection response in recipients' body.
[46] Medawar, "A Second Study," p. 174.
[47] See F. M. Burnet, *The Production of Antibodies* (Melbourne: Macmillan, 1949), pp. 74-77.

the tissue regeneration process which he investigated extensively at that time as a wartime project.[48]

As a theoretical tool for studying this problem, he adopted cytoplasmic inheritance theories, particularly those proposed during the 1940s, which explained embryonic development in quite a satisfactory way. As many exponents of cytoplasmic inheritance emphasized and Medawar agreed, the hereditary materials in the cytoplasm were probably the agent responsible for cell differentiation during embryogenesis, since "all the cells of the individual have the same components of nuclear genes," and it should thus be the cytoplasm rather than the nucleus that made the difference among distinct types of cells.[49] To support this idea, Medawar and his colleague R. E. Billingham studied melanogenesis in guinea pig's skin and argued that it was caused by a self-replicating cytoplasmic hereditary entity in skin cells and that such an entity would probably cause cell differentiation during embryogenesis as well.[50] Although I will not discuss the role of cytoplasmic inheritance theories in immunology's development any more, it should be noted that in his 1949 theory of "self" and "tolerance" Burnet also used cytoplasmic inheritance theories which Medawar supported through his 1953 experiment. It is interesting to see that the two Nobel laureates used the same theories in their research on embryogenesis and immunity development.[51] But what is more significant for this chapter is the fact that Medawar considered it very important to understand embryogenesis as a kind of changes the living organism underwent over time.

Medawar was interested in explaining homograft rejection in a longer time scale, that is, in evolutionary terms. He wrote that the rejection response toward a different individual's tissue was a byproduct of evolution, during which animals developed

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[48] P. B. Medawar, "Biological Aspects of the Repair Process," *British Medical Bulletin* 3 (1945), pp. 70-71.
[49] Medawar, "Biological Aspects," p. 72. For a general review of the history of cytoplasmic inheritance theories, see Jan Sapp, *Beyond the Gene: Cytoplasmic Inheritance and the Struggle for Authority in Genetics* (Oxford: Oxford University Press, 1987), esp. pp. 87-122.
[50] R. E. Billingham and P. B. Medawar, "The 'Cytogenetics' of Black and White Guinea Pig Skin," *Nature* 159 (1947), pp. 115-117.
[51] Burnet, *Production of Antibodies* (1949), pp. 86-106. Medawar's copy of Burnet's *Production of Antibodies* (1949) can be found in Box 43, Folder C.154, PBM. Medawar's hand-written memos and underlines in this copy indicate that Medawar read Burnet's book very thoroughly, especially the part concerning cytoplasmic inheritance theories. Also see Park, "Germs, Hosts, and the Origin," pp. 518-522, 529-531.

mechanisms of protecting themselves against invading microbes.[52] While such mechanisms, one of which was the immune response, successfully increased the rate of survival of the individual and was thus selected during evolution, it came to frustrate surgeons' efforts to transplant homograft, even though the tissues of distinct persons did not "invade" the recipients' body as microbes did. But this did not mean that homograft transplantation was completely impossible. Just as organisms in their embryonic periods failed to resist extrinsic agents, animals located at low levels in the evolutionary scale did not reject homografts.[53] Medawar wrote that "the rule that skin cannot be transplanted between individuals of the same species is known to be true only of higher vertebrates—from adult frogs and upwards…..though it sometimes seems to work in adult birds."[54] This phenomenon meant that "individuality" or "self-specificity," which was employed to distinguish one organism from another in infection and tissue transplantation, was something that developed over time during both embryogenesis and evolution.[55] In a language reminiscent of the old biogenetic law of nineteenth century biology, he thus argued, "As self-specificity develops [during embryogenesis], so also it evolves."[56]

But Medawar, who was sensitive to the new trends and developments in contemporary biology, did not seriously consider the biogenetic law which postulated that an organism's embryo development repeated its evolutionary history.[57] Indeed, he wholeheartedly accepted the new evolutionary biology after the Modern Evolutionary

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[52] P. M. Medawar, "The Theory of the Differences between Individuals," *The Substance of a Lecture Given to the Oxford Summer School of the British Social Hygiene Council*, pp. 102-103, 1946, Box 36, Folder E.23, PBM.

[53] Medawar, "Notes," p. 4; "The Theory of the Differences between Individuals," *The Substance of a Lecture Given to the Oxford Summer School of the British Social Hygiene Council*, p. 103, 1946, Box 36, Folder E.23, PBM.

[54] Medawar, "The Theory of the Differences between Individuals," *The Substance of a Lecture Given to the Oxford Summer School of the British Social Hygiene Council*, p. 103, 1946, Box 36, Folder E.23, PBM.

[55] Medawar, "The Theory of the Differences between Individuals," *The Substance of a Lecture Given to the Oxford Summer School of the British Social Hygiene Council*, pp. 103, 107, 1946, Box 36, Folder E.23, PBM.

[56] Medawar, "The Theory of the Differences between Individuals," *The Substance of a Lecture Given to the Oxford Summer School of the British Social Hygiene Council*, p. 103, 1946, Box 36, Folder E.23, PBM.

[57] Medawar's sensitiveness toward new developments in biological sciences can be seen in P. B. Medawar, "Zoology," in A. E. Heath (ed.), *Scientific Thought in the Twentieth Century* (London: Watts, 1951), pp. 163-189.

Synthesis, which emerged during the 1930s and 1940s through the efforts of a number of scientists, many of whom were in Britain and America.

The Modern Synthesis was a series of new developments and changes in evolutionary science and biology in general. Although there are several distinct historical accounts of the process, significance, and impact of the Synthesis, historians have generally agreed on the following factors as the most important constituents of the new evolutionary biology formulated through the Synthesis. First, the notion of Lamarckian inheritance of acquired characteristics was denied in favor of the hereditary mechanism based on Mendelian genetics. Second, natural selection became the most important, or probably, the only, mechanism of evolution. Third, no inherent orthogenetic tendency toward a specific evolutionary goal was accepted, because mutation, which introduced variations in a population, was basically a random process, and the environment which favored a specific variant also changed in an unpredictable way. Therefore, evolution itself was defined as the *contingent* changes in gene frequencies in a population. With this definition, contingency, rather than any trend, came to be accepted as the fundamental feature of evolution.[58]

Medawar's institutional position exposed him to the ideas of the architects of the Synthesis. As historian Jack Morrell has shown, there was a growing community of zoologists at Oxford who were deeply committed to evolutionary studies, such as Edward Poulton, E. S. Goodrich, Julian Huxley, J. B. S. Haldane, and E. B. Ford.[59] In particular,

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[58] According to philosopher of science Michael Ruse, even the most important contributors of the Modern Synthesis did not wholly reject the notion of the goal-directed progress. For Ruse, the idea of progress continuously and implicitly influenced the mainstream professional evolutionary biology even though the concept of contingency emerged following the acceptance of natural selection as the mechanism of evolution. See Michael Ruse, *Monad to Man: The Concept of Progress in Evolutionary Biology* (Cambridge, Mass.: Harvard University Press, 1996), pp. 410-455. I would like to add to these generalizations the arguments of Ernst Mayr and Vassilika B. Smocovitis that the Modern Synthesis brought about a firm connection between diverse biological disciplines that had developed separately, such as population genetics, paleontology, biogeography, and ecology. See Ernst Mayr, "Prologue: Some Thoughts on the History of the Evolutionary Synthesis," in Ernst Mayr and William B. Provine (eds.), *The Evolutionary Synthesis: Perspectives on the Unification of Biology* (Cambridge, Mass.: Harvard University Press, 1980), pp. 1-48; Vassiliki Betty Smocovitis, *Unifying Biology: The Evolutionary Synthesis and Evolutionary Biology* (Princeton: Princeton University Press, 1996), pp. 122-138.

[59] Haldane left Oxford in 1923 to become Dunn Reader in Biochemistry of Cambridge. Yet he was one of the scientists who carefully read and gave comments to Medawar's paper on the evolutionary theory of aging.

Morrell has written that Goodrich, the Linacre Zoology Professor, "prepared favourable ground for the evolutionary synthesis" by encouraging research on evolution among his students and associates.[60] Goodrich and other Oxford zoologists advocated natural selection as the sole mechanism of evolution and criticized alternative mechanisms such as the Lamarckian theories based on the inheritance of acquired characters. Although not as deeply committed to evolution as these scholars, ecologist C. S. Elton was another important Oxford scientist whose ideas influenced Medawar's evolutionary thinking. Medwar's 1944 article titled "Oxford Zoology" shows that he deeply respected these biologists and their research, and regarded himself as a member of this active scientific community.[61]

Some of Medawar's notes written during the 1940s show how he used these scholars' thoughts in his ideas on the time-dependency of organismic change, especially evolution and development. In particular, Medawar read and accepted Ford's, Huxley's, and Haldane's concepts of the "time genes" which were activated only at or after a specific phase in an organism's life course.[62] Medawar also seriously considered their argument that these genes were important in evolution, since their changes altered an organism's developmental process that in turn influenced its fitness in its habitat. Furthermore, Medawar noted Huxley's idea that this time-dependency and developmental implication of gene expression was a departure from the traditional "views of certain of

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[60] Morrell, *Science at Oxford*, p. 272. According to William Provine, Haldane also took Goodrich's class. See William B. Provine, *The Origins of Theoretical Population Genetics* (Chicago: University of Chicago Press, 1971), p. 168.

[61] P. B. Medawar, "Oxford Zoology," *Biology* Autumn Term (1944), pp. 1-4. In his 1986 autobiography, however, Medawar wrote that the department was not appropriate for his new biomedical research project, since it was attached "to a cavernous and dusty museum full of old bones and doubtless bacterial spores." This was the reason that he went to Howard Florey's laboratory. See Medawar, *Memoir*, p. 66.

[62] A series of Medawar's undated and untitled manuscripts in Box 17, Folder C.26, PBM includes his notes written after reading several books and articles related to aging, growth, and evolution. I will call these notes "Demography: notes," following the Wellcome Library's catalogue of the Medawar Papers. Although the notes were not paginated, it is possible to page it from 1 to 14, since it was written consecutively. These notes were probably composed in 1945 or early 1946, because all the papers and monographs cited in them were published in or before 1945. In these notes, Medawar mentioned Julian Huxley, *Evolution: The Modern Synthesis* (New York: Harpers, 1942); J. B. S. Haldane, "The Time of Action of Genes, and Its Bearing on Some Evolutionary Problems," *American Naturalist* 66 (1932), pp. 5-24; E. B. Ford and Julian Huxley, "Genetic Rate-Factors in Gammarus," *Archiv für Entwicklungsmechanik der Organismen* 117 (1929), pp. 67-79.

Morgan's school" that constructed classical genetics with more static understanding of the gene's function.[63]

Medawar's notes also show how carefully he studied the works of other scientists involved in the Modern Synthesis and used them in his own research. In particular, he thought that Theodosius Dobzhansky's *Genetics and the Origin of Species* (1941) and an article by Sewall Wright published in 1940 "[discussed] matter that is relevant to one aspect of the evolution of death."[64] While "Weisman and the classical evolutionists" thought that "genetic changes which dispose of a superfluous organ entail a saving of effort and thereby acquire a positive adaptive value," Dobzhansky, citing Wright, argued that "mutation pressure…might lead to rudimentation" rather than total disappearance of an organ, when it ceased to function due to the changes of the environment and came to be uninfluenced by the force of natural selection.[65] Indeed, Medawar found in George Simpson's *Tempo and Mode in Evolution* (1944) how this idea of rudimentation could be applied to the problem of aging and death. Simpson wrote that "hereditary factors that reach their expression only after adults cease to breed have little bearing on natural selection" and could thus be subject to mutations that might bring about random variations in the form and function of the organism. "In any case," Simpson wrote, "only a fraction of the individuals survive" at that time.[66] According to Medawar, the changes occurring in this later phase of life, might be "'rudimentary' in a way essentially similar to" what Dobzhansky and Wright pointed out.[67] Since Ford, Huxley, and Haldane already showed that gene expression depended on time, Dobzhansky's, Wright's, and Simpson's idea indicated that the genes expressed in later phases of life could mutate without the influence of natural selection, making the changes occurring in these periods a kind of rudimentary alteration that had little to do with reproduction.

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[63] P. B. Medawar, "Demography: notes," p. 4, undated, Box 17, Folder C.26, PBM. Also see Huxley, *Evolution*, p. 528.
[64] P. B. Medawar, "Demography: notes," pp. 7-8, undated, Box 17, Folder C.26, PBM.
[65] Theodosius Dobzhansky, *Genetics and the Origin of Species*, 2nd edition (New York: Columbia University Press, 1941), p. 344.
[66] G. G. Simpson, *Tempo and Mode in Evolution* (New York: Columbia University Press, 1944), p. 174. Also see P. B. Medawar, "Demography: notes," p. 12, undated, Box 17, Folder C.26, PBM.
[67] P. B. Medawar, "Demography: notes," p. 7, undated, Box 17, Folder C.26, PBM.

But from which period did the "later phases of life" begin? When were the phases of life after which "adults cease to breed"? Medawar did not think such phases could be clearly distinguished from earlier periods of life. Through his mathematical studies of growth inspired by Thompson, Minot, and Huxley, Medawar had already concluded that the distinction between growth and senescence was not meaningful, since developing organisms underwent aging as well, as could be seen in their continuously decreasing rate of growth. His reading of Elton's *Voles, Mice, and Lemmings* (1942) reconfirmed this conclusion. From Elton's statement that voles "drop off at all times of life," probably due to the process of "wear and tear," Medawar inferred that the "curve of the specific mortality of a group" was similar to the "specific growth rate of the individual," in that both curves showed that the process of aging occurred throughout an individual's whole life, even during its earliest stages.[68] Admittedly, Medawar thought that a more important cause of the mortality during the early periods in life might be infectious diseases that primarily attacked infants rather than any factors related to aging. "But in general," Medawar stated, "the fact remains that both in the [specific growth rate] of the individual and in the mortality curve (life table) of the population, there is a smooth trend towards extreme old age with never a hint that the senile state begins with the close of, for example, reproduction."[69]

The first step in Medawar's construction of a mathematical model of aging population was taken from the above "fact" that both the specific growth rate and the mortality in a population revealed the progress of senile changes over time. From this "fact," Medawar inferred that aging in a changing population could be expressed in a mathematical formula like the "senescence" of tissues during their growth. For him, phenomena with similar nature could be described using mathematics in a comparable way. As I have mentioned, this was a typical way mathematical statistics developed. Like the statisticians, Medawar used similar mathematical formulas to describe different phenomena.

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[68] P. B. Medawar, "Demography: notes," p. 1, undated, Box 17, Folder C.26, PBM; Charles Elton, *Voles, Mice, and Lemmings: Problems in Population Dynamics* (Oxford: Clarendon, 1942), p. 202.
[69] P. B. Medawar, "Demography: notes," p. 1, undated, Box 17, Folder C.26, PBM.

By using this analogical reasoning, Medawar was following the footsteps of the architects of the Modern Synthesis who embraced statistics as a primary method for describing and measuring the evolutionary changes occurring at the population level. As Smocovitis has pointed out, "the critical moment for evolutionary studies" during the Modern Synthesis was the "quantification of evolution" through the mathematical and statistical models of Fisher, Haldane, Wright, and others, which could be used by experimenters and field naturalists as well.[70] Medawar, too, thoroughly used their methods in his theoretical work.

Medawar paid special attention to Fisher's idea among them, and incorporated it into his work along with Lotka's concepts. In both Fisher's and Lotka's books, Medawar found the following statistical formula for the definition of μ_x, which Fisher called the "death rate" and Lotka called the "force of mortality."[71]

$$\mu_x = -\frac{1}{l_x}\frac{d}{dx}l_x = -\frac{d}{dx}(\log l_x)$$

If l_x, "the number of living to age x," was replaced by W, the size or mass of an individual or a tissue, then μ_x can be converted into Medawar's specific growth rate, $\phi(W)$.[72]

Fisher's 1930 book contained another important concept, the "reproductive value," which Medawar used in a simplified form in his research. If the death rate could be defined by the above equation, then the "birth rate," b_x, could also be defined in a similar way, and the total expectation of offspring for all the age groups in a static population could be described in the following way.[73]

$$\int_0^\infty l_x b_x dx$$

[70] Smocovitis, *Unifying Biology*, pp. 119, 122.
[71] Fisher, *Genetical Theory*, p. 23; Lotka, *Elements*, p. 102; Medawar, "Demography: notes," p. 7, undated, Box 17, Folder C.26, PBM.
[72] Fisher, *Genetical Theory*, p. 23; Medawar, "The Growth," p. 335.
[73] Fisher, *Genetical Theory*, p. 25.

Fisher reasoned that this could be changed into the next formula if the size of the population increased at the rate of e^{mt} with the progress of time t.

$$\int_0^\infty e^{-mx} l_x b_x dx$$

With this form, the reproductive value of a group of people aged x was obtained.

$$\frac{e^{mx}}{l_x} \int_x^\infty e^{-mt} l_t b_t dt$$

This value was the extent to which "persons of this age, on the average, contribute to the ancestry of future generations" *per head*.[74] It was an important factor in evolution, since "the direct action of Natural Selection must be proportional to this contribution."[75]

After carefully perusing these formulas, Medawar defined the reproductive value in his own terms, which were slightly different from Fisher's. Rather than lx, he used p_x which was defined as l_x/l_0, and supposed that the population did not increase. This supposition simplified the formula by eliminating e^{-mt}. Hence, the reproductive value R_x was defined by this equation.[76]

$$R_x = \frac{1}{p_x} \int_x^\infty p_x b_x dx$$

[74] Fisher, *Genetical Theory*, p. 27.
[75] Fisher, *Genetical Theory*, p. 27.
[76] P. B. Medawar, "Definitions in Vital Statistics," p. 2, undated, Box 17, Folder C.23, PBM. Although these mathematical papers in Box 17, Folder C.23 are "undated," the archival catalogue says that they were composed in the 1940s. This dating is reliable, because Medawar did not publish any paper related to these mathematical works after he delivered his inaugural lecture on the evolution of aging at University College London in 1951. While it is still not certain whether he wrote these manuscripts before or after he published his first paper on the evolutionary theory of senescence in 1946, from logical perspectives it was probably before 1946 that he wrote them.

In his research memo, Medawar explained how this reproductive value varied according to the age of humans in the real world. It was "certainly higher at age 10 than at age 0" in the case of humans, because newly born babies had a higher probability of death than those who survived to become ten years old.[77] Therefore, "a sublethal disease affecting children at 10 will be more serious than a disease affecting a similar fraction of children at 0, because some of those affected at 0 will have died anyway. Those aged 10 have a higher potential for parenthood."[78] The reproductive value was also related to the actual reproductive capacity of the individual, which depended upon its age. Basically, the capacity should be zero before it reached puberty and adolescence.

However, Medawar spent more time and energy in defining and using the reproductive value in a "special case" than the above general case.[79] In this special case, he postulated that the age-specific birth rate, as well as the total population size, did not change. Furthermore, the number of individuals within an age group decreased at a certain fixed rate, due to accidents such as predation or disease. Therefore, the death rate or the force of mortality μ_x was set at a value μ, and $l_x = \quad l_0 e^{-\mu x}$. Then p_x could be defined as $l_x/l_0 = e^{-\mu x}$. The variable b_x was also replaced by the constant b, and Fisher's e^{-mt} was eliminated because it was not necessary. In this condition, the reproductive value, R_x, of a group aged x was determined by the following formula that always had the constant value, b/μ.[80]

$$R_x = \frac{1}{p_x}\int_x^\infty p_x b dx \quad = \quad \frac{b}{e^{-\mu x}}\int_x^\infty e^{-\mu x} = \frac{b}{\mu}$$

For Medawar, this equation meant that "the contribution made by each age-group of the population to the total births, and therefore to the ancestry of future generations, depends

[77] P. B. Medawar, "Definitions in Vital Statistics," p. 3, undated, Box 17, Folder C.23, PBM.
[78] P. B. Medawar, "Definitions in Vital Statistics," pp. 3-4, undated, Box 17, Folder C.23, PBM.
[79] P. B. Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[80] P. B. Medawar, "Population with Constant Force of Mortality," undated, Box 17, Folder C.23, PBM.

only upon the [number] of individuals within the age group," since reproductive value per head was always constant.[81]

An origin of this idea on the "special case" can be found in Minot's thesis on cellular aging which Medawar had already accepted. Admittedly, Medawar felt that Minot's "general thesis leans too heavily on mere [cell] multiplication rates: very important they are indeed, but not all important," because "there are other physiological characters which do not start to age at zero."[82] Nevertheless, Minot's argument that aging began right after birth rather than after passing the period of reproductive maturity was reflected in Medawar's "special case" where the peculiarities of reproductive maturity and infant mortality was temporarily neglected and the organisms of all ages were subject to the same constant force of mortality. Indeed, this was not the first time that Medawar abolished the distinction between growth and aging, since he did the same thing in his earlier work on the decline of growth energy in embryonic chicken heart cells.

But a more immediate origin of Medawar's "special case" is found in Lotka's 1925 monograph. After defining the force of mortality, Lotka supposed a special and non-real condition, in which the force did not change over time and death occurred only by accidents, such as predation and disease. In his note, Medawar copied the following statement of Lotka.

> Such a simple life curve as this is not to be expected in a species of living organisms. It implies that the individual does not *age*, that his chance of living another year is just as good at ninety years of age as at fifty or at ten or at five; he can die, as it were, only by accident; he is perpetually young.[83]

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[81] P. B. Medawar, "Special Case," undated, Box 17, Folder C.23, PBM. Actually, he crossed out "number" in the above sentence and wrote "fraction" above it. But this does not make difference, because "fraction" applies to p_x, while "number" applies to l_x. p_x becomes l_x if divided by the constant l_0.

[82] Medawar, "Demography: notes," p. 6, undated, Box 17, Folder C.26, PBM.

[83] Lotka, *Elements*, p. 106; Medawar, "Demography: notes," p. 7, undated, Box 17, Folder C.26, PBM.

After copying this passage, Medawar wrote that the degree of departure of the force of mortality from constancy….is a measure of the degree to which senescence contributes to the causes of death."[84]

How, then, was this departure made possible? In his notes on the "special case," he wrote that "under these circumstances, it should be possible to draw the following genetic conclusions."[85] The first of them is that "the selective advantage of a mutant character varies inversely with the age in life at which the gene determining that character expresses itself phenotypically."[86] This statement, which already took Ford's, Huxley's, and Haldane's idea of the time gene for granted, depended on the concept of the reproductive value, which remained constant over time. Hence the contribution of each age group to the ancestry of future generations depended only upon the number of its members, which decreased as time passed under the constant force of mortality. In this state, even the mutation which might confer a great survival value could give little actual selective advantage to the organism, if it was expressed at an age when most members in that age group had died. Indeed, since the occurrence of a mutation was simply random, it was also possible that the genes expressed at this age would be subject to deleterious mutations, although they, too, had little chance of being activated. Therefore, the second conclusion was deduced—"in a future population, there will be a certain age beyond which the selective value of any modification, however 'good' or 'bad,' will be null and void, i.p. selection pressure will be insufficient to counterbalance mutation pressure."[87] Medawar then tried to show that this "certain age" could be determined in relation to the proportion of the age group in the total population. Since the death rate should equal to the birth rate in his "special case," μ could be replaced by b.[88] Then the "$1/n^{th}$ of the population" that "reaches or exceeds an age x" would be[89]

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[84] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[85] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[86] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[87] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[88] Since the total population size did not change in Medawar's "special case," the birth rate and death rate should be the same.
[89] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.

$$\frac{1}{n} = \int_{x}^{\infty} be^{-bx} dx$$

To put it more precisely, this value was the fraction that the age groups above *x* years old could contribute to the next generation. But since the total population and age-specific birth rate did not change, this value did not differ from the fraction of the current generation. Under this condition, the theoretical "old age" *x* could be calculated as follows.

$$x = \frac{\log n}{b}$$

He estimated that *n* was "of the order of 10^5 or 10^6."[90] But what was more important than such arbitrary numbers was that "a theory of the 'cause of senescence'" could be constructed from the above reasoning. Medawar wrote the following statements which contained a core element in his theory of aging.

> That the genes controlling 'good' characters will be so modified in time of phenotypic expression, that they come into action earlier and earlier in life; while, conversely, the phenotypic expression of 'bad' genes will be progressively slowed on in life….Lethal genes, if they reach expression late in life, will spread through a population simply because counter-selection is ineffective. Ordinary mutation pressure is sufficient to account for the accumulation of ultimately lethal degenerative changes late in life, because mutations….usually have ill effects.[91]

The influence of Wright and Dobzhansky's concept of evolutionary rudimentation, which Medawar found applicable to old age through Simpson's book, can be found in this idea.

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[90] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.
[91] Medawar, "Special Case," undated, Box 17, Folder C.23, PBM.

The genes expressed only in old age when few individuals would survive could mutate freely and spread into the population without being subject to the force of natural selection. In this way, old age could evolve into a kind of rudimentary period of life with many deleterious symptoms. In contrast, the genes activated in earlier phases will be effectively subject to natural selection, and most of these genes should evolve into a direction that could contribute to the survival of the organism. This difference based on age explained the departure from the constant force of mortality Medawar suggested in his notes. Even in a non-real state with a constant death and birth rate, evolutionary process, through the accumulation of deleterious mutant genes that would become activated only in later phases of life, would inevitably bring about the force of mortality that would become stronger with aging.

Medawar did more than proposing this theory in this abstract form. He tried to calculate the changes in a hypothetical population according to his theory to see whether his idea could be applied to an actual example. He first supposed that the constant birth and death rate b was 0.1. This meant that 10% of the original population would perish and be replaced by the same number of newly born organisms within a certain period of time. Therefore, if the replacement period was one month, 90% of the original population would survive after the first month, 81% after the second month, and 72.9% after the third month. This meant that only a tiny portion of the original cohort group would remain alive after, say, five years, and that the genes that were expressed in these periods would mutate randomly without being controlled by the force of natural selection. Interestingly, Medawar found that if he used 0.105 rather than 0.1 as the death or birth rate, almost the same result could be obtained in $p_x = e^{-bx}$. If b was 0.105 in this equation, then $p_x = e^{-1.05x} \approx (0.9)^x$. The value of $p_{x=60}$ after five years, 0.001836, was very close to $(0.9)^{60}$, which was 0.001797. Medawar wrote that this was "a good enough correspondence."[92]

But this equation using p_x did not appear in Medawar's first paper on the evolutionary theory of aging published in *The Modern Quarterly* in 1946.[93] As the

[92] P. B. Medawar, "Natural Examples," undated, Box 17, Folder C.23, PBM.
[93] P. B. Medawar, "Old Age and Natural Death," *The Modern Quarterly* 2 (1946), pp. 30-49.

journal's name indicates, Medawar's article aimed at broad audience including both professional scientists and the general public. The first part of this article summarized in plain languages previous research on aging and other relevant studies by Minot, Elton, Carrel, Weismann, Elie Metchnikoff, Herbert Spencer Jennings, Raymond Pearl, and Clive McCay. While the second portion did discuss Medawar's own evolutionary theory of aging in relation to the works of these scientists, it never used any of the mathematical formulas he employed in his previous publications and unpublished notebooks. The only "mathematics" he used in the article was the calculation of the number of surviving individuals when the death and birth rate was 10% in a given period.[94]

Nevertheless, Medawar's 1946 paper introduced a new idea, which was novel for both professional scientists and the general public. Whereas it had generally been thought that aging and "natural death" was an inevitable phenomenon as a part of the organic life cycle, Medawar introduced the concept of *contingency* in his new theory of aging. According to him, senescence and death were not the part of the natural course all living organisms had to follow, but "an artifact of domestication."[95] Although the genes responsible for senile changes in later life could spread in the population without being influenced by natural selection, most of them didn't have any chance to express their phenotypes in the wild nature, where most individuals would die of accidents, predation, or disease before becoming aged. Yet domestication of humans and their animals completely changed this state. Due to the protective environment humans constructed around them, they and their animals began to live longer than their counterparts in the wild, and usually long enough to see the phenotypic effects of the mutant genes that would be activated only in their later life. Senescence was thus introduced into human life and its vicinity as a contingent byproduct of domestic life, which was far from an inevitable phenomenon in nature.

The post-reproductive phase was another contingent product of evolution. Whereas Simpson suggested that the phase functioned as the given last stage of life when genetic change did not have any meaning for evolution, Medawar thought that the phase

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[94] Medawar, "Old Age," p. 45.
[95] Medawar, "Old Age," p. 42.

itself was a consequence, rather than any predetermined causal factor, for the evolutionary changes leading to senility. Medawar wrote, "the existence of a post-reproductive phase of life is not causally relevant to the problem of ageing, for it is just that very ingredient of the ageing process—decline and eventual loss of fertility—which it is our business to explain."[96] According to him, the loss of reproductive capacity in old age was caused by the mutant genes that spread into the population without being influenced by natural selection. Since the course of evolution could not be predicted and could be changed by many unexpected factors, the mode and time of appearance of post-reproductive phase could also be influenced by these random factors.

An important theoretical background of this notion of contingency was the Modern Synthesis. The scientists involved in the Synthesis, many of whom were cited by Medawar in his writings, tried to explain evolution as a process driven by gene mutation and natural selection which were basically haphazard events occurring in a specific habitat in which an organism *happened* to live. Although the Synthesizers did not completely abandon the traditional notion of evolution as a progress along a predetermined direction, the new evolutionary theory that they constructed became much less goal-oriented than previous ideas on evolution. By accepting and using this novel theory, Medawar was able to postulate that the emergence, the length, and the time of onset of post-reproductive phase in evolution were determined through evolutionary contingency.

Medawar could also depart from the older ideas on evolution and aging with the new evolutionary theory after the Synthesis. As historian Robert J. Richards has shown, while the term "evolution" had been traditionally used to designate an individual organism's development, it eventually came to mean a species' development as well through the recapitulation theory of the nineteenth century.[97] This notion of evolution, in which the theory of natural selection hardly played any role, was highly teleological, since it meant a change toward a certain goal, the adult human body. The term

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[96] Medawar, "Old Age," p. 43. Also see P. B. Medawar, *An Unsolved Problem of Biology: An Inaugural Lecture Delivered at University College London, 6 December 1951* (London: Lewis, 1952), p. 15.
[97] Robert Richards, *The Meaning of Evolution: The Morphological Construction and Ideological Reconstruction of Darwin's Theory* (Chicago: University of Chicago Press, 1992), pp. 5-61.

"involution" was also goal-oriented, since it designated the inevitable senile decline of an individual organism's function and structure as well as the decline of a species which usually ended up as the species' extinction.[98] Indeed, for many nineteenth century evolutionary scientists—such as A. Packard, Alpheus Hyatt, Joseph LeConte, and Henry Osborn—"evolution" and "involution" were the terms to designate the unidirectional course of change of both individual and species from their birth to death.[99] As the species-level process was thought to be an inevitable, and even cosmic, event caused by the gradual diminution of "evolutionary energy," individual organisms' senescence and death was also considered to occur through the unavoidable decrease of mysterious internal principles, such as innate heat or vital force.[100] Medawar's theory was very different from these older ideas, since it did not assume anything about the involution at the species level or unavoidable decrease of vital energy. While aging still remained a determined destiny for every individual human, it was an artifactual, not natural process from the new evolutionary perspective.

Likewise, Medawar rejected the German biologist August Weismann's older evolutionary theory of aging, which he thought was based on a faulty logic. According to Medawar, Weismann had argued that natural selection tends to weed out the old and decrepit members of a species to reserve more resources and food for the newly born and young members of that population. If the elderly continued to survive and consume resources, their descendents might not be able to live in a good condition for their life and proliferation. Therefore, an inherent mechanism causing senescence and death of the older individual had been evolved to provide more space and better nutrition for the next generation. For Medawar, however, this idea was based on a "vicious cycle" or circular logic. In his inaugural lecture delivered in 1951 at University College London, he pointed out that "Weismann….assumes that the elders of his race are worn out and decrepit—the

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[98] Hans-Joachim von Kondratowitz, "The Medicalization of Old Age: Continuity and Change in Germany from the Late Eighteenth Century to the Early Twentieth Century," in Margaret Pelling and Richard M. Smith (eds.), *Life, Death, and the Elderly: Historical Perspectives* (London and New York: Routledge, 1991), p. 143; Peter Bowler, *Life's Splendid Drama: Evolutionary Biology and the Reconstruction of Life's Ancestry, 1860-1940* (Chicago: University of Chicago Press, 1996), pp. 353, 357.

[99] Bowler, *Life's Splendid Drama*, pp. 353-358; Ruse, *Monad to Man*, pp. 251-257.

[100] Bowler, *Life's Splendid Drama*, p. 437.

very state of affairs whose origin he purports to be inferring—and then proceeds to argue that because these dotard animals are taking the place of the sound ones, so therefore the sound ones must by natural selection dispossess the old!"[101]

But Medawar's criticism on Weismann' "circular logic" was not completely fair, since Weismann claimed that decrepitude with aging occurred even among individuals which would not undergo senescence by themselves. According to Weismann, this phenomenon was due to the accumulation of "slight injuries" that were unavoidable in any natural environment.[102] Since an individual would inevitably become weaker and physically worn-out with aging due to these injuries, natural selection should eliminate the aged during their competition with the young. With this process, more resource and food would be available to younger and healthier individuals that could thereby continue the proliferation of the species. Medawar's argument that Weismann tried to explain the origin of decrepitude by assuming its existence in the first place was thus based on an incomplete understanding of Weismann. Furthermore, Medawar was not aware that these slight injuries were not the only cause of senile decrepitude for Weismann. There was another cause, namely, the inherent limitation of somatic cell division that had emerged through natural selection. According to Weismann, the number of divisions that the somatic cells of an individual could undergo was limited, and because of this limitation, somatic cells, along with the whole organism, would eventually die of old age. This restriction in cell proliferation was due at least to two factors. First, somatic cells had to shape the form of the organism that was adapted to its environment through natural selection.[103] An organism's attainment of a certain size and shape through proliferation of its somatic cells accompanied the restriction of their proliferative capacity. Second, the unlimited proliferation of somatic cells was "lost when it ceased to be of use."[104] Since even an immortal individual would eventually be eliminated in its old age, the capacity for indefinite proliferation was not necessary and was lost during its evolutionary process.

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[101] Medawar, *An Unsolved Problem*, p. 14.
[102] Weismann, *Essays upon Heredity*, p. 24.
[103] Weismann, *Essays upon Heredity*, p. 31.
[104] Weismann, *Essays upon Heredity*, p. 25.

While Medawar did not seem to understand these subtleties of Weismann's theory, there are certain features in his theory which were selectively accepted by Medawar. It is important to notice that Medawar employed Weismann's notion of the contingency of aging. Even though Medawar's idea was based on a very different theoretical basis, he accepted Weismann's notion that senescence was a contingent product of evolutionary process rather than a predetermined phenomenon of life. (See Chapter 1.) In adopting this notion, Medawar also employed, like Weismann, natural selection as the mechanism of evolution. Although Weismann regarded the species as the unit of selection and Medawar favored the individual, what was important for both scientists was the primacy of natural selection which engendered senescence in almost all multicellular living organisms.

The factors that contributed to the development of Medawar's ideas can be found in the social conditions in Great Britain of the 1930s and 1940s as well as the intellectual resources he used. The next section will discuss how Medawar's ideas could be understood in the context of contemporary British social problems and how these problems, along with his attitude toward his own work, guided his roles in gerontology in the United Kingdom. The next section also includes an account of Medawar's protégé Alex Comfort's experimental research on senescence as well as the American biologist George Williams' new evolutionary theory of aging.

Evolutionary Theory, Gerontology, and the Aging Population in Great Britain after World War II

When Medawar published his theory in 1946, aging had already become an important national issue in Great Britain. From the 1930s, statisticians, eugenicists, and economists had argued that the decline of both birth and death rates would make Britain a country of elders and would eventually depopulate the nation.[105] In particular, they worried that the "ageing of Britain" would worsen the already unsound economic condition of the country during the Great Depression, because elderly people were

[105] Richard A. Soloway, *Demography and Degeneration: Eugenics and the Declining Birthrate in Twentieth Century Britain* (Chapel Hill: University of North Carolina Press, 1990), pp. 226-258, esp. p. 240.

thought to be less active participants in capitalistic economy.[106] It was also feared that aged people's reduced physical fitness would weaken the military prowess of the country, especially when the outbreak of World War II was imminent.[107] While such fears were somewhat lessened during the War through the widespread mobilization of aged workers and the increase of the birth rate, the warnings could still be heard, and because of this, the Royal Commission on Population surveyed the national population trends and the state of elderly people from 1944.[108] For the same reason, the Nuffield Foundation also initiated its research program on aging.[109] (See Chapter 4.)

Medawar's published articles show that his evolutionary theory of aging was influenced by this national problem. In the last paragraph of his 1946 paper, his basic thesis on the evolution of aging was articulated in relation to the contemporary social issue in the country. Here, he pointed out that due to the "reduced mortality in infancy and childhood" the "life of many who would otherwise have died" was saved, and thus "the age-spectrum of the population….is in many civilized countries shifting slowly towards old age."[110] The artificial environment of these "civilized countries" thus made more apparent the symptoms of aging which were caused by the mutant genes that had spread through the population without being influenced by the force of natural selection. In his 1951 inaugural lecture, he stated the relevance of his theory to the social issues more clearly. "Fifty years ago," he said in front of the audience of University College London, "the major killing diseases were pneumonia and tuberculosis, both of infective origin; to-day they are cancer and what is compendiously called cardiovascular

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[106] Pat Thane, *Old Age in English History: Past Experiences, Present Issues* (Oxford: Oxford University Press, 2000), pp. 336-342.
[107] Richard M. Titmus, *Poverty and Population: A Factual Study of Contemporary Social Waste* (London: Macmillan, 1938).
[108] Thane, *Old Age*, p. 343.
[109] See, for example, B. Seebohm Rowntree, *Old People: Report of a Survey Committee on the Problems of Ageing and the Care of Old People* (London: The Nuffield Foundation, 1947); J. H. Sheldon, *The Social Medicine of Old Age: Report of an Inquiry in Wolverhampton* (London: The Nuffield Foundation, 1948).
[110] Medawar, "Old Age," p. 49. This argument was supported by his citation of several articles concerning the population change, including the British biologist Lancelot Hogben's article. See Lancelot Hogben, "The Measurement of Human Survival," in M. L. Johnson and M. Abercrombie (eds.), *New Biology*. Vol. 1 (London: Penguin Books, 1945), pp. 29-44.

disease."[111] Yet the latter two diseases were "affections of middle and later life," the periods which had not been subject to the force of natural selection in the past.[112] The growing significance of these chronic diseases showed that "in the last seventy-five years, the whole pattern of the incidence of selective forces on civilized human beings has altered."[113]

Clearly, this changing pattern of population and the concomitant increase of chronic diseases in Britain and other Western countries brought about "a problem of conspicuous sociological importance" that demanded more systematic research.[114] Medawar argued,

> Everyone now knows that the proportion of older people in our population is progressively increasing, that the centre of gravity of the population is shifting steadily towards old age. Using a plausible combination of hypotheses, one among several, the Statistics Committee of the Royal Commission on Population predicts that in half-a-century's time one quarter of our population will be not less than 60 years of age. The economic consequences of such an age-structure are all too obvious. Now biological research is by no means uninfluenced by the economic importunities of the time, and there can be little doubt that the newly-awakened interest of biologists in ageing…..is a direct reaction to this economic goad.[115]

The changing population trends thus demanded the scientific study of aging, gerontology. He argued that "something must be done," and gerontology should be pursued as a serious scientific field like pediatrics that had developed through the enhanced concerns about children's health.[116]

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[111] Medawar, *An Unsolved Problem*, p. 4.
[112] Medawar, *An Unsolved Problem*, p. 4.
[113] Medawar, *An Unsolved Problem*, p. 5.
[114] Medawar, *An Unsolved Problem*, p. 3.
[115] Medawar, *An Unsolved Problem*, pp. 3-4.
[116] Medawar, "Old Age," p. 49.

Indeed, Medawar knew the early contributors to gerontology quite well. As I have written in the previous sections, Medawar used the works of Minot and Carrel for the construction of his theory. Medawar also read other major publications of early researchers of aging, such as Elie Metchnikoff's *The Prolongation of Life* (1910), C. M. Child's *Senescence and Rejuvenescence* (1915), Raymond Pearl's *The Biology of Death* (1922), and Herbert Spencer Jennings' 1945 article on the senescence of protozoa.[117] Moreover, Medawar carefully studied *Problems of Ageing* (1939), the first comprehensive handbook on aging research, edited by the American cytologist Edmund Vincent Cowdry. (See Chapter 5.) Medawar also knew the research of Vladimir Korenchevsky, the Russian-British scientist who made a substantial contribution to the birth of gerontology in the United Kingdom. (See Chapter 4.)

However, Medawar was quite critical about some of these scientists' works and accepted them only in a selective way. He criticized Pearl's arguments that aging and natural death of metazoan organisms was initiated when some of their mutually dependent parts of the body broke down, leading to the destruction of the whole body's balance. For Medawar, Pearl did not explain how such a breakdown happened in the first place.[118] Nevertheless, it cannot be denied that Medawar, like Pearl, used statistical approaches and accepted his experimental conclusions that longevity was substantially guided by hereditary factors. Indeed, as I have pointed out in chapter one, both Pearl and Medawar were heirs of British statistical tradition and genetic research, even though the character of their work was very different.

Medawar did not like other scientists' ideas and publications on aging, except those of Carrel and Minot who heavily influenced him. He wrote that Metchnikoff's aging theory was "disputed at all stages," and Child drew "a great many vague and amorphous inferences from experiments of a technically obsolete type."[119] Medawar also stated that Jenning's experiments on the aging of protozoa were "most unfortunate; the

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[117] Herbert Spencer Jennings, "*Paramecium bursaria*: Life History. V. Some Relations of External Conditions, Past or Present, to Ageing and to Mortality of Exconjugants, with Summary of Conclusions on Age and Death," *Journal of Experimental Zoology* 99 (1945), pp. 15-31.
[118] Medawar, "Demography: notes," p. 14, undated, Box 17, Folder C.26, PBM.
[119] Medawar, "Demography: notes," pp. 3, 9, undated, Box 17, Folder C.26, PBM.

problem seems to have been driven back again into the penumbra of speculation."[120] Medawar also criticized some chapters in Cowdry's book, which was made of "essays of very unequal merit."[121] While there were several "authoritative" and interesting articles in Cowdry's handbook—such as Clive McCay's chapter on the chemical aspects of aging—their influence upon Medawar's work were not evident, since he did not say anything about the relation of these articles to his theory of aging.[122]

But what is more surprising was the fact that Medawar did not discuss much about the research on aging being done in his home country, since all the scientists addressed above were Americans, except Metchnikoff. Although Medawar did mention Korenchevsky's name, he did not write anything about his research. Indeed, Korenchevsky's name appears in Medawar's correspondence only in relation to the organization created by Korenchevsky, the Club for Research on Ageing.[123] Medawar did not think that the existence of this "so-called Club" was enough to say that there was any "organized work on the biology of the ageing process going on in England."[124]

In fact, Medawar did not consider his own theory an important contribution to professional gerontology in Great Britain. This attitude can be found, first of all, in the fact that he published his first article on aging in *The Modern Quarterly*, a semi-popular magazine rather than a peer-reviewed professional scientific journal. Medawar also wrote in his later autobiography that his theory of aging was a product of his "intellectual pastime," which he was not able to abandon even while he extensively studied the problem of skin homograft during the War.[125] His letter to his mentor Fisher shows that this retrospective account was basically true, since Medawar wrote that his 1946 paper was "a popular article on the subject of ageing."[126]

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[120] Medawar, "Demography: notes," p. 2, undated, Box 17, Folder C.26, PBM.
[121] Medawar, "Demography: notes," p. 8, undated, Box 17, Folder C.26, PBM.
[122] Medawar, "Demography: notes," p. 8, undated, Box 17, Folder C.26, PBM.
[123] Medawar to Alex Comfort, 24 April 1950, Box 2, File 3, ACF.
[124] Medawar to Alex Comfort, 24 April 1950, Box 2, File 3, ACF. This statement was correct in some sense, because, as I will show in the next chapter, the state of gerontology was certainly less strong in Britain than in America.
[125] Medawar, *Memoir*, p. 84.
[126] Medawar to Fisher, undated, RAF. This letter is undated, but probably written in late 1946 or early 1947. This letter is followed by Fisher's reply written in March 18, 1947.

But Medawar knew that even this "popular article" was based upon sophisticated mathematical, statistical, and evolutionary reasoning which was far from being "popular." He wrote to Fisher that "the theory outlined is simply a development of a broad hint dropped in Chapter 2 of The Genetical Theory of Nat. Sel.," and this was the reason why Medawar was "bothering" Fisher.[127] Medawar asked Fisher to read the article and "tell [him] if….the ideas contained in the second part are worthy of quantitative development."[128] Fisher replied that he carefully studied the article "with a great deal of interest," and thought that Medawar's "approach by considering a hypothetical organism with a constant death rate [was] quite excellent as a step to understanding the situation that has come about in….practically all animals, and to a quite surprisingly large extent in perennial plants."[129] Medawar also showed his theory to Haldane, who wrote a detailed comment on it, particularly on its mathematical aspects.[130]

Yet the evolution of aging never became Medawar's major research topic, although he did attempt some "quantitative development" in his 1951 lecture[131] and hoped to "take up [aging research] again in a big way in a year or so's time."[132] One possible reason for his not taking up the research as he hoped can be found in the fact that aging was not a major issue in any scientific organizations in which Medawar participated as a member, including "the Anatomical, Physiological, Linnean, Royal, Genetical, Experimental Biology and probably a good many more societies."[133] While he was very busy in the matters concerning these various organizations and the research topics discussed in their publications, there is no indication that he was able to receive any stimulus for further gerontological research from these societies and their journals, since they did not deal with aging as a serious subject. Another, perhaps more important,

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[127] Medawar to Fisher, undated, RAF.
[128] Medawar to Fisher, undated, RAF.
[129] Fisher to Medawar, 13 March 1947, RAF.
[130] Haldane to Medawar, undated, Box 17, Folder C.23, PBM. Medawar appreciated Haldane's comment as well as that of L. S. Penrose in his 1951 lecture. See Medawar, *An Unsolved Problem*, p. 3.
[131] Medawar, *An Unsolved Problem*, p. 17. He expressed with mathematical formula the constant reproductive value and the fraction of the population within a certain age interval.
[132] Medawar to Comfort, 13 November 1950, Box 2, File 3, ACF.
[133] Medawar to Fisher, 13 March 1951, RAF.

reason is that his experimental research on immune tolerance was immensely successful and eventually earned him the Nobel Prize in 1960. The success of his immune tolerance research led him to spend most of his time in extending the scope of the application of his discovery in many biomedical fields.[134] In this situation, aging research could hardy be a priority for him.

Nevertheless, Medawar did make contributions to the development of gerontology in Great Britain. Indeed, his place in the science of senescence in Britain can be understood in the context of the emergence of aging as a national problem during the 1930s and 1940s and the British responses to it. As I will discuss further in the next chapter, British scientists' part in this response was the formation of the British Society for Research on Ageing. British philanthropies like the Nuffield Foundation also did their part through their support of gerontological research. As a biologist who proposed a theory that reflected this important social issue, Medawar too participated in these scientists' and philanthropies' responses to the problem. First of all, he did attend some of the meetings of the British Society for Research on Ageing, although it is not known how deeply he was involved in the Society's activities.[135] Moreover, he gave his comments to the scientists who had read his papers on aging and contacted him to seek his advice on the plan for their research on senescence.[136] Medawar was also appointed a member of the advisory committee of the Nuffield Gerontological Research Fellowship, which was established in 1953 "as a part of [the Foundation's] programme for the care of old people."[137] As a member of the committee, he read and selected applications and research proposals written by various British researchers of aging.[138]

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[134] For the extension of the research on immune tolerance, see Ilana Löwy, "The Strength of Loose Concepts—Boundary Concepts, Federative Strategies and Disciplinary Growth: The Case of Immunology," *History of Science* 13 (1992), pp. 376-382. Indeed, Medawar's research publications after his 1953 tissue transplantation experiment are almost exclusively about immune tolerance.

[135] Medawar to K. Gerhard Brand, 16 April 1962, Box 18, Folder C.48, PBM.

[136] See Brand to Medawar, 10 April 1962, Box 18, Folder C.48, PBM; Brand to Medawar, 25 April 1962, Box 18, Folder C.48, PBM.

[137] "Nuffield Gerontological Research Fellowship," Box NF AGE1, Folder Nuffield Gerontological Fellowship, NF.

[138] Medawar to W. A. Sanderson, 24 June 1953, Box NF AGE1, Folder Nuffield Gerontological Fellowship, NF.

But Medawar made a more important contribution to gerontology in Great Britain by supporting Alex Comfort (1920-2000), a poet, novelist, anarchist, sex reformer, and leading British gerontologist. Comfort resembled Medawar in many respects, since both had prestigious academic backgrounds, broad interests, and intellectual acuity. Just as Medawar was a "First" in Oxford zoology graduation exam, Comfort was the first in Cambridge's Natural Science Tripos Part I and the second in the Part II.[139] After that, Comfort left Cambridge and was trained as a physician and physiologist in London, and wrote several poems and novels such as *The Power House* (1945) that made him known as an ingenious writer, too. He then worked as a lecturer in physiology at London Hospital Medical College and studied the pigments of mollusks.[140] At the same time, he read Medawar's article on aging as well as other publications by Carrel, Minot, and McCay, which deeply impressed him.[141] Since Comfort knew that the Nuffield Foundation was supporting gerontology, he contacted the Foundation to acquire funding for his "training and research experience in the biochemical side of geriatrics, with a view to doing some work….on the nature of the process of ageing."[142] He also wrote to Medawar to ask whether it was possible to study aging with him in his laboratory at the University of Birmingham. While Medawar first replied that he did not have any vacant space in his lab and he might be moving to another school in a year or so, he changed his mind and decided to include Comfort in his team after a long overnight discussion.[143] Medawar, as a member of the "biological advisory group" of the Nuffield Foundation wrote to the Foundation that Comfort was "exceedingly quick witted, widely read, and enthusiastic."[144] Since Medawar had seen that even "a person of half Comfort's intelligence" had succeeded as a scientist, there was no reason that the Foundation should

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[139] See Comfort's brief resume in Comfort to the Nuffield Foundation, 27 July 1949, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[140] J. Z. Young to Sanderson, 5 August 1949, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[141] Alex Comfort, "Research in Ageing," *Discovery* (September 1950), pp. 295-299.
[142] Comfort to the Nuffield Foundation, 27 July 1949, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[143] Medawar to J. H. Sheldon, 7 November 1950, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[144] Medawar to Sanderson, 20 November 1950, Box NF AGE1, Folder Dr. Alex Comfort, NF. On Medawar's membership in the "Biological Advisory Group," see Medawar to Sheldon, 13 November 1950, Box NF AGE1, Folder Dr. Alex Comfort, NF.

not support Comfort.[145] Since other scholars' evaluation of Comfort was also very positive, the Foundation, on November 1950, decided to award him a research grant, provided that Medawar offered lab space and any necessary instructions for the young scientist.[146] When Medawar moved to University College London in 1951, Comfort followed him and the Foundation continued the support.

Comfort's research on aging began from Medawar's ideas in his 1946 paper on aging. Here, Medawar proposed that the nature of aging could be approached by transplanting old animals' tissues to young animals and vice versa.[147] He expected that this experiment, if performed with highly inbred animal strains, would show how the senescence of tissues was influenced by the environment whose age was different from their own. Actually, as I have written in the previous section, Medawar did conduct a tissue transplantation experiment between animals of different ages, although he did not use genetically homogeneous organisms at that time.[148] Comfort's proposal submitted to the Nuffield Foundation discussed precisely this sort of experiment. He wrote that he hoped to unite "rats of identical strain but different ages" and examine "the effect of substances present in tissue fluids and plasma upon the process of ageing."[149] While this experiment was not pursued further for some unknown reason, Comfort started his research on another problem Medawar pointed out in his 1946 paper. In that article, Medawar suspected the validity of G. P. Bidder's argument that marine organisms did not undergo aging because they were able to grow without any limit.[150] As far as growth continued, Bidder thought, aging did not occur. But Medawar suspected that this argument might not be very trustworthy, since it was not based on a careful observation of how long marine species could actually survive in their natural environment. Comfort pursued this research further after 1953, and published a series of articles from 1960

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[145] Medawar to Sanderson, 20 November 1950, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[146] In 1950, the Foundation offered £1,000 per annum as the personal grant, £650 for apparatus, and £200 for experimental materials. See L. Farrer-Brown to Comfort, 30 November 1950, Box NF AGE1, Folder Dr Alex Comfort, NF.
[147] Medawar, "Old Age," p. 48.
[148] Medawar, "A Second Study," pp. 161-163.
[149] Alex Comfort, "Synopsis of Proposed Research," October 1949, Box NF AGE1, Folder Dr. Alex Comfort, NF.
[150] Medawar, "Old Age," pp. 40-42; G. P. Bidder, "Senescence," *British Medical Journal* 2 (1932), p. 583.

which revealed that aging of a marine organism, *Lebistes reticulatus*, did occur even while it was still growing.[151] This research, which was later reconfirmed by other scientists, became one of his most important contributions to gerontology.[152] With this and other experimental studies, Comfort gradually became a leading gerontologist in the United Kingdom.

While Comfort furthered the experimental side of Medawar's aging research, the American evolutionary biologist George Williams intensified its theoretical side with some new ideas. William's "antagonistic pleiotrophy theory," proposed in 1957, contained an idea Medawar briefly suggested in his 1946 article—there could be a "genetic endowment which can favour young animals only at the expense of their elders."[153] While Medawar did not actually further this idea, and focused on the possibility that different genes were activated in different ages, Williams employed that idea along with new experimental research on the alteration of many *Drosophila* genes' phenotypic effects with growth and aging, including a work by geneticist Raymond Pearl's colleague at Johns Hopkins.[154] Williams also used a mathematical equation borrowed from a contributor to the Modern Synthesis—Sewall Wright. According to Wright, a gene usually had "mixed effects on fitness" that could be described by the following equation.[155]

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[151] Alex Comfort, "Research Grant, October 1953-Octobr 1954," Box NF AGE1, Folder Dr. Alex Comfort, NF; Alex Comfort, "The Effect of Age on Growth Resumption in Fish (*Lebistes*) Checked by Food Restriction," *Gerontologia* 4 (1960), pp. 177-186; "The Longevity and Mortality of a Fish (*Lebistes reticulatus*, Peters) in Captivity," *Gerontologia* 5 (1961), pp. 209-222; "Effect of Delayed and Resumed Growth on the Longevity of a Fish (*Lebistes reticulatus*, Peters) in Captivity," *Gerontologia* 8 (1963), pp. 150-155.

[152] Avril D. Woodhead, "Aging, The Fishy Side: An Appreciation of Alex Comfort's Studies," *Experimental Gerontology* 33 (1998), pp. 39-51.

[153] Medawar, "Old Age," pp. 45-46.

[154] A paper by Bienvenido Gonzalez, a colleague of Raymond Pearl, was cited by Williams. But Gonzalez' article includes a very short mention of such differential phenotypic effects, and the genes (*black and speck*) mentioned by Williams do not seem to have a strong age-differential expressions pattern. See Bienvenido Maria Gonzalez, "Experimental Studies on the Duration of Life. VIII. The Influence upon Duration of Life of Certain Mutant Genes of Drosophila melanogaster," *American Naturalist* 57 (1923), pp. 289-325, esp. pp. 302, 314. Also see C. B. Bridges and K. S. Brehme, *The Mutants of Drosophila melanogaster* (Washington, D.C.: Carnegie Institute of Washington, 1944); Ernst Caspari, "Pleiotropic Gene Action," *Evolution* 6 (1952), pp. 1-18.

[155] Sewall Wright, "Modes of Selection," *American Naturalist* 90 (1956), pp. 5-24.

$$W = (1 + m_1p_1)(1 + m_2p_2)\ldots(1 + m_np_n)$$

Here, *W* was the overall fitness, *n* was the age, and *m* was the magnitude of the contribution of a gene to the fitness. But this magnitude had to be multiplied by the factor *p*, which was the "relevant proportion of the reproductive probability."[156] Williams wrote,

> Obviously a gene that conveyed a slight increase in fitness with a high *p*-value might be favorably selected in spite of a serious decrease in fitness with a low *p*-value. This, I believe, is the key to understanding the evolution of senescence, because any genic effect that arises late in life will have a low *p*-value. Natural selection may be said to be biased in favor of youth over old age whenever a conflict of interests arises.[157]

Natural selection hardly affected the impacts of genes in old age, when most organisms would have already been dead due to diverse natural hazards and thus "the relative proportion of the reproductive probability" was extremely low. In this case, without regard to the value of *m*, low *p* value made the whole factor containing such *p* insignificant for the overall fitness expressed as the value of *W*. If the effects of such genes initiated the evolution of senescence, then these genes would increase the death rate and further lower the *p* value of other genes in old age. Therefore, the evolution of senescence should proceed in a "self-aggravating" way.[158]

Williams' contribution to the extension of Medawar's idea was not limited to broadening its theoretical side. His article suggested many interesting "testable deductions" that could be confirmed by experiments or observations.[159] The first example of such deductions was found among the organisms that reproduced asexually.

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[156] George Williams, "Pleiotropy, Natural Selection, and the Evolution of Death," *Evolution* 11 (1957), p. 401.
[157] Williams, "Pleiotropy," p. 401.
[158] Williams, "Pleiotropy," p. 402.
[159] Williams, "Pleiotropy," p. 403.

Here, Williams distinguished the asexual organisms which reproduced by making clones—e. g. by binary division—from those that made their offspring by budding.[160] While the former did not show any symptoms of senescence, the latter did, because there was clear morphological and physiological distinctions between parent and offspring, and such distinctions could be caused by genes whose effects differed according to age. Indeed, the American protozoologist Tracy Sonneborn demonstrated that the flatworms reproducing by budding revealed definite signs of senescence when they were artificially cultured without predators.[161] Williams also suggested that "the evolutionary cause of the low rate of bird senescence must be that birds can fly, are thereby less liable to predation and accidents, and therefore have lower mortality rates."[162] Since the lower adult mortality rate of birds slowed down the rate of decrease of reproductive potential by letting more organisms survive in old age, it eventually lengthened the reproductively active period that was subject to natural selection and thus delayed the onset of senile phases, as could be seen in some contemporary observations.[163] Williams also suggested that repeated experimental castration at a certain age over many generations would hasten senescence after that age, because it eliminated evolutionary force at that point. Indeed, similar experiments have been done by later experimental biologists, who found that the artificial evolution of fruit flies within a harsh environmental condition substantially shortened their inherent longevity, while the selection of the flies reproducing later than others eventually led to the evolution of the fruit flies with longer lifespan and slower rate of senescence.[164] Currently, many experimental gerontologists are studying the evolution of aging, inspired by Medawar and Williams.

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[160] Williams, "Pleiotropy," p. 403.

[161] Tracy Sonneborn, "Genetic Studies on *Stenostomum incaudatum* (nov. spec.). I. The Nature and Origin of Differences among Individuals Formed during Vegetative Reproduction," *Journal of Experimental Zoology* 57 (1930), pp. 57-108.

[162] Williams, "Pleiotropy," p. 405.

[163] David Lambert Lack, *The Natural Regulation of Animal Numbers* (Oxford: Clarendon, 1954); W. Frank Blair, "Population Density, Life Span, and Mortality Rates of Small Mammals in the Blue-Grass Meadow and Blue-Grass Field Associations of Southern Michigan," *American Midland Naturalist* 40 (1948), pp. 395-419.

[164] Michael R. Rose, *Evolutionary Biology of Aging* (New York: Oxford University Press, 1991); S. C. Stearns, M. Ackermann, M. Doebeli, and M. Kaiser, "Experimental Evolution of Aging, Growth, and

Conclusion

Williams' article and the later developments in evolutionary research on aging show how the notion of evolutionary *contingency* was applied and expanded into gerontology: Senescence was no longer a cosmic phenomenon whose course could never be changed. It was highly contingent upon a species' evolutionary pathway and the pattern of natural selection in its environment. Therefore, as gerontologists Michael Rose and Joseph Graves wrote in 1989, there is "no physiological necessity about senescence," and a "universal biochemical mechanism" of aging cannot be found.[165] This concept of contingency, while differing from Carrel's version, was similar to it in some sense, because both enabled scientists to manipulate aging through experimental means. Whereas the scientists after Carrel began to manipulate senescence at the cell level, the researchers following Medawar's and Williams' ideas did the same thing at the population level. Along with McCay's caloric restriction experiments that made possible the control of the rate of the aging process, Medawar's and Williams' works created a new line of research in experimental gerontology.

In this sense, Medawar, Williams, and later researchers constructed a new and important part of the multidisciplinarity of modern gerontology. They constructed the mathematico-experimental approach to aging from an evolutionary viewpoint which was not advocated by any contributors to Cowdry's *Problems of Ageing* or any other early gerontologists in Britain and America.[166] Hence, I conclude that Medawar and others enlarged the scope of gerontology's multidisciplinarity by incorporating in it the evolutionary study of aging that employed mathematical and theoretical approach supported by experimental means. Since mathematical statistics, which had already been extensively used across disciplinary boundaries, was a core element in this approach, it may also be stated that gerontology became subject to the multidisciplinary expansion of

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Reproduction in Fruitflies," *Proceedings of the National Academy of Sciences of the United States of America* 97 (2000), pp. 3309-3313.

[165] Rose and Graves, "What Evolutionary Biology Can Do," p. B28.

[166] A review in *American Naturalist* pointed to this issue. See, for example, "Problems of Aging," *American Naturalist* 76 (1942), pp. 616-617. Also see Vladimir Korenchevsky, "Gerontology in the United Kingdom," *Journal of Gerontology* 6 (1951), pp. 275-287.

statistics, which in turn came to be included within gerontology's multidisciplinary structure.[167]

Another peculiar feature of Medawar's evolutionary theory is that it may reflect a British style of doing science during the first half of the twentieth century. His theory contained mathematical statistics which had primarily developed in Britain in the past. It also incorporated Britons' concerns on the aging of their population, which was encouraged during the 1940s through the statistical surveys of which Britain had a long tradition. Since some of the mathematical scholars whose ideas Medawar used, such as R. A. Fisher, were also social thinkers on the issue of population, we could regard Medawar's theory as a construct that reflected both British science and society at that time.

However, the development of the theories of aging initiated by Medawar could not be continued for almost ten years after Williams' proposal of a new version in 1957. It was not until 1966 that another British biologist, William Donald Hamilton, extended Medawar's line of evolutionary investigation with advanced mathematical approaches,[168] and it was only in the 1970s that more systematic evolutionary studies of aging were resumed by several scientists.[169] Why, then, didn't the publication of Medawar's theory bring about a more active response at that time? While American gerontologists' silence about Medawar's theory was strange as well, the fact that few British scientists became interested in the theory during the 1940s and 1950s is even more curious, if we consider that these scholars were physically closer to the influential and prestigious British scientist, Medawar. Indeed, the responses toward his theory of aging reprinted in his *The Uniqueness of the Individual* (1957) indicate that some Britons misunderstood the major point of the theory and failed to regard it as a starting point for further experimental inquiry on the evolution of aging.[170] As has been discussed in the previous section, this

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[167] About the multidisciplinary character of statistics, see, for example, Porter, *The Rise of Statistical Thinking*, p. 8.
[168] W. D. Hamilton, "The Moulding of Senescence by Natural Selection," *Journal of Theoretical Biology* 12 (1966), pp. 12-45.
[169] Martin, "How Is the Evolutionary Biological Theory of Aging," p. 2.
[170] See E. Ashby, "Growth, Development, and Ageing," *British Medical Journal* 2 (1957), p. 147. In this article, Ashby took the post-reproductive phase as a predetermined period, neglecting Medawar's argument

was partly due to the fact that Medawar himself was never very enthusiastic about continuting his research on the evolution of aging. He never published his theory in a professional journal, and *The Uniqueness of the Individual* was also a semi-popular monograph. Another reason for this poor initial reception was that aging did not become an important scientific research subject in Britain during the postwar years, especially if compared with the state of gerontology in America. Britain had smaller number of researchers of aging, less well-organized professional societies, no academic journals devoted to aging research, and less stable systems for funding gerontology. The next chapter will discuss the factors that contributed to this state of gerontology in the United Kingdom.

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that this phase was also a product of evolution. Also see Jean Howard, "Biologist's Lens," *The Spectator* (23 August 1957).

Chapter 4

"The Work Was Not, from Its Nature, Likely to Be Exciting" The Troubled Beginning of the Science of Aging in the United Kingdom

This chapter aims at explaining the issue mentioned at the end of the last chapter. Why did Peter Medawar's evolutionary theory of aging fail to initiate a major research program in Great Britain? Certainly, Medawar's theory was not seriously considered by British scientists during the 1940s and 1950s. What is more remarkable is the fact that there were few other research programs on aging actively pursued in Great Britain. Basically, there were not many scientists in the country who were enthusiastic about senescence as a scientific research subjects during these decades.

While there could be several explanations of this phenomenon, this chapter analyzes three interrelated factors that were mainly responsible for this state of early gerontology in Britain. First, I will discuss the life of the Russian-British scientist Vladimir Korenchevsky (1880-1959) who tried to build gerontology in the United Kingdom. His efforts and the response he received reveal some reasons why gerontology could not fare well in this side of the Atlantic. Second, I will analyze the works of other scholars who joined the field of aging during its early years, especially in relation to their actual research interest and the size of the community. This will provide a more detailed picture of the state of the scientific practitioners on aging in the country and show their limitations. Finally, I will examine the British society's primary way of caring for its aged members and its impact on the growth of gerontology in terms of funding and institutional support. An analysis of the British sponsors of gerontology, including the Nuffield Foundation and the Medical Research Council, within the social context of the 1940s and 1950s will indicate reasons why aging research could not have priority at that time.

The first two sections of this chapter deal with the activities of the scientists of aging. In the first section, the works of Korenchevsky are discussed in detail in relation to the characteristics and the difficult early years of British gerontology in which he was a major player. I argue that his concerns on his own troubled older years—characterized by the impending retirement, deep economic hardship, and low academic status—contributed to his decision to study senescence as a major research topic and to construct gerontology as a scientific field. Simultaneously, I also show, his problems regarding his aging and socioeconomic condition hindered any further growth of the science of senescence into a mature scientific field. While he did make a crucial contribution to building the British Society for Research on Ageing, the Club for Research on Ageing, and the International Association of Gerontology, his work for these organizations had clear limitations due to his own personal problems regarding his aging and other factors. Unfortunately, there were few other investigators who tried to develop gerontology. In contrast to the United States which had a number of scientists who studied senescence intensively and tried to construct gerontology as a scientific field, there were not many scholars in Britain who were deeply committed to aging research and willing to play leading roles in fundraising, enhancing publicity, and recruiting sympathetic scholars.

The third and fourth sections of this chapter analyze the social condition surrounding gerontology to examine why there were not strong institutional supports for aging research and why not many researchers were interested in senescence. I will argue that the emerging welfare state in Great Britain created an unfavorable environment for the growth of the science of aging during the 1940s and the 1950s. While the British government and private philanthropies were struggling to enhance the welfare of older Britons, scientific research on senescence, except for some fields that had a direct bearing upon practical issues, did not attract much attention. Gerontology was considered too far from the immediate and actual needs of the elderly whose health, employment, and retirement had to be taken care of by the government and foundations.

This social context needs more explanation from comparative perspectives. As the next two chapters will show, the United States provided a more favorable environment for the growth of the science of gerontology than the United Kingdom. A major factor in

this difference was the role of federal governmental agencies such as the National Institutes of Health (NIH) which supported both intramural and extramural research projects on aging since the 1940s. (See Chapter 6.) Interestingly, this focus on research was found to be connected to the neglect of another, perhaps equally important subject on the citizen's health and well-being. According to Stephen Strickland, the federal government's support of biomedical scientific research at that time was the only possible means of improving the health of Americans when national health insurance could not be introduced.[1] Indeed, gerontological research was one of these biomedical research programs that received increasingly abundant support from the NIH after the end of World War II. In contrast, the actual healthcare for the elderly did not receive as much attention. The United States, before establishing the Medicare program in 1965, chose to spend money for biomedical research on aging rather than providing better and free healthcare for the elderly. The program for the livelihood of older people through pensions followed a similar pattern. Unlike Britain that had a long tradition of caring for the aged and passed the Old Age Pensions Act in 1908, America did not have such a tradition and established the Social Security Act only in 1935. For a long time, old age has been an individual concern in America, while in Britain it has been a collective social issue that had to be managed by the government.

Although this gross national difference would change with progress of time, it was certainly reflected in the state and characteristics of the early science of aging in the two countries. As I have shown through the popular reception of Clive McCay's research on longevity and caloric restriction, aging was considered something that had to be managed by individuals in America. (See Chapter 2.) As historian Thomas Cole has pointed out, "civilized old age" could be achieved through consistent self-care, including meticulous examination of dietary caloric intake. What is remarkable is that this concern about aging as an individual problem accompanied more stabilized and better-funded gerontology in the United States which included both basic research on aging process and

[1] Stephen P. Strickland, *Politics, Science, and Dread Disease: A Short History of United States Medical Research Policy* (Cambridge, Mass.: Harvard University Press, 1972), pp. 154-156, 213. Also see Victoria A. Harden, *Inventing the NIH: Federal Biomedical Research Policy, 1887-1937* (Baltimore: Johns Hopkins University Press, 1986), p. 182.

the projects that had practical implications. (See Chapters 5 and 6.) In some sense, Americans expected that more scientific knowledge on aging would help individuals find a better way of getting old. In contrast, the science of aging did not get much support from either the government or private philanthropies in Britain. If there were some moderately successful projects, these were always ones that had strong practical values, relevant to the current social measures for the elderly. Many clinical investigations supported by the Nuffield Foundation belonged to this category, especially because they had connection to geriatrics that was in a much better state after the establishment of the National Health Service (NHS) in 1948. Psychological research on the work capacity of the elderly was another project which was supported by both a foundation and a governmental agency.

The next section will discuss the birth of gerontology through the career of Korenchevsky from the 1920s to the 1940s. While this section is longer than others, I feel that this is necessary considering his role in the birth of the science of aging in Britain. The following sections will deal with the developments of the field after the late 1940s, by examining the state of the British Society for Research on Ageing and the "Care of Old People" program of the Nuffield Foundation. The Medical Research Council's support for the science of aging will also be examined in the last section.

The Troubled Old Age of Vladimir Korenchevsky and the Troubled Beginning of the Science of Aging in Great Britain

Korenchevsky was born in Russia in 1880, and studied medicine at the Imperial Medical Academy and earned his M.D. degree at the University of Moscow in 1909.[2] (See Figure 4.1.) He was trained as an experimental pathologist and worked with several eminent Russian biomedical scientists, including Ivan Pavlov and Elie Metchnikoff. After

[2] While his obituaries say that he was born in Lida, which is currently a part of Belarus rather than Russia, his short autobiography states that he was born in the town of "Oshmiani" in Russia, whose exact location is unknown. See "Autobiography of Dr. Vladimir Korenchevsky," Box 82, Folder 1109, WBC. See also Kathleen Hall, "Vladimir Korenchevsky," *Journal of Pathology and Bacteriology* 80 (1960), pp. 451-461; F. V., "V. Korenchevsky," *Gerontologia* 3 (1959), pp. 117-118; E. V. Cowdry, "V. Korenchevsky: Father of Gerontology," *Science* 130 (1959), pp. 1391-1392; Geoffrey H. Bourne, "Preface," in Vladimir Korenchevsky, *Physiological and Pathological Ageing* (New York: Hafner, 1961), pp. v-vi.

finishing his training, he was appointed professor of experimental pathology at the Imperial Medical Academy in Petrograd. But the Russian Revolution in 1917 completely changed his professional career. He left Petrograd after the outbreak of the Revolution and joined the White Army as a medical officer. When the White Army was defeated, he fled to Paris, France, where he sent a letter to the Lister Institute of Preventive Medicine in London. This letter, which showed his strong will to continue his scientific research as well as his desperate situation as a political refugee, was favorably considered by British medical scientists, especially by director of the Lister Institute, Charles James Martin (1866-1955).[3] In 1920, Korenchevsky was invited to the Lister Institute to study endocrinology and dietary deficiency under the financial sponsorship of the Medical Research Council (MRC). While his and his assistants' stipends were paid by the MRC, his research expenses and facilities were provided by the Lister Institute.

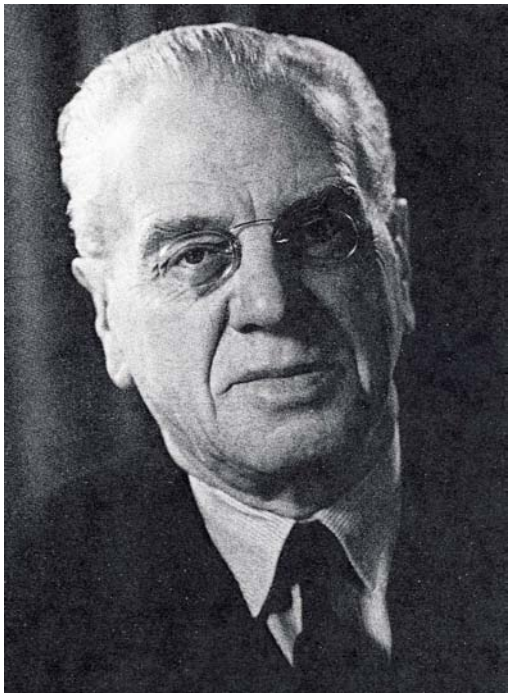

Figure 4.1. *Vladimir Korenchevsky. By Lettice Ramsey and Helen Muspratt, Oxford. From "Vladimir Korenchevsky, 30th January 1880-9th July 1959," Journal of Pathology and Bacteriology 80 (1960), p. 450.*

[3] Korenchevsky to Unnamed, 1 June 1920, FD 1/671 (Korenchevsky's Research 1920-32), NAUK; Walter Fletcher to Parkes, 13 February 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

During the first ten years, the relationship between Korenchevsky and his patrons was very good. Even though he was offered a professorship at the University of Zagrel, he decided to stay in London because the MRC extended Korenchevsky's grant, and he also appreciated the "atmosphere in which everyone becomes [imbued] with the same love of science and the same enthusiasm for scientific work."[4] The Medical Research Council and the Lister Institute evaluated his work very favorably. For them, Korenchevsky was a "senior and distinguished man" who was "doing sterling work of great importance, and with immense industry."[5] Since it was "almost 'sweating' him to give" only £600 per year, the MRC decided to raise his annual stipend to £800 in 1922.[6] The fact that the British Medical Association invited Korenchevsky to their annual meeting as a "distinguished foreigner" showed how respectful British physicians and medical researchers were toward him and his scientific work at that time.[7]

But the relationship between Korenchevsky and his patrons began to change after the early 1930s when several interrelated events occurred. First of all, the Great Depression brought about deep financial woes in almost every sector in British society including its medical research establishments like the MRC and the Lister Institute.[8] Due to their severe budget cuts in the 1930s, these institutions began to change their attitude toward Korenchevsky and their support for his research. Indeed, Walter Fletcher, secretary of the MRC, questioned whether it was desirable to continue the personal grant for Korenchevsky which had become a "heavy burden."[9] Furthermore, they claimed, Korenchevsky had been receiving his grant for more than ten years although he was in his fifties. In fact, the usual term of the MRC grant was three years, and it tended to be

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[4] Korencehvsky to C. J. Martin, 16 May 1921; Unnamed to Martin, 27 May 1921, FD 1/671 (Korenchevsky's Research 1920-32), NAUK.
[5] Unnamed to Hopkins, 26 January 1922, FD 1/671 (Korenchevsky's Research 1920-32), NAUK.
[6] Unnamed to Hopkins, 26 January 1922, FD 1/671 (Korenchevsky's Research 1920-32), NAUK.
[7] Jean Agrew to L. Thomson, 22 March 1922, FD 1/671 (Korenchevsky's Research 1920-32), NAUK.
[8] Korenchevsky to Walter Fletcher, 4 November 1931, FD 1/671 (Korenchevsky's Research 1920-32), NAUK; "I discussed with Ledingham," 28 February 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[9] Fletcher to Ledingham, 21 March 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

awarded to younger researchers.[10] To make matters worse, Martin, director of the Lister Institute and a person favorable to Korenchevsky's research, left the Institute in 1930.[11]

Martin's resignation from the institute revealed another problem that had not been seriously considered for the past ten years. Korenchevsky's position was temporary at best, and it was Martin, according to Fletcher, who offered the position with an assumption that Korenchevsky would "get a professorship in Eastern Europe before long."[12] Yet Fletcher said that "all hope of that, of course, has long disappeared, and perhaps partly because of the political activities that [Korenchevsky] pursued in his spare time."[13] Indeed, Korenchevsky was a refugee who was deeply concerned about his home country and his fellow Russians who had to flee from the communist government. This concern prompted him to participate in organizing the Russian refugees and spending a large portion of his income and time in this work. Fletcher believed that this work made it difficult for Korenchevsky to get a professorship in Eastern Europe, which was increasingly coming under Russian influence. Where, then, could Korenchevsky get a permanent job? The MRC was certainly not willing to offer Korenchevsky any permanent staff position, because "it [was] contrary to their present policy," which was to give "staff status….only as a part of a definite scheme for promoting work in some subjects for which there is otherwise inadequate provision."[14] Unfortunately, there is no evidence that any other academic institutions in the United Kingdom were willing to offer him a professorship.

But it was not possible to lay off Korenchevsky and to discontinue his grant, because that could "mean starvation for him."[15] In any case, J. C. G. Ledingham, the new director of the Lister Institute, argued that the MRC had had "their money's worth" because Korenchevsky was a very productive researcher who had been publishing quite a

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[10] Edward Mellanby to Ledingham, 26 February 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[11] Unnamed to Carr, 18 February 1931, FD 1/671 (Korenchevsky's Research, 1920-32), NAUK.
[12] Fletcher to Ledingham, 21 March 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[13] Fletcher to Ledingham, 21 March 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK. See also Unnamed, 1 June 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[14] Unnamed to Ledingham, 6 March 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[15] Fletcher to Parkes, 13 February 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

large number of scientific articles.[16] Yet Edward Mellanby, secretary of the MRC after Fletcher, wrote, "That might be so….If the work was as good as [Ledingham] said, why could not the Lister pay some part of the salary themselves"?[17] Indeed, Mellanby felt that the grant of £800 per year was a great burden, because the MRC was then suffering from a severe financial crisis incurred by the Depression. The MRC thus had to negotiate with the Lister Institute and succeeded in persuading the Institute to pay £400, which was half of Korenchevsky's annual grant, while the MRC would pay the rest. In this way, they decided to continue the total grant of £800 "no longer than the attainment by Professor Korenchevsky of his 60th birthday."[18]

Mellanby's reluctance to pay for the full grant seemed to be related to his doubts about Korenchevsky's ability as a scientist. In fact, Mellanby was not very different from his predecessor, Walter Fletcher, who wrote that while it was certain that Korenchevsky was "a competent and industrious worker," he "hardly [seemed] to be able to supply himself with intellectual direction."[19] In a similar sense, Mellanby wrote to Korenchevsky that he "never [seemed] inclined to tackle any particular problem in detail."[20] For instance, Mellanby knew that Korenchevsky, while finding that cortical extracts increased the longevity of adrenalectomized rats and improved their general conditions, observed that these extracts did not affect the size of the rats' sexual organs that had been enlarged after the surgery. According to Mellanby, however, Korenchevsky did not study what *chemical* substance in the cortical extracts caused these phenomena, even though Korenchevsky said that there were some "active principles" responsible for them.[21] Mellanby thought that the absence of chemical investigation revealed that Korenchevsky's research was not very thorough or rigorous.

But this criticism on Korenchevsky was not based on a proper understanding of his training and methodology. In fact, since 1920, Korenchevsky had been pursuing

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[16] Unnamed, 28 February 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[17] Unnamed, 28 February 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[18] Ledingham to Mellanby 15 March 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[19] Fletcher to Parkes, 13 February 1933, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[20] Mellanby to Korenchevsky, 10 July 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK. This letter was written in response to Korenchevsky's request of review.
[21] Mellanby to Korenchevsky, 10 July 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

experimental research on the effects of hormones and vitamins on the growth, function, and pathogenesis of the animal body. Using the rat as the primary experimental organism, he studied the dietary factors causing or preventing rachitic disease,[22] physiological functions of various sexual hormones,[23] and the combined effects of vitamins and hormones on the development and metabolism in castrated animals.[24] But Korenchevsky did not employ any chemical approaches in his research, since, as he wrote to Mellanby, he had been trained primarily as an experimental pathologist rather than as a biochemist. While he did want to use chemical methods to complement his main approach, and even asked the MRC to provide him with a biochemist for his lab, his hope was not realized, and thus he did not have any opportunities to analyze his rats using chemical methods.[25]

Yet it was during this research using more traditional scientific methods that Korenchevsky began to realize that age of the body was an important factor in physiological functions. From 1932 to 1933, he studied the differential responses of rats to testicular extracts according to their age, finding that adult rats showed smaller changes in the histology of the testes and the weight of other internal organs than younger rats.[26] He also examined the alterations in the weight of various organs with aging, classifying them into four groups in accordance with the changes of their actual weight and the "weight per unit of body weight."[27] During this research, age emerged as an important factor that needed to be considered and controlled in experiments. Korenchevsky also became interested in a highly controversial issue—rejuvenation through sexual hormone treatment. From 1934 to 1935, he studied whether aged rats

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[22] V. Korenchevsky: References, 15 August 1926, FD 1/671 (Korenchevsky's Research 1920-32), NAUK; Professor Korenchevsky's Report to the Medical Research Council, 1925, FD 1/671 (Korenchevsky's Research 1920-32), NAUK.

[23] Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1934-1935, pp. 4-6, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

[24] Dr. Korenchevsky's Proposed Scheme of Research, 13 July 1931, FD 1/671 (Korenchevsky's Research 1920-32), NAUK; Report of Dr. Korenchevsky, 13 October 1931, pp. 1-2, FD 1/671 (Korenchevsky's Research 1920-32), NAUK; Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1931-1932, pp. 1-8, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

[25] According to Korenchevsky, the MRC neglected Korenchevsky's request. See Korenchevsky to Mellanby, 13 July 1934, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

[26] Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1932-1933, p. 4, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

[27] Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1933-1934, pp. 1-2, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

injected with the testicular hormone showed any improvements in their symptoms of old age. In contrast to Charles Edouard Brown-Séquard (1817-1894), Serge Voronoff (1866-1951), and others, who argued that youth can be brought back through the manipulation of sex hormones such as injection of testis extract or testis-grafting, Korenchevsky showed that no such effects were observed in his experiments. Like many scholars in the 1930s who became more suspicious of gland transplantation and rejuvenation, he argued that the "so-called rejuvenating influence" of testicular hormones was not confirmed.[28]

While Korenchevsky had still considered age and aging a subsidiary research subject during these decades, he declared in 1938 that he would pursue research on senescence as his major project. In his letter to Mellanby, Korenchevsky wrote, "From the time when I started my scientific training and work with Prof. Pavloff and later in Paris with Prof. [Metchnikoff], my ultimate aim has always been to fit myself for an investigation of the causes of senility and this aim has been also present during my work at the Lister."[29] While it is not certain how much influence he received from Pavloff and Metchnikoff, and it is unclear whether he really had been trying to "fit [himself] for an investigation of the causes of senility" since his years in Russia, it was true that he had been studying aging at the Lister Institute.[30] He then continued to write that he planned to "organize the investigation of senility" which might take at least two years.[31]

Why, then, did Korenchevsky suddenly declare this substantial change in direction? First of all, we can examine the social context. As historians Pat Thane and Richard Soloway have written, British demographic investigations during the 1930s showed that the birth rate had fallen considerably, and that this would cause, along with

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[28] Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1934-1935, pp. 6-7, FD 1/672 (Korenchevsky's Research 1932-38), NAUK. In his later letter to Cannon, Korenchevsky wrote that gerontologists should be "very skeptical about the possibilities of "rejuvenation." See Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC. On the glandular transplantation of the 1930s, see David Hamilton, *The Monkey Gland Affair* (London: Chatto and Windus, 1986), pp. 120-142.

[29] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

[30] It is probably certain that Metchnikoff's research on aging influenced Korenchevsky. See Korenchevsky's mention of Metchnikoff's books in Vladimir Korenchevsky, "Some books and papers on senility," 28 August 1939, Box 41, Folder 12, EVC. Yet it is not clear whether Korenchevsky regarded Metchnikoff's arguments as a serious claim that needed further examination and extension, since it is not possible to see Metchnikoff's influence in Korenchevsky's ideas.

[31] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

increased longevity, a more aged and a less productive population.[32] It was even argued that the senile population would eventually bring about a national crisis or "race suicide" and impose a heavy burden upon younger generations who had to support aged citizens through pension payments.[33] Furthermore, the 1930s was the decade of the Depression during which almost everybody, including the elderly, had financial difficulties. The burden upon younger generation was thus expected to increase, and the elderly's hardship amid the Depression was becoming a serious social problem. Did his observation of these problems lead him to study aging systematically to do something about their solution? Although this is certainly possible, there is no record revealing that the above issues led Korenchevsky to choose senescence as his main research subject. Unlike the American gerontologist Edmund Vincent Cowdry's case which will be discussed in chapter five, there is no definite evidence that Korenchevsky's observation of the state of the British society during the Depression prompted him to study aging systematically and to organize researchers of aging. His writings composed at that time do not show any social concerns on his part.

On the other hand, we may also examine Korenchevsky's personal experience as a factor that prompted him to study senescence, as historian Thomas Söderqvist has done in his biography of the Danish immunologist Niels Jerne.[34] For instance, Cowdry and Geoffrey Bourne, who were Korenchevky's professional colleagues, wrote that Korenchevsky's interest in aging stemmed from his visit in 1906 to a Russian infirmary in Moscow that housed many aged patients.[35] There, it was argued, he was shocked by the severe pathological states of elderly patients in the Infirmary and began to think that scientific research could find the ways to help people become old without suffering from

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[32] Pat Thane, *Old Age in English History: Past Experiences, Present Issues* (Oxford: Oxford University Press, 2000), pp. 333-342; Richard Soloway, *Demography and Degeneration: Eugenics and the Declining Birthrate in Twentieth Century Britain* (Chapel Hill: University of North Carolina Press, 1990), pp. 226-258, esp. pp. 239-241.

[33] For example, see William Brian Reddaway, *The Economics of a Declining Population* (London: Allen & Unwin, 1939).

[34] Historian Thomas Söderqvist, through his biography of the Danish immunologist Niels Jerne, argued that scientists' imaginations and decisions related to their research can be influenced by their *personal* factors. See Thomas Söderqvist, *Science as Autobiography: The Troubled Life of Niels Jerne* (New Haven: Yale University Press, 2003).

[35] Bourne, "Preface," p. vi; Cowdry, "V. Korenchevsky," p. 1391.

such diseases. But this story, which was probably told by Korechevsky himself, does not have any documentary support. Even if this was true, it still cannot explain why Korenchevsky declared that aging should be studied as a major research subject only in 1938 rather than earlier.

Another personal factor can explain Korenchevsky's motives in a better way, although it may still not be possible to know his intention completely. A close reading of the above letter he wrote to Mellanby reveals that Korenchevsky decided to devote the remainder of his life to aging research when he was deeply worrying about his own aging and old age. He wrote to Mellanby,

> On 28th. January of this year I became 58 years old and according to the note received a few years ago, I shall be expected to retire in about two years time. In about one years time I should like to make official application to the Medical Research Council and the Lister Institute for extension of my personal grants for two extra years i.e. up to my age of 62.[36]

He then stated the specific reasons why he needed to retire at the age of sixty-two rather sixty, which was the age of his official retirement stipulated in the 1934 contract. First of all, he had not saved for his old age due to his semi-political activities for the Russian refugees for which he had spent a lot of money.[37] He also claimed that while he could finish his current endocrinology projects before his sixtieth birthday, he needed two more years to organize researchers of aging by creating professional societies. This statement was followed by his explanation of how he could continue his research work, although his age was approaching sixty. Korenchevsky claimed, "At my present age I am still physically strong and I am able to do a long days work without fatigue and frequently on into the late evening."[38] He also emphasized that "the quality of my recent published

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[36] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[37] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK. An earlier letter written in 1925 also says that Korenchevsky had a debt. See Unnamed to Martin, 2 June 1925, FD 1/671 (Korenchevsky's Research 1920-1932), NAUK.
[38] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.

work is better…than before" and his papers "seem to arouse more interest, the application for reprints is large and increasing and I am frequently consulted as one of the specialists on sexual hormones and organs, whose opinion might be considered of value."[39] Why did he care to emphasize his physical and mental vigor like this? Certainly, he was worrying about the possibility that Mellanby or other people would think that Korenchevsky should retire because he was too old. He was also concerned about the possibility that his aging would really influence his health and vigor and ultimately make him unfit for continued research activities.[40] He wanted to stress to them—and probably to himself as well—that his body and mind were far from being senile and the continued funding for him was thus worthwhile. Interestingly, what he wanted to do with this extended funding was research on aging and organizing scientists of senescence. He did not plan to pursue any other kinds of research. This plan, namely, a plan to study aging as his main research subject when he was actually worrying about his own old age, indicates that his scientific interest in senescence was closely associated with the state of his own old age and his desire to continue to work after sixty years old. For him, his own body and mind was a proof that mandatory retirement was not right, since chronological aging did not necessarily lead to physical or mental deterioration. In this sense, aging was an important subject that was related to both his personal concerns about his body and the professional activities that he wanted to carry out with it. Although it is difficult to make a strong causal connection between Korenchevsky's senescence and his aging research, the rest of this chapter will show that his struggles and concerns in his later years were closely intertwined with his project for promoting the science of senescence.

The relationship between Korenchevsky's study of aging and his own aging can be more clearly seen through a comparison of his case with that of American gerontologists. His aim was not the study of rejuvenation, as his previous conclusion on that issue had shown. Rather, it was basic research on the nature of senescence in general,

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[39] Korenchevsky to Mellanby, 2 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK.
[40] There is indeed a correspondence from H. A. Hoy, an elderly man in Texas, which was probably written as a reply to Korenchevsky's earlier letter. Hoy wrote about a special exercise that can help overcome a declining health in old age, about which Korenchevsky would certainly be concerned. See H. A. Hoy to Korenchevsky, 19 April 1949, ff. 450 Add. 50526B, GBS.

which might also produce knowledge on how humans' old age could be better and less tragic than before. Indeed, the same motives could be seen in the thoughts of American scientists of aging, which chapter five will discuss. However, there was a crucial difference between Korenchevsky and the gerontologists in the United States. He was personally suffering from the problems of aging—including ageism, financial insecurity, and retirement—while the same problems were conceived as social rather than personal problems by relatively younger and financially more secure American gerontologists such as Cowdry, McCay, and Shock. The meaning of the Depression was also different. The American scientists viewed the Depression as a problem in society which was particularly severe to the aged rather than to themselves, and this view was connected to their relatively stable posts in their institutions which enabled them to observe the chaos with a calm and analytic mind. In contrast, the economic crisis at that time was the very factor that initiated Korenchevsky's financial insecurity, especially when he was preparing for his old age and impending retirement.

In this state, Korenchevsky succeeded in extending his grants for two more years in 1939 and began his works for organizing researchers of aging. Although Mellanby wrote to Korenchevsky that "you would be wise to assume that your personal remuneration will cease after" his sixtieth birthday, the Lister Institute and the MRC eventually agreed to offer the grant for two more years.[41] By any standard, he was a very productive researcher who published more than fifty scientific articles after 1930.[42] Moreover, before beginning his organization activity in Europe and America, he also heard from Mellanby that his work would have Mellanby's "blessing as a private individual" while "not as Secretary of the Medical Research Council."[43] Korenchevsky wrote to Mellanby that it was "a great relief to know that you will consider the research

[41] Mellanby to Korenchevsky, 4 March 1938, FD 1/672 (Korenchevsky's Research 1932-38), NAUK; A. White to Mellanby, 20 June 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK.
[42] Medical Research Council, Private and Confidential: Applications for Renewal or Adjustment of Existing Grants, 5 July 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK.
[43] Mellanby, 17 May 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK.

on senility with sympathetic interest and would be willing to help the group with your good word as much as your official position will allow it."[44]

Korenchevsky was quite successful in his first summer trips to organize scientists interested in aging. During the spring of 1939, he already met and persuaded a number of British scientists—including G. Roy Cameron, F. A. E. Crew, and Francis Fraser—to join the "Informal Group on Research of Senility."[45] Later, the Group was able to recruit more prestigious scientists, such as the renowned chemist Robert Robinson who would be awarded the Nobel Prize in 1947. After finishing his work in Britain, Korenchevsky went to France and Switzerland during the early summer of 1939 and met with several biologists and physicians who responded favorably to his suggestion. There he recruited eight French and three Swiss members in his "International Club for Research on Senility."[46] Korenchevsky then went to the United States, meeting with the people who had scientific interests in senescence, especially the contributors to *Problems of Ageing* (1939) edited by Cowdry. Korenchevsky received highly enthusiastic responses from the American scientists of aging, including McCay, William deB. MacNider, Earl T. Engle, and Jean Oliver, who agreed to join the Club.[47] A letter written in December, 1939 shows that the Club changed its name into the "Club for Research on Ageing."[48]

As the organizer of the Club, Korenchevsky set its initial rules and aims. First of all, the Club's members should be limited to sixty, because "the Club is an informal and intimate group of research workers who are interested in the problem of senility and who

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[44] Korenchevsky to Mellanby, 19 May 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK.

[45] Vladimir Korenchevsky, "Members of the Informal Group on Research of Senility," undated, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK. Although this document is undated, it is the earliest document listing the members of the group that would become the Club for Research on Ageing, because it shows the smallest number of people among all the documents listing the current members of the Club. The members in this document were mentioned by Mellanby in his memorandum in May 17, 1939. See Mellanby, 17 May 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK.

[46] Korenchevsky to Unnamed, 27 May 1939, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK; "Members of the International Club on Research of Senility," undated, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK. While it is unclear whether Korenchevsky visited Denmark, in a later paper, he mentioned the Danish members as well. See "The Club on Research of Senility," undated, FD 1/673 (Korenchevsky's Research, 1939-1940), NAUK. While "undated," this paper was written after he organized the American Club.

[47] Korenchevsky to McCay, 11 July 1939, Box 6, Folder McCay, Clive [Comments listed], NWS; Jean Oliver to McCay, 12 July 1939, Box 6, Folder McCay, Clive [Comments listed], NWS; MacNider to McCay, 19 July 1939, Box 6, Folder McCay, Clive [Comments listed], NWS.

[48] Korenchevsky to Alfred Cohn, December 1939, Box 19, Folder 5, Record Group 450C661-U, AEC.

are willing to co-operate in research work for advancement of the problems."[49] To create this cooperative environment, he added a more specific rule—the nomination of new members should be unanimously approved by all the current members. This procedure would maintain the intimacy among the members and prevent "'difficult' colleagues" from entering the Club.[50] At the same time, he tried not to give an impression that the current members should spend a large amount of their time in studying aging, probably because he knew that senescence was not a major research subject at that time and that he should be more modest in his goal at the early stage of gerontology's development. He thus wrote, "Nobody who is invited to join the Club, is expected to devote his whole time and laboratory to research on senility….for this purpose part of the usual line of work of the member would be adjusted to the question of senility."[51] If a scientist, he continued, "is working on young and adults, he is expected also to include in his research some experiments on old animals or patients."[52] He also emphasized that the Club should be "very [skeptical] about the possibility of 'rejuvenation,'" which was highly controversial and was disproven by his and others' experiments.[53] Nevertheless, while rejuvenation was not feasible, extending the human's lifespan by minimizing premature or pathological aging was seen as possible. According to him, "the changes which at present accompany ageing" were "in most cases pathological and that the usual span of human life should be perhaps at least 100 years and not about 70-72."[54] Therefore, "the present development of science and medicine," he argued, would ultimately realize the human's natural lifespan by preventing the pathological changes of aging.[55] One of the aims of the Club should thus be to facilitate scientific progress toward this goal.

For Korenchevsky, the actual means of implementing this progress was a "friendly co-operation" among the members of the Club, although he did not yet

[49] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.
[50] Korenchevsky to Cowdry, 28 August 1939, Box 41, Folder 12, EVC.
[51] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.
[52] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.
[53] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.
[54] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.
[55] Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC.

explicitly advocate the ideal of multidisciplinary research.[56] On the issue of the Club's conferences and interaction among the members, he argued,

> It is desirable that at the general conferences, or by correspondence in the interval between them, members who are specialists in the same branch of Medicine or Science should endeavour to divide the various parts of the problem to be investigated between themselves; or to co-operate, if desired, in the same particular question. Similarly, specialists in different branches of Science or Medicine might do the same (so that, if possible and members are willing, a particular question might be attacked simultaneously by a clinician, biochemist, pathologist, experimentalist, statistician, etc.,).[57]

These statements, except the last sentence within the parentheses, did not necessarily argue for cross-disciplinary or multidisciplinary approaches. Korenchevsky wanted to emphasize that the members in the *same* field should cooperatively tackle their common problem, while he also said that it is possible for the scholars in different disciplines would investigate a subject in a collaborative manner.

By including a broad range of subjects in the Club's potential study topics, however, Korenchevsky opened the way for a multidisciplinary scientific research. Indeed, he argued that aging in various organs should be investigated, including that of the bone, cartilage, brain, pancreas, and spleen, as well as the effects upon senescence of various dietary factors, hormones, and environmental factors.[58] He also stressed that diverse scientific and medical approaches—including those in genetics, immunology, statistics, biochemistry, neurology, and clinical medicine—should be adopted in the study of senescence by the Club members.

The books and papers recommended by Korenchevsky for the "members who are making their first approach to the problem" of aging also showed that he was interested

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[56] Korenchevsky to Cowdry, 28 August 1939, Box 41, Folder 12, EVC.
[57] Korenchevsky to Cowdry, 28 August 1939, Box 41, Folder 12, EVC.
[58] Korenchevsky to Cowdry, 28 August 1939, Box 41, Folder 12, EVC.

in encouraging a broad range of scientific approaches. He included most books which made significant impacts on the researchers of senescence since the 1900s, such as Cowdry's *Problems of Ageing* (1939), Metchnikoff's *Old Age* (1904), Alexis Carrel's "The Problems of the Prolongation of Life" (1937), Ludwig Aschoff's "Zur normalen und pathologischen Anatomie des Greisenalters" (1937), C. M. Child's *Senescence and Rejuvenescence* (1915), and Raymond Pearl's *The Biology of Death* (1922).[59] Although Korenchevsky did not yet make an explicit statement, the Club was expected to be multidisciplinary, since the approaches and subjects dealt with in these works were very diverse and did not belong to a single discipline.

Yet Korenchevsky's scope of relevant disciplines did not include non-biomedical fields such as anthropology, botany, or the social sciences, and this rather limited scope contributed to settling the character of the science of aging in Britain.[60] As I will show in chapter five, these fields were included in the multidisciplinary field of gerontology in America, especially through the efforts of Cowdry who had the ideal of broad multidisciplinary cooperation and appreciated the societal relevance of scientific research. His expertise in textbook editing was a means of realizing this ideal. Korenchevsky, who did not have this ideal or expertise, contacted only biomedical scientists and physicians he knew well to organize the Club for Research on Ageing. This initial work of Korenchevsky indeed left a long-lasting mark on gerontology in his own country. In fact, biomedical scientists and social scientists made separate professional organizations in Britain. The British Society for Research on Ageing, which was created by Korenchevsky in 1947, was only for biological and medical researchers, while the British Society of Gerontology, which was established in 1971, was mostly for social scientists.

But Korenchevsky did have a serious interest in the social problems of aging, although he still did not think that social scientists could become his professional colleagues. In 1939, he wrote to Mellanby to ask whether "in some curious and unexpected way my organizing work on senility may find some connection with the

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[59] Vladimir Korenchevsky, "Some books and papers on senility," 28 August 1939, Box 41, Folder 12, EVC.

[60] "Psychology" itself was mentioned by Korenchevsky as a part in gerontology. But he meant clinical rather than experimental psychology. See Korenchevsky to Cowdry, Box 41, Folder 12, EVC.

present problems" concerning World War II.[61] Although no official defense-related position was offered to Korenchevsky, he wrote a long article, titled "The War and the Problem of Ageing" that advocated that gerontological research could assist the British military. According to him, those "who hold the highest and most responsible key-positions during the present war" were mostly aged people.[62] But since aging was found to have a lesser influence on mental faculties than physical strength, these aged people's accumulated wisdom and experience could be fully employed for successful war efforts. Nevertheless, it was true that the senile processes, especially when distorted by pathological changes, could substantially weaken both mental and physical capacities of these persons. Therefore, the efforts of the scientists of aging were important, because they could develop the means to alleviate the symptoms of aging and prevent premature senescence of the elderly, especially those who were principally in charge of conducting the warfare.

This idea stemmed from his preliminary study of sexual hormones and other scientists' works on the relation of vitamins to mammals' coat color and aging. These researchers discovered that vitamin deficiency could cause some symptoms of aging such as gray hair, and Korenchevsky himself found that sexual hormones were not in a balanced state in elderly men's body.[63] It was then necessary, he thought, to make a more systematic study of the effects of hormones and vitamins on human aging, especially if the War lasted longer than expected and "the reconstruction period after the

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[61] Korenchevsky to Mellanby, 4 September 1939, FD 1/673 (Korenchevsky's Research, 1939-40), NAUK.

[62] Vladimir Korenchevsky, The War and the Problems of Ageing, undated, p. 1, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK. The same article was published in two journals. See Vladimir Korenchevsky, "The War and the Problem of Aging," *Journal of the American Medical Association* 119 (1942), pp. 624-630; "The War and the Problem of Ageing," *Annals of Eugenics* 11 (1942), pp. 314-332.

[63] Report to the Medical Research Council on the Work Carried out by Dr. V. Korenchevsky during the Year 1934-1935, p. 8, FD 1/672 (Korenchevsky's Research 1932-38), NAUK. Also see, for example, Martin Yavorsky, Philip Almaden, and C. G. King, "The Vitamin C Content of Human Tissues," *Journal of Biological Chemistry* 106 (1934), pp. 525-529; Agnes Fay Morgan and Helen Davison Simms, "Adrenal Atrophy and Senescence Produced by a Vitamin Deficiency," *Science* 89 (1939), pp. 565-566; Agnes Fay Morgan, Bessie B. Cook, and Helen G. Davison, "Vitamin B2 Deficiencies as Affected by Dietary Carbohydrate," *Journal of Nutrition* 15 (1938), pp. 27-43. Also see Vladimir Korenchevsky to Unnamed, 10 April 1940, FD 1/673 (Korenchevsky's Research, 1939-40), NAUK; "The Scheme of the Clinical Trial of Vitamin B Complex on Human Seniles" undated, FD 1/673 (Korenchevsky's Research, 1939-40), NAUK.

war [would] be at least as long."[64] Since the works during these periods would "tax severely the mental and physical energy of all men in responsible positions," the scientific measures using the proper use of hormones and vitamins could prevent premature senility and promote health of the national and military leaders who tended to be aged.[65]

Fortunately, Korenchevsky was able to find money, collaborators, and the "clinical material" to put this idea into practice. In 1941, Korenchevsky and his colleagues conducted a preliminary study of the effects of vitamins B and C upon fifty-eight aged patients in Littlemore Hospital at Oxford. The result was modest, but it indicated that vitamins B and C could "prevent or improve, in some cases to a striking degree, certain of those senile features which could be considered as pathological, because they appear prematurely or in an extreme degree."[66] The next year, with a donation of £1,500 from the renowned British millionaire Lord Nuffield (1877-1963), Korenchevsky began a clinical research project on a much larger scale using 2,500 elderly patients in Tooting Bec Hospital at London.[67] It was the first official project of the British Club for Research on Ageing in which several members of the Club and other physicians and psychologists participated, including E. F. Scowen of St. Bartholomew's Hospital and William Stephenson in the Institute of Experimental Psychology at Oxford. Korenchevsky himself worked as secretary of the project and directed the physiological and psychological examination of the aged patients who were given various hormones and vitamins.[68] In 1943, with Korenchevsky's encouragements and the Josiah Macy Jr.

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[64] Korenchevsky to Ross Harrison, 27 July 1942, Box 82, Folder 1117, WBC.
[65] Korenchevsky to Ross Harrison, 27 July 1942, Box 82, Folder 1117, WBC. Also see Korenchevsky to Walter Cannon, 10 July 1941, Box 82, Folder 1112, WBC.
[66] W. Stephenson, C. Penton, and V. Korenchevsky, "Some Effects of Vitamins B and C on Senile Patients," *British Medical Journal* 2 (1941), pp. 839-844.
[67] Korenchevsky to Frank Fremont-Smith, 7 December 1942, Box 16, Folder 620, WDM. Also see "Korenchevsky" in "List of Donations 1926-52," Box 47, MLN. After Lord Nuffield donated the initial grant, the Nuffield Foundation supported the project. In 1945, the Foundation awarded £2,820 to the British Club for Research on Ageing for the clinical project at Tooting Bec Hospital. See Nuffield Foundation, *The Nuffield Foundation: Report on Grants Made during the Ten Years April 1943 to March 1953* (Oxford: Oxford University Press, 1954), p. 160.
[68] Report of Dr. V. Korenchevsky to the Medical Research Council for 1941-1942, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.

Foundation's funding, American gerontologists began a similar project using the elderly patients at Elgin State Hospital in Illinois.[69]

Korenchevsky also began more extensive experimental research on aging using rats, especially after he moved his laboratory to the University of Oxford in 1939.[70] In a room offered by zoologist E. S. Goodrich, he studied how sexual, thyroid, and adreno-cortical hormones along with vitamins affected senile processes in aging rats. He also measured the changing weights of various organs in developing rats, which showed that relative hypoplasia or atrophy of organs occurred at quite an early stage in their life, as early as two weeks after birth. Korenchevsky thought that this result showed that the senile process started at very early phases of life, supporting the American scientist C. S. Minot's argument that "the prime of life appears to be in early infancy and the process of ageing starts from this age."[71]

While the problem of aging as a scientific subject began to be rather successfully tackled in this way, Korenchevsky's own problem of aging was bringing a serious crisis to him and his works, especially as the year of his grant termination was approaching. Although the previous contract stipulated that he would retire in 1942 when he became sixty-two years old, he was not yet prepared to do so. First of all, his "research work on ageing will be interrupted and ruined" and the "successful results of [his] [organizing] activity in founding an international team….will be seriously handicapped" if he had to retire in 1942.[72] Since the whole project on aging was still at a fledgling stage, his sudden retirement would disrupt the professional organization that he was actively

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[69] Josiah Macy, Jr. Foundation, *Twentieth Anniversary Review*, p. 37; William MacNider to Walter Cannon, 30 November 1942, Box 16, Folder 618, WDM; MacNider to Vladimir Korenchevsky, 4 January 1943, Box 16, Folder 624, WDM; MacNider to Korenchevsky, 4 August 1942, Box 15, Folder 607, WDM; Korenchevsky to Ross Harrison, 27 July 1942, Box 82, Folder 1117, WBC.

[70] Korenchevsky to Mellanby, 15 September 1939, FD 1/673 (Korenchevsky's Research, 1939-40), NAUK. But Korenchevsky's lab was not a part of the University of Oxford. The funding for Korenchevsky's research still came from the Lister Institute. Including Korenchevsky's stipend of £400, the Institute paid £600-£700 per year for his scientific works. See Memorandum by the Director of the Lister Institute Relating to Dr. Korenchevsky's Application for Extension of His Grant and for a Pension, undated, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.

[71] Report of Dr. V. Korenchevsky to the Medical Research Council for 1941-1942, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK. Also see Charles S. Minot, *The Problem of Age, Growth, and Death: A Study of Cytomorphosis* (New York: Putnam, 1908), pp. 86-130, esp. pp. 116, 126.

[72] Korenchevsky to Mellanby, 29 March 1941, FD 1/674 (Korenchesvky's Research, 1941), NAUK.

constructing. But this was not the only reason. It was wartime, and if he had to retire amid the War, it would almost be impossible for him to "continue [his] research work elsewhere and to obtain a satisfactory position and salary."[73] He thus wrote to Mellanby, "In addition to my official enclosed letter to you, I would like to add that I shall be literally in the street, without money saved for my old age, if I have to retire during the war."[74] It was crucially necessary for the MRC and the Lister Institute to delay Korenchevsky's retirement and to continue his personal grant until he became sixty-five years old.[75]

Indeed, Korenchevsky was very desperate at that time, and he did everything he could to delay his retirement or to find a job elsewhere. For example, he forwarded Mellanby three letters delivered to Korenchevsky from overseas that showed that he was a very competent specialist in sex hormone research.[76] In particular, a letter from E. C. Hamblen, chief of the Endocrine Division at Duke Medical School, said that they needed a recent photograph of Korenchevsky that could be hung on the wall of his Division, because he was one of the major contributors to the development of endocrinology.[77] If Korenchesvky was such a great scientist, how, then, could the MRC and the Lister Institute lay him off simply because he was too old? In 1940, he also sent a letter to William MacNider, chairman of the American Club for Research on Ageing, to ask for a salaried position and assistants in any academic institution in the United States. However, this letter surprised MacNider and other members of the Club, although they had been very favorable to Korenchevsky and his works until that time. According to MacNider, Korenchevsky's letter contained "a pretty big order," which required a lot of effort on American gerontologists' part and had uncertain prospects, especially during the War.[78] While Korenchevsky later explained that he had wanted to come to America not to stay there permanently but to offer a temporary help to American gerontologists for their

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[73] Korenchevsky to Mellanby, 29 March 1941a, FD 1/674 (Korenchesvky's Research, 1941), NAUK. Korenchevsky wrote two letters on March 29, 1941.
[74] Korenchevsky to Mellanby, 29 March 1941b, FD 1/674 (Korenchesvky's Research, 1941), NAUK.
[75] Edward Mellanby, 28 April 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[76] Korenchevsky to Mellanby, 29 March 1941b, FD 1/674 (Korenchesvky's Research, 1941), NAUK.
[77] Hamblen to Korenchevsky, 5 April 1940, FD 1/674 (Korenchesvky's Research, 1941), NAUK.
[78] MacNider to Cannon, 23 August 1940, Box 82, Folder 1109, WBC.

cooperative research, it was certain that he was searching for a job in America under the impending threat of the grant termination in 1942.[79]

But Korenchevsky did not go to the United States, because the MRC and the Lister Institute eventually agreed to extend the term of his grant. He was allowed to stay with the grant of £800 per year from the two institutions until he became sixty-four years old, although this decision was not made easily. Indeed, Korenchevsky's request for a grant extension was very embarrassing for Mellanby and Ledingham. Mellanby wrote to Ledingham that "I gather that we are both in the same frame of mind and do not know what to do about this man."[80] Mellanby had "not the slightest doubt in [his] mind that, were there no war, Korenchevsky would have to go as far as the Medical Research Council [is] concerned."[81] Yet Ledingham and the Lister Institute were more sympathetic to Korenchevsky, and decided to extend the grant until "he is 64 (or perhaps 65) particularly as he continues to be active mentally and physically."[82] Although Mellanby did not "feel very happy about [Korenchevsky] and his work," the MRC also agreed to follow the Lister Institute in paying half of the grant until 1944.[83]

However, there was an important condition added to the extension of the grant, which frustrated Korenchevsky and aggravated the relationship between him and his patrons that was already highly strained. When Mellanby informed Korenchevsky that the grant was extended provided that that he should concentrate on experimental endocrinology, not the "clinical investigations on factors affecting senility in human beings," Korenchevsky claimed that the condition implied a serious misunderstanding of his position as secretary of the clinical project at Tooting Bec Hospital.[84] According to

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[79] There was no other reason for Korenchevsky to come to America than getting a job. Although he wrote that American gerontologists needed his help because "most American members of the Club do not realize or do not believe that the most important and promising feature of the Club is its aim to do research work in close and intimate cooperation," there is no evidence that the American Club was doing less well than its British counterpart in its cooperative approach. See Korenchevsky to Cannon, 8 January 1941, Box 82, Folder 1112, WBC.

[80] Mellanby to Ledingham, 17 April 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

[81] Mellanby to Ledingham, 17 April 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

[82] Ledingham to Mellanby, 3 July 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

[83] Mellanby to Ledingham, 7 July 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK; Mellanby to Ledingham, 24 July 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

[84] Mellanby to Korenchevsky, 31 July 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

him, he, as secretary, did not directly conduct the clinical research and thus his part on the project took "so little of [his] time, that…no damage could be done to [his] experimental work on animals."[85] But what was more disturbing to Korenchevsky was the MRC's attitude toward aging research itself that was reflected in the act of adding a special condition. He felt that such an attitude would threaten the survival of the field of aging and placed him "in a humiliating position and perhaps [limited] the freedom of [his] research work."[86] Hence, he asked Mellanby, who was primarily responsible for adding the condition, to meet with him in person to discuss the issue. While Mellanby never responded to this request, Korenchevsky came to suspect through his colleagues that L. J. Witts at the Radcliff Infirmary at Oxford was one of the people who made the decision. Korenchevsky thus wrote to Witts that he was deeply humiliated by the misinformed decision by the MRC and asked him not to "handicap our research on one of the biggest problems."[87] While Witts' reply was very brief, indicating that he did not "have any personal prejudice against" aging research and that Korenchevsky overestimated his influence on the MRC, Korenchevsky was still not satisfied with the reply and wrote back to him that he did not properly understand the problem.[88] What was really significant was the fact that Witts and others made the decision based on the wrong information about Korenchevsky's role in the project and thereby inflicted damage upon both the science of aging and his reputation as a researcher. Since Witts did not reply to this letter, Korenchevsky then turned to Mellanby and asked him to help him resolve the issue with Witts and the special condition attached to the grant. But what he received from Mellanby was a very short reply that he had "no desire to continue discussing with [him] the subject."[89] Deeply frustrated, Korenchevsky wrote,

> I had not the slightest desire to discuss the incident with Witts, because even to think about it disgusts me. The letters have been sent to you simply in order to

[85] Korenchevsky to Mellanby, 4 August 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[86] Korenchevsky to Mellanby, 4 August 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[87] Korenchevsky to Witts, 20 October 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[88] Witts to Korenchevsky, 21 October 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK; Korenchevsky to Witts, 27 October 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[89] Mellanby to Korenchevsky, 17 December 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.

> show that Russians, just like Englishmen, defend their honour, and the honour of their work. The symbolic animal of Russia is a peaceful bear which never attacks unless provoked, but when attacked fights against any odds.[90]

Certainly, this statement would not please many Englishmen, including those in the MRC.

The troubles between Korenchevsky and his patrons over the issue of gerontology continued. While he tried to convince the MRC and the Lister Institute of the importance of the science of aging and the necessity of long-term support for it, few were persuaded. Indeed, he reported to the MRC that he was very successful in organizing the International Club for Research on Ageing, and its American members were particularly highly active.[91] He also stated that the federal government of the United States had already begun to realize the significance of the problems of aging in relation to the War.[92] If Americans were supporting gerontological research, why then couldn't Britons do the same? However, the MRC did not show any interest in these issues. It did not accept any applications related to gerontology, including William Stephenson's application for a small grant for the clinical gerontology project initiated by Korenchevsky.[93] The MRC also disapproved of Korenchevsky's attempts to include more hospitals in his clinical research. For instance, when he asked the Royal Eastern Counties Institution to participate in his clinical project, the Institution contacted the MRC to know more about Korenchevsky and his projects.[94] Receiving this inquiry, A. Landsborough Thompson of the MRC replied that they did not sponsor gerontology and Korechevsky's research on aging was just his personal project.[95] Knowing that this reply prompted the Institution to turn down his proposal, Korenchevsky wrote again to Mellanby, "May I hope that you,

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[90] Korenchevsky to Mellanby, 21 December 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[91] Korenchevsky to Mellanby, 20 October 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK; Korenchevsky to Mellanby, 6 March 1942, FD 1/675 (Korenchevsky's Rsearch, 1942-53), NAUK.
[92] Korenchevsky to Mellanby, 9 March 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.
[93] "Application by W. Stephenson," 17 September 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK; Unnamed to Stephenson, 29 October 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[94] F. Douglas Turner to Landsborough Thompson, 22 December 1941, FD 1/674 (Korenchevsky's Research, 1941), NAUK.
[95] Thompson to Turner, 5 January 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.

dear Sir Edward, will help me, officially and unofficially, to do my duty, as I consider it, in the present war, or at least not to make it more difficult for me."[96]

Korenchevsky also asked for an interview with Mellanby and Ledingham to discuss his recent article, "the war and problems of ageing," and its relation to his gerontology projects. While Ledingham agreed to meet with him, Mellanby refused to do so, especially if he was going to talk about aging research.[97] After receiving Mellanby's response with great disappointment, Korenchevsky wrote to him,

> Therefore, if not to you, to whom have I to send it? And why write to me a letter which could have only been addressed to an inexperienced student? I am a mature scientist with a modest, but nevertheless international reputation in my field of research.[98]

To say nothing of rejecting Korenchevsky's continued request for supporting gerontology, Mellanby and Ledingham did not even want Korenchevsky to write in his published article on the war and aging that he was sponsored by the MRC and the Lister Institute.[99] Mellanby also said, "I regard his interest in the ageing problem, and especially in its clinical aspects, as a hobby."[100]

What, then, was the cause of all these problems? As I will show in the next section, scientific research on senescence, despite Korenchevsky's efforts, had a low priority in Britain during and after World War II, and Mellanby and Ledingham's negative attitude toward aging research reflected the general opinion among British

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[96] Korenchevsky to Mellanby, 27 February 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK. The Royal Eastern Counties Institution was already receiving a grant from the MRC. Since none of the MRC grants could be used for gerontology, the Institution could not participate in the project. See Turner to Thompson, 3 January 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK; Turner to Thompson, 6 January 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.

[97] Mellanby to Korenchevsky, 11 March 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.

[98] Korenchevsky to Mellanby, 21 March 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.

[99] Ledingham to Mellanby, 28 September 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK; Mellanby to Ledingham, 9 October 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK. Ultimately, Korenchevsky ended up in writing in his published article that he expressed "*his personal opinions only*, and not those of any official body or laboratory with which he is connected." See Korenchevsky, "The War and the Problem of Ageing," p. 314.

[100] Mellanby to Ledingham, 9 October 1942, FD 1/675 (Korenchevsky's Research, 1942-53), NAUK.

scientists and bureaucrats at that time. As I have shown in the above paragraphs, however, there was another factor which was at least as important as the British attitude on gerontology: Korenchevsky's difficult personality, combined with his old age, financial problems, and unstable status as a grant recipient, led him to offend many people around him, making the acceptance of his argument even more difficult. Indeed, although he was a senior scientist, he was financially insecure and was going to be more so due to his old age. Moreover, he did not have a stable position in the British academia and nevertheless had a great pride in himself and his scientific achievements which other people did not always respect. Perhaps we might think that Mellanby's conflicts with Korenchevsky were merely due to their problematic relationship regarding the MRC's attitude on aging research or Mellanby's intolerant character. But Korenchevsky's reputation among other scientists indicated that Mellanby was not the only person who had trouble with him.

For instance, Korenchevsky did not have a good relationship with the renowned British biochemist Henry Dale. In 1935, Korenchevsky took offense at Dale after receiving a letter from him which asked whether Korenchevsky is "inclined to attend the meeting (of the International Conference on Sex Hormones) as an 'Assessor.'"[101] For Korenchevsky, this letter was very insulting because it invited him only as an "assessor," which, according to him, was "of so little importance that it is left to [his] 'inclination' to attend."[102] Simply put, Dale made a mistake in ignoring the fact that Korenchevsky was a great endocrinologist in the field of sex hormone research and gave him such an insignificant position as "assessor" in the important international conference. While Dale, surprised by this angry response, wrote a long letter of explanation to Korenchevsky, he also said to Ledingham about the "temperamental freaks" of Korenchevsky, who "does not yet quite understand the ordinary forms of English correspondence."[103] Dale also recollected a meeting during which Korenchevsky had spent so much time in discussing

[101] Korenchevsky to Dale, 20 June 1935, HD/12/1/147, HHD. The words within the parentheses were written by Korenchevsky. The letter from Dale to Korenchevsky before June 20, 1935 was not found.
[102] Korenchevsky to Dale, 20 June 1935, HD/12/1/147, HHD.
[103] Dale to Ledingham, 24 June 1935, HD/12/1/150, HHD; Dale to Korenchevsky, 29 June 1935, HD/12/1/151, HHD.

his own research that other participants "had completely lost patience and gone away."[104] Reflecting these and other episodes in the past, Dale did not want to "involve [himself] in an endless argument with [Korenchevsky]" any more.[105] Some of the members of the American Club for Research on Ageing did not have a good feeling about Korenchevsky, either. For them, Korenchevsky was sometimes quite embarrassing with his inordinate request for money. For example, he asked Cowdry to pay $1,000 "to cover all [the] living expenses" for his prospective visit to the United States, while Cowdry's initial offer was only $200.[106] Although it might be true that the budgetary state of academic institutions in America was better than those in Britain right after the War, Korenchevsky, with his desperate financial condition, often overestimated their wealth and embarrassed many American gerontologists.[107] In a similar sense, Frederick Hisaw at Harvard University complained that Korenchevsky "is going to continue to be somewhat of an annoyance" due to his "paternalistic attitude" toward the American members.[108] Hisaw wrote to Cowdry, "If this were backed up by a very large research program on his part it would not be so bad, but as a matter of fact I fail to find anything in his publications that really indicate a spark of originality with regard to the problems of Aging."[109] In terms of both his character and scientific achievements, Korenchevsky was not considered an ideal leader of a new field.

In this state, Korenchevsky's clinical project at Tooting Bec Hospital could not proceed very well, especially when every material for research was scarce during the War. The problem occurred when Korenchevsky asked the Ministry of Food to provide the Club seventeen hundred grams of ascorbic acid for the continuation of the project. Upon receiving this request, the Ministry of Food asked the MRC whether it approved what

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[104] Dale to Ledingham, 24 June 1935, HD/12/1/150, HHD
[105] Dale to Ledingham, 24 June 1935, HD/12/1/150, HHD.
[106] Korenchevsky to Cowdry, 28 September 1948, Box 41, Folder 12, EVC.
[107] See Cowdry to Frank Fremont-Smith, 24 May 1946, Box 41, Folder 9, EVC; MacNider to Cowdry, 26 September 1947, Box 42, Folder 7, EVC; Cowdry to Korenchevsky, 12 October 1948, Box 41, Folder 12, EVC.
[108] Hisaw to Cowdry, 27 September 1947, Box 42, Folder 2, EVC.
[109] Hisaw to Cowdry, 27 September 1947, Box 42, Folder 2, EVC.

Korenchevsky was doing with such a large amount of a valuable dietary substance.[110] Mellanby wrote that he, as an MRC officer, had never endorsed the project and as a scientist had never thought that Korenchevsky's work was "likely to be particularly profitable."[111] For Mellanby, Korenchevsky was not following the MRC's condition of the grant by undertaking an unapproved project, and the scheme of the project was based on his inattention to the fact that such a huge amount of ascorbic acid would be merely excreted by the body.[112] When the Ministry refused Korencehvsky's request after receiving this answer, Korenchevsky wrote a long letter to justify why such a large amount of ascorbic acid was necessary for the project and why the project itself was valuable.[113] Although Korenchevsky did not get any response from the MRC, E. C. Dodds, a member of the Club for Research on Ageing and Professor of Biochemistry at the University of London, changed the situation. Dodds wrote to Mellanby that the project needed an overdose of ascorbic acid that was definitely over what the normal body could absorb because "a number of workers have demonstrated abnormalities in the distribution of ascorbic acid in old people" and there was "a fairly extensive bibliography on the subject" which he could send Mellanby upon request.[114] After receiving this letter from Dodds, Mellanby wrote again to the Ministry that he had "had the impression that this experiment was one of Korenchevsky's efforts, and did not know that it had any more reputable backing."[115] What moved Mellanby was not only an explanation of the scientific basis of the request but also the fact that the explanation was given by a distinguished British scientist, Dodds. While the full amount was not given, six hundred

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[110] Korenchevsky to E. M. Hugh-Jones, 28 February 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK; Jack Drummond to Mellanby, 7 March 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

[111] Mellanby to Drummond, 10 March 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

[112] "Letter sent to Professor Dodds and Sir Robert Robinson," 29 March 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

[113] Korenchevsky to Mellanby, 27 March 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

[114] Dodds to Mellanby, 5 April 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

[115] Mellanby to Drummond, 6 April 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-45), NAUK.

and eighty grams of ascorbic acid was offered to the Club for the completion of the project.

Korenchevsky and his reluctant patrons continued to be unhappy with each other until his retirement. Indeed, the MRC, which hoped to terminate its relationship with him on his sixty-fourth birthday, had to receive his application for an extension of his grant once again, this time until he became sixty-five years old.[116] He also asked the MRC and the Lister Institute to pay his pension after his retirement. But his patrons did not fully accept his request, and extended his grant only for three more months, terminating it in June, 1944.[117] Yet it was not possible to ignore Korenchevsky's deep concern about his financial insecurity.[118] Moreover, it was significant that he was not eligible for a government pension because he had not paid five percent of his income per year to the public pension fund.[119] Therefore, considering Korenchevsky's lifetime service, the MRC and the Lister Institute decided to offer £300 annually for his old age from their own budget. However, despite his patron's favorable decision on his pension issue, Korenchevsky retired with a deep regret and frustration due to his repeated failure to prompt the MRC and the Lister Institute to support the science of aging. He wrote to Mellanby on June 1944, "I am very sorry indeed and the future will show whether I was right or wrong."[120]

Korenchevsky's "future" was not coming soon. Although he continued his research after 1944 without actually retiring, only a small number of people in the country gave serious attentions to his arguments for the importance of gerontology. There were several reasons for this unsuccessful outcome of his efforts. While this section has pointed to Korenchevsky's difficult character and lack of leadership as a factor, the next

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[116] Korenchevsky to A. N. Drury, 26 May 1943, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.
[117] Landsborough-Thompson to Korenchevsky, 2 October 1943, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.
[118] Memorandum by the director of the Lister Institute relating to Dr. Korenchevsky's application for extension of his grant and for a pension," p. 2, undated, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.
[119] England had a long tradition of public pension for government employees. The 1925 amendment of the Old Age Pensions Act introduced a general contributory pension system that covered almost 79% of all British citizens. See Thane, *Old Age in English History*, pp. 308-332.
[120] "Extract from Prof. V. Korenchevsky's letter to Sir Edward Mellanby," 26 June 1944, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.

sections will illuminate two other causes. Initially, I will show that the number of Brtish scientists involved in the study of aging was quite small and that even these people had little interest in basic research on senescence. Medical care and investigation was their major field, and consequently, most of them identified themselves as geriatricians rather than as gerontologists. In fact, this issue was related to another factor that contributed to the difficulties of gerontology's early development in Britain—the rise of welfare state during the early twentieth century. The following sections will analyze how the rise of welfare system, which included the establishment of the National Health Service, became a factor for the unsuccessful outcomes of Korenchevsky's organizational works.

The Scientists of Aging in Britain, the BSRA, and Its Problems after World War II

While Korenchevsky could not hold the first scientific conference of the British Club for Research on Ageing during the War, the Club's inherent problems were already clear. There was no scientist who might become a leader of the new field of gerontology in terms of scientific achievements and professional recognition. Basically, few scholars except Korenchevsky were deeply involved in research on aging. Indeed, most members of the British Society for Research on Ageing—including the executive committee members of the Club, such as Dodds, Robinson, and Cameron—did not study senescence as their main research subject, although their work always had some relation to gerontology. For example, Cameron, Professor of Morbid Anatomy at University College Hospital Medical School, was primarily interested in arteriosclerosis rather than aging which could be regarded as its underlying cause. As far as the published records are concerned, his only work which was directly connected to senescence during the 1940s was his support of his fellows' and students' works on senescence at University College, including F. A. Denz's research on the age changes in lymph nodes.[121] Similarly, Dodds, while studying age-related symptoms such as menopause and carcinoma of the prostate that could be treated by his synthetic hormone "stilboestrol," was interested in senescence

[121] F. A. Denz, "Age Changes in Lymph Nodes," *Journal of Pathology and Bacteriology* 59 (1947), pp. 575-591.

in general only as a research subject of distant future.[122] The research topics of Robinson, who was basically a chemist rather than a biomedical scientist, were farther from gerontology. Indeed, he even said that he "entered the Club because of an interest in hormones but seem to have become ever more deeply involved in matters which….[he does] not understand."[123] Admittedly, these scholars could have been encouraged to study aging more deeply if there had been a strong leader of the field or those who could suggest a profitable research program. Yet, due to the several problems I have mentioned, Korenchevsky could not become such a leader, and Peter Medawar was too busy in pursuing other projects. Unfortunately, there were no other researchers who would form a core group of the fledgling field to incorporate gerontology into the current scientific establishment in Britain and to attract more investigators and research funds. Certainly, this state of the British Club was very different from its American counterpart, which included a number of enthusiastic researchers on aging such as Cowdry, McCay, Shock, MacNider, and Edward J. Stieglitz.

While the Club was transformed in 1947 into a more formal organization—the British Society for Research on Ageing (BSRA)—to facilitate its fundraising, legal, and international organization activities, the lack of strong leadership and enthusiastic researchers continued to hamper the growth and institutionalization of the science of aging in the United Kingdom.[124] Even after the establishment of the BSRA, Korenchevsky still thought that there were not many researchers who were seriously studying aging.[125] As chapter one has shown, most early researchers of senescence who brought about the changes in the ideas and research practices concerning aging did their work in the United States. Even British scientists of aging, such as Korenchevsky or Medawar, cited in their manuscripts or publications far more works by American scientists than those by British or European researchers. This indicates that the absolute

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[122] For a summary of Dodds' works, see Unnamed (probably Alexander Fleming), Untitled, ff. 159 Add. 56116, AF. Dodds' writings do not reveal that he was seriously interested in aging. As far as the records show, his sole explicit mention of aging appears in his 1950 lecture in which he said that it was a research topic of the distant future. See E. C. Dodds, "Bertram Louis Abrahams Lecture," 1950, MS 2030/1, ECD.
[123] "Extract from Sir Robert Robinson's letter to Sir Edward Mellanby," 28 March 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-1945), NAUK.
[124] Korenchevsky to Cowdry, 8 January 1947, Box 41, Folder 12, EVC.
[125] Vladimir Korenchevsky, "European Gerontologic Activities," *Journal of Gerontology* 4 (1949), p. 315.

number of scholars seriously interested in aging was small in Britain and that research traditions and professional networks could not be easily created in the country. Admittedly, it seems quite remarkable that the number of the BSRA members continuously increased, from twenty-eight in 1946 to forty-five in 1949, which included several distinguished British scientists such as Medawar, R. A. Fisher, and Julian Huxley.[126] But Fisher and Huxley were not active members as far as the records show, and Medawar, although having published an important paper on the evolutionary aspect of aging in 1946, did not pursue any further works in gerontology. (See Chapter 3.)

After the War, however, several relatively active researchers of aging joined the BSRA and constructed new research programs.[127] First of all, Frederick Charles Bartlett (1886-1969), Professor of Psychology at the University of Cambridge, initiated a research project on aging and work capacity by founding the Nuffield Unit for Research into Problems of Ageing, which was heavily funded, as its name indicates, by the Nuffield Foundation. With Lord Nuffield's donation, F. A. E. Crew at the University of Edinburgh also constructed the Edinburgh Gerontological Research Unit which focused on atherosclerosis in relation to growth and aging.[128] Moreover, several renowned physicians began to study clinical aspects of senescence, including Marjory W. Warren at West Middlesex Hospital, Trevor H. Howell at St. John's Hospital, and L. Z. Cosin at Orsett Hospital. William Hobson and his colleagues at the University of Sheffield also investigated clinical manifestations of senescence through hemoglobin levels and blood pressure in the aged people.[129] After the end of the war, Korenchevsky himself continued

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[126] "The Club for Research on Ageing," "List of Members," "British Branch of the Club," Box 5, Folder European and American Societies for Research, NWS; "List of Members," "British Commonwealth," "British Society for Research on Ageing," in British Society for Research on Ageing, *Societies for Research on Ageing* (Oxford, The British Society for Research on Ageing, 1949).

[127] For a summary account, see Vladimir Korenchevsky, "Gerontology in the United Kingdom," *Journal of Gerontology* 6 (1951), p. 275-287.

[128] "Edinburgh Gerontological Research Unit" in "List of Donations 1926-52," Box 47, MLN. Lord Nuffield donated £1,500 to the Edinburgh Unit in about 1949. For the Unit's work on atherosclerosis, see, for example, Erich Geiringer, "The Gerontological Aspects of Atheroma: An Approach to a Pathology of Senescence," *British Journal of Social Medicine* 2 (1948), pp. 132-138.

[129] See, for example, W. Hobson and E. K. Blackburn, "Haemoglobin Levels in a Group of Elderly Persons Living at Home Alone or with Spouse," *British Medical Journal* 1 (1953), pp. 647-649; Hugo Droller, John Pemberton, Cissie Roseman, and J. L. A. Grout, "High Blood Pressure in the Elderly," *British Medical Journal* 2 (1952), pp. 968-970.

his experimental study of the influence of hormones and vitamins upon senile changes in his small laboratory, which he called the Oxford Gerontological Research Unit.

But the names of these researchers did not necessarily mean that gerontology was growing rapidly in Great Britain. As I will show in the next sections, the Nuffield Unit at Cambridge was disbanded after the Nuffield funding was discontinued, and the Edinburgh Unit, which was much less productive than the one in Cambridge, relied only on Lord Nuffield's temporary gift.[130] The state of the Oxford Unit was not different, especially since the support from the MRC and the Lister Institute had ceased in 1944. After that, its funding relied only upon Lord Nuffield's unpredictable donation of £1,200 per year, which was hardly adequate for beginning any substantial research program.[131] Moreover, the condition of the lab was far from excellent. According to an observer, Korenchevsky's lab was located in a cellar, where "no reputable scientist would consent to work."[132] Admittedly, if the Unit had been an integral part of the University of Oxford, it could have expected the University's support. But the Unit never became incorporated into the University, and Korenchevsky remained a guest researcher rather than a regular faculty member at Oxford.

In this state, the BSRA could not maintain itself as an active organization, especially as compared with the Gerontological Society in the United States. Whereas many members of the Gerontological Society were employees of the National Institutes of Health or were receiving NIH extramural grants, few members of the BSRA were supported by the British government. Moreover, the BSRA did not have its own professional journal until 1972, while the *Journal of Gerontology*, the first official periodic publication of the Gerontological Society, was increasing the number of its

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[130] See "Edinburgh Gerontological Research Unit" in "List of Donations 1926-52," Box 47, MLN. While Crew did apply for a grant from the Nuffield Foundation, it was not successful. See the Nuffield Foundation, Minutes of the Twenty-first Meeting of the Trustees, 14 January 1948, (797), p. 60, NF.

[131] For Lord Nuffield's donation to the Oxford Unit, see "Oxford Gerontological Research Unit 49" in "List of Donations 1926-52," Box 47, MLN. Korenchevsky's funding applications to the Nuffield Foundation were often turned down. See the Nuffield Foundation, Minutes of the Seventh Meeting of the Trustees, 5 October 1944, (173), p. 28, NF. Yet the Nuffield Foundation, with Lord Nuffield's "special request," awarded about £2,400 for "the completion of Dr. V. Korenchevsky's work….in 1952-3." See Nuffield Foundation, *Report on Grants, 1943-1953*, p. 160.

[132] Douglas Veale, untitled, 17 May 1951, UR6/NF/2, UOA.

subscribers after being launched in 1946.[133] Although the BSRA's activities during the 1950s are not clearly known, an account of MRC secretary Harold Himsworth in 1962 indicates that the BSRA "was apparently flagging rather badly" during the past decades.[134]

There was another important difference between British and American gerontology after World War II. Whereas the Gerontological Society was highly multidisciplinary, including a number of non-medical scientists, there were no such scholars in the BSRA which was dominated by doctors and medical researchers.[135] As I have written in this chapter, Korenchevsky did not try to include social scientists in the BSRA and, unlike the Gerontological Society, no sociological approaches to the problems of aging were discussed during its regular meetings.[136] In fact, many of its members—such as Warren, Howell, Cosin, Hobson, Dodds, and Crew—were medical researchers and physicians who were deeply involved in clinical rather than experimental study of aging. In particular, some of them, especially Warren and Cosin, were basically geriatricians rather than gerontologists, and were interested in medical care of older people rather than in undertaking research using them. Perhaps psychologist Frederick Bartlett and his colleagues at the Nuffield Unit at Cambridge were the only active scientists of aging in the BSRA who were not affiliated with hospitals or medical research institutes. Korenchevsky himself, who also had a medical degree, wrote in 1949

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[133] The BSRA's official journal, *Mechanisms of Ageing and Development*, was launched only in 1972.

[134] Himsworth to James Mountford, 1 June 1962, FD 23/1826 (British Society for Research in Ageing and the Preparation for Retirement Committee, 1962), NAUK. Unfortunately, no records of the BSRA's activities during the 1950s can be found.

[135] As will be discussed in chapter five, the authors of Cowdry's *Problems of Ageing* (1939) included many non-medical scholars, such as Clive McCay (animal husbandry), Herbert Spencer Jennings (protozoology), Walter Miles (psychology), Clark Wissler (anthropology), Louis Dublin (statistics), William Crocker (botany), and A. J. Carlson (physiology). Cowdry was also trained in anatomy and cytology rather than medicine, although he was appointed in Washington University Medical School. Lawrence Frank of the Macy Foundation, who himself participated in the group of gerontology, was a social scientist. Many of these people became the founding member of the Gerontological Society.

[136] Vladimir Korenchevsky, "Ageing in Man and Other Animals," *Nature* 158 (1946), pp. 276-280; "Second Gerontologic Conference of the British Society for Research on Ageing," *Journal of Gerontology* 3 (1948), pp. 294-300.

that there were three gerontology units in the country while the number of geriatric centers was eight.[137]

Independently of Korenchevsky's efforts, old age was becoming an important medical subject in postwar Britain with the growth of geriatrics as a specialty. In his comparative study of the British and American ways of providing medical care for the elderly, David K. Carboni has concluded that geriatrics was more successfully institutionalized in the United Kingdom after World War II, especially under the sponsorship of governmental organizations such as the Department of Health and Social Security.[138] While health care was primarily controlled by physicians in America, Carboni has pointed out, the government regulated medical practice in Britain, and the establishment of the National Health Service in 1948 provided strong institutional leverage for doing so.[139] In this state, the medical personnel and resource in Britain could be distributed not so much by the interests and will of physicians—as was the case in the United States—as by the government which was more concerned about the actual needs of its citizens, including aged people. Fortunately for geriatricians, the expansion of the elderly population and the concomitant increase of chronic diseases enhanced such needs, which could be met by the large number of doctors who returned to peacetime practice and sought for consultant positions after the War. Despite several problems and difficulties, geriatrics in Britain thus began to be established as a legitimate specialty in hospitals, medical schools, and professional societies such as the Royal College of

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[137] Korenchevsky, "European Gerontologic Activities," p. 315.

[138] David K. Carboni, *Geriatric Medicine in the United States and Great Britain* (Westport, Conn.: Greenwood, 1982). It is also noticeable that an American observer in 1952 extensively discussed as British "gerontologic problems" their ways of medical care for the elderly rather than any scientific research activities. See Georgia France McCoy, "Gerontologic Problems in England as Seen by an American Observer," *Journal of Gerontology* 7 (1952), pp, 579-583. On the factors hindering the growth of American geriatrics, see Carole Haber, "Geriatrics: A Specialty in Search of Specialists," in David Van Tassel and Peter N. Stearns (eds.), *Old Age in a Bureaucratic Society: The Elderly, the Experts, and the State in American History* (Westport, Conn.: Greenwood, 1986), pp. 66-84; Charles E. Rosenberg, "The Aged in a Structured Social Context: Medicine as a Case Study," *Old Age in a Bureaucratic Society*, pp. 231-245.

[139] Sociologist Paul Starr has also pointed out that the American health care was dominated by the "professional sovereignty" of physicians, especially during the first half of the twentieth century. See Paul Starr, *The Social Transformation of American Medicine* (New York: Basic Books, 1982), pp. 3-29.

Physicians and the British Medical Association.[140] In 1947, British geriatricians themselves formed their own professional organization, the Medical Society for the Care of the Elderly, which changed its name to the British Geriatrics Society (BGS) in 1959.

The BSRA held an awkward position during these years, and this awkwardness was related to the question on its practical dimensions. Can basic biomedical research on senescence really benefit aged people? Americans' answer to this question was definitely affirmative, while Britons responded that at least some kinds of basic research, despite their ultimate importance, might not be very useful for fulfilling the immediate needs of the elderly. Interestingly, this British response was at least partially reflected in the fact that Britons established the National Health Service in 1948 when the United States federal government began to expand the National Institutes of Health.[141] Whereas Americans promoted more thorough biomedical investigation as a major way of enhancing the well-being of old as well as young Americans—especially when the national health insurance plans could not be introduced—Britons created government-sponsored medical insurance for all the citizens including the elderly for the same purpose, thereby helping geriatrics' growth and institutionalization.[142] Even if any biomedical research on aging was needed in Britain, it was still not clear whether the scientists involved in this research should be affiliated with the BSRA. If the BGS represented medical practitioners and researchers interested in aging, why, then, did they need to make another professional society whose member composition was very similar?

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[140] While better than the situation in America, the state and development of geriatrics in Britain after World War II should not be exaggerated. While being much more successful in Scotland, British geriatrics suffered from inadequate funding, building, supply, and personnel. See Pat Thane, "Old Age," in Roger Cooter and John Pickstone (eds.), *Medicine in the Twentieth Century* (Amsterdam: Harwood Academic Publishers, 2000), pp. 620-631; *Old Age in English History*, pp. 444-452.

[141] Charles Webster, "The Elderly and the Early National Health Service," in Margaret Pelling and Richard M. Smith (eds.), *Life, Death, and the Elderly: Historical Perspectives* (London and New York: Routledge, 1991), pp. 165-193. But Webster has argued that the NHS did not bring a transformation of health care for the elderly due to the lack of leadership, insufficient funding, and the inertia of traditional systems inherited from the Poor Law.

[142] On the relationship between the rise of medical research and the failure of introducing the national health insurance in America, see Strickland, *Politics, Science, and Dread Disease*, pp. 154-156, 213; Harden, *Inventing the NIH*, p. 182.

Of course, it is not true that the making of the National Health Service was unrelated to, or even prevented, the growth of medical research in general in Britain.[143] Yet the British government's support for more basic biomedical research was relatively less ample than the U. S. federal government sponsorship, and the major avenues for promoting the health of British citizens were sought in the national health insurance. In particular, the British priority was clear, in the case of aging issues. There was little substantial support for basic biomedical study of senescence by the British government.

In retrospect, this British way of responding to the problems of aging stems from the traditional systems of dealing with old age in the United Kingdom which did not exist on the other side of the Atlantic. Since the seventeenth century, England and Wales had had the Poor Law to take care of their aged, sick, poor, and disabled people. The high government officers also had their own systems of pension which gradually came to include other public employees in the lower strata.[144] Of course, the Poor Law, especially after the 1834 amendments, was often regarded as a harsh way of treating these people rather than any means of offering help, and the public employee pensions also had a number of consistent problems including abuse and corruption during their implementation. Yet the development of the new social systems during the first half of the twentieth century—including the modern non-contributory and contributory pensions introduced after the enactment of the Old Age Pensions Act in 1908 and its later amendments as well as the making of the National Health Service in 1948—provided more adequate and reliable means of achieving the welfare for the elderly.[145] Although the United States finally established the Social Security Act in 1935, Britain had a much longer and and more extensive tradition in the area of old age pension, which was already occupying a secure place as a means to help the elderly. The rise of geriatrics and the

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[143] For the role of the NHS for the development of clinical research in Britain, see Carsten Timmermann, "Clinical Research in Postwar Britain: The Role of the Medical Research Council," in Caroline Hannaway (ed.), *Biomedicine in the Twentieth Century: Practices, Policies, and Politics* (Amsterdam: IOS, 2008), pp. 231-254.

[144] See Thane, *Old Age in English History*, pp. 236-255.

[145] Whereas it has generally been known that the Poor Law, especially after the amendment of 1834, was harsh and authoritarian, David Thomson has argued that it was quite adequate for the livelihood of the elderly. See David Thomson, "The Welfare of the Elderly in the Past: A Family or Community Responsibility?" *Life, Death, and the Elderly*, pp. 200-205.

relatively poor state of gerontology in the United Kingdom could be understood in this historical context.

But Korenchevsky and others' hopes for gerontology did not lead to a complete failure during the postwar period. The Nuffield Foundation became a major supporter of the science of aging, and the Medical Research Council, despite its stubborn unwillingness to sponsor anything related to Korenchevsky, also began to support some gerontology programs after Mellanby retired in 1949. This weak yet continued tradition of gerontology, which still showed peculiar British characteristics, constituted a basis of its later growth. The next section will review the development of the science of aging from the late 1940s to the late 1950s through this institutional sponsorship.

Lord Nuffield, the Nuffield Foundation, and the "Care of Old People"

William Richard Morris, who later became the first Viscount Nuffield, was an English millionaire who founded the Morris Motor Company in 1910. With the huge amount of money he earned through his lucrative automobile business, he launched the Nuffield Foundation in 1943 for various reasons. First of all, he had in mind several philanthropic aims, which included the advancement of "health," "education," and "the care and comfort of the aged poor."[146] He believed that science and technology were making substantial progress at that time, and that the Foundation could function as an organization encouraging the use of new scientific knowledge for the benefit of humanity. Yet according to Ronald Clark, a historian of the Nuffield Foundation, there were other, more informal reasons for creating a foundation. One of them was the necessity of a legal body to take care of Nuffield's major share of the Morris Motors, because he had no children who could inherit his company's stocks in case of his unexpected death. By making the Foundation a major shareholder of the Morris Motors, Nuffield could be more prepared for the uncertain future of his wealth.[147] Nuffield's decision to launch a

[146] Nuffield Foundation, *The Nuffield Foundation: Review of the First Ten Years, 1943-53* (Oxford: Oxford University Press, 1953), p. 4.
[147] Ronald W. Clark, *A Biography of the Nuffield Foundation* (London: Longman, 1972), pp. 6-7. According to Clark, Nuffield was worrying about a possibility that his shares could not be sold after his death because he had no children who formally inherit them.

foundation also reflected his will to promote private initiatives and individualistic spirits when Britain was gradually becoming a welfare state. According to Clark, Nuffield, who did not like socialism or communism, wanted to contribute to the social betterment by private means when the government was searching for more organized and centralized measures for social welfare.[148] The Nuffield Foundation, through its support for science, health-related research, and the social work programs, could certainly become an important means to do so.

The Nuffield Foundation's "Care of Old People" program was formulated through such private initiatives. Clark has written that Nuffield's interest in aging stemmed from his various personal commitments and experiences, such as his "fear of loneliness and old age," the Victorian paternalistic attitude which influence him, and his observation of the re-employment of many elderly people during the War due to labor shortage.[149] For Nuffield, the elderly began to live differently amid the War, and the welfare measure for old people which would expand after the 1940s might change their life further. According to Clark, however, Nuffield thought that this movement toward the elderly's material comfort might neglect their deeper needs, especially their psychological and mental health, about which the Foundation could do something different.

Korenchevsky's influence upon Nuffield was also important in leading him to create the "Care of Old People" program. Although it may not be possible to estimate how strong Korenchevsky's influence was upon Nuffield's plans for his old age project, their contemporaries testified that Korenchevsky did play a significant role in prompting Nuffield to support research programs on senescence. For example, Douglas Veale, University Registrar at Oxford, as well as Mellanby at the MRC, stated that Korenchevsky persuaded Nuffield of the importance of aging as a scientific research subject.[150] Indeed, Nuffield had already financially sponsored the clinical project at

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[148] Clark, *A Biography of the Nuffield Foundation*, pp. 5, 8, 20.
[149] Clark, *A Biography of the Nuffield Foundation*, pp. 38-39.
[150] Douglas Veale, untitled, 17 May 1951, UR6/NF/2, UOA; Mellanby to C. R. Harington, 20 December 1943; Mellanby to Frederic Bartlett, 12 January 1945, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-1945), NAUK.

Tooting Bec Hospital Korenchevsky proposed in 1942. The new Foundation Nuffield created in 1943 could thus become a more stable means to support gerontology.

However, the Nuffield Foundation' standpoint on the issue of senescence reflected the current social consensus on the problems of aging in Britain, and was thus not the same as Korenchevsky's viewpoint. Basically, the trustees of the Foundation wanted to promote the "care of old people" rather than supporting gerontology. As noted above, Nuffield and the trustees he had chosen aimed at doing something which the welfare government was not currently implementing well, especially for the health and well-being of the aged. Admittedly, scientific research on aging could be considered one of such subjects ignored by the government, and thus it might be included in the Foundation's program on the "care of old people." However, gerontology was always marginal in the list of programs supported by the Foundation, just as the MRC treated aging only as a minor topic for research. If gerontology was ever included in the list, the Foundation preferred medical and practical investigations rather than more basic research projects. This policy was very different from that of the Josiah Macy Jr. Foundation, a major patron of gerontology in America. (See Chapter 6.) While the Macy Foundation supported gerontology as a part of its "Life Cycle" program which promoted multidisciplinary approach to birth, growth, and aging, the Nuffield Foundation was focused on less diversified and more practical aspects of senescence.[151]

Indeed, the first project the Foundation supported for the "care of old people" was a thorough survey of the state of the elderly in the country rather than any systematic funding program for gerontology. During the second meeting of the Foundation's trustees held on July 22nd, 1943, William Goodenough, a trustee and the chairman of the meeting, stated that "there is a serious and hampering lack of authoritative and comprehensive information with regard to the problems of old age."[152] He thus argued that "a survey which would provide a complete picture from the medical, social, and other aspects of what is being done at present, of the resources and facilities available, and of the

[151] Josiah Macy, Jr. Foundation, *The Josiah Macy, Jr. Foundation, 1930-1955: A Review of Activities* (New York: 1955), pp. 71-87.
[152] The Nuffield Foundation, Minutes of the Second Meeting of the Trustees, 22 July 1943, (48), p. 11, NF.

problems which need to be dealt with, would not only provide a sound basis for further action by the Foundation in this field, but that the results, if published, would be of great value to those who are concerning themselves with the problems of the aged."[153] With the trustees' general approval of this proposal, the Foundation appointed B. Seebohm Rowntree, a renowned social scientist, as chairman of the committee for conducting an extensive survey of various aspects of the elderly's life in the country, especially their income, housing, employment, mental state, and available health care resources. The result of the survey, which was published in 1947 as *Old People: Report of a Survey Committee on the Problems of Ageing and the Care of Old People*, was the first comprehensive study of the elderly population in Britain and was favorably received by the general readers as well as by professional scientists in the country.[154] Later, the Foundation sponsored two more survey projects, which focused on more specific issues or particular places—J. H. Sheldon's survey of the elderly in Wolverhampton (1948) and F. Le Gros Clark and Agnes Dunne's study of the aged workers in industry (1955).

For the Foundation, Rowntree's report became an important basis of its series of efforts to promote the welfare of the elderly, especially through the National Corporation for the Care of Old People (NCCOP) established in 1947. The NCCOP, based on the information in Rowntree's report, systematically supported "the provision of many…voluntary homes and welfare services for old people; and by experiment and demonstration, [influenced] the growing amount of official provision" for the elderly.[155] It provided a number of grants to voluntary homes, while establishing by itself several institutions for the aged and the poor.[156] The fund which the Foundation approved for the NCCOP took a major part of the total budget, which was approximately £4,200,000 during its first ten years since 1943.[157] Indeed, according to the Foundation's report, it

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[153] The Nuffield Foundation, Minutes of the Second Meeting of the Trustees, 22 July 1943, (48), p. 11, NF.
[154] Stephen Katz, *Disciplining Old Age: The Formation of Gerontological Knowledge* (Charlottesville, Virginia: University Press of Virginia, 1996), p. 114.
[155] Nuffield Foundation, *Review of the First Ten Years*, p. 39.
[156] Thane, *Old Age in English History*, pp. 393-394.
[157] Nuffield Foundation, *Review of the First Ten Years*, p. 10.

reserved about £1,000,000 for the operation of the NCCOP from 1947 to 1958.[158] The Nuffield also funded the National Old People's Welfare Committee, which, with the cooperation of various experts in medicine and social work as well as many local committees dispersed across the country, studied "the needs of old people and [encouraged and promoted] measures for their well-being."[159] At the same time, the Foundation created and sponsored several homes for the elderly, including the Elizabeth Nuffield Home at Oxford that received from the Foundation about £11,500 for five years after 1950.[160]

This series of efforts shows that the Foundation was highly interested in aging as a social problem and attempted to do something for the welfare of the elderly. Did the Foundation give an equal attention to the science of aging? The Nuffield Foundation trustees thought that it might be necessary to support gerontology as a science. However, they also thought that gerontology was "in a neglected and rather confused state," and "felt strongly that" any fellowships or grants toward research on aging

> should not be used to encourage people to go to special gerontological departments but might be made available to suitable persons wishing to research into the problems of ageing, more particularly from the angle of the chronological development of growth, and who could do so at some department dealing with the general field of which their particular problem is a part.[161]

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[158] Nuffield Foundation, *Review of the First Ten Years*, p. 39. Also see Clark, *A Biography of the Nuffield Foundation*, p. 148. The NCCOP already spent £820,000 from 1947 to 1953. See Nuffield Foundation, *Report on Grants, 1943-1953*, p. 156.

[159] D. Ramsey, "The National Old People's Welfare Committee of the United Kingdom," *Journal of Gerontology* 7 (1952), p. 500; The Nuffield Foundation, Minutes of the Seventeenth Meeting of the Trustees, 14 January 1947, (560), p. 44, NF.

[160] The Nuffield Foundation, Minutes of the Thirty-third Meeting of the Trustees, 20 June 1950, (II. 320), p. 20, NF; Minutes of the Forty-seventh Meeting of the Trustees, 26 February 1953, (II. 1054), p. 56, NF; Minutes of the Fifty-second Meeting of the Trustees, 10 December 1953, (II. 1420), p. 75, NF; Minutes of the Fifty-sixth Meeting of the Trustees, 15 October 1954, (III. 151), p. 40, NF

[161] The Nuffield Foundation, Minutes of the Eleventh Meeting of the Trustees, 24 July 1945, (306), p. 29, NF

A similar line of thought can be found in some NIH officers' statements when the Gerontology Study Section was disbanded in 1949. (See Chapter 6.) For both American and British patrons of science, gerontology could not be a single discipline that should be pursued by an independent university department.

In fact, the Nuffield trustees were less interested in the science of aging than NIH officers, especially when it came to basic research on senescence. Most notably, the amount of money the Nuffield Foundation spent for gerontology was much smaller than what was reserved for the welfare programs such as the NCCOP whose budget amounted to £1 million. The use of these small sums allotted to gerontology was also strictly limited for a set of specified purposes, as can be seen in the unsuccessful application by Korenchevsky.[162] Indeed, the fact that Korenchevsky's application in 1943 was declined even though it was accompanied by a strong letter of recommendation by Robert Robinson—another BSRA member and the Nobel laureate in 1947—reveals the Foundation's negative view of Korenchevsky's projects and perhaps basic research on aging in general.[163] Interestingly, the Foundation approved a proposal on the aging of common duckweed by Eric Ashby, a botanist at the University of Manchester.[164] Yet Ashby's proposal was classified by the Foundation in the category of "Biological Studies" rather than that of the "Care of Old People." This reveals the nature of the Foundation's program on the "Care of Old People," particularly in comparison with the broadly multidisciplinary viewpoint of American gerontologists and their patrons. The "Care of Old People" program had to deal with *humans*' problem of aging, especially its

[162] Crew's funding application for his Edinburgh Unit was rejected, too. See the Nuffield Foundation, Minutes of the Seventh Meeting of the Trustees, 5 October 1944, (173), p. 28, NF; Minutes of the Twenty-first Meeting of the Trustees, 14 January 1948, (797), p. 60, NF.

[163] See "Application for Support of Dr. Korenchevsky's Experimental Laboratory," Paper F. 4/9, in The Nuffield Foundation, Minutes of the Fourth Meeting of the Trustees, 7 December 1943, p. 60, NF. Clark has mentioned that the Foundation did not favorably evaluate Korenchevsky. See Clark, *A Biography of the Nuffield Foundation*, pp. 40-41.

[164] The Nuffield Foundation, Minutes of the Thirty-second Meeting of the Trustees, 19 April 1950, (II. 271), p. 7, NF; Minutes of the Forty-third Meeting of the Trustees, 19 March 1952, (II. 821), p. 86, NF. As far as the record is concerned, Eric Ashby was not a member of the BSRA at least till 1949. See "List of Members," "British Commonwealth," "British Society for Research on Ageing," in British Society for Research on Ageing, *Societies for Research on Ageing*.

medical and industrial aspects. If the program should ever support research projects on senescence, it had to be about practical aspects of aging related to health care and labor.

Indeed, during the mid-1940s to the mid-1950s, the Foundation supported several clinical studies of aging, which could be considered geriatrics, rather than gerontology, projects. The first clinical project the Foundation supported was the research on the influence of hormones and vitamins on the symptoms of aging conducted at Tooting Bec Hospital. After this project, the Foundation spent approximately £2,000 from 1946 to 1949 for the renowned geriatrician Trevor Howell's clinical investigation into chronic bronchitis using elderly patients at St. John's Hospital.[165] G. P. Xuereb in the Nuffield Department of Surgery at Oxford also received £3,680 from 1951 to 1953 for his study of senile changes of blood vessels,[166] and L. Z. Cosin, an eminent geriatrician and a founder the BGS, was awarded the grant of £7,500 for three years from 1953 for his research on mental deterioration of the aged patients.[167] The Nuffield Foundation also enabled Woodford Williams to study the senescence of the kidney, heart, and brain with a grant of £7,500 for five years from 1953 in Dr. O. Olbrich's Geriatrics Unit at Sunderland General Hospital.[168]

The research project on which the Foundation spent the largest amount of money was Frederic Bartlett's psychological study of the aging of work performance at the University of Cambridge.[169] From 1946 to 1955, the Foundation awarded £6,000 per year, in addition to £2,000 for the initial expenses for setting up the laboratory.[170] After

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[165] The Foundation's official record says that the total amount of the money Howell received was £2,300, while the Foundation's manuscripts states that it was £2,000. See The Nuffield Foundation, Minutes of the Thirteenth Meeting of the Trustees, 14 January 1946, (384), p. 55, NF; Minutes of the Seventeenth Meeting of the Trustees, 14 January 1947, (548), p. 41, NF; *Report on Grants, 1943-53*, p. 158.

[166] Nuffield Foundation, *Report on Grants, 1943-53*, p. 159; Minutes of the Forty-seventh Meeting of the Trustees, 26 February 1953, (II. 1055), p. 56, NF.

[167] The Nuffield Foundation, Minutes of the Fifty-second Meeting of the Trustees, 10 December 1953, (II. 1418), p. 75, NF; Minutes of the Fifty-sixth Meeting of the Trustees, 15 October 1954, (III. 150), p. 40, NF.

[168] The Nuffield Foundation, Minutes of the Fifty-second Meeting of the Trustees, 10 December 1953, (II. 1419), p. 75, NF; Unidentified to R. Dobbin, 2 June 1954, Box NF AGE1, Folder Sunderland General Hospital Geriatric Unit Dr. O. Olbrich, NF.

[169] The Department of Psychology at Cambridge had a strong experimental tradition and was funded by the Rockefeller Foundation and the MRC since the 1930s. See "Laboratory of Experimental Psychology, Cambridge, England," D.1, FB.

[170] The Nuffield Foundation, Minutes of the Eleventh Meeting of the Trustees, 24 July 1945, (305), p. 29, NF.

Bartlett retired in 1951, A. T. Welford, a lecturer in psychology, was appointed director of the Unit and kept investigating the subject until 1955. The total amount of the grant the Foundation awarded the Unit was £56,000 for the ten years.[171]

This long-term project explicitly aimed at investigating the practical problems in industry by studying the change in the capacity of perception and complex motions in accordance with age. According to Bartlett, the traditional experimental psychology had not adequately examined how humans's ability to perform a particular task required in a certain type of work in industry changed with aging, since "the function concerned [had] been studied in isolation."[172] Actual performances needed in a factory were combinations of many distinct types of motions, and the target of psychological research on aging had to be these coordinated motions rather than each isolated movement. Hence, "the crying need is for accurate methods of measuring the efficiency of co-ordinated functions" and their changes with aging.[173] These methods would reveal the actual degree of senile alteration in diverse kinds of performances required for a particular job. This research was necessary, because "retraining for new industrial and technical operations is likely to become of increasing" importance in "a highly mechanized age" which accompanied the "rapid development of scientific invention."[174]

The Nuffield Foundation evaluated Bartlett's proposal very favorably. To the Foundation, Bartlett's project had "probable applications to industrial and social problems, certainly directly concerned with age development."[175] The Foundation thus approved funding for the project, and Bartlett was able to construct the Nuffield Research Unit for Research into Problems of Ageing at Cambridge. The Unit hired total eighteen researchers at various levels of training from 1946 to 1955. The Foundation

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[171] Nuffield Foundation, *Report on Grants, 1943-53*, p. 160.
[172] Bartlett to A. S. Parkes, 23 November 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-1945), NAUK.
[173] Bartlett to A. S. Parkes, 23 November 1944, FD 1/676 (Survey of Problems of Ageing and Care of Old People 1943-1945), NAUK.
[174] "Care of the Aged: Scheme Submitted by Professor F. C. Bartlett," Paper F. 12/10, in The Nuffield Foundation, Minutes of the Twelfth Meeting of the Trustees, 26 November 1945, p. 124, NF.
[175] "Care of the Aged: Professor Bartlett's Proposed Scheme of Research Resolution Submitted by the Sub-Committee on Medical Research into Causes and Results of Ageing," Paper F. 9/9, in The Nuffield Foundation, Minutes of the Ninth Meeting of the Trustees, 6 March 1945, p. 75, NF.

recommended that "an experimental study should be made of medical and psychological aspects of employability with special reference to age groups in industry."[176]

In contrast to this affluent Unit at Cambridge, Korenchevsky's Oxford Unit did not fare very well. After his unsuccessful application in 1944, he turned to Nuffield himself again, who, quite fortunately for Korenchevsky, agreed to donate £1,200 per year from 1945 to 1950.[177] After the end of this period, through Nuffield's "special request," Korenchevsky's Unit could continue to receive the same amount of money from the Foundation from 1951 to 1952.[178] Indeed, while Nuffield seldom attended the trustees' meetings, he was still highly influential upon the decision making of the Foundation, and his "special items" could never be easily ignored by any trustees.[179] Unlike Nuffield, however, the trustees did not think that the Oxford Unit was doing a very meaningful job. According to an observer, his Unit had "just Korenchevsky, who has fire and drive, and two dull technicians who carried out dull thirty-year-old techniques, and a cage of rats."[180] Korenchevsky retired from the directorship of his impoverished Unit in 1953.

But Korenchevsky, with Nuffield's patronage, left a legacy of gerontological research in Great Britain after that. When he requested the Foundation's continued support for his Oxford Unit after 1953, the Foundation asked whether Oxford University would allow the Unit to continue. When the University replied that it could no longer accommodate the Unit because the Department of Physiology in which the Unit was housed was moving, the Foundation chose two alternative ways to fund gerontology.[181] The first was the funding of K. J. Franklin's laboratory at St. Bartholomew's Hospital to

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[176] "Care of the Aged: Professor Bartlett's Proposed Scheme of Research Resolution Submitted by the Sub-Committee on Medical Research into Causes and Results of Ageing," Paper F. 9/9, in The Nuffield Foundation, Minutes of the Ninth Meeting of the Trustees, 6 March 1945, p. 75, NF.

[177] "British Society for Research on Ageing: Oxford Gerontological Research Unit," Paper F. 37/1, in The Nuffield Foundation, Minutes of the Thirty-seventh Meeting of the Trustees, 24 January 1951, p. 178, NF. Actually, the Nuffield Foundation did not completely reject Korenchevsky's application. It did award £270 which could be spent by the Oxford Unit from July to December, 1944. See The Nuffield Foundation, Minutes of the Seventh Meeting of the Trustees, 5 October 1944, (173), p. 28, NF.

[178] The Nuffield Foundation, Minutes of the Thirty-third Meeting of the Trustees, 20 June 1950, (II. 321), p. 20, NF.

[179] But Nuffield's "special items" often brought about frustrations and complaints among the trustees. See Clark, *A Biography of the Nuffield Foundation*, p. 21.

[180] Clark, *A Biography of the Nuffield Foundation*, p. 41.

[181] The Nuffield Foundation, Minutes of the Thirty-eighth Meeting of the Trustees, 18 April 1951, (II. 541), p. 6, NF.

which Korenchevsky's research facilities were moved. Franklin declared that he would begin gerontological research following Korenchevsky's ideal, although he would not necessarily follow Korenchevsky's methodologies and research programs. The second yet more expensive way was the establishment of the Nuffield Gerontological Research Fellowship, whose initial budget was about £51,000.[182] This fellowship was to be awarded to a few promising scientists who would pursue extensive research on aging for an extended period of time. While the fellowship was "tenable at any University….(but preferably at Oxford)" considering Korenchevsky's legacy, it was eventually awarded to two scholars who had their own institutional homes—Peter Leslie Krohn, reader in endocrinology at the University of Birmingham as the senior fellow, and David Alan Hall, lecturer in biochemistry at the University of Leeds as the junior fellow.[183]

This series of grants and fellowships, along with Alexander Comfort's grants from the Foundation in 1951 (see Chapter 3), might suggest that gerontological research, especially its basic and experimental side, was getting adequate resources for its development as a mainstream scientific field. But the situation was still unfavorable to basic research on aging. In Franklin's case, the Nuffield grants of £15,000 from 1954 to 1960 enabled him to implement various experimental projects related to aging, such as the effects of brushing upon aging rats' masticatory mucosa, the kidney's differing responses to stress with senescence, and the impact of irradiation upon mice' aging and longevity.[184] But these projects did not lead to the institutionalization of gerontology at the University of London to which St. Bartholomew's Hospital was affiliated. Ironically, the department of radiobiology rather than gerontology was created at the University after the completion of these projects, probably because the last project using irradiation was

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[182] Nuffield Foundation, *Reports on Grants, 1943-53*, p. 162. But W. A. Sanderson, an assistant secretary of the Foundation, mentioned a slightly smaller amount. See W. A. Sanderson, "Nuffield Gerontological Research Fellowship," 13 July 1953, Box NF AGE 1, Folder Age 18 Nuffield Gerontological Research Fellowship, NF.

[183] "The Nuffield Foundation: Revised Draft Regulations for NUFFIELD GERONTOLOGICAL FELLOWSHIP," March 1952, UR6/NF/2, UOA; W. A. Sanderson, "Nuffield Gerontological Research Fellowship," 13 July 1953, Box NF AGE 1, Folder Age 18 Nuffield Gerontological Research Fellowship, NF.

[184] "Summary Report for Nuffield Grant"; Unidentified to Franklin, 25 June 1954; Unidentified to Dean, 2 April 1957; Unidentified to Dean, 30 January 1958, Box NF AGE1, Folder St. Bartholomew's Hospital Medical College Prof. K. J. Franklin, NF.

considered important.[185] The continued funding for gerontology ended up in making a department in a completely different field.

The results of the Nuffield Gerontological Research Fellowship did not meet the initial expectation, either. Krohn planned several interesting experimental projects in gerontology, such as research on immortality of cultured cells and the study of age-hybrids using tissue transplantation.[186] After these projects were pursued with the long-term Fellowship, however, the Foundation stated in 1966 that "rather like Alex Comfort, [Krohn] has come to be too much of an accepted authority in his own field and he finds that his scientific environment is not sufficiently challenging."[187] Although the situation surrounding this statement needs to be further studied, what it means seems to be clear enough. While a competent researcher, Krohn was isolated as a scientist of aging, since the professional community of gerontologists was still not very strongly established in Britain. In the same year, Hall also came to a similar dead-end after studying aging for more than ten years with the Foundation's sponsorship. Indeed, he became a senior lecturer in biochemistry in 1966, and planned to build a "Gerontological Institute" within the University of Leeds to promote more systematic study of aging. But his Nuffield Fellowship had already been terminated and the Foundation did not want to be involved in any further funding for initiating such an expensive project.[188]

In retrospect, such unhappy endings of several ambitious funding programs reveal the limitations of small private philanthropies like the Nuffield Foundation. The trustees of the Foundation decided that they should be regarded as "a pump primer rather than as an engine" and that they should not make grants for institution-building or chair-establishments.[189] Unlike the NIH in the United States, the Nuffield Foundation was a

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[185] Patricia J. Lindop to Leslie Farrer-Brown, 1 March 1960, Box NF AGE1, Folder St. Bartholomew's Hospital Medical College Prof. K. J. Franklin, NF.
[186] Krohn, "The Nuffield Foundation: Nuffield Gerontological Research Fellowship, Proposals for Research," pp. 3-6, Box NF AGE1, Folder Age 18 Nuffield Gerontological Research Fellowship, NF.
[187] "Professor P. L. Krohn," 11 October 1966, Box NF AGE1, Folder Age 18 Gerontological Fellowships, NF. Indeed, Krohn suddenly resigned from his position at the University of Birmingham in 1966.
[188] J. W. McAnuff, "Note on Discussion with Dr. D. A. Hall, at University of Leeds," 23 November 1966, Box NF AGE1, Folder Dr Hall Gerontological Research Fellowship Junior Status, NF.
[189] Clark, *A Biography of the Nuffield Foundation*, p. 20; The Nuffield Foundation, Minutes of the Ninth Meeting of the Trustees, 6 March 1945, (218), p. 4, NF.

private institution and thus could not make the grants for gerontology as a regular part of national scientific funding system. The next section will show that the British government and the MRC were not very different in this respect, since scientific research on aging had a low priority in the welfare-state.

The Medical Research Council, the British Government, and Aging as a Social Problem

In 1952, seven years after the end of World War II, the "National Advisory Committee on the Employment of Older Men and Women" was formed under the sponsorship of the Ministry of Labour and National Service. This committee's goal was to give professional advice on the employment problems of the elderly which had already become a significant issue during the War. As the proportion of elderly people in the population increased, their health and impact on national efficiency emerged as a serious social problem during the interwar period, and their reemployment amid the War due to labor shortage gave Britons an occasion to think about the elderly's proper role in the country again.[190] In this sense, the introductory paper of the first meeting stated that

> During the war and in the period of labour scarcity since, the Ministry of Labour and National Service has urged upon employers and workers through the National Joint Advisory Council the need for older people to continue at work as long as they are fit and willing. The report of the Royal Commission on Population presented to Parliament in June, 1949 (Omd. 7695) drew attention to this need and emphasized that the "arrangements"—employment conditions, social services, pension schemes, etc.—should be such as to facilitate and not impede continued work by older people. Since then various steps have been taken….and the Minister of Labour has now decided, in consultation with his colleagues in the Government concerned, that the time has come for a thorough investigation of the

[190] Thane, *Old Age in English History*, pp. 333-342; Soloway, *Demography and Degeneration*, pp. 226-258, esp. pp. 239-241; "National Advisory Committee on the Employment of Older Men and Women: Introductory Paper," p. 1, undated, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women—First Meeting, 1950-1952), NAUK.

problem in all its aspects and that a National Advisory Committee should be set up for the purpose.[191]

In the era of the welfare state in which the basic needs concerning health, employment, and retirement were supported by the government, the National Advisory Committee on the Employment of Older Men and Women was made "not to deny people the right of retirement when they are ready for it," but to prevent the cases of people's being "denied the opportunity [for employment] on account of their age alone."[192]

If the employer should not reject a person based on her or his age alone, what, then, were the other factors that could give meaningful information about an elderly person's employability and job fitness? Scientific and medical research was certainly considered to provide knowledge about these factors, and it was argued that "no policy to promote the employment of older men and women can hope to succeed if it is not founded on a proper appreciation of the effects of ageing on capacity to work and of the medical and social effects of ageing."[193] Hence, several scientific experts on aging were invited to join the Committee, including Bartlett, Welford, and Sheldon. During the meetings of the Committee, these researchers discussed the practical implication of their research, especially about better ways of adapting to changed work environments in old age. For instance, Welford argued for "the need for workers, in certain occupations where retirement was caused by reduced capacity, to consider a change of occupation in middle age….in order to enable them to defer retirement to a later age."[194] This argument was

[191] "National Advisory Committee on the Employment of Older Men and Women: Introductory Paper," p. 1, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women – First Meeting, 1950-1952), NAUK.
[192] Ministry of Labour and National Service: National Advisory Committee on the Employment of Older Men and Women: Minutes of the First Meeting, 2 April 1952, p. 2, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women – First Meeting, 1950-1952), NAUK.
[193] First Meeting of the National Advisory Committee on the Employment of Older Men and Women – 2nd April, 1952: Notes for the Minister's Address to the Committee, p. 2, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women – First Meeting, 1950-1952), NAUK.
[194] Ministry of Labour and National Service: National Advisory Committee on the Employment of Older Men and Women: Minutes of the Third Meeting, 16 October 1952, p. 3, LAB 8/2004 (National Advisory Committee on the Employment of Older Men and Women – Third Meeting, 1952), NAUK.

seriously considered by the other members of the Committee, and further discussions on the issue were followed.

Yet research on aging was hardly a major issue during the Committee meetings. In fact, the Committee did not include many scientific experts. Most members came from organizations related to the elderly's welfare, health, and labor—such as the NCCOP, the Ministry of Health, the Ministry of National Insurance, and the British Employers' Confederation. What they needed to do was to "review the various problems involved in promoting the employment of older men and women" rather than to "'work through' the problem and present [the Minister of Labour] with a final and comprehensive report."[195] More precisely, the Minister of Labour Peter Bennett emphasized, the Committee was expected to be "a standing body to which older workers' problems as they arise in the day-to-day work of the Employment Exchange and Appointments Office can be referred for advice and help in their solution."[196] Scientific knowledge could be useful in this kind of work, but it could not be of a major importance, since the Committee had to make a quick response to the actual daily problems related to old age. The Committee was disbanded in 1959 after establishing an interdepartmental committee on the employment of the elderly.[197]

In fact, research on aging had to be dealt with by another governmental organization, the Medical Research Council, if it was dealt with at all. After being established in 1911 with the passage of the National Insurance Act, the MRC aimed at supporting medical research related to the health of British citizens as well as more basic scientific investigations. It assisted nutrition studies, tropical medicine, and public health, while at the same time sponsoring experimental projects in physiology, genetics, and

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[195] First Meeting of the National Advisory Committee on the Employment of Older Men and Women – 2nd April, 1952: Notes for the Minister's Address to the Committee, p. 1, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women – First Meeting, 1950-1952), NAUK.

[196] First Meeting of the National Advisory Committee on the Employment of Older Men and Women – 2nd April, 1952: Notes for the Minister's Address to the Committee, p. 1, LAB 8/2002 (National Advisory Committee on the Employment of Older Men and Women – First Meeting, 1950-1952), NAUK.

[197] See the papers in LAB8/2492 (Employment of Older Men and Women: Winding Up of the National Advisory Committee on the Employment of Older Men and Women, Nov 1958; Setting Up of a Small Inter-Departmental Committee to Co-ordinate the Work of Departments in Identifying Problems Connected with the Employment of Older Workers, 1958-1961), NAUK.

immunology.[198] It was then quite natural to hope that the MRC would be interested in supporting the science of aging, which was anticipated to deal with several significant social issues concerning pension, health insurance, and retirement.

But the MRC did not give much attention to gerontology during the 1940s when Edward Mellanby was its secretary. As I have shown in the earlier part of this chapter, Korenchevsky's reputation and his unhappy relationship with Mellanby effectively hindered the MRC from supporting any gerontology-related projects during his tenure. The only project the MRC paid for during the 1940s was a social survey on the "Employment of Older Persons" which examined the current state of the elderly's employment, housing, and pension. This project, implemented by the Wartime Social Survey with the MRC funding, investigated the elderly people in twelve thousand randomly chosen households in England and Wales.[199] But even this project was authorized very reluctantly, because it was originally suggested by the Nuffield Foundation whose program for old age was influenced by the person the MRC did not like, Korenchevsky.[200] In any case, this survey, which was similar to many statistical investigations commissioned by the government since the nineteenth century, had to be only an introductory act for more comprehensive programs of the future.[201]

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[198] Joan Austoker and Linda Bryder (eds.), *Historical Perspectives on the Role of the MRC: Essays in the History of the Medical Research Council of the United Kingdom and Its Predecessor, the Medical Research Committee, 1913-1953* (Oxford: Oxford University Press, 1989).

[199] See "The Employment of Older Persons," undated, FD 1/677 (Survey of Problems of Ageing and Care of Old People, 1946-1953), NAUK. This survey resulted in a publication. See Geoffrey Thomas, *The Employment of Older Persons: An Inquiry Carried Out in Mid 1945 for the Industrial Health Research Board of the Medical Research Council* (London: Central Office of Information, 1947).

[200] For Mellanby, it was quite embarrassing that Korenchevsky prompted Nuffield to support gerontology and the MRC was sponsoring a project that originated from the Nuffield Foundation. Moreover, it was possible that the Foundation could use the MRC for its own private initiative. While the precise course of events is not known, the MRC's survey on the elderly was initiated after the Foundation asked the MRC to assist its own survey project. See Mellanby to Bartlett, 12 January 1945, FD 1/676 (Survey of Problems of Ageing and Care of Old People, 1943-1945), NAUK; "The Employment of Older Persons," undated, FD 1/677 (Survey of Problems of Ageing and Care of Old People, 1946-1953), NAUK.

[201] On the British health statistics and subsequent public health reform measures, see John Eyler, "Health Statistics in Historical Perspective," in Daniel J. Friedman, Edward L. Hunter, and R. Gibson Parrish II (eds.), *Health Statistics: Shaping Policy and Practice to Improve the Population's Health* (Oxford: Oxford University Press, 2005), pp. 29-38. The MRC also conducted several survey projects. See, for example, Celia Petty, "Primary Research and Public Health: The Prioritization of Nutrition Research in Inter-war Britain," in *Historical Perspectives on the Role of the MRC*, p. 89.

Yet the MRC, unlike the Nuffield Foundation, did not support many such programs after finishing its survey.[202] To the MRC, the biomedical aspect of aging was distinct from its social aspect, and "whatever may be said of the medical aspect," the "practical issue" concerning aging "is largely a social one, and there is no reason why action should be made entirely contingent upon further research."[203] For the MRC, this was true because "existing knowledge is capable of application on a suitable scale by purely social measures."[204] Therefore, from this standpoint, it was not wise to begin a new scientific project on aging. Even if any project had to be sponsored by the MRC, it should be something which had strong practical utility that had been confirmed by its previous patrons. In fact, even these projects chosen by the MRC for support suffered from many troubles that hampered their continuous progress.

One of these projects, probably the only one during the 1950s, was the psychological research on work capacity of aged employees which the Nuffield Foundation had funded from 1946 to 1955. When the term of this Nuffield grant was about to be terminated, Bartlett enquired whether the MRC could continue funding the project. However, while the MRC's response to this inquiry was quite positive, it was not possible for the research facilities and staff members to stay within the Cambridge campus, because psychology professor O. L. Zangwill opposed to the Unit's contunued existence. According to Zangwill, the Nuffield Unit was not suitable for Cambridge in many aspects—its director would not be one of his staff members and the quality of its scientific research was not very good.[205] In particular, no longitudinal study had been done which Zangwill considered important, and it was better for the Unit to be located in a major industrial city than a college town like Cambridge. But the Unit's departure from Cambridge was not the best option, either. A. T. Welford, director of the Unit, had a

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[202] See Joan Faulkner to Korenchevsky, 2 Novembere 1950, FD 7/1434 (Nuffield Research Group on Ageing 1953-1955), NAUK.
[203] "Research on Ageing: Employment of Older Persons," 9 November 1948, FD 7/1434 (Nuffield Research Group on Ageing 1953-1955), NAUK.
[204] "Research on Ageing: Employment of Older Persons," 9 November 1948, FD 7/1434 (Nuffield Research Group on Ageing 1953-1955), NAUK.
[205] "Nuffield Foundation Research Unit into the Problems of Ageing: Note of Interview with Dr. Mackworth on the 29th September, 1954," FD 7/1434 (Nuffield Research Group on Ageing 1953-1955), NAUK.

permanent position at Cambridge, and other senior staff members would not be satisfied with the financial insecurity of the newly constituted Unit whose funding was contingent solely upon the MRC grant.[206] Therefore, the only possible option was to make a completely new research program in a different university with totally different staff members. This resulted in closing down the Nuffield Unit at Cambridge and losing its accumulated research experience and tradition.

The new unit created in 1955 in the Department of Psychology at the University of Liverpool had similar problems throughout the 1960s. According to a report, the Unit's laboratory was "hopelessly cramped," since adequate accommodation was not provided by the University.[207] Moreover, Alastair Heron, director of the Unit, was thought to have an "over-ambitious drive" which set "fairly severe limits on any collaborative projects" in the campus.[208] Meanwhile, the MRC's staff members were thinking that the Unit's "work was not, from its nature, likely to be exciting" and that it might be better for the Unit to be disbanded or moved to another school.[209] But the MRC decided to continue the funding for the Liverpool Unit because it was still interested in the Unit's research and its practical prospects.

Here, the persistent problems of British gerontology could be seen again: There were always some conflicts between gerontologists and other scholars in the same institution who did not share a favorable outlook on aging research. Moreover, neither academic institutions nor their patrons provided adequate funding for the science of aging, because it was not considered a critical field that needed immediate and abundant support. While aging might be an important social issue, it was hardly regarded as a significant

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[206] Welford to F. H. K. Green, 21 April 1955, FD 7/1434 (Nuffield Research Group on Ageing 1953-1955), NAUK.

[207] L. S. Hearnshaw, "Accommodation for the Medical Research Council Unit for Research on Occupational Aspects of Ageing," 15 January 1962, FD 12/53 (Future of the Unit and Consideration of Possible Relocation, 1961-1963), NAUK.

[208] Unnamed to Himsworth, 2 November 1962, FD 12/53 (Future of the Unit and Consideration of Possible Relocation, 1961-1963), NAUK. This reputation could be related to Heron's argument for multidisciplinary research, which he thought, was necessary for the adequate operation of the Unit and should be supported by faculty members of the University from many distinct fields. See Alastair Heron, "Unit for Research on Occupational Aspects of Ageing," 12 February 1962, FD 12/53 (Future of the Unit and Consideration of Possible Relocation, 1961-1963), NAUK.

[209] "Future of the Unit for Research on the Occupational Aspects of Ageing," p. 1, FD 12/53 (Future of the Unit and Consideration of Possible Relocation, 1961-1963), NAUK.

research subject that demanded systematic funding from either the governmental agencies or private philanthropies. Indeed, even the few fields that did succeed in obtaining support amid this general neglect—the projects related to geriatric care and worker's adaptation— often failed to obtain sufficient and stable long-term support.

The research on osteoporosis and its relation to aging did not have adequate support from the MRC, either. When Harriet Chick, a Cambridge medical researcher, proposed a project on the "frequency of fractures, especially of the femur, in old people" in relation to nutritional deficiency, Harold Himsworth, who was appointed secretary of the MRC in 1949, responded that it might be better to "call a conference, predominantly composed of people who knew something about this condition."[210] For Himsworth, there was no direct evidence that nutritional deficiency was related to osteoporosis in old age, and it was thus necessary to have a meeting of experts in relevant fields to see what could be done about this problem.[211] Acting upon this idea, the MRC invited more than twenty biomedical experts to the two meetings held in London in 1956 and 1957. During the 1957 meeting, it was decided that two working groups should be formed—the first was a survey group studying "the incidence and sites of fractures in elderly people" in relation to their age, and the second was a group of scholars who regularly gathered to exchange information on their recent research on pathology and morphology of aging bones.[212] While the MRC paid for the first group's survey project, the second, which was considered an informal gathering, did not get any support and remained as a voluntary discussion group. Admittedly, three medical projects connected to osteoporosis were eventually chosen for funding by the MRC in the early 1960s as a consequence of these working groups' efforts.[213] But the total amounts of their grants were extremely small

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[210] Chick to Rudolph Peters, 21 January 1955; Chick to Himsworth, 12 December 1955; Himsworth to H. J. Seddon, 22 December 1955, FD 23/1812 (Fractures in the Elderly, 1st Meeting, 1955-1956), NAUK.
[211] Himsworth to A. M. Cooke, 15 June 1955; Himsworth to Cooke, 14 February 1956, FD 23/1812 (Fractures in the Elderly, 1st Meeting, 1955-1956), NAUK.
[212] Conference on the Problem of Fractures in Elderly People: Minutes of Second Meeting, 21 January 1957, FD 23/1813 (Fractures in the Elderly, 2nd Meeting, 1956-1957), NAUK.
[213] B. E. C. Nordin at the University of Glasgow was awarded £4,155 for his project on pathogenesis of osteoporosis and vitamin D's role in it. J. V. G. A. Durnin at the same university also got £3,735 for his research on food intake and energy expenditure of young and old people. C. E. Dent at University College Hospital Medical School received £1,200 for his study of plasma calcium and metabolism. See "Calcium

and, as far as the records were concerned, no substantial research program on osteoporosis and aging emerged thereafter.

Conclusion

This chapter has discussed the effort to create a research agenda and support for the emerging field of gerontology in the United Kingdom. While there was increasing support and a growing number of researchers interested in this area, these were limited by at least three factors. First, Korenchevsky, who created the field and the professional society in the country, was not positioned to provide strong leadership. This was due to several interrelated personal factors, including his difficult character, low academic status, old age, and financial insecurity. If Korenchevsky's own aging and money problems led him to study senescence as a scientific subject, these very factors also served to hamper the growth of the science of aging in the country. Second, there were not many researchers seriously interested in aging. There was no strong research tradition on senescence in Britain, and consequently, few scientists could become the early organizers of the field. Third, since the early twentieth century, Britain was already becoming a welfare state which took care of its elderly citizens through various policies on employment, pension, and free medical care. In this state, gerontology could not have a high priority in either the government's or private philanthropies' funding, especially if compared with geriatrics which fared much better in the welfare state of Britain.

The next chapter will discuss the situation in the United States which was far more favorable to the development of gerontology. Unlike those in the United Kingdom, the federal government and several philanthropies in America were deeply interested in aging as a scientific subject. Moreover, there were many scientists of aging who, unlike Korenchevsky, had a solid position and status in the academia. Among them, the next chapter will discuss the life of cytologist Edmund Vincent Cowdry and his principal role in the birth of gerontology in America.

Metabolism and Osteoporosis," undated, FD 23/1825 (Working Group on a Survey of Fractures in the Elderly Correspondence Concerning Future Meetings, 1961-1964), NAUK.

Chapter 5

"Many Aged and Dead Cells Are Not Consigned to Oblivion" Constructing Gerontology as a Multidisciplinary Scientific Field in the United States

While Vladimir Korenchevsky's efforts to establish gerontology in Great Britain did not easily lead to a satisfactory result, the Canadian-American cytologist Edmund Vincent Cowdry was more successful in establishing gerontology as a scientific field in the United States.[1] With the support of the Josiah Macy Jr. Foundation, he edited *Arteriosclerosis: A Survey of the Problem* (1933), in which he and other eminent biomedical scientists presented their current understanding of and approach to the disease that afflicted many aged people. After publishing this book, he chaired the first academic conference on aging in 1937 at Woods Hole, Massachusetts under the Foundation's sponsorship. The scientists who gathered there contributed to the publication of *Problems of Ageing: Biological and Medical Aspects* (1939), which contained a comprehensive

[1] A shorter version of this chapter was published as Hyung Wook Park, "Edmund Vincent Cowdry and the Making of Gerontology as a Multidisciplinary Scientific Field in the United States." *Journal of the History of Biology* 41 (2008), pp. 409-587. With kind permission of Springer Science+Business Media, this article has been included in the current dissertation. For other literature on Cowdry, see W. Andrew Achenbaum, *Crossing Frontiers: Gerontology Emerges as a Science* (Cambridge: Cambridge University Press, 1995), pp. 52-89; Stephen Katz, *Disciplining Old Age: The Formation of Gerontological Knowledge* (Charlottesville: University Press of Virginia, 1996), pp. 93-103; Hannah Landecker, "Edmund Vincent Cowdry," in Noretta Koertge (ed.), *The New Dictionary of Scientific Biography* vol. 2. (Farmington Hills, Mich.: Gale, 2007); "Cowdry, Edmund Vincent," *The National Cyclopedia of American Biography* 61 (1982), pp. 145-146; Joseph T. Freeman, "Edmund Vincent Cowdry, Creative Gerontologist: Memoir and Autobiographical Notes," *The Gerontologist* 24 (1984), pp. 641-645; Jane Maienschein, "Cytology in 1924: Expansion and Collaboration," in Keith Benson, Jane Maienschein, and Ronald Rainger (eds.), *The Expansion of American Biology*, (New Brunswick: Rutgers University Press, 1991), pp. 23-51; Richard B. Calhoun, *In Search of the New Old: Redefining Old Age in America, 1945-1970* (New York: Elsevier, 1978), pp. 68-69; James E. Birren, "A Brief History of the Psychology of Aging," in Gerald J. Gruman (ed.), *Roots of Modern Gerontology and Geriatrics* (New York, Arno, 1979), pp. 75-76; "Edmund Vincent Cowdry," in W. Andrew Achenbaum and Daniel M. Albert (eds.), *Profiles in Gerontology: A Biographical Dictionary* (Westport, Conn.: Greenwood, 1995), pp. 90-91. Also see the obituaries, Albert I. Lansing, "Edmund Vincent Cowdry, 1888-1975," *The Gerontologist* 15 (1975), p. 477; "Edmund V. Cowdry, B.S., Ph.D., 1888-1975," *International Journal of Leprosy* 46 (1978), p. 216.

survey of current scholarship on the problem in various disciplines. As the editor of this volume, he encouraged the contributors to join the "Club for Research on Ageing," an informal discussion group that consisted of approximately twenty scientists. In 1945, core members of this Club eventually established the Gerontological Society, Inc., which changed its name to the Gerontological Society of America (GSA) in 1981. He also played a major role in organizing the International Association of Gerontology and served as its second president.

As historian W. Andrew Achenbaum and sociologist Stephen Katz have pointed out, these works of Cowdry contributed to the rise of gerontology as a respected *multidisciplinary* scientific field pursued by eminent scientists with distinct academic training and norms.[2] From the beginning, the Gerontological Society opened its membership to scholars in various fields, including biology, clinical medicine, psychology, and the social sciences. The *Journal of Gerontology* also accepted research articles from these and other disciplines. The National Institute on Aging is another body that has supported both biomedical and social scientific approach to aging.

In this chapter, I trace the birth and development of gerontology and its multidisciplinary character by focusing on Cowdry's thoughts and activities. Admittedly, multidisciplinarity is a complex and controversial notion, which has often been used interchangeably with "interdisciplinarity" or "transdisciplinarity."[3] The scholars who constructed gerontology during the 1930s and 1940s argued that the science of aging needed to be a field that consisted of multiple disciplines, which maintained close cooperative relationships with one another, just as the atomic bomb project during World War II was a closely integrated effort of physicists, engineers, and military personnel.[4]

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[2] Achenbaum, *Crossing Frontiers*, pp. 52-89; Katz, *Disciplining Old Age*, pp. 93-103.

[3] Julie Thompson Klein, *Interdisciplinarity: History, Theory, and Practice* (Detroit: Wayne State University Press, 1990), pp. 55-73. Klein tries to distinguish "interdisciplinarity" from "multidisciplinarity" and "transdisciplinarity." For other literature on inter/cross/multidisciplinarity, see Ellen Messer-Davidow, David R. Shumway, and David J. Sylvan (eds.), *Knowledges: Historical and Critical Studies in Disciplinarity* (Charlottesville: University Press of Virginia, 1993); Peter Weingart and Nico Stehr (eds.), *Practising Interdisciplinarity* (Toronto: University of Toronto Press, 2000). Also see the essays in Peter Galison and Bruce Hevly (eds.), *Big Science: The Growth of Large-Scale Research* (Stanford: Stanford University Press, 1992).

[4] Vladimir Korenchevsky, "Proceedings of the Research Session on Endocrinologic Aspects of Aging," *Journal of Gerontology* 7 (1952), p. 291; Vladimir Korenchevsky, "Conditions Desirable for the Rapid

Yet later scholars have thought that such a close integration and cooperation has not been feasible in gerontology, although some think that it is increasingly becoming possible. It is even said that gerontology is an applied field for helping the aged rather than a formal scientific discipline, since it has few paradigmatic theories or methodologies which are shared by every member in the field.[5] Sociologist Julie Thompson Kline has decribed the nature of multidisciplinarity shown in these descriptions—it merely "signifies the juxtaposition of disciplines….essentially additive, not integrative."[6] In this chapter, I argue that while the later scholars' accounts of gerontology do reveal some aspects of its current state, they fail to show what the early gerontologists actually did. Although the cooperation among physicists, engineers, and military personnel for constructing the atomic bomb was quite different from what gerontologists could do at that time, they nevertheless tried to develop their field in that direction and were successful in a large measure. The early gerontologists, many of whom contributed to Cowdry's *Problems of Ageing*, shared a broader social concern for the elderly and helped other researchers from different fields during their research and writing on aging. Eventually, the gerontologists formed professional societies through which they discussed various aspects of senescence across disciplinary boundaries. I argue that the multidisciplinarity of early gerontology shown in this series of developments could hardly be called an "additive" "juxtaposition of disciplines."

But this process of gerontology's growth can raise another important question about multidisciplinarity. Why and how did Cowdry and his colleagues decide to make their field multidisciplinary? Indeed, a multidisciplinary approach is demanded when a problem is considered too complex to be solved through a single discipline's efforts. Although this was true for gerontology as well, gerontology's multidisciplinary character

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Progress of Gerontological Research," *The British Medical Journal* 2 (1946), p. 468; Lawrence K. Frank, "Gerontology," *Journal of Gerontology* 1 (1946), pp. 1-11.

[5] Katz, *Disciplining Old Age*, pp. 104-119. Katz' book cites the following references. See Ira S. Hirschfield and David A. Peterson, "The Professionalization of Gerontology," *Gerontologist* 22 (1982), pp. 215-220; R. D. Bramwell, "Gerontology as a Discipline," *Educational Gerontology* 11 (1985), pp. 201-205; and David A. Peterson, *Career Paths in the Field of Aging* (Lexington, Mass.: Lexington Books, 1987).

[6] Klein, *Interdisciplinarity*, p. 56. Interestingly, Klein classifies gerontology as an *interdisciplinary* field, which was made through more integrative teamwork between disciplines. See Klein, *Interdisciplinarity*, p. 44.

was created by many factors rather than merely through his realization of the complexity of the problem. Perhaps it might not have been very difficult for Cowdry, who had interacted with both biological and medical scientists, to think that physiology and clinical medicine should be a part of his project. But how did he conclude that anthropology, psychology, and the social sciences could also contribute to gerontology? Indeed, the Russian immunologist Elie Metchnikoff, who coined the term "gerontology," did not mention social science or psychology as a part of his new field.[7] Gerontology could thus have become a single biomedical discipline and the Gerontological Society of America could have accepted only the scientists from biology and medicine as its members. But this did not happen, and one of my aims in this chapter is to explain how and why Cowdry, along with other scholars, contributed to making gerontology a multidisciplinary field encompassing a broad spectrum of scholars from biological, medical, psychological, and social sciences.

In this chapter, I will argue that Cowdry's training and experience as a biologist in America—more specifically a cytologist—and as a textbook editor guided his endeavor to construct gerontology in this direction, especially amid the Great Depression. First of all, I show that his interaction with a number of eminent contemporary American biologists became a basis of his efforts to make gerontology a multidisciplinary field and to include sociological and psychological approaches as well as biology and medicine in his new scientific field. In particular, I argue that the problems of aging that emerged during the Depression stimulated him to practice what he learned from these biologists and to garner cooperation from these and other scholars. I also point out that his experience in editing textbooks—including *General Cytology* (1924), *Special Cytology* (1928), and *Human Biology and Racial Welfare* (1930)—provided the model of the actual implementation of this cooperation.[8]

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[7] Metchnikoff first mentioned "gérontologie" in 1903. See Elie Metchnioff, *Études sur la nature humaine: Essai de philosophie optimiste* (Paris: Masson, 1903), pp. 294-339, 386.

[8] This aspect of Cowdry's experience was briefly suggested by Achenbaum, Katz, and Landecker. See Achenbaum, *Crossing Frontiers*, p. 63; Katz, *Disciplining Old Age*, pp. 97-103; Landecker, "Edmund Vincent Cowdry."

Indeed, the common experience Cowdry and his biologist colleagues shared reveals how and why he made efforts to establish gerontology as a scientific field. Cowdry's teachers and friends—including Walter Cannon (1871-1945), Herbert Spencer Jennings (1868-1947), Edwin Conklin (1863-1952), and Charles Judson Herrick (1868-1960)—belonged to the generation that experienced race riots, World War I, and the rise of fascism and communism during the early twentieth century.[9] As Sharon Kingsland and other historians have argued, these biological scientists, worried deeply about such political turmoil, tried to offer a new vision of social betterment through the knowledge gained from their research on living organisms that solved their own problems through intimate cooperation and ingenious and dynamic social organization.[10] Cowdry conceptualized the problem of aging in a similar way, after he came in contact with his teachers and colleagues at the University of Chicago, where he received his Ph.D. degree, and at Woods Hole, Massachusetts, where many American biologists regularly gathered for cooperative research and leisure activities.[11] Through his training and research there as well as through his experience during the Depression, Cowdry came to think that while elderly people were suffering from social isolation and economic hardships due to the strengthened age discrimination and destruction of private pensions, the aged cells in the body were still actively contributing to the survival of the whole organism as its important members.[12] Therefore, Cowdry thought, it was necessary to devise ways to promote the welfare and social participation of the elderly following the wisdom of the body's cellular community. Cowdry and his colleagues constructed gerontology as an

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[9] In this sense, they belonged to what sociologist Karl Mannheim has labeled the "generation-unit." See Karl Mannheim, "The Problem of Generations," in Paul Kecskemeti (ed.), *Essays on the Sociology of Knowledge* (London: Routledge and Kegan Paul, 1952), pp. 286-322.

[10] Sharon Kingsland, "Toward a Natural History of Human Psyche: Charles Manning Child, Charles Judson Herrick, and the Dynamic View of the Individual at the University of Chicago," *The Expansion of American Biology*, pp. 195-230; Stephen J. Coss and William R. Albury, "Walter B. Cannon, L. J. Henderson, and the Organic Analogy between the Wars," *Osiris* 3 (1987), pp. 165-192; Gregg Mitman, *The State of Nature: Ecology, Community, and American Social Thought, 1900-1950* (Chicago: University of Chicago Press, 1992).

[11] Philip J. Pauly, *Biologists and the Promise of American Life: From Meriwether Lewis to Alfred Kinsey* (Princeton: Princeton University Press, 2000), pp. 145-164; Maienschein, "Cytology in 1924," pp. 23-51.

[12] On the plight of the elderly during the Depression, see W. Andrew Achenbaum, *Old Age in the New Land: The American Experience since 1790* (Baltimore: Johns Hopkins University Press, 1978), pp. 127-141; *Social Security: Visions and Revisions, A Twentieth Century Fund Study* (Cambridge: Cambridge University Press, 1986), pp. 13-37.

important means to do so, at the same time as bureaucrats and politicians established and developed the Social Security Act during and after the 1930s.

The actual means of constructing gerontology in its early years came from Cowdry's experience in textbook editing that began during his research at Woods Hole. Historians have recently discussed science textbooks as an important agent of constructing disciplines and their practices under various social, political, and pedagogic constraints.[13] I argue that the multiauthored textbooks and their editorial process offered the framework of building gerontology when few people knew how this new science should be organized.[14] Cowdry began editing his textbooks while participating in the American biological community at Woods Hole and other places. There he recruited the contributors to his cytology textbooks from various subspecialties in biology, and successfully encouraged interaction and cooperation among them during the editorial process. Cowdry continued this editorial work in an advanced manner to produce *Problems of Ageing*, whose contributors, despite distinct background and institutional affiliation, were able to discuss senescence in a cooperative manner. Although there were certain conflicts, and some of the authors were not very concerned about what others studied, the contributors, through the editorial process, came to feel that their cooperation would eventually help solve the problems of aging which were considered highly complex and multidimensional issues. While these authors remained as specialists in their own fields, they gradually began to think that they also belonged to a new field, gerontology. I argue that this was the beginning of the multidisciplinary science of aging in the United States.

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[13] A classic historical work on science textbook is Owen Hannaway, *The Chemists and the Word: The Didactic Origins of Chemistry* (Baltimore: Johns Hopkins University Press, 1975). For sophisticated analyses of modern chemistry textbooks, see the articles in Anders Lundgren and Bernadette Bensaude-Vincent (eds.), *Communicating Chemistry: Textbooks and Their Audiences, 1789-1939* (Canton, Massachusetts: Science History Publications, 2000); Antonio García-Belmar, José Ramón Bertomeu-Sánchez, and Bernadette Bensaude-Vincent, "The Power of Didactic Writings: French Chemistry Textbooks of the Nineteenth Century," in David Kaiser (ed.), *Pedagogy and the Practice of Science: Historical and Contemporary Perspectives* (Cambridge, Mass.: MIT Press, 2005), pp. 219-251; Andrew Warwick, *Masters of Theory: Cambridge and the Rise of Mathematical Physics* (Chicago: University of Chicago Press, 2003); David Kaiser, *Drawing Theories Apart: The Dispersion of Feynman Diagrams in Postwar Physics* (Chicago: University of Chicago Press, 2005), pp. 253-279.

[14] I do not argue that Cowdry initiated the tradition of multiauthored textbooks. Textbooks written by multiple contributors had begun to be published especially in medicine long before Cowdry's works started.

In at least three aspects, this early history of American gerontology contrasts with that of its counterpart on the other side of the Atlantic. First, Korenchevsky, who organized gerontology in Great Britain, did not have experience in textbook editing and was not exposed to the ideal—that biologists could contribute to social progress by finding better forms of social organization among living organisms—shared by many American biological scientists at that time. As I have briefly mentioned in chapter four, this was an important reason, I think, why the British Society for Research on Ageing included biomedical scientists and physicians. Second, the size of the group of scientists interested in aging who were invited by Cowdry as the contributors to *Problems of Ageing* was bigger and their commitment to aging was deeper than those of the similar group of scholars in Britain Korenchevsky contacted. In the United States, there were already a larger number of scientists who were seriously interested in aging than in Britain, and many of these Americans participated in Cowdry's new handbook project. Third, Cowdry's leadership, which was supported by the Macy Foundation, was also important in furthering these scholars' academic interest in aging and prompting them to create gerontology. While Cowdry might not have been a charismatic leader, he was a respected and renowned scholar who had a permanent position at a strong research university and was thus successful in leading his colleagues to participate in the new field. This contrasts with the situation of Korenchevsky, a foreign-born scientist who never got a stable job and had to beg continuously for the renewal of his grants throughout his later years when he created the Club as an international organization. Interestingly, the nature of the Great Depression's impact upon Cowdry and Korenchevsky was different due to this reason. Whereas aging was his own personal problem for Korenchevsky who experienced job insecurity and economic instability during the Depression, it was a social rather than a personal problem in the same period for Cowdry and other American scholars, who were younger and had more stable institutional appointments. This chapter describes these Americans' intertwined views and efforts, especially those of Cowdry, which brought about the creation of the science of aging in the United States.

Chicago, Woods Hole, Cytology, and the Art of Editorship, 1909-1932

Cowdry was born in MacLeod, Alberta, Canada in 1888 and earned his bachelor's degree at the University of Toronto in 1909.[15] He then went to the University of Chicago to study anatomy and cytology with Robert R. Bensley and Charles Judson Herrick. In 1913, he received his Ph.D. degree from the Department of Anatomy and moved to the Johns Hopkins University as an associate in anatomy. His dissertation, "The Relations of Mitochondria and Other Cytoplasmic Constituents in Spinal Ganglion Cells of the Pigeon," reveals his expertise in the precise description and identification of microscopic objects with advanced staining techniques.[16] In his dissertation, he aimed at clarifying two highly technical issues in cytology—the first one was the identification of the "neurosome" observed by a German cytologist Hans Held and the second was the characterization of intracellular organelles within the neuronal cell. For the first problem, he argued that the "neurosome," which Held saw within the cell, was indeed a mixture of two kinds of organelles, the mitochondrion and an organelle of unknown identity.[17] He also gave his answer to the second problem. He argued that there were four kinds of "morphologically independent" organelles, "which are not transformed one into another"—the mitochondria, the Nissl bodies, the canalicular system and the neurofibrils.[18]

At Chicago, Cowdry learned the ideal of cooperation in biological research as well as an expertise in microscopic morphology. As historian of science Jane Maienschein has pointed out, the biology departments at the University of Chicago were the places where "the Chicago Style" of biology developed under Charles O. Whitman who had built them during and after the late nineteenth century.[19] It emphasized cooperative and comparative studies of heredity, development, and evolution of diverse organisms and their interactions. The Marine Biological Laboratory (MBL) at Woods

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[15] His bachelor thesis was published. See E. V. Cowdry, "The Colour Changes of Octopus Vulgaris," in *University of Toronto Studies: Biological Series* (Toronto: University Library, 1911), pp. 5-53.
[16] E. V. Cowdry, "The Relations of Mitochondria and Other Cytoplasmic Constituents in Spinal Ganglion Cells of the Pigeon," Ph.D. Dissertation, University of Chicago (Leipzig: Verlag von Georg Thieme, 1912).
[17] Cowdry, "The Relations of Mitochondria," p. 17.
[18] Cowdry, "The Relations of Mitochondria," p. 25.
[19] Jane Maienschein, "Whitman at Chicago: Establishing a Chicago Style of Biology?" in Ronald Rainger, Keith Benson, and Jane Maienschein (eds.), *The American Development of Biology* (Philadelphia: University of Pennsylvania Press, 1988), pp. 151-184.

Hole, Massachusetts was another place where professors and graduate students from Chicago gathered and interacted with biologists from other institutions. Philip Pauly has shown that the professional identity of the American biologists and the direction of their study were formed through their academic and leisure activities at the MBL.[20] Cowdry as a Chicago biologist was a member of this professional community and interacted with a number of eminent contemporary American biologists—including Conklin, Raymond Pearl, Herbert Spencer Jennings, and Edmund B. Wilson—many of whom would offer him a substantial assistance and cooperation when he edited his textbooks and organized the first conference on aging at Woods Hole.[21] In particular, *General Cytology*, edited by Cowdry, was a product of such a cooperation of various experts interested in functions and structures of diverse types of cells.[22] Maienschein, and Cowdry himself, stated that this book reflected the interactive style of biological research that was being pursued at the Marine Biological Station.[23]

At Chicago and Woods Hole, Cowdry was also influenced by contemporary biologists' broad vision on the relation of biological science to human society. According to historian Sharon Kingsland, two biology professors at the University of Chicago, Charles Manning Child and Charles Judson Herrick, promoted the outlook of democracy and progress based on their biological investigation.[24] Living organisms' dynamic, holistic, and cooperative mode of survival and evolution in nature could teach humans a way of reorganizing their societies in an age of war, economic depression, communism, and fascism. Kingsland also pointed out that the socio-biological ideas of these two scientists were shared by, and influenced by, many other scholars at that time—including philosopher John Dewey, physiologist Walter Cannon, protozoologist Herbert Spencer

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[20] Pauly, *Biologists*, pp. 152-160.
[21] See W. E. Garrey and E. V. Cowdry, "Marine Biological Laboratory Increases Activities," *The Nation's Health* 7 (1925), pp. 805-808; E. V. Cowdry, "Preface," in E. V. Cowdry (ed.), *General Cytology: A Textbook of Cellular Structure and Function for Students of Biology and Medicine* (Chicago: University of Chicago Press, 1924), p. v. Also see Cowdry to Pearl, July 21, 1926, Box 158, Folder 12, EVC.
[22] Maienschein, "Cytology," pp. 46-49.
[23] Cowdry, "Preface," *General Cytology*, p. v.
[24] Kingsland, "Towards a Natural History of Human Psyche," pp. 195-230. See, for example, Charles Manning Child, *Physiological Foundations of Behavior* (New York: Holt, 1924), esp. pp. 267-300; Charles Judson Herrick, *Neurological Foundations of Animal Behavior* (New York: Holt, 1924), esp. pp. 295-309.

Jennings, and entomologist William Morton Wheeler—who also tried to show how knowledge gained from biological science could lead to better philosophy and more productive scientific research as well as humans' deepened understanding of their society.[25]

Cowdry knew the above scholars' writings well and kept in touch with them during his scientific career. Herrick was one of Cowdry's thesis advisors and Child a faculty member at Chicago, with whom Cowdry maintained his relationship even after he finished his degree. Particularly, Cowdry was familiar with Child's biosocial philosophy and cited it in his later writing. [26] During and after his doctoral training, Cowdry also met other scholars who interacted with his Chicago professors, such as Dewey, Cannon, and Wheeler. Cowdry first met Dewey while teaching anatomy at Peking Union Medical College in Beijing, China from 1917 to 1921,[27] and asked him to write the chapter on education in *Human Biology* and the introduction to *Problems of Ageing*.[28] Cowdry also kept in touch with Cannon on the matters of research and administration and asked him to contribute the chapter on the aging of homeostatic mechanisms in *Problems of Ageing*.[29] Jennings was a regular participant of the MBL, and helped Cowdry by writing the chapter on the senescence of protozoa in his handbook on aging.[30] It is also important to note that Cowdry read and cited the writings of Wheeler, and asked him to author "Societal

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[25] Kingsland, "Towards a Natural History of Human Psyche," pp. 196, 213-220. Also see, for example, John Dewey, "The Need for a Recovery of Philosophy," in Dewey et al., *Creative Intelligence: Essays in the Pragmatic Attitude* (New York: Holt, 1917), pp. 3-69; Herbert Spencer Jennings, "Diverse Doctrines of Evolution, Their Relation to the Practice of Science and of Life," *Science* 65 (1927), pp. 19-25; William Morton Wheeler, *Emergent Evolution and the Development of Societies* (New York: Norton, 1928); Walter B. Cannon, *The Wisdom of the Body* (New York: Norton, 1932), pp. 287-306.

[26] E. V. Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 4 (3)-5(4), undated, Box 142, Folder 1, EVC. Although this document is "undated," its mention of totalitarianism and the situations during the Great Depression reveals that it was written during the 1930s.

[27] See, for example, Cowdry to John Dewey, 15 March 1920, Box 6, Folder 40, EVC; Dewey to Cowdry, 9 March 1923, Box 6, Folder 40, EVC.

[28] John Dewey, "Influence of Education," in E. V. Cowdry (ed.), *Human Biology and Racial Welfare* (New York: Hoeber, 1930), pp. 468-488; "Introduction," in E. V. Cowdry (ed.), *Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1939), pp. xix-xxvii.

[29] For example, see Cowdry to Cannon, 18 June 1921, Cannon to Cowdry, 21 June 1921, Box 4, Folder 7, EVC; Walter B. Cannon, "Ageing of Homeostatic Mechanisms," *Problems of Ageing* (1939), pp. 623-641.

[30] Herbert Spencer Jennings, "Senescence and Death in Protozoa and Invertebrates," *Problems of Ageing* (1939), pp. 32-52.

Evolution" in Cowdry's *Human Biology*.[31] As will be seen in the next section, Cowdry's interaction with these scholars broadened his perspective on the potential role of biological science in social advancement and human welfare.

While carefully maintaining his relationship with these people, Cowdry kept investigating various problems in biology and medicine using his expertise in detailed description of microscopic objects. In particular, he came to study microbes as well as eukaryotic cells after he returned from China and was appointed as an associate member of the Rockefeller Institute for Medical Research.[32] For example, he studied the distinct staining properties of mitochondria and various types of bacteria as well as the difference between rickettsia and intracellular organelles.[33] He also investigated, through careful staining and observation, where in the nervous system of animals was affected by botulinus poisoning.[34]

But Cowdry was not completely satisfied with cytology's traditional mode of research which was employed in investigating the above problems. To him, the issues he studied were barely related to one another, except for the fact that most of them were about the cell and its various features. Unlike more experimental fields like physiology, Cowdry thought, there was no single unified view or paradigm in these cytological studies which, therefore, hardly led to any rigorous conclusions on the nature of living organisms.[35] Indeed, later scholars continued to view cytology in this way. For them, cytology was a kind of morphology, which aimed at precise description of various structural features of living organisms rather than understanding and theorizing about

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[31] William Morton Wheeler, "Societal Evolution," *Human Biology*, pp. 139-155; Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 5(4)-6(5), undated, Box 142, Folder 1, EVC.

[32] See Cowdry to Bensley, 22 October 1923, Box 3, Folder 6, EVC.

[33] E. V. Cowdry and Peter K. Olitsky, "Differences between Mitochondria and Bacteria." *The Journal of Experimental Medicine* 36 (1922), pp. 521–533; E. V. Cowdry, "The Independence of Mitochondria and the Bacillus Radicicola in Root Nodules," *American Journal of Anatomy* 31 (1923), pp. 339-343; "The Distribution of Rickettsia in the Tissues of Insects and Arachnids," *Journal of the Experimental Medicine* 37 (1923), pp. 431-456.

[34] E. V. Cowdry and F. M. Nicholson, "An Histological Study of the Central Nervous System in Experimental Botulinus Poisoning," *Journal of Experimental Medicine* 39 (1924), pp. 827-836.

[35] See, for example, E. V. Cowdry, "The Value of the Study of Mitochondria in Cellular Pathology," *Proceedings of the Pathological Societies of Philadelphia* 25 (1923), pp. 80–86. Cowdry thought that histology was not different from cytology in this respect, especially from physiology. See E. V. Cowdry, "Teaching of Histology," *Journal of the Association of American Medical Colleges* 11 (1936), p. 292.

essential biological phenomena such as heredity, development, and evolution. This made the accumulation of observational facts, which engendered endless controversy and confusion on the nature of the cell, the major activity of cytologists.[36]

However, cytology of the 1920s had ties to other more experimental biological disciplines because its subject, the cell, was a basic structural element of most biological phenomena. Geneticists such as Thomas Hunt Morgan needed cytological expertise to describe the behavior of the chromosome which they regarded as the material basis of heredity. Embryologist Conklin also had to be familiar with cytology to appreciate cell growth and differentiation occurring in embryogenesis. Cowdry himself was an advocate of biochemical approaches to cytology and always considered experimental methods a way to make breakthroughs in biomedicine.[37]

This state of cytology—apparent lack of a paradigm and its linkage to more experimental subfields in biology—was reflected in *General Cytology*, edited by him and published in 1924. As Jane Maienschein has mentioned, this book shows that cytology apprehended by Cowdry had received contributions by various specialists in distinct biomedical fields, who seldom had "a single unified view."[38] Cowdry noted that his colleague Wilson "freely [expressed] the opinion that the time is passed when one man alone can adequately cover the field" and he and other cytologists "feel the same way about it."[39] Therefore, Cowdry thought that "the best plan [for the book] would be to emphasize the results obtained in different lines of work" which could be written through each contributor's "full responsibility."[40] The author of each chapter needed to be an

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[36] See, for example, William Bechtel, *Discovering Cell Mechanisms: The Creation of Modern Cell Biology* (Cambridge: Cambridge University Press, 2006), pp. 88-89; Aryn Martin, "Can't Any Body Count? Counting as an Epistemic Theme in the History of Human Chromosomes," *Social Studies of Science* 34 (2004), pp. 923-948; Marina Bentivoglio and Paolo Mazzarello, "The Pathway to the Cell and Its Organelles: One Hundred Years of the Golgi Apparatus," *Endeavour* 22 (1998), pp. 101-105.

[37] See E. V. Cowdry, "Comparison of a Virus Obtained by Kobayashi from Cases of Epidemic Encephalitis with the Virus of Rabies," *Journal of Experimental Medicine* 45 (1927), pp. 799-806; "Studies on the Etiology of Heartwater. I. Observation of a Rickettsia, Rickettsia ruminantium (N. Sp.), in the Tissues of Infected Animals. II. Rickettsia ruminantium (N. Sp.) in the Tissues of Ticks Transmitting the Disease." *Journal of Experimental Medicine* 42 (1925), pp 231–273. Also see Bechtel, *Discovering Cell Mechanisms*, p. 77.

[38] Maienschein, "Cytology in 1924," p. 24.

[39] Cowdry to Bensley, 28 September 1922, Box 3, Folder 6, EVC.

[40] Cowdry, "Preface," *General Cytology*, p. v.

expert in one subject to which cytology had relevance, such as the cell's reactivity, differentiation, heredity, and chemical constitution. Of course, the detailed description of cellular morphology, which was cytology's traditional domain, was not neglected.

These experts' contributions to *General Cytology* showed how cytology was being transformed from the traditional morphological field aimed at accumulating observational facts to a new science of the cell supported by novel experimental and physico-chemical methods. Morgan wrote a chapter on "Mendelian inheritance in relation to cytology" and Conklin contributed to the part on cell differentiation during embryogenesis.[41] Warren and Margaret Lewis from the Carnegie Institution of Washington also discussed the "behavior of cells in tissue culture," while Albert P. Matthews from the University of Cincinnati studied cell biochemistry.[42]

Cowdry's goal was to make cytology a *cooperative* discipline with contributions from these researchers. While they came from many different subfields in biology and medicine, the cell, Cowdry thought, was relevant to all of them as "the fundamental unit in health and disease."[43] Although each chapter was their "independent contribution," Cowdry wanted to achieve "a certain coherence resulting from friendly cooperation" in the making of the book.[44] He wrote to his Chicago advisor Bensley that during the summer of 1922 at Woods Hole "a strong sentiment developed in favor of co-operation in the writing of a textbook of general cytology." The scientists who agreed to cooperate in this textbook project were among the regular attendees of the MBL. Cowdry asked these contributors to submit their "brief and tentative outline of two or three pages" before they began to write. These outlines would then be "grouped and a synopsis of the entire book" would be sent to all the contributors to help them complete their chapters in accordance with the general outline and aim of the book.[45] Admittedly, this editorial work alone did not make *General Cytology* a coherent book organized around a "general outline and

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[41] Thomas Hunt Morgan, "Mendelian Heredity in Relation to Cytology," *General Cytology*, pp. 691-736; Edwin G. Conklin, "Cellular Differentiation," *General Cytology*, pp. 537-608.
[42] Warren H. Lewis and Margaret R. Lewis, "Behavior of Cells in Tissue Cultures," *General Cytology*, pp. 383-448; Albert P. Mathews, "Some General Aspects of the Chemistry of Cells," *General Cytology*, pp. 13-96.
[43] Cowdry, "Preface," *General Cytology*, p. v.
[44] Cowdry to E. G. Conklin, 23 October 1922, Box 4, Folder 63, EVC.
[45] Cowdry to Conklin, 23 October 1922, Box 4, Folder 63, EVC.

aim." Each chapter was related to some, but not all, other portions of the book.[46] Yet Cowdry emphasized that "several of the contributors had developed their lines of study by availing themselves year after year of the facilities for investigation offered at Woods Hole," and in this sense, *General Cytology* could be regarded "as a contribution from the Marine Biological Laboratory" where cooperative research was an accepted norm.[47]

This character of Cowdry's cytology textbook was intensified in *Special Cytology* (1928), another study of the cell edited by Cowdry. This book, which aimed at an exhaustive investigation of almost all kinds of cells known to biologists at that time, was written by 35 contributors. Interestingly, many of these people were not "cytologists" by training and institutional position.[48] For example, Alexis Carrel of the Rockefeller Institute was a surgeon and an expert of tissue culture. Alfred E. Cohn from the same institute was a cardiologist and Leo Loeb of Washington University was a professor of pathology and a transplantation biologist. But these scientists could contribute to cytology by writing chapters dealing with a specific type of cells they knew well, such as erythrocytes, lymphocytes, cardiac cells, muscle cells, and nerve cells. To Cowdry, this was the way to relieve the book from "amateurishness" and to enhance "accuracy," although "unity and coherence" was sacrificed in some measure.[49] Nevertheless, Cowdry tried to make the book as coherent as possible through the same method he used to edit *General Cytology*—asking each contributor to submit a short summary of the chapter and sending it to other authors. "Although each writer is solely responsible for his own work," Cowdry wrote, "this helps to reveal gaps, to avoid duplication, to weld the presentations together, and to foster the cooperative aspect of the enterprise."[50]

Cowdry's editorial style which aimed at enhancing the collaborative character of the book continued in *Human Biology and Racial Welfare* (1930) which dealt with

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[46] For example, Cowdry and Morgan did not cite each other while Wilson was cited by both. Conklin cited Cowdry, Morgan, and Wilson, but not the Lewis or Mathews. Mathews cited none of the contributors to the book.

[47] Cowdry, "Preface," *General Cytology*, p. v.

[48] E. V. Cowdry, "Preface," in E. V. Cowdry (ed.), *Special Cytology, The Form and Functions of the Cell in Health and Disease: A Textbook for Students of Biology and Medicine* (New York: Hoeber, 1928), p. vii.

[49] E. V. Cowdry, "Suggestions for Contributors to 'Special Cytology,'" circ. 1926, p. 1, Box 5, Folder 22, EVC; "Preface," *Special Cytology*, p. viii.

[50] Cowdry, "Suggestions for Contributors to 'Special Cytology,'" circ. 1926, p. 1, Box 5, Folder 22, EVC.

various aspects of humans' biological, physical, and social environments. Here, he interacted with a number of distinguished scholars from various disciplines, including the two leading scientists in the editorial committee—Conklin of Princeton University and William K. Gregory of the American Museum of Natural History—and the twenty-five prestigious contributors—including Cannon, Carrel, Dewey, and geneticist and eugenicist Charles Davenport. Even Robert Milikan, who was awarded the Nobel Prize in Physics in 1923, contributed to the book by writing a chapter on "the relation of science to industry." Cowdry also maintained a close cooperative relationship with the publisher Paul B. Hoeber to deal with many difficult problems that occurred during the editorial and publication process. Edwin Embree, director of the Division of Studies of the Rockefeller Foundation, was another important supporter who initiated the project and wrote the introduction to the book.

But the names of these contributors and the Rockefeller Foundation can mislead us. Davenport, Conklin, Carrel, and Milikan have been criticized for their eugenic thinking and racist ideology, despite the wide divergence of their standpoints concerning the issue.[51] Moreover, the Rockefeller Foundation's support of some projects—such as their sponsoring of eugenics, social hygiene, and the reorganization of American medicine according to capitalistic system—has been critically described by later historians, even though the Foundation's activities were not confined to them and and the contexts surrounding these projects were quite complex and multilayered.[52] In this sense, it might be possible for some later scholars to assume that their book, which was about

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[51] Charles E. Rosenberg, *No Other Gods: On Science and American Social Thought* (Baltimore: Johns Hopkins University Press, 1976), pp. 89-97; Lily E. Kay, *The Molecular Vision of Life: Caltech, the Rockefeller Foundation, and the Rise of the New Biology* (New York: Oxford University Press, 1993), pp. 67-68; Melinda Cooper, "Resuscitations: Stem Cells and the Crisis of Old Age," *Body and Society* 12 (2006), p. 16. But historians begin to reevaluate Conklin's view on eugenics. See Kathy J. Cooke, "Duty or Dream? Edwin G. Conklin's Critique of Eugenics and Support for American Individualism," *Journal of the History of Biology* 35 (2002), pp. 365-385; "A Gospel of Social Evolution: Religion, Biology, and Education in the Thought of Edwin Grant Conklin," (Ph.D. Dissertation: University of Chicago, 1994).

[52] For example, see E. Richard Brown, *Rockefeller Medical Men: Medicine and Capitalism in America* (Berkeley: University of California Press, 1979); Kay, *Molecular Vision of Life*; Diane B. Paul, "The Rockefeller Foundation and the Origins of Behavior Genetics," *The Expansion of American Biology*, pp. 262-283; *Controlling Human Heredity: 1865 to the Present* (Amherst, N.Y.: Humanity Books, 1998).

the "Racial Welfare," should contain "the full panoply of sexist, racist, anti-Semitic, and ethnocentric stereotypes of their time."[53]

However, the scope, contents, and perspectives contained in the book were much wider and more diverse than such an assumption indicates. This reflects Cowdry's way of editing the book he had used for his previous projects—asking the best scholars in each field to write their chapters according to what they knew about the field. The choice of the contributors and topics also represented his broad view on the role of biological sciences in the progress and welfare of the *human race* that he learned at Chicago and Woods Hole. Admittedly, Davenport's chapter did deal with the social and racial problems from a rather conservative standpoint, which can be shown in his warning against the "mingling of races."[54] However, anthropologist Aleš Hrdlička pointed out in another chapter that interbreeding between different racial groups had seldom produced biologically undesirable consequences. Moreover, most human racial groups that had been deemed "pure"—such as Germans and Norwegians—were actually products of complex racial mixtures.[55] Conklin, while generally supporting the necessity of eugenics, also criticized "simplistic" eugenicists who regarded a whole racial group such as blacks and Asians as a biologically inferior stock.[56] Instead, he called for a more sophisticated approach to eugenic problems through a thorough understanding of human heredity, development, and environment. The chapter written by Cowdry himself also aimed at an enhanced understanding of the relation of biology to humans in social organizations. Although his was more a general review of basic cytological knowledge and its potential applications than an article with explicit political arguments, it made some remarks on the similarity between cells and individual humans in their societal organizations, which would later develop into his idea on the place of the elderly cells and people in their societies.[57] He also discussed how cytology had contributed to the human welfare

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[53] Katz, *Disciplining Old Age*, p. 97.
[54] Charles B. Davenport, "The Mingling of Races," *Human Biology*, pp. 553-565.
[55] Aleš Hrdlička, "Human Races," *Human Biology*, pp. 156-186. But Hrdlička thought that the white are generally more advanced and talented than the black due to their different evolutionary process in distinct environments.
[56] Edwin G. Conklin, "The Purposive Improvement of the Human Race," *Human Biology*, pp. 578-579.
[57] E. V. Cowdry, "The Vital Units Called Cells," *Human Biology*, pp. 188-192.

through the applications in medical and pharmaceutical research.[58] While holding such social implications in a remote sense, however, the chapters by Carrel, Cannon, J. F. Fulton, A. B. Macallum, and Hans Zinsser were summaries of their recent investigations in their discipline that contained little explicit mention of its relation to "human welfare" or politics. Indeed, *Human Biology* was a book which included a broad spectrum of disciplines, approaches, and political standpoints.

This character of *Human Biology* was what Edwin Embree had conceived when he established the Division of Studies in 1924 within the Rockefeller Foundation, as "an umbrella for a number of small projects."[59] According to historian Robert Kohler, Embree's "human biology," which he tried to develop as a main project for his new Division, was "a fashionable but ill-defined rubric for a congeries of related fields: human heredity, growth, and development, psychology, anthropology, constitutional medicine, and others."[60] But editing a book on such a "congeries of related fields" was Cowdry's specialty. On June 2, 1925, Embree wrote to Cowdry that the "Foundation….through the Division of Studies is exploring a little the subject that might be included under the term 'human biology.'"[61] Embree asked Cowdry to meet with him and to talk about this project further. Although there was little progress on this project afterwards, Embree was able to receive Cowdry's proposal for the contents of the proposed book during the summer of 1926, which he thought was "alluring."[62] However, the Division of Studies was eliminated by 1927 and its human biology program was terminated.[63] Probably for this reason, Embree ceased to sponsor the project, although he promised to write the introduction to the book. Thereafter, Cowdry came to take the full editorial responsibility for the project.

Despite such problems, the book on human biology was edited through the consistent efforts of Cowdry and his colleagues. At first, however, he was not sure about

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[58] Cowdry, "The Vital Units Called Cells," *Human Biology*, pp. 202-204.
[59] Robert E. Kohler, *Partners in Science: Foundations and Natural Scientists, 1900-1945* (Chicago: University of Chicago Press, 1991), p. 125.
[60] Kohler, *Partners in Science*, p. 126.
[61] Edwin Embree to Cowdry, 2 June 1925, Box 7, Folder 17, EVC.
[62] Embree to Cowdry, 3 June 1926, Box 7, Folder 17, EVC.
[63] Kohler, *Partners in Science*, pp. 126-127.

the scope, level, and the kinds of contributors and prospective readers of the book he was editing. But embryologist Conklin, one of his close colleagues at Woods Hole, was able to help Cowdry in these matters. Moreover, Conklin and Cowdry gained the support of another expert in human biology, William K. Gregory at the American Museum of Natural History, by asking him to join the editorial committee when Gregory was elected as a member of the National Academy of Sciences through the help of Conklin.[64] These three scientists did their best to garner the cooperation of the scholars who were "really eminent" in each field.[65] Most of them were colleagues they had met at their home institutions and professional societies, such as Davenport, Dewey, Carrel, Cannon, and Wheeler. Raymond Pearl was another important contributor as a major recipient of the Rockefeller fund awarded through Embree's substantial help.[66] The other contributors could be recruited through the recommendations of the people who had already decided to join Cowdry's project. For example, the renowned criminologist William Healy at Yale University was recommended by Davenport[67] and John F. Fulton of Oxford was asked by Cannon to co-author the chapter on neurophysiology with Charles S. Sherrington when Sherrington abruptly refused to participate in the project.[68]

Cowdry, Conklin, and Gregory closely cooperated in adjusting the book's level and choosing the topics as well as in recruiting the contributors. One of the most important issues they had to decide was the level of the scientific discussion in their book. While Conklin thought that it was not appropriate to "popularize" their book too much by excluding more specialized and technical knowledge in human biology,[69] it was decided

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[64] Edwin G. Conklin to Cowdry, 20 May 1927, Box 159, Folder 3, EVC.
[65] Cowdry to Conklin, 12 November 1926, Box 157, Folder 7, EVC.
[66] Kohler, *Partners in Science*, p. 126; Sharon Kingsland, "Raymond Pearl: On the Frontier in the 1920s," *Human Biology* 56 (1984), p. 7. Also see Embree to Pearl, 16 February 1925, 28 May, 1925, Folder Embree, Edwin #1, Series 1, BP 312, RP.
[67] Charles Davenport to Cowdry, 10 November 1926, Box 6, Folder 30, EVC.
[68] Cowdry to Sherrington, 15 October 1928; Cannon to Cowdry, 14 December 1928, Box 178, Folder 10, EVC. Indeed, it was Fulton rather than Sherrington who actually wrote the chapter, even though both are listed as co-authors. Sherrington was merely requested to read Fulton's manuscript and give comments on it. Since Sherrington was an eminent neurophysiologist, his name was necessary to add prestige to the book. Also see Cannon to Cowdry, 6 December 1928; Cowdry to Fulton, 29 January 1929, Box 178, Folder 10, EVC.
[69] Conklin to Cowdry, 21 October 1926, Box 157, Folder 7, EVC; Conklin to Cowdry, 26 August 1926, Box 157, Folder 7, EVC.

that the chapters should "explain in simple language just what is being done in lines of research directly affecting man."[70] Nevertheless, it was "unwise to overbalance on the side of simplicity and insult the reader by underestimating his capacity to comprehend things that interest him."[71] The editorial committee also had to decide whether they needed to assign a chapter on cancer or not, and whether they should include a discussion of the biochemical aspect of evolution. Eventually, the chapter on cancer cells was eliminated because Cowdry's article partially included it, while the biochemical aspect of evolution was assigned a separate chapter, since it had been neglected by biologists despite its importance.[72]

Another significant issue was the problem of Henry Osborn's chapter on "the antiquity of man," which could support and complement Gregory's chapter on "the animal ancestry of man."[73] Although it was Gregory who recommended that Osborn join the project, he himself found that Osborn's chapter was "a hopelessly confusing and misleading production" with numerous "unverifiable assumptions."[74] On this problem, Conklin commented that the editors should "be prepared to decline articles that are not up to standard."[75] Yet it could be very embarrassing for both the editors and Osborn to write to him that they should turn down his chapter for such a reason, because he was Gregory's mentor and a respected senior scholar.[76] Therefore, after discussing this issue with the editorial committee, Cowdry sent a letter to Osborn to ask "what course of action should be pursued" about his manuscript, since his "views….differ materially from those of our other contributors" and his chapter "consists almost wholly of clippings from [his] previous published papers."[77] The publisher wanted a completely new article rather than these "clippings" that might cause copyright problems. Osborn replied that the difference

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[70] "Suggestions as to Writing," Box 178, Folder 6, EVC.
[71] "Suggestions as to Writing," Box 178, Folder 6, EVC.
[72] Cowdry to Conklin, 23 October 1926, Box 157, Folder 7, EVC.
[73] Gregory recommended that Osborn should write this chapter. Gregory to Conklin, 29 July 1927, Box 5, Folder 18, EVC.
[74] Gregory to Cowdry, 28 April 1928, Box 178, Folder 7, EVC.
[75] Conklin to Cowdry, 3 May 1928, Box 178, Folder 6, EVC.
[76] See Ronald Rainger, "Vertebrate Paleontology as Biology: Henry Fairfield Osborn and the American Museum of Natural History," *The American Development of Biology*, pp. 219-256.
[77] Cowdry to Conklin, 2 May 1928, Box 178, Folder 6, EVC; Conklin to Cowdry, 3 May 1928, Box 178, Folder 6, EVC; Cowdry to Osborn, 5 May 1928, Box 161, Folder 3, EVC.

of views Cowdry mentioned could be quite interesting to some readers, and the copyright problems would not occur, because Osborn himself owned the copyright of his previous publications and his chapter did not consist of "clippings" from his former works.[78] Nevertheless, Osborn withdrew his chapter, since he knew that his text "in its present form….embarrasses [the editors]" and he did not have enough time to revise it.[79]

The publisher, Paul B. Hoeber, was another important collaborator in this project. For *Human Biology*, Cowdry worked with him on various issues, such as the amount of royalty paid to the authors, the ways to boost sales, and the means to eliminate the words that might provoke antivivisectionists.[80] The title of the book, which brought about substantial misunderstandings among later readers, was another important issue that had to be decided through the cooperation between them. Many prospective titles were suggested by both Cowdry and Hoeber, such as "Human Biology and Civilization: The Past, Present, and Future of Man," "Human Biology and the Future of Man," "Human Biology and Society," and "Human Biology and Social Welfare."[81] Among them, Cowdry and Hoeber liked the last one, "Human Biology and Social Welfare." For some unknown reason, however, this title was changed into "Human Biology and Racial Welfare" at some point of time before March 21, 1930.[82]

Cowdry's interaction with the contributors also shows his cooperative way of working. As a scientist who developed a broad view on society and biology through his contact with his professional colleagues, he tried to discuss each chapter's topic with the contributors even if it was not closely related to his expertise. For example, he asked William Healy to deal with the following questions in his chapter on criminology.

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[78] Osborn to Cowdry, 10 May 1928, Box 161, Folder 3, EVC.
[79] Osborn to Cowdry, 10 May 1928, Box 161, Folder 3, EVC
[80] Cowdry to Hoeber, 13 October 1928, Box 178, Folder 1, EVC; Cowdry to Hoeber, 19 November 1928, Box 178, Folder 1, EVC; Cowdry to Hoeber, 31 October 1928, Box 178, Folder 1, EVC. The reaction of antivivisectionists led science journal editors to modify vocabulary in their articles. See Susan E. Lederer, "Political Animals: The Shaping of Biomedical Research Literature in Twentieth-Century America," *Isis* 83 (1992), pp. 61-79.
[81] Hoeber to Cowdry, 23 June 1928, Box 178, Folder 1, EVC; Hoeber to Cowdry, 27 June 1929, Box 178, Folder 2, EVC.
[82] Hoeber to Cowdry, 21 March 1930, Box 28, Folder 59, EVC.

1. How would you define antisocial behavior, delinquency, and crime? In what do they differ?
2. To what primary factors may they be due? Is it a case of social maladjustment? Is an hereditary factor involved?
3. To what extent are they remediable?
4. In what countries is the situation most effectively met, and how?
5. What is about the proportion of state budget involved? Is this on the increase and, if so, how rapidly?
6. What are crime waves and have they an etiology which can be at all defined?
7. Is there any a racial factor in crime? In other words, are certain races by reason of their characteristic temperaments more prone than others to commit crimes of different kinds, violence for instance?
8. Is the attitude of the newspapers toward antisocial behavior, delinquency, and crime helpful or the reverse? In what way should it be altered, if at all?[83]

He asked similar questions of other contributors, who usually responded with constructive feedback.[84] Moreover, as he had done before, he sent them the general outline of the entire book which was produced from each chapter's abstract.[85] This time, however, he tried to become more actively engaged with the authors and to enhance the degree of cooperation in the book production. First, he encouraged the authors to read a few particular chapter synopses or full articles written by other authors that were closely related to theirs. For example, he asked Healy to read Wheeler's and Dewey's chapter synopses and recommended Cannon's article to Haven Emerson, who wrote about "the

[83] Cowdry to Healy, 30 January 1928, Box 178, Folder 8, EVC.
[84] For example, see Cowdry to Ellsworth Huntington, 30 January 1928, Box 178, Folder 8, EVC; Cowdry to Hrdlička, 30 January 1928, Box 178, Folder 8, EVC; Cowdry to Milikan, 31 March 1928, Box 178, Folder 9, EVC; H. A. Overstreet to Cowdry, 12 October 1928, Box 178, Folder 9, EVC. Some contributors did not wholly accept Cowdry's comments. For an example, see Pearl to Cowdry, 23 January 1928, Box 178, Folder 9, EVC.
[85] See Cowdry to Wheeler, 25 November 1927, Box 162, Folder 1, EVC.

Influence of Urban and Rural Environment" with Earle B. Phelps.[86] Second, he tried to hold an "informal conference" of the contributors and editors to discuss the contents and direction of the book.[87] Although it is not certain whether Cowdry really held that conference at that time, this attempt shows how his efforts to encourage cooperation among the authors could be translated into an actual scientific meeting. This translation would become important when he organized the first conference on aging while editing *Problems of Ageing*.

Cowdry participated in another cooperative handbook project, *Sex and Internal Secretions* (1932), which has been regarded as "the American Bible of Reproductive Endocrinology."[88] Although he did not directly edit the book, Edgar Allen, the editor and dean of the medical school of the University of Missouri, wrote that "this project saw its inception in a proposal by Dr. E. V. Cowdry, then Chairman of the Medical Division of the National Research Council," who helped "not only during the initial phases of the project, but throughout its progress and consummation."[89] Robert M. Yerkes at Yale, who contributed a chapter to Cowdry's *Human Biology*, concurred. According to him, "the Committee for Research in Problems of Sex, on suggestion of Dr. E. V. Cowdry, member ex-officio as Chairman of the Division of Medical Sciences of the National Research Council," decided to publish the book.[90] Indeed, Cowdry gave Allen many helpful suggestions for a better book editing, which required "much diplomacy, hard work and continual attention."[91] Even the organization of *Sex and Internal Secretions* was the same as the books Cowdry had edited: It was contributed by various experts across disciplines such as embryology, gynecology, obstetrics, anatomy, biochemistry, psychology, and dairy husbandry. As Yerkes wrote, these experts intended to "organize a coöperative survey of recent advances in research on internal secretion in relation to

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[86] Cowdry to Healer, 30 January 1928; Cowdry to Emerson, 18 May 1928, Box 178, Folder 7, EVC.
[87] Cowdry to Paul A. Lewis, 24 August 1927, Box 178, Folder 8, EVC.
[88] Adele E. Clarke, *Disciplining Reproduction: Modernity, American Life Science, and the Problems of Sex* (Berkeley: University of California Press, 1998), p. 136.
[89] Edgar Allen, "Preface," in Edgar Allen (ed.), *Sex and Internal Secretions: A Survey of Recent Research* (Baltimore: Williams and Wilkins, 1932), p. xx.
[90] Robert M. Yerkes, "Foreword," *Sex and Internal Secretions,* p. xvii.
[91] Cowdry to Allen, 12 December 1932, Box 24, Folder 4, EVC.

sex."[92] According to sociologist Adele E. Clarke, this survey represented the nature of American reproductive science which was made through the contributions of various fields in their institutional and social worlds that had the "mutual disciplining, reciprocal relations, and negotiations."[93]

Gerontology resembled American reproductive science in that both consisted of various fields which interacted with one another and were represented in multiauthored handbooks. While *Sex and Internal Secretions* was the first such book for reproductive science, Cowdry's *Problems of Ageing* was the one for the science of aging. It is important to note that the first conference on aging at Woods Hole, which developed into the Club for Research on Ageing and the Gerontological Society, was originally planned as a discussion forum for the multidisciplinary contributors to *Problems of Ageing*. While he might not have been able to have an "informal conference" while editing *Human Biology*, he could hold a more formal conference in 1937 when he worked for *Problems of Ageing*. The only difference between this new project and previous ones was the fact that it developed in a new social and economic condition during the Great Depression. The next section will discuss how Cowdry conceived and edited his new handbook in this novel social environment.

Problems of Aging during the Great Depression: The Body Politic, the Body Anatomic, and Aging as a Scientific Problem, 1930-1936

When Cowdry published *Human Biology* in 1930, the severe economic recession after the stock market crash on October 24, 1929 already cast a gloomy shadow on every aspect of American's life. The long lines of hungry job seekers in one place and huge heaps of abandoned agricultural products in other places led scholars to rethink the nature of capitalist economy and the social structure. What went wrong? What should be done to restore the disrupted economy and reorganize society in a way that could be more stable in the future?

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[92] Yerkes, "Foreword," *Sex and Internal Secretions*, p. xvii.
[93] Clarke, *Disciplining Reproduction*, p. 31.

The establishment of the Social Security Act in 1935 was the federal government's response to the problem of old age that emerged during the Great Depression, although historians have not agreed upon what factor or issue at that time led to the making of the Act. Achenbaum has argued that it was the elderly people's grave poverty and unemployment during the Depression that motivated the policy makers to institute the Act.[94] While aged Americans were already becoming marginalized in the urbanizing and industrializing American society of the early twentieth century, the disaster of the 1930s cut their work opportunities and means of support more sharply than any other age groups and thus threatened their survival in the severest way. These acute and deep economic woes thus became a basis for establishing national pension plan for helping the livelihood of aged Americans. However, William Graebner argued that the New Dealers in the 1930s were not very concerned about the welfare of older Americans. The Social Security Act, which furthered the mandatory retirement over 65 in many workplaces, was constructed merely as a means of reorganizing labor force in a more "efficient" way. The logic was that more jobs could be available for the younger and perhaps more efficient workers when these old people were forced out of the labor market.[95] But Carole Haber and Brian Gratton in a more recent publication claimed that the policy makers of 1935 did have concern for the welfare of the aged. Yet it was not the old people's destitution but their elevated expectation for a more comfortable life that prompted the New Dealers to establish the Act.[96] According to Haber and Gratton, the developing industrial capitalism in the early twentieth century brought more money and security to the elderly people's life through increased wages, family savings, and private pensions. Since the Depression destroyed these sources of stability in old age, people

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[94] Achenbaum, *Old Age in the New Land*, pp. 127-141; *Social Security*, pp. 13-37.
[95] William Graebner, *A History of Retirement: The Meaning and Function of an American Institution, 1885-1978* (New Haven: Yale University Press, 1980), pp. 181-214.
[96] Carole Haber and Brian Gratton, *Old Age and the Search for Security: An American Social History* (Bloomington: Indiana University Press, 1994), pp. 172-185.

demanded an alternative one through the federal government, which was realized as the Social Security Act in 1935.[97]

In my dissertation, I do not attempt to decide whose view is closer to the historical truth. What is more important for my study is the *discourse* on the state of the elderly, which was certainly problematized as one of the most important social and political concerns for the bureaucrats in the federal government. The bureaucrats felt that they needed to do something for elderly people, because their life was becoming miserable due to the insecurity incurred by the loss of their jobs and pensions during the Depression. On the other hand, the bureaucrats thought, aged Americans were inefficient and conservative and perhaps a hindrance to the social and economic reorganization needed by the New Dealers. Although depicting a quite different picture on the economic reality of the elderly, Haber and Gratton have also agreed with this negative perception of old age, which was represented by the state of aged poor institutionalized in almhouses.[98] But it was not only the bureaucrats who worried about the "problems" of old age. Biological and medical experts also began to deal with this problem in their own way. Yet their response reflected issues and problems generated through their scientific research as well as the general perception of old age during the 1930s.

Cowdry's *Arteriosclerosis: A Survey of the Problem* (1933) was a treatise written by these biomedical experts on one of the most prevalent diseases of old age. After moving to Washington University in 1928 as professor of cytology and being appointed as chairman of the Division of Medical Research of the National Research Council, Cowdry was approached by the Josiah Macy, Jr. Foundation for his professional advice on arteriosclerosis.[99] On this request, Cowdry investigated who the most renowned experts on this disease were and who among them could contribute to the publication of a thorough summary on the contemporary medical and biological understanding of the disease. The result of this effort was a book which was quite similar to his previous

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[97] In a recent monograph, sociologist John Macnicol has criticized this assertion. But this issue needs to be investigated more thoroughly. See John Macnicol, *Age Discrimination: An Historical and Contemporary Analysis* (Cambridge: Cambridge University Press, 2006), pp. 209-223.

[98] Haber and Gratton, *Old Age and the Search for Security*, p. 179.

[99] E. V. Cowdry, "Preface," in E. V. Cowdry (ed.), *Arteriosclerosis: A Survey of the Problem* (New York: Macmillan, 1933), p. ix.

publications—a handbook written through the cooperation of multiple expert authors recruited from various fields, including histology, pathology, neurology, cardiology, cytology, and anatomy. The only difference in this book was the predominance of medical researchers and clinicians among the authors and the support of a philanthropic organization.

Cowdry's chapter on "the Structure and Physiology of Blood Vessels," in *Arteriosclerosis* reflected the newly emerging view—aging as a localized process—supported by several leading researchers such as Charles Minot, Alfred Cohn, and Raymond Pearl, and was strongly implied in Alexis Carrel's tissue culture experiments.[100] Particularly, Cowdry was heavily influenced by Carrel, his former colleague at the Rockefeller Institute. Cowdry mentioned the importance of tissue culture as early as 1920[101] and kept corresponding with Carrel over various issues, including Cowdry's plan for setting up tissue culture facility at Washington University.[102] Cowdry learned through Carrel that each type of cells needed distinct media to be cultured and the rate of cell senescence differed according to these environments in which the cell was placed. From this, Cowdry inferred that cells' rates of senescence were determined by their location within the body, which had a distinct local fluid environment.[103] He made this point clear in his chapter in *Arteriosclerosis*, which reviewed the morphology and physiology of blood vessels from arteries to veins. He argued,

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[100] Charles S. Minot, *The Problem of Age, Growth, and Death: A Study of Cytomorphosis* (New York: Putnam, 1908), pp. 214-216; Alfred E. Cohn and Henry A. Murray, Jr., "Physiological Ontogeny I. The Present Status of the Problem," *Quarterly Review of Biology* 2 (1927), pp. 482, 490; Raymond Pearl, *The Biology of Death* (Philadelphia: Lippincott, 1922), pp. 138-149, 225; Alexis Carrel, "Tissue Culture and Cell Physiology," *Physiological Reviews* 4 (1924), pp. 1-20; Alexis Carrel, "The New Cytology," *Science* 73 (1931), pp. 297-303. For later scientists' ideas on this issue, see Clive M. McCay, "Chemical Aspects of Ageing," *Problems of Ageing* (1939), p. 574; Nathan W. Shock, "Ageing of Homeostatic Mechanism," in Albert I. Lansing (ed.), *Cowdry's Problems of Ageing*, third edition (Baltimore: Williams and Wilkins, 1952), pp. 421, 429-31, 436, 438; A. J. Carlson to Cowdry, 28 June 1937, Box 10, Folder 397, WDM.

[101] E. V. Cowdry, "Anatomy in Japan," *Anatomical Record* 18 (1920), p. 94. Indeed, the lack of tissue culture research was one of the few problems Cowdry found in Japanese biomedical science programs. Also see Cowdry, "The Value of the Study of Mitochondria," p. 85.

[102] See, for example, Cowdry to Carrel, 13 April 1929, Box 159, Folder 5, EVC.

[103] Cowdry, "Ageing of Tissue Fluids," *Problems of Ageing* (1939), pp. 643, 685, 689.

> Since their local environments vary as well as their duties, the muscular arteries themselves exhibit peculiar and interesting modifications. The uterine artery is almost made anew with each pregnancy. The umbilical artery is a highly special structure designed to serve a temporary and unique function. The arteries of the placenta become old and senile in less than nine months.[104]

The rate of generation, development, and senescence of each blood vessel differed depending on its local environment. Although the entire body might be far from being senile, its cells constituting blood vessels at a particular environment tended to undergo senescence at their own rate.

Indeed, Cowdry had been interested in cellular senescence as early as in 1916, when he wrote a review article on mitochondria,

> [The Mitochondria] are most abundant in the active stages of the life of the cell. They diminish progressively in number as the cells become senile. The most striking example of this is seen in sections of the skin as one passes from the cells of the deeper layers, which contain many mitochondria, to the more superficial, desquamating cells, which are dead or dying and which often are quite devoid of mitochondria. Moreover, the mitochondria decrease in number as one passes from nucleated to nonnucleated red blood cells.[105]

Like his chapter in *Arteriosclerosis*, his 1916 article focused on cell aging at a particular location within the body rather than the whole individual organism's senescence.[106]

Cowdry's interest in local dimensions of aging became more explicit in *Human Biology and Racial Welfare*. In his chapter, he repeated his 1916 observation on the aging of the skin cell, which, according to him, could be accounted for through an ironical

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[104] Cowdry, "The Structure and Physiology of Blood Vessels," *Arteriosclerosis*, p. 63.
[105] E. V. Cowdry, "The General Functional Significance of Mitochondria," *American Journal of Anatomy* 19 (1916), p. 432.
[106] He expressed the same kind of view in another review article on mitochondria published in 1923. See Cowdry, "The Value of the Study of Mitochondria," p. 76.

statement used in funerals: "While we are in life we are in death."[107] That is, senescence occurred constantly in every portion of our body even when we were youthful individuals. And this process of aging was more closely related to each cell's local environment than its genetic constitution, which was uniform throughout the body.

Cowdry was thinking about this issue and its meaning for the social place of the elderly when he asked Ludwig Kast, president of the Macy Foundation, to support his new handbook on aging. On October 9, 1935, recollecting his arteriosclerosis project with the Foundation, Cowdry wrote to Kast that he had "an idea which may or may not appeal to" him.[108] Cowdry said,

> The problem of ageing in relation to arteriosclerosis often confronted us. Would it not be a good plan to make a similar study of this problem of ageing viewed from many angles? I think that the factors involved in "growing old" have been sadly neglected. Interest has centered in helping the young. Old age is inevitable and so, as with arteriosclerosis, nothing is done to postpone it or to render it less tragic. This is not a small matter; it is a serious [indictment] of our body politic.[109]

Indeed, since the publication of *Human Biology* in 1930, Cowdry had been pondering the similarity between this "body politic"—the society of human beings—and the "body anatomic"—the society of cells within a living organism—and the way to improve the body politic through the knowledge gained from the study of the body anatomic. Like humans in the body politic, cells in the body anatomic lived in communities and went through a series of life stages, such as birth, growth, maturity, and senescence.[110] The cell community also had "criminals," such as cancer cells, just as human society did.[111] Through his cytological research, however, he came to think that the cell community was

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[107] Cowdry, "The Vital Units Called Cells," *Human Biology*, p. 189. This statement came from "In the midst of life we are in death" in "The Order for the Buriall of the Dead," *The Book of Common Prayer and Administration of Sacraments: And Other Rites and Ceremonies in the Church of England* (London: 1642).
[108] Cowdry to Ludwig Kast, 9 October 1935, Box 31, Folder 9, EVC.
[109] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.
[110] Cowdry, "The Vital Units Called Cells," *Human Biology*, p. 188.
[111] Cowdry, "The Vital Units Called Cells," *Human Biology*, p. 192.

much better than the human community in solving social problems in an effective way. Most of all, the aged and dead cells in their specific localities still played an important role in the maintenance of the whole body whereas elderly people in their societies were suffering from the loss of their place during the Depression. For example, the senile and dying cells in the epidermis maintained their status as a significant member of their local communities through their role as a "shield between the living delicate tissues beneath and the environment outside" like a "shock absorber."[112] In contrast, as Cowdry wrote to Kast, aged Americans during the 1930s were "wrongly [considered] to be past their usefulness" and became "the forgotten ones."[113]

In his two manuscripts written at that time—"the Biological Basis of the New Deal" and "Citizen Cells: How Cells Manage Their Social Problems"—Cowdry developed this idea further.[114] Although he was not successful in publishing these writings,[115] they reflected his broad view on the place of biological science in society, which had matured through his contact with his fellow biologists at Chicago and Woods Hole and his own reflection on the cause of the Great Depression.

In the first chapter of "Citizen Cells," he cited the ideas of several biologists with whom he had interacted since the 1910s—Conkin, Pearl, Wheeler, Wilson, and Child. Cowdry cited Conklin's argument that "the animal body has always been regarded as the ideal for the organization of society" along with Wilson's claim that "the multicellular organism may be regarded as a 'cell-state.'"[116] For Cowdry, these scientists of the twentieth century reconfirmed the validity of the old analogy between the human body and society through their biological research. Yet Cowdry knew that many social scientists did not accept such analogies, which they thought, were "no proof of anything"

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[112] Cowdry, "The Vital Units Called Cells," *Human Biology*, p. 189.
[113] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.
[114] From many different kinds of pagination and an extensive addition of paragraphs, I think that they were written and revised many times after he wrote his chapter for *Human Biology* in 1930. Unfortunately, the precise date of first draft and subsequent revision could not be determined.
[115] Although Cowdry submitted his manuscripts to the Williams and Wilkins Company for publication, the editor thought that it was very "formidable" and "not an easy subject for the reader to sustain his interest in." See Robert S. Gill to Cowdry, 19 April 1939, Box 42, Folder 22, EVC.
[116] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 1(2)-2(3), undated, Box 142, Folder 1, EVC. The number within the parenthesis designates a different sort of pagination Cowdry added to each page. Numbers in parentheses indicates a different pagination on a single page.

and potentially even "vicious because they are unscientific and likely to lead the unwary astray."[117] Moreover, according to a renowned sociologist R. M. McIver, "the territory which the sociologist explores changes even as he explores it," while "human nature was not very different thousands of years ago."[118] However, Cowdry claimed that "a human being, while it lives, is always changing so that the material of a cytologist is in point of fact much closer to that of a sociologist than is that of a physicist or chemist."[119] As he would show in the rest of his manuscript, there were indeed numerous examples of how a living organism dynamically changed its state according to environments, just as human societies did. Therefore, it was still quite useful to find the similarities between the human body and society. Indeed, he wrote, "We join a distinguished company of biologists….when we compare social integration at the cellular and human levels."[120]

Cowdry found a number of such similarities, many of which were drawn from the contemporary issues engendered during the Depression. He pointed out that the body anatomic had in the cardiovascular system a much better way of distributing energy sources than the body politic which was then suffering from "the burning of grains in Kansas, urgently demanded in industrial areas; the allowing of oranges to rot in Florida, which could be used to great advantage elsewhere…and the letting of coal heap up at the mine heads, while people suffer from the cold in other parts of the country."[121] Another example was the problem of unemployment which was "unknown" in the body anatomic with its effective use of the labor force that had developed during its long evolutionary process.[122] He wrote, "[n]ever in the body anatomic is the risk incurred of disrupting established condition by the sudden introduction of some new invention permitting one to

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[117] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 7(8)-8(9), undated, Box 142, Folder 1, EVC.
[118] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 11(13), undated, Box 142, Folder 1, EVC.
[119] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 11(13), undated, Box 142, Folder 1, EVC.
[120] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 25(54), undated, Box 142, Folder 1, EVC.
[121] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 105(8), undated, Box 142, Folder 1, EVC.
[122] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 55, 74(74), undated, Box 142, Folder 1, EVC.

do the work of many" as was the case in the body politic. For instance, Cowdry pointed out that industrialists and scientists, including Charles Kettering of General Motors, had argued that the advancement of science and technology could create more jobs than those eliminated by the introduction of new machines.[123] Yet Cowdry knew that many of his contemporaries thought differently. In the case of the automobile industry, the rise of mass production technology eliminated jobs related to the traditional means of transportation—such as manufacturers of harnesses, carriages, wagons, and those who drove and took care of horses—even though big corporations like GM created some positions in their factories.[124] Cowdry wrote that the United States federal government already took action upon this problem by initiating the New Deal and establishing the National Resources Committee.[125] Rather than letting the problem be dealt with solely by scientists and industrialists, Americans realized the importance of managing the resources of their society in a systematic way to avoid the same economic problems. As Walter Cannon wrote in *The Wisdom of the Body* (1932), however, the body anatomic had already solved the problem and this might provide "the biological basis of the New Deal."[126]

Yet the body anatomic was not always the best model for reorganizing the society. In cell communities, "government is largely automatic" and "many citizen cells are without direct representation."[127] Moreover, "laws, or codes of behavior, are to maintain order not to provide equal treatment for all."[128] Indeed, "class distinctions are definite

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[123] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 55(80), undated, Box 142, Folder 1, EVC.

[124] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 55(80), undated, Box 142, Folder 1, EVC. This shows that Cowdry also felt what historians Daniel Kevles has called a "revolt against science" during the Great Depression, as was described by Daniel Kevles. See Daniel J. Kevles, *The Physicists: The History of a Scientific Community in Modern America* (Cambridge, Mass.: Harvard University Press, 1995), pp. 236-266. Also see A. Hunter Dupree, *Science in the Federal Government: A History of Policies and Activities* (Baltimore: Johns Hopkins University Press, 1986), pp. 344-368. For a more recent work, see Amy Sue Bix, *Inventing Ourselves out of Jobs? American's Debate over Technological Unemployment, 1929-1981* (Baltimore: Johns Hopkins University Press, 2000).

[125] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 55A(81), undated, Box 142, Folder 1, EVC.

[126] E. V. Cowdry, "The Biological Basis of the New Deal," p. 4, undated, Box 172, Folder 8, EVC. See Cannon, *The Wisdom of the Body* (1932), pp. 287-306.

[127] Cowdry, "The Biological Basis of the New Deal," p. 6, undated, Box 172, Folder 8, EVC.

[128] Cowdry, "The Biological Basis of the New Deal," p. 6, undated, Box 172, Folder 8, EVC.

because division of labor must be maintained" and thus each cell's "position in community is fixed."[129] In this sense, the body anatomic was similar to the totalitarian state" as described by the seventeenth century philosopher Thomas Hobbes.[130] (See Figure 5.1.)

Figure 5.1. *Totalitarianism. Cowdry's signature can be seen at the right bottom corner. This was a part of a holiday card from Charles and Dorothea Singer which Cowdry planned to use for his book. From Box 142, Folder 2, The Edmund Vincent Cowdry Papers, Bernard Becker Medical Library, Washington University. The original picture can be found in Thomas Hobbes, Leviathan or the Matter, Forme, & Power of a Common-wealth Ecclesiasticall and Civill (London: Andrew Crooke, 1651).*

But the body anatomic was different from the actual totalitarian states that began to rise in Europe at that time. Cowdry wrote that "there are two fundamental differences between the community of cells and the totalitarian state as ordinarily conceived."[131] First, the nerve cells in the body anatomic, despite its ruling power, were not dictators like those in real totalitarian states, because their ability to rule the whole body had

[129] Cowdry, "The Biological Basis of the New Deal," p. 6, undated, Box 172, Folder 8, EVC.
[130] Cowdry, "The Biological Basis of the New Deal," p. 8, undated, Box 172, Folder 8, EVC.
[131] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 158(158), undated, Box 142, Folder 1, EVC.

evolved for millions of years during which they became able to respond to the suggestions and recommendations of other citizen cells. Second, the body anatomic was regulated according to its "constitution" and "principles," "which do not change suddenly at the behest of politicians," unlike Nazi Germany and Fascist Italy.[132] Moreover, cells retained their ability to live independently if they were detached from the body anatomic, as Carrel's tissue culture experiments had showed.[133] Indeed, many citizen cells were enjoying some sort of independence even when they were living within the body anatomic, because, as Carrel's experiments and Cowdry's own observations indicated, most cells lived in their own distinct local fluid environment rather than in blood or lymph that was tightly regulated by the homeostatic mechanisms.[134]

For these reasons, Cowdry thought that the body anatomic could be a good guide for solving various problems in the human society during the Depression, including those of old age. According to him, "virile people between 35-45 only enjoyed half the chance [in reemployment after losing job] as compared with individuals only a little younger. And what of the decades 45-55 and 55 to 65?"[135] But the body anatomic was different. It did not have any "age discrimination," because

> All cells begin to work in particular ways, gradually, when they become able to do so…. Many cells function during reproductive maturity, others

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[132] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 90(127), 158(158), undated, Box 142, Folder 1, EVC.

[133] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 20(49), undated, Box 142, Folder 1, EVC.

[134] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 107(2)-108(3), undated, Box 142, Folder 1, EVC. In this respect, Cowdry followed the new view on "in vitro life" initiated by Alexis Carrel's tissue culture experiments. Hannah Landecker has argued that Carrel's demonstration of in vitro life made a substantial departure from Claude Bernard's concept of *milieu intérieur*, which was based on the traditional notion of organic integrity. See Hannah Landecker, "New Times for Biology: Nerve Cultures and the Advent of Cellular Life in Vitro," *Studies in History and Philosophy of Biological and Biomedical Sciences* 33 (2002), pp. 667-694.

[135] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56(83), undated, Box 142, Folder 1, EVC.

> (polymorphonuclear leucocytes) after it, and still others (red blood cells) after they have died.[136]

While Cowdry's estimate of the rate of unemployment of older age groups might not have been quite precise, historians have generally agreed that age discrimination in the job market was a reality, particularly during the 1930s.[137] The above paragraph shows how this social reality was combined with his biosocial vision, which had developed through his contact with his colleagues at Chicago and Woods Hole and his cytological research on cellular aging. Nature was a source of wisdom and guidance, even though not every feature of it was acceptable as a model. He continued,

> Many aged and dead cells are not consigned to oblivion. They still serve the rest and are given positions of great importance. Firmly bounded together in a dense layer on the surface of the skin, dead epidermal cells act as a shield and protect the living cells within. "While we are in life we are in death" is a true saying….To summarize, as far as labor is concerned, the body anatomic is a community of cells in a kind of moving equilibrium as it passes through phases of youth, maturity and old age. The division of labor is stabilized….and the labor is spread fairly evenly among all of them so that there is no division into employed and unemployed. Far greater equality is provided among cells in a given labor class than among laborers of the same sort in the body politic.[138]

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[136] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 55B(82), undated, Box 142, Folder 1, EVC.

[137] Even Haber and Gratton have agreed on the reality of age discrimination in the job market, although they have not emphasized it in their book. See Haber and Gratton, *Old Age and the Search for Security*, p. 114. Also see MacNicol, *Age Discrimination*, pp. 211-216; Achenbaum, *Old Age in the New Land*, pp. 127-131; Achenbaum, *Social Security*, pp. 14-18; Graebner, *A History of Retirement*, pp. 181-214. For a study of age discrimination in earlier periods, see Judith C. Hushbeck, *Old and Obsolete: Age Discrimination and the American Worker, 1860-1920* (New York: Garland, 1989).

[138] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 58(86)-59(87), undated, Box 142, Folder 1, EVC.

Cowdry argued that the body politic's neglect of its elderly members could be easily accounted for. "There is a taboo," he wrote.[139] The elderly "are on the downward path and we don't like to think that we shall follow in their footsteps."[140] Hence, people tended to "pay them a small dole" and "shrug our shoulders, saying death is inevitable anyway and pass by on the other side."[141] Indeed, "we turn from them to beautiful, starry eyed children full of promise for the future."[142]

This was the problem of aging Cowdry mentioned in his letter to Ludwig Kast of the Macy Foundation. According to Cowdry, "the passing generation, in its "second childhood"….is expected to retire gracefully without complaint and with no assistance."[143] "Obviously this is all wrong," Cowdry argued.[144] What was urgently demanded was "another project logically following" the arteriosclerosis project.[145] As he wrote in "Citizen Cells," "what we need is a systematic study of the problem of the aged" in order to "profit from the many ways that aged persons can serve and then with proper safeguards to ease their departure."[146] He stated that Kast would "appreciate the magnitude of the task and how fruitful a propitious beginning might be."[147] Yet Kast did not immediately promise his support. Although Cowdry's letter was "most interesting" and "has been constantly in [his] mind," there were also "a few "cons" which [he wanted] to think over."[148] So Cowdry wrote again on October 28, 1935, this time with a list of questions on aging. This list included social, psychological, medical, and biological queries, such as

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[139] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56(83), undated, Box 142, Folder 1, EVC.
[140] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56(83), undated, Box 142, Folder 1, EVC.
[141] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56A(84), undated, Box 142, Folder 1, EVC.
[142] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56A(84), undated, Box 142, Folder 1, EVC.
[143] Cowdry to Kast, October 9, 1935, Box 31, Folder 9, EVC.
[144] Cowdry to Kast, October 9, 1935, Box 31, Folder 9, EVC.
[145] Cowdry to Kast, October 9, 1935, Box 31, Folder 9, EVC.
[146] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 56A(84), undated, Box 142, Folder 1, EVC.
[147] Cowdry to Kast, 9 October 1935, Box 31, Folder 9, EVC.
[148] Kast to Cowdry, 15 October 1935, Box 31, Folder 9, EVC.

8. What arrangements are made for the care of the aged by (1) private organizations, religious and otherwise, and by (2) municipal, state and federal governments?
14. What are the shining examples of great service in government by the aged?
16. In what ways does the mind of an aged person react differently to the same situation from the minds of a mature and of a youthful person?
32. Does the body age as a unit, or may a youthful thyroid, a mature pituitary and a senile liver be forced to labor together for the preservation of the whole?
35. Why is cancer often less malignant in extreme old age?
38. Do studies on senescence in lower forms….afford any clues as to the process in humans?[149]

He further wrote, "The real and obvious reason why old age is so tragically neglected is that its contemplation is depressing, even painful."[150] People had often turned "from [a] sad-eyed and driveling old man believing, perhaps honestly, that but little can be done for him."[151] However, "the great advantage of a Foundation like yours is that it can foster an unpopular cause if convinced that it affords a real opportunity for service."[152] These statements, along with the above questions for further research, finally moved Kast. He replied, "The problem as you conceive it in its major implications is of course a very timely and in many ways an urgent problem and an inquiry into this problem may lead to a very fine piece of work."[153] This problem was timely, Kast wrote later, because of "the anxieties and insecurities of our present social-economic situation" as well as the increase of "the proportion of our population over 40 years of age" suffering from inadequate care.[154] With this approval of Kast, Cowdry began his new handbook project on the current state of research on senescence.

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[149] Cowdry to Kast, 28 October 1935, Box 31, Folder 9, EVC.
[150] Cowdry to Kast, 28 October 1935, Box 31, Folder 9, EVC.
[151] Cowdry to Kast, 28 October 1935, Box 31, Folder 9, EVC.
[152] Cowdry to Kast, 28 October 1935, Box 31, Folder 9, EVC.
[153] Kast to Cowdry, 31 October 1935, Box 31, Folder 9, EVC.
[154] Kast to MacNider, 17 March 1937, Box 10, Folder 380, WDM.

It was this project that initiated the Macy Foundation's long-term support of gerontology, which continued for more than twenty years after the 1930s.[155] Fortunately, Cowdry and the Foundation were ideal partners, since the Foundation valued communication and cooperation of the people involved in the projects, as Cowdry did in editing his textbooks.[156] The next section will describe how Cowdry, with the Foundation's assistance, constructed gerontology as a multidisciplinary scientific field after 1935.

Problems of Ageing, Woods Hole Conference, and the Making of Multidisciplinarity in Gerontology, 1936-1940

With support of the Josiah Macy Foundation, Cowdry edited *Problems of Ageing* (1939), a monumental book in the history of American gerontology. For Katz, Cowdry's book was one of the first successful "textual formations" in gerontology, which brought together diverse institutions, curricula, scientific expertise, and academic power relations in one place and "naturalized" their associations.[157] Achenbaum has also argued that the contributors to *Problems of Ageing* "helped to establish professional organizations and research institutes that remain in operation to this day."[158] As he has correctly pointed out, this remarkable book reflected Cowdry's "own professional style" that had been developing since he had edited *General Cytology*.[159] Indeed, he continued his editorial job in an advanced form to produce *Problems of Ageing*.

Basically, the chapters in *Problems of Ageing* were similar to those of Cowdry's previous books in terms of the subjects and organization. As his *Special Cytology* dealt with the cells in the skin, blood, heart, bone, ovary, testes, thyroid, renal system, and

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[155] Admittedly, the Foundation was supporting several small projects on arteriosclerosis. But these were not gerontology programs in a strict sense. See Achenbaum, *Crossing Frontiers*, p. 64. See Josiah Macy, Jr. Foundation, *Twentieth Anniversary Review of the Josiah Macy, Jr. Foundation* (New York: The Josiah Macy, Jr. Foundation, 1950), p. 31.

[156] For Macy Foundation's emphasis on cooperation and interaction, see Josiah Macy, Jr. Foundation, *A Review of Activities, 1930-1955* (New York: The Josiah Macy, Jr. Foundation, 1955), pp. 17-18; Fremont-Smith to Cowdry, 21 November 1947, Box 41, Folder 8, EVC.

[157] Katz, *Disciplining Old Age*, pp. 77-103.

[158] Achenbaum, *Crossing Frontiers*, p. 53.

[159] Achenbaum, *Crossing Frontiers*, p. 63.

nerve system, *Problems of Ageing* included the chapters on the aging of the skin, cardiovascular system and blood, skeleton, female and male reproductive system, thyroid, urinary system, and brain. The mission of Cowdry's cytology project—precise description of various portions of the living organism through the cooperation of specialists on each part—was transferred to the study of senescence.

Another notable point of this continuity could be found in Cowdry's choice of contributors. Many of them were recruited from those who had already participated in his previous book projects—including Walter Cannon (*Human Biology*), Alfred Cohn (*Special Cytology*, *Arteriosclerosis*), John Dewey (*Human Biology*), Clark Wissler (*Human Biology*), E. B. Krumbhaar (*Special Cytology*), and T. Wingate Todd (*Special Cytology*). Edgar Allen, who edited *Sex and Internal Secretions* (1932) with Cowdry's substantial assistance, wrote the chapter on the aging of the female reproductive system. The other contributors usually recruited through the above scholar's recommendations. For example, Walter R. Miles, a psychologist who had initiated the Stanford Studies of Later Maturity in 1928,[160] was recommended by Allen as a contributor to Cowdry's project.[161] William deB. MacNider of the University of North Carolina also joined the project as an expert on the senile changes of tissue susceptibility and stability. Cowdry also recruited Clive McCay of Cornell University, who discovered in 1935 that a reduced caloric intake increased the longevity of animals, and Herbert Spencer Jennings of the Johns Hopkins University, who investigated the aging of protozoa since the 1920s.

A remarkable feature of these scholars is that many of them were already conducting research on senescence before being invited by Cowdry. Besides McCay and Jennings whose works have been discussed in chapters one and two, there were a number of scientists in the United States who had studied aging as a serious research subject.

Alfred Cohn was among these scientists.[162] As a renowned cardiologist trained under Ludwig Aschoff—a German scientific authority on arteriosclerosis—he joined the newly established Rockefeller Institute Hospital in 1910 and studied electrocardiography,

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[160] This study has barely been studied by historians of science. See Birren, "A Brief History," pp. 74-75.
[161] Cowdry to Allen, 22 January 1937, Box 34, Folder 4, EVC.
[162] For a brief biography of Cohn, "Alfred E. Cohn," *Profiles in Gerontology*, p. 86.

heart diseases, and the effects of digitalis and other therapeutic drugs upon the heart. Cohn became interested in aging because his specialty dealt with age-related diseases in the heart and blood vessels. To him, the age of patients was an important factor in diagnosis and research, which was dependent upon the definition of the "normal" heart and blood vessels at least in a statistical sense. Yet the problem was that the category of the normal in the structure and function of the cardiovascular system varied widely with senescence.[163] Therefore, through his own and other scholars' research, he studied this issue further, such as the age-related alterations in the heart's weight, the valves' rigidity, and the thickness of the endocardium.[164] Probably through this study, Cohn also came to be interested in the biological changes accompanying aging in general. He indeed became familiar with the current state of research on senescence by reading the works of Jennings, Minot, Child, Cowdry, Pearl, and Conklin.[165] In his own article, Cohn agreed with the new perspectives on aging that were promoted by these scholars, especially the notion that aging began with fertilization and proceeded in distinct localities at different rates. Cohn also respected Carrel, his close colleague in the Rockefeller Institute, and accepted his argument that it was possible to culture tissues indefinitely without causing aging and death.[166]

William MacNider had also been conducting various experiements on senescence before being invited by Cowdry.[167] As a professor of pharmacology at the University of North Carolina, he investigated diverse biomedical issues—the diseases of the liver and kidney, the cellular resistance of the organs to chemicals, and the acid-base balance of the blood. In all these investigations, he regarded age as a critical factor that needed to be controlled. MacNider pointed out that "the various ways in which the age of an organism expresses itself has received little consideration in the interpretation of many reactions

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[163] Alfred Cohn, "The Aging of the Heart Muscle Regarded from a General Biologic Point of View," Address Delivered at Annual Graduate Fortnight—The Problems of Aging and of Old Age, New York Academy of Medicine, 3 October 1928, p. 625; "Cardiovascular System and Blood," *Problems of Ageing* (1939), pp. 120-148.
[164] Cohn, "Cardiovascular System and Blood," pp. 126-141.
[165] Cohn and Murray, "Physiological Ontogeny I.," pp. 469-493; "The Aging of the Heart Muscle."
[166] See Cohn to Carrel, 15 October 1935, Box 41, Folder 22, AC.
[167] A brief biographical sketch of MacNider can be found in "William deB. MacNider," *Profiles in Gerontology*, p. 222.

that occur naturally and that are induced experimentally."[168] Indeed, as early as in 1914, he found that senile dogs inoculated with uranium nitrate, if compared with younger dogs with the same inoculation, showed a larger amount of fat deposits in the liver and kidney and a higher glucose level in blood.[169] He also found that anesthetics such as ether and chloroform disturbed the acid-base balance in blood more severely in aged dogs whose "depletion in the alkali reserve of the blood" following "the use of these anesthetics is more marked than in younger animals."[170] Yet his research showed that old animals were not necessarily "weaker" than younger ones. After a series of experiments which revealed that uranium nitrate produced altered liver epithelial cells that conferred resistance against the intoxication by alcohol and chloroform, he found that many senile animals already had some types of liver cells which were similar to these altered cells, and this condition made the liver more resistant to the harmful action of chloroform.[171]

MacNider, who would become the first president of the Gerontological Society and the Club for Research on Ageing, played a more important role in terms of the human network than other contributors chosen by Cowdry. He was able to share the results of his experimental studies of aging and his enthusiasm about them with many researchers who would later contribute to Cowdry's book and to other gerontology projects. He came to know them through various national scientific and medical organizations he participated in, such as the National Academy of Sciences, the American Academy of Arts and Sciences, and the Society for Experimental Biology and Medicine for which he served as president in 1941.[172] In addition, he knew well and regularly

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[168] William deB. MacNider, "A Consideration of the Relative Toxicity of Uranium Nitrate for Animals of Different Ages. I.," *Journal of Experimental Medicine* 26 (1917), p. 1.

[169] William deB. MacNider, "On the Difference in the Response of Animals of Different Ages to a Constant Quantity of Uranium Nitrate," *Proceedings of the Society for Experimental Biology and Medicine* 11 (1914), pp. 159-162.

[170] William deB. MacNider, "A Preliminary Paper on the Relation between the Amount of Stainable Lipoid Material in the Renal Epithelium and the Susceptibility of the Kidney to the Toxic Effect of the General Anesthetics," *Journal of Pharmacology and Therapeutics* 42 (1921), p. 317.

[171] William deB. MacNider, "The Resistance of Fixed Tissue Cells to the Toxic Action of Certain Chemical Substances," *Science* 81 (1935), pp. 601-605; "Concerning the Naturally Acquired Resistance of the Livers of Certain Senile Dogs to Alcohol and to Chloroform," *Proceedings of the Society for Experimental Biology and Medicine* 30 (1932), pp. 237-238.

[172] Moreover, MacNider was president of the American Society for Pharmacology and Experimental Therapeutics from 1931 to 1933, and the honorary president of the International Anesthesia Research

corresponded with Walter Cannon, Henry Simms, George Lawton, A. Baird Hastings, J. A. Gunn, Raymond Pearl, Edwin Conklin, and Anton Carlson.[173] Moreover, MacNider maintained a close relationship with the officers of the Macy Foundation which funded Cowdry's new volume, namely, Ludwig Kast, Lawrence K. Frank, and Frank Fremont-Smith.[174]

Cowdry sometimes could not garner a full agreement about his way of editing the book from all these contributors, and MacNider was a case in point. As has been stated, the chapters in *Problems of Ageing* were organized according to the types of cells, tissues, and organs, following the structure of *Special Cytology*. But MacNider had a different idea about this issue as a pharmacological physiologist. He claimed, "My feeling is that it would be unfortunate for an author to pick out and isolate an organ or tissue and try to see it in a detached fashion in terms of senility."[175] He continued, "If we are to understand the ageing processes, it strikes me that we should attempt to see them as a whole."[176] He then requested that at least his chapter would approach aging from a general physiological standpoint on the whole body rather than focusing on a particular organ or tissue. Cowdry consulted Frank Fremont-Smith of the Josiah Macy Foundation on this matter and allowed MacNider to write about "aging processes considered in relation to tissue susceptibility and resistance" rather than about certain organs or tissues.[177]

Society from 1933 to 1934. See "Academic Record of William deBernier MacNider," Box 8, Folder 320, WDM; Edmond J. Farris to MacNider, 2 September 1941, Box 15, Folder 569, WDM.

[173] See, for example, MacNider to Cannon, 25 February 1936, Box 9, Folder 337, WDM; Simms to MacNider, 30 July 1937, WDM; MacNider to Lawton, 2 November 1938, WDM; Hastings to MacNider, 7 April 1936, Box 9, Folder 341, WDM; MacNider to Gunn, 28 February 1936, Box 9, Folder 337, WDM; Carlson to MacNider, 4 February 1935, WDM; MacNider to Pearl, 4 June 1937, Box 10, Folder 393, WDM; MacNider to Conklin, 28 January 1938, Box 11, Folder 424, WDM; MacNider to Conklin, 21 January 1943, Box 16, Folder 629, WDM. Gunn was a member of the British Club for Research on Ageing. Carlson, MacNider, and Pearl were members of the Commission for Scientific Investigation of the Effects of Alcohol. See Carlson to MacNider, undated, Box 12, Folder 463, WDM.

[174] In particular, Fremont-Smith was a close colleague of MacNider and discussed with him various issues on aging and other scientific topics. See, for example, Fremont-Smith to MacNider, 10 June 1938, Box 11, Folder 439, WDM; MacNider to Fremont-Smith, 14 June 1938, Box 11, Folder 440, WDM.

[175] MacNider to Cowdry, 22 February 1937, Box 31, Folder 8, EVC.

[176] MacNider to Cowdry, 22 February 1937, Box 31, Folder 8, EVC.

[177] Cowdry to MacNider, 3 March 1937, Box 31, Folder 8, EVC; William deB. MacNider, "Ageing Processes Considered in Relation to Tissue Susceptibility and Resistance," *Problems of Ageing* (1939), pp. 695-716.

Cowdry's interactive and cooperative editorial style, which had been developing since his editing of *General Cytology*, led him to be more flexible on the organization of the book.

Cowdry continued to interact actively with the contributors on various issues. For example, Cowdry asked the following questions after receiving A. J. Carlson's chapter on the aging of internal secretion system.

> There are just two questions I want to ask. You say that the blood calcium appears to remain within normal limits up to 80 years or beyond. Greisheimer et al….describe a fall in serum Ca. from 11.6 mg. to 10.0 mg. in men and from 11.8 mg. to 9.7 mg. in women as age progresses. Do you question this decrease or do you interpret it as within normal limits? Is there any evidence that the reactivity, or responsiveness of various tissues to hormones, other than reproductive, decreases with age? The reason why I am curious is that Allen and Engle both describe decrease in responsiveness of the female and male reproductive systems to pituitary gonadotropic and ovarian follicular hormones.[178]

Carlson replied to this question very briefly: "1. Serum calcium of from 10-11 is essentially within the normal limit. 2. The evidence for decreased response of tissues to hormones other than the gonad hormones is hardly worth considering at present, because of the fragmentary data and uncontrolled material."[179] But Carlson could not understand why Cowdry was interested in such details of his work. In fact, Carlson was not familiar with Cowdry's style of editorship since he had not participated in any of Cowdry's previous projects. So he added after his answer to Cowdry's questions, "Brother, you are just too enthusiastic about my chapter."[180]

Collecting and distributing the summaries of all the chapters to encourage cooperation was another aspect of Cowdry's editorial work that continued since he had edited *General Cytology*. Through this, the contributors assisted, and were also helped by,

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[178] Cowdry to Carlson, 19 July 1938, Box 25, Folder 23, EVC.
[179] Carlson to Cowdry, 20 July 1938, Box 25, Folder 23, EVC.
[180] Carlson to Cowdry, 20 July 1938, Box 25, Folder 23, EVC.

others with different background in writing their chapter. For example, physiologist Walter Cannon asked cardiologist Alfred Cohn whether the aged heart responded to stress "by dilating and beating more rapidly than normally, whereas the effective athletic heart responds by a greater degree of emptying and not so much by acceleration."[181] Cohn answered that his current research project was dealing with this question, and wrote that the more aged the heart was, the more the degree of increase of the pulse rate during exercise.[182] Psychologist Walter Miles also sent pharmacologist William MacNider an article on aging that he thought "might be of some service to [MacNider] in the preparation of [his] chapter."[183] MacNider thanked Miles for sending the paper.[184] At the same time, MacNider asked botanist William Crocker about "mitotic figures of an abnormal order" in plant cells, which could lead to modified cell types.[185] MacNider was interested in this phenomenon, because he observed in animals that similar mitotic figures were associated with altered cell types, which persisted longer in older cells and contributed to enhancing the overall defense of the organism against toxic chemicals.[186] Crocker wrote to MacNider what he thought about this phenomenon and detailed his current and future research on the topic.[187]

But Cowdry's efforts sometimes engendered a clash rather than cooperation among the contributors, and he did his best to lead the debate to a constructive conclusion. For example, MacNider was not very satisfied by Jean R. Oliver's abstract of his chapter on the aging of the urinary system. On August 30, 1937, MacNider wrote, "It strikes me that the trouble that he is having is dependent upon his assumption that all tissue changes which depart from a hypothetical norm are essentially pathological."[188] On September 22, MacNider criticized Oliver again because he did not "realize the type of readers which [Cowdry] and the Macy people—and I trust the other contributors to the book—are

[181] Cannon to Cohn, 26 October 1937, Box 5, Folder 5, AEC.
[182] Cohn to Cannon, 29 October 1937, Box 5, Folder 5, AEC.
[183] Miles to MacNider, 8 December 1937, Box 11, Folder 417, WDM.
[184] MacNider to Miles, 29 June 1938, Box 11, Folder 441, WDM.
[185] MacNider to Crocker, 1 July 1937, Box 10, Folder 398, WDM.
[186] MacNider to Fremont-Smith, 14 June 1938, Box 11, Folder 440, WDM.
[187] MacNider to Crocker, 29 June 1938, Box 11, Folder 441, WDM.
[188] MacNider to Cowdry, 30 August 1937, Box 31, Folder 8, EVC.

hoping to reach through their chapters."[189] While the book was targeted to more general educated readers as well as professional scientists, Oliver's chapter was too technical for the former to appreciate. Yet it did not mean that his chapter would be impressive for the professionals, since Oliver failed to "incorporate in his abstract an adequate amount of experimental material, as indicated by the relatively few references he gives to the changes in the kidney which he is in the process of discussing."[190] Cowdry did not send this critical note of MacNider to Oliver, even though he usually transmitted each contributor other's comments.[191] But MacNider himself personally sent Oliver a telegram, requesting the list of reference he mentioned in his letter to Cowdry.[192] Although Oliver immediately sent MacNider a list of relevant publications as far as he knew on the topic,[193] such an abrupt act of MacNider embarassed Cowdry. He gently wrote to MacNider, "It is my impression that you are perhaps taking your task too seriously."[194] What was more appropriate for *Problems of Ageing* was a general summary of the current state of research rather than "a very long or detailed account of the subject."[195] At the same time, he wrote to Oliver that "it is unavoidable that there will be duplications and also omissions" and this was "the weakness in the kind of presentation we are making."[196] Cowdry's job was to reduce this weakness "to promote cooperation between the authors."[197]

But a more heated controversy occurred between Walter Cannon and Cowdry himself as a contributor. Although Cowdry wrote to Cannon that Cowdry's chapter on the aging of tissue fluids was "built upon [Cannon's] very interesting account of the ageing of homeostatic mechanisms,"[198] Cannon suddenly criticized Cowdry's "fantastic hypothesis" that "if the environment of cells were uniform the division of labor among

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[189] MacNider to Cowdry, 22 September 1937, Box 31, Folder 8, EVC.
[190] MacNider to Cowdry, 22 September 1937, Box 31, Folder 8, EVC.
[191] Cowdry to Oliver, 28 September 1937, Box 33, Folder 4, EVC. But Cowdry sent Oliver Walter Cannon's and Alfred Cohn's comments.
[192] See Oliver to Cowdry, 3 January 1938, Box 33, Folder 4, EVC.
[193] Oliver to MacNider, 3 January 1938, Box 11, Folder 421, WDM.
[194] Cowdry to MacNider, 5 January 1938, Box 11, Folder 421, WDM.
[195] Cowdry to MacNider, 5 January 1938, Box 11, Folder 421, WDM.
[196] Cowdry to Oliver, 5 January 1938, Box 33, Folder 4, EVC.
[197] Cowdry to Oliver, 5 January 1938, Box 33, Folder 4, EVC.
[198] Cowdry to Cannon, 6 May 1938, Box 25, Folder 22, EVC.

them would be quite impossible."[199] He argued that "this assumption seems to contradict all we know about the relations of structure and function," and asserted that "if cells are different in structure they will be different in function, even if the environment is the same."[200] Basically, Cowdry wrote that each cell matured and aged at a distinct rate because it was immersed in its peculiar local fluid environment.[201] This argument had developed from his earlier cytological interest in cell aging and the influence of Alexis Carrel's experiments. However, Cannon, a physiologist, was interested in how the old age of an organism brought about the gradual disruption of its homeostatic mechanisms—such as the acid-base equilibrium of the blood plasma—which allegedly controlled *all* portions of the body through blood and lymph.[202] He indeed delivered a talk on this issue in the William Henry Welch Lectures and included a discussion on the senile disruption of homeostasis in the second edition of *The Wisdom of the Body* published in 1939.[203] Yet if the homeostatic mechanisms and their aging were deeply related to the regulation of the life and senescence of every cell in the body, how then could each cell develop and maintain its peculiar identity? Cannon wrote that as different protozoa in a pond could keep their self despite the same fluid environment where they lived, cells in multicellular organisms could also maintain their distinct character although they constantly contacted the blood and lymph.[204] But Cowdry pointed out that this statement of Cannon missed the point because each protozoan organism in a pond had a distinct genetic constitution while the cells in a metazoan animal had the same gene sets.[205] The somatic cells nevertheless differentiated into distinct types, because they lived in their unique local fluid environment.

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[199] Cannon to Cowdry, 6 June 1938, Box 94, Folder 1302, WBC; Cannon to Cowdry, 24 June 1938, Box 94, Folder 1302, WBC. While reporting this criticism to Fremont-Smith of the Macy Foundation, Cowdry wrote that his "position as editor is rather embarrassing" and it is "with great reluctance that [he disagrees] with any contributor." See Cowdry to Fremont-Smith, 18 June 1938, Box 94, Folder 1302, WBC.
[200] Cannon to Cowdry, 6 June 1938, Box 94, Folder 1302, WBC; Cannon to Cowdry, 24 June 1938, Box 94, Folder 1302, WBC.
[201] Cowdry, "Ageing of Tissue Fluids," *Problems of Ageing* (1939), pp. 642-694.
[202] Walter B. Cannon, "Ageing of Homeostatic Mechanisms," *Problems of Ageing* (1939), pp. 623-641.
[203] MacNider to Cannon, 25 January 1940, Box 82, Folder 1108, WBC.
[204] Cannon to Cowdry, 24 June 1938, Box 94, Folder 1302, WBC.
[205] Cowdry to Cannon, 27 June 1938, Box 25, Folder 22, EVC.

Cannon, however, was not persuaded. He wrote again that Cowdry did not yet provide "any convincing evidence that the environment is different for many different kinds of cells in many different parts of the body."[206] He also asserted that he failed "to see how [Cowdry] could expect the tissue fluid which escapes through the capillary wall to be very different in one region as compared with another, unless there is demonstrable difference in the structure of the cells in the capillary wall."[207] Moreover, Cannon asked, "Even in the 'same fluid blood serum environment,'….do not the various cells of tissue cultures 'maintain their distinctive structure'?"[208] Through his cytological knowledge, Cowdry defended his position from these questions. First—while pointing out that he did not use the word "very" or "many" in his chapter—he cited recent cytological research which indicated that cells in the spleen and connective tissue were surrounded by unique fluid environments which chemically differed from other portions of the body.[209] Second, he pointed out that capillaries were not the sole blood vessel through which fluids could escape the blood stream into the local tissue environment. Other larger blood vessels also allowed the exchange of fluids between tissue and blood and each of them had different permeability due to its distinct structure.[210] Third, he argued that his colleague Alexis Carrel had already shown that different types of cells needed different fluid media to be cultured since blood plasma was not an adequate medium for any kind of cells.[211] For Cowdry, Carrel's experiment was an *in vitro* proof of Cowdry's idea that local tissue fluid environment controlled the cellular differentiation and aging. As an example, he wrote that red blood cells and lymphocytes matured and aged in their distinct local surroundings apart from the blood plasma and were released into the blood stream only

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[206] Cannon to Cowdry, 6 July 1938, Box 25, Folder 22, EVC.
[207] Cannon to Cowdry, 6 July 1938, Box 25, Folder 22, EVC.
[208] Cannon to Cowdry, 12 July 1938, Box 25, Folder 22, EVC.
[209] Cowdry to Cannon, 8 July 1938, Box 25, Folder 22, EVC. For the original sources of this evidence, see Sylvia H. Bensley, "On the Presence, Properties, and Distribution of the Intercellular Ground Substance of Loose Connective Tissue," *Anatomical Record* 60 (1934), pp. 93-110; M. H. Knisely, "Spleen Studies. I. Microscopic Observations of the Circulatory System of Living Unstimulated Mammalian Spleens," *Anatomical Record* 65 (1936), pp. 23-50.
[210] Cowdry to Cannon, 8 July 1938, Box 25, Folder 22, EVC; Cowdry to Cannon, 16 July 1938, Box 25, Folder 22, EVC.
[211] Cowdry to Cannon, 16 July 1938, Box 25, Folder 22, EVC.

after they became very aged or nearly dead.[212] Red blood cells could do their function "when they are dead or nearly so," and lymphocytes, when they were allowed to work, "are so old that they have lost their ability to multiply."[213] Receiving these answers, Cannon responded that he would reply to Cowdry after "some further examination of data."[214] But Frank Fremont-Smith of the Macy Foundation, who knew of the controversy, asked both of them to stop, since "controversial material is out of place in this cooperative venture" and "the question is largely one of emphasis" rather than something in need of a definite answer.[215] While it is not certain whether Cannon and Cowdry agreed with Fremont-Smith on this matter, the debate was not continued thereafter.

In retrospect, controversies of this kind are quite common in scientific communities and often indicate that they are in a healthy state. But controversies can also be damaging to a community, especially during its early phase when its institutional norms have not been established. In this sense, Fremont-Smith's involvement could be thought to be an appropriate way to protect the fledgling community of researchers of aging from being disrupted due to a heated internal debate.

One of the most important causes of this internal debate between Cowdry and Cannon was the multidisciplinarity of gerontology which included the two scientists who had different scientific backgrounds and distinct prescriptions on the problems of the "body politic." As a cytologist, Cowdry was interested in the study of *local* objects while Cannon as a physiologist studied the changes in the *whole* body for which homeostatic mechanisms were responsible. Interestingly, this difference was related to what they thought about desirable societies. Based on his physiological research, Cannon argued in *The Wisdom of the Body* that the homeostatic mechanism of the "body physiologic" could be a model of ensuring the stability of the "body politic."[216] While Cowdry agreed with this idea of Cannon in many respects, he slightly differed on why the "body anatomic"—

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[212] Cowdry to Cannon, 16 July 1938, Box 25, Folder 22, EVC.
[213] Cowdry to Cannon, 16 July 1938, Box 25, Folder 22, EVC.
[214] Cannon to Cowdry, 2 August 1938, Box 25, Folder 22, EVC.
[215] Fremont-Smith to Cannon, 21 July 1938, Box 94, Folder 1302, WBC.
[216] Cannon, *The Wisdom of the Body* (1932), pp. 298-306.

rather than the "body physiologic"—could be a good model for human's social reorganization. Whereas Cannon thought that homeostatic mechanisms controlling the internal stability of the body could be referred to in maintaining an order in the human society, Cowdry thought that this aspect of the cell community, despite several strong points, was too similar to a "totalitarian state."[217] To Cowdry, what was more important than homeostatic mechanisms as a model of a better body politic was the *diversity* of *local* environments in the body anatomic. As a cytologist, he observed that most cells "live outside the [bloodstream] in what is called tissue fluid" which provided peculiar local fluid environment to its resident.[218] The cells in such environments cooperatively contributed to the making of their own living conditions and the survival of the whole body while satisfying their own needs. In these aspects, particularly in terms of fulfilling the needs of their local residents, the body politic was far behind the body anatomic.[219]

The two scientists drew different conclusions on the problems of old age through these distinct models of analogy. Cannon, supporting the value of the "sacrifice of lesser for greater values" and "lessening of the independence" of the individual for larger social benefits,[220] thought that aged cells were not useful members of the body physiologic, because their death eventually contributed to the demise of the whole body. While the body politic usually didn't need to worry much about death like the body physiologic, the aged individuals were still not useful members and their death was "a means of ridding society of old members in order to yield places for the new."[221] Cowdry, who valued local diversity, had a very different idea on this issue. For him, many kinds of aged cells, such as red cells and lymphocytes, were produced from the diversified local environments and served important functions for the whole body. Indeed, red cells and

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[217] Cowdry, "Biological Basis of the New Deal," p. 8, undated, Box 172, Folder 8, EVC.
[218] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 107(2), undated, Box 142, Folder 1, EVC.
[219] Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," pp. 108(3)–110(5), undated, Box 142, Folder 1, EVC.
[220] Cannon, *The Wisdom of the Body* (1932), pp. 304-305. Cannon did not change this standpoint in the second edition of the book. See Walter B. Cannon, *The Wisdom of the Body* (New York: W. W. Norton, 1939), pp. 322-323.
[221] Cannon, *The Wisdom of the Body* (1932), p. 302. Also see Cannon, *The Wisdom of the Body* (1939), p. 320.

lymphocytes were already old at the time of release into bloodstream, and therefore, the former could live only for 40 days before death, and the latter less than 20 hours. Yet they carried out significant functions such as carrying oxygen and defending the body from parasites.[222] To Cowdry, what could be learned about the problems of aging from the body anatomic was this aspect which ensured and demanded the continuous use of its elderly members.

Such a view on aged people and cells, which was absent in Cannon's view on the relation of the body politic and physiologic, was perhaps less influential than Cannon's because Cowdry did not publish it.[223] However, Cowdry's worries about the social place of elderly people which appeared in his unpublished writings led him to recruit to his project the scholars with similar concerns. For example, John Dewey, in his introduction to *Problems of Ageing*, wrote how the employment in old age became a social problem during "the recent economic crisis."[224] He wrote, "persons above fifty are experiencing ever greater difficulty in finding employment" as well as getting a new job after being laid off from the workplace.[225] However, Dewey continued, since "conservatism increases with age….at just the time when measures of social readjustment are most needed, there is an increasing number of those whose habits of mind and action incline them to resist policies of social readjustment."[226] For Dewey, the problem was both biological and social in nature, because elderly people's decline of biological capacity occurred in the social space. Therefore, the solution required knowledge about "the ways in which social contexts react back into biological processes as well as.….the ways in which the biological processes condition social life. This is the problem to which

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[222] Cowdry to Cannon, 16 July 1938, Box 25, Folder 22, EVC. Also see Cowdry, "Citizen Cells: How Cells Manage Their Social Problems," p. 58(86), undated, Box 142, Folder 1, EVC.

[223] On the influence of Cannon's theory of social homeostasis, see Cross and Albury, "Walter B. Cannon, L. J. Henderson, and the Organic Analogy," pp. 165-192. Actually, Cannon's view on elderly people was changing. While neglecting the problem of social place of elderly people in the above statement drawn from the first and second editions of *The Wisdom of the Body*, he did discuss "the social and industrial needs of the older man," particularly the "speed-up neurosis" of aged factory workers, during the first meeting of the Club of Research on Aging in 1940. See "Meeting of the Club for Research on Ageing," p. 5, 11-12 January 1940, Box 41, Folder 2, EVC.

[224] Dewey, "Introduction," *Problems of Ageing* (1939), p. xxi.

[225] Dewey, "Introduction," *Problems of Ageing* (1939), p. xx.

[226] Dewey, "Introduction," *Problems of Ageing* (1939), p. xxi.

attention is invited."[227] Cowdry wrote to Dewey that his mention of elderly people's socio-economic situation was "of utmost importance."[228] Clark Wissler of the American Museum of Natural History was another contributor who was also interested in larger social and cultural aspects of aging. In his chapter on "Human Cultural Levels," Wissler wrote how different cultures had distinctive customs concerning the treatment of their elderly members. He argued that "all societies have formulated concepts of age capacity and treated the individual accordingly. Since no society ignores age changes, it seems safe to assume that they are deeply enmeshed in every form and state of culture."[229] He then described the customs concerning old age in many different societies, including those of Ainu, Eskimo, and Tasmanian. He argued that aging was not a pure biological phenomenon, because it was defined and explained in different ways according to the culture of a society.

Louis I. Dublin, a statistician and vice-president of the Metropolitan Life Insurance Company, was also invited as a contributor, because he was a prominent statistician interested in aging and his view was similar to Cowdry's. Dublin had become deeply interested in aging through his job in the life insurance business. As one of the earliest vital statisticians in the country, he closely traced population trends in America, noticing the increasing proportion of the elderly people over the first three decades of the twentieth century due to the reduced birthrate, the decrease of infant mortality, and the passing of a stringent immigration law in 1924.[230] Because these factors would influence the future policy of the Metropolitan Life Insurance Company for which he worked, it was necessary to know more about old age and the changes it brought about in terms of health and social environment. Of course, an immediate corporate response to the

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[227] Dewey, "Introduction," *Problems of Ageing* (1939), p. xxvi.
[228] Cowdry to Dewey, 6 May 1938, Box 26, Folder 42, EVC.
[229] Clark Wissler, "Human Cultural Levels," *Problems of Ageing* (1939), p. 98.
[230] During the 1920s and 1930s, numerous articles were published in the Metropolitan Life Insurance Company's *Statistical Bulleting* on the decrease of infant and childhood mortality, the reduction of birth rate and immigration, and the aging of the American population. Dublin was among those who were primarily responsible for these publications. See, for example, "Another Drop in the Birth Rate," *Statistical Bulletin* 17:9 (1936), pp. 1-2; "A Quarter Century of Progress in Longevity," *Statistical Bulletin* 17:9 (1936), pp. 2-4; "World's Fairs: As Milestones of American Longevity," *Statistical Bulletin* 20:3 (1939), pp. 1-3. Also see Louis I. Dublin, "Old Age and What It Means to the Community," *Bulletin of the New York Academy of Medicine* 4 (1928), pp. 1077-1086.

changed age structure would be to adjust its system of charging premiums. Yet finding the ways to improve the elderly's health was also important due to its long-term consequences for the future of the company and the whole country. In fact, the company had been conducting a campaign of teaching healthy ways of living to its young policyholders. Since the body of the aged was different from that of the young, however, a new need for more "knowledge of the processes which produce aging and degeneration" emerged.[231] The company actually studied the relationship between body weight and longevity and published its results, which were cited by later gerontologists.[232] The vital statistics published in the *Statistical Bulletin* of the Metropolitan Life Insurance Company was also favorably received by scientists of senescence.[233] With the coming of the Great Depression, Dublin became interested in another matter, namely, the displacement of the elderly in industries and the need for their proper means of livelihood, which the insurance programs of his company could provide.[234] The displacement problem was actually what interested Cowdry, too, and probably because of this shared view, Cowdry invited Dublin to write a chapter in his new book.[235]

To Cowdry, Lawrence K. Frank and Frank Fremont-Smith of the Josiah Macy, Jr. Foundation were also valuable partners with whom Cowdry should cooperate. As well as paying the contributors the royalty for their chapters, these officers of the Foundation read the drafts of the chapters and gave comments before approving them and reconciled

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[231] Dublin, "Old Age and What It Means to the Community," p. 1086.

[232] See "Conference on Nutritional Requirements for the Ageing Population," p. 10, November 1 and 2, 1941, Folder Nutritional Requirements Conference 1942, Box 1, GS; "Further Facts on Body-Weight and Longevity," *Statistical Bulletin* 4:3 (1923), pp. 2-4.

[233] See, for example, Cohn to Carrel, 5 December 1940; Carrel to Cohn, 10 December 1940, Box 5, Folder 7, AEC. Indeed, Dublin and the Metropolitan Life Insurance Company were the earliest collectors of vital statistics in the United States. See John M. Eyler, "Health Statistics in Historical Perspective," in Daniel J. Friedman, Edward L. Hunter, and R. Gibson Parrish II (eds.), *Health Statistics: Shaping Policy and Practice to Improve the Population's Health* (Oxford: Oxford University Press, 2005), pp. 38-39.

[234] Louis Dublin, "The Care of the Aged," *Forum* (December, 1933), pp. 361-366.

[235] After that, Dublin continued his study of pension, age discrimination, and older people's healthy way of life in relation to insurance business. Louis Dublin, "Economics of the Aged," Address before the American Association of Industrial Physicians and Surgeons, Atlantic City, N. J., 24 April 1951, Box 11, Folder Economics of the Aged, LID; "Retirement Policies and Practices: A Report Prepared by Louis I. Dublin," 21 September 1955, Box 11, Folder Changes of Our Aging Population, LID.

any controversy occurring among contributors.[236] They also helped Cowdry recruit the scholars who were hesitating to join the project. For example, Wissler refused Cowdry's first invitation but accepted it after "some conversation with Mr. Frank" who led Wissler to have "a more intelligent view of what [Cowdry contemplated] in [his] new volume."[237] Cannon also promised to cooperate with Cowdry only after receiving a letter from Ludwig Kast and Frank Fremont-Smith of the Macy Foundation.[238]

With these officers' assistance, Cowdry was able to do what proved to be the most critical event in creating the field of gerontology—holding the first conference on aging at Woods Hole, Massachusetts. Indeed, Cowdry had planned an "informal conference" among some contributors to *Human Biology* and tried to do the same thing while editing *Problems of Ageing*.[239] With the Foundation's support for travel expenses and other costs, however, he was able to hold a formal conference on June 25 and 26, 1937 at Cape Codder Hotel in Woods Hole, where most contributors convened—including Carlson (physiology), Cohn (cardiology), Crocker (botany), Jennings (protozoology), Oliver (pathology), McCay (animal husbandry), MacNider (physiology), Todd (anatomy), Wissler (anthropology), Dublin (statistics), Miles (psychology), E. T. Engle (reproductive science), J. S. Friedenwald (ophthalmology), E. B. Krumbhaar (immunology), and Cowdry himself. (See Figure 5.2.) As the representatives of the Foundation, Lawrence K. Frank and Frank Fremont-Smith participated as well. Moreover, since Cowdry wanted to promote this conference as a national scientific meeting, he invited W. S. Hunter as the person representing the Union of American Biological

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[236] Frank to Cannon, 31 March 1938, Box 94, Folder 1302, WBC; Fremont-Smith to Cannon, 21 July 1938, Box 94, Folder 1302, WBC.

[237] Wissler to Cowdry, 27 February 1937, Box 38, Folder 21, EVC.

[238] Cannon to Cowdry, 17 May 1937, Box 25, Folder 22, EVC; Cowdry to Cannon, 20 May 1937, Box 25, Folder 22, EVC; Kast to Cannon, 14 May 1937, Box 94, Folder 1298, WBC; Cannon to Kast, 17 May 1937, Box 94, Folder 1298, WBC; Fremont-Smith to Cannon, 18 May 1937, Box 94, Folder 1298, WBC; Cannon to Kast, 4 January 1938, Box 94, Folder 1302, WBC. Kast's letter to Cannon was particularly instrumental in changing Cannon's mind, since they were had been close friends, and Cannon had helped Kast in his work for the Foundation since 1931. See Cannon to Dave Morris, 1 Oct 1941, Box 94, Folder 1296, WBC.

[239] Cowdry to Oliver, 17 February 1937, Box 33, Folder 4, EVC.

Societies and E. D. Merrill as the scholar from the National Research Council, even though many contributors already belonged to either or both of the organizations.[240]

The diversity of the fields these people represented was similar to that of the scientists who regularly attended the Marine Biological Station every summer.[241] Their composition was also similar to that of the "cytologists" in Cowdry's previous textbooks, who were actually specialists in their own domains rather than professional cytologists. Moreover, in some sense, the participants were like the cells in Cowdry's "body anatomic," which matured and aged at a distinctive rate in their local environment while contributing to the welfare of the whole body.

During the formal sessions of the conference, these multidisciplinary scholars discussed various biological, medical, psychological, and social issues concerning aging. According to Clive McCay's recollection, they also thoroughly enjoyed leisure activities in their free times, just as biologists and doctors had done at Woods Hole.[242] McCay playfully wrote that Anton Carlson had some problems in joining these leisure activities, since he forgot to bring his swimming suit.

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[240] "Woods Hole Conference on Aging: Preliminary Schedule for Morning and Evening Sessions," Box 94, Folder 1298, WBC; E. V. Cowdry, "Woods Hole Conference on the Problems of Aging," *The Scientific Monthly* 45 (1937), pp. 189-191; Cowdry to MacNider, 10 May 1937, Box 10, Folder 389, WDM. Indeed, he himself was president of the Union and had been chairman of Division of Medical Sciences of the National Research Council during the early 1930s.

[241] Although not mentioned by historians of biology, Cowdry wrote that many physicians, as well as professional biologists, also participated in the research activities at the MBL. See Garrey and Cowdry, "Marine Biological Laboratory Increases Activities," pp. 805-808.

[242] C. M. McCay, "A Student of Aging Looks at the Macy Foundation for Seventeen Years," p. 2, Box 30, Folder Macy Foundation Contribution, NWS. Also see Josiah Macy, Jr. Foundation, *A Review of Activities*, p. 15; *Twentieth Anniversary Review of the Josiah Macy, Jr. Foundation*, p. 33.

Figure 5.2. *The Woods Hole Conference on Aging, 1937, Woods Hole, Massachusetts. Cowdry is sitting on the third chair from the right in the front row. Box 44, Folder Photographs, Professional, 1937-1958, The Nathan W. Shock Papers, Bentley Historical Library, University of Michigan.*

Cowdry intended to use this conference as a forum for furthering discussion among the contributors. Indeed, a few issues brought forth during the conference—such as whether aging was a result of "endless repetition of injury" or "supervention of degenerative disease"—continued to be discussed after it, during the final phase of the book editing.[243] The "editorial policy" Cowdry composed at this time described how his job as an editor contributed to the making of the cooperative multidisciplinary project.

> Summaries of contents of most [chapters] have been received. Summaries, which are still outstanding are urgently requested[,] so that they may be multiplied and sent to all contributors. The contributors receiving them are asked not merely to read them but also to make constructive suggestions leading to improvements. At the Woods Hole Conference an opportunity was afforded for the exchange of

[243] Cowdry to Todd, 2 July 1937, Box 36, Folder 13, EVC; Todd to Cowdry, 12 July 1937, Box 36, Folder 13, EVC.

views. We hope that contributors will pull together by corresponding with each other freely.[244]

Although the official duties of the editor and the contributors ended with the publication of *Problems of Ageing* in January, 1939, their sense of belonging to a community continued. While they had been trained in various different disciplines and worked in distinct institutions, they began to feel that they needed to study aging in a cooperative way. Under the leadership of Cowdry, some of these people—MacNider, Carlson, McCay, Crocker, Jennings, and Krumbhaar—formed the "Committee on the Biological Processes of Ageing" within the National Research Council during March of 1938.[245] (See Chapter 6.) Anton Carlson also suggested that it might be necessary to have a second conference for further investigations after the contributors finished their chapters.[246] Although this suggestion was not immediately realized, an opportunity came when Vladimir Korenchevsky, the Russia-born British gerontologist visited America during the July of 1939. (See Chapter 4.) He had already formed the Club for Research on Ageing in Great Britain, a small discussion group of biomedical scientists and physicians interested in aging. With his recommendation, American scientists of aging also formed a similar organization in the United States—"the American Division of the Club for Research on Ageing."[247] The next section will briefly discuss the early phase of this club and the second and third editions of *Problems of Ageing*.

The Early Years of Gerontology as a Multidisciplinary Scientific Field in America

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[244] E. V. Cowdry, "Editorial Policy," pp. 7-8, 28 July 1937, Box 33, Folder 31, EVC.
[245] Cowdry was the first president of this committee. See MacNider to Cowdry, 21 March 1938, Box 31, Folder 8, EVC; Cowdry to MacNider, 25 March 1938, Box 31, Folder 8, EVC; Cowdry to E. F. Williams, 18 August 1938, Box 42, Folder 22, EVC. The precursor of this committee is the "Committee on Cellular Physiology," which was authorized in June, 1937. Its first meeting was held during the Woods Hole Conference. See Cowdry to MacNider, 17 June 1937, Box 31, Folder 8, EVC; Cowdry to MacNider, 17 June 1937, Box 10, Folder 397, WDM; Cowdry to MacNider, 6 July 1937, Box 10, Folder 393, WDM.
[246] It is highly probable that Carlson's suggestion was made during the Woods Hole Conference. See MacNider to Cowdry, 30 June 1937, Box 31, Folder 8, EVC.
[247] MacNider to McCay, 19 July 1939, Box 6, Folder McCay, Clive [comments listed], NWS.

When the first meeting of the Club was held on January 11 and 12, 1940 at the Willard Hotel, Washington, D.C., many of the contributors to *Problems of Ageing* met again—Cannon, MacNider, Crocker, Cohn, Engle, Hastings, McCay, Oliver, and Cowdry.[248] Although Jennings did not come after moving to California, Cowdry invited another prestigious biologist, Ross Harrison of Yale University, who was a founder of tissue culture techniques and chairman of the National Research Council. Robert A. Moore, Cowdry's colleague at Washington University and the scholar who would become the first editor of the *Journal of Gerontology*, was also present. Moreover, the Club invited Lewis Thompson, director of the National Institute of Health, along with Edward J. Stieglitz, who would be appointed the first head of the NIH gerontology unit which was established through the Macy Foundation's short-term grant. The Club also asked R. E. Coker to join the discussion as Chairman of the Division of Biology and Agriculture of the National Research Council to which the Committee on the Biological Processes of Ageing belonged. Lawrence K. Frank and Frank Fremont-Smith from the Macy Foundation paid these participants' travel expenses and participated in the discussion.

These multidisciplinary scholars discussed various issues. How and why should aging be studied as a scientific and social problem? What were the appropriate experimental organisms to investigate senescence? What did the plant's potential immortality suggest about the nature of aging in general? What was the difference between chronic illness and "normal" aging? What was the change in the reproductive systems during aging and what was the cause of menopause? How did nutrition affect the rate of aging in animals? How did the aging of the population affect industry, and what were American corporations' responses to their aged employees?

Along with these academic problems, the members of the Club discussed several issues related to its policy and administration. First of all, they agreed to maintain the

[248] Minutes of the Meetings of the Club for Research on Ageing, p. 1, 11-12 January 1940, Box 41, Folder 2, EVC.

name suggested by Korenchevsky—the Club for Research on Ageing.[249] At Cowdry's recommendation, MacNider was appointed as the first president of this Club.[250] Cowdry also asked a question that would become important for the future direction of gerontology's development: Was it appropriate and necessary to include social scientists in the Club?[251] Although this question did not lead to any immediate action during the first meeting of the Club, other scholars such as Cannon, Stieglitz, and C. Hartman, showed interest in the social aspects of aging,[252] and Cohn emphasized the importance of understanding the "social background" of senescence as well as its biomedical aspects.[253] It was also decided that the Club should be a small and informal discussion group of multidisciplinary scholars who were seriously concerned about aging.[254] Their annual meetings had to be a roundtable discussion of these scholars, rather than formal presentations of papers by individual scientists.[255] In this sense, the Club was very different from other scientific societies. As Robert Moore aptly put it, this Club was "regarded as an experiment."[256]

Meanwhile, Cowdry kept editing new versions of *Problems of Ageing*. In 1942, its second edition was published with contribution by the members of the Club. In this edition, several influential figures in gerontology newly joined, including psychologist George Lawton, pathologist Robert Moore, and clinician Edward Stieglitz, who returned to his private medical practice after leaving the NIH Gerontology Unit to Nathan Shock. The third edition of *Problems of Ageing* came out ten years later, with a number of new contributors. After Cannon died in 1945, Nathan Shock at the NIH took charge of the

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[249] Minutes of the Meetings of the Club for Research on Ageing, p. 18, 11-12 January 1940, Box 41, Folder 2, EVC.
[250] Minutes of the Meetings of the Club for Research on Ageing, p. 3, 11-12 January 1940, Box 41, Folder 2, EVC.
[251] Minutes of the Meetings of the Club for Research on Ageing, p. 8, 11-12 January 1940, Box 41, Folder 2, EVC.
[252] Minutes of the Meetings of the Club for Research on Ageing, pp. 5, 21, 22, 11-12 January 1940, Box 41, Folder 2, EVC.
[253] Cohn to Frank, 2 February 1939, Box 10, Folder 8, AEC.
[254] Minutes of the Meetings of the Club for Research on Ageing, p. 17, 11-12 January 1940, Box 41, Folder 2, EVC.
[255] Minutes of the Meetings of the Club for Research on Ageing, p. 1, 11-12 January 1940, Box 41, Folder 2, EVC.
[256] Minutes of the Meetings of the Club for Research on Ageing, p. 17, 11-12 January 1940, Box 41, Folder 2, EVC.

chapter on the aging of homeostatic mechanisms. The "St. Louis Group of Gerontology"—John E. Kirk, William B. Kountz, and Albert I. Lansing, who were colleagues of Cowdry and Moore—was another new addition to the list of authors, and Lansing among them actually edited the book even though the book's title was *Cowdry's Problems of Ageing*. But the most remarkable development was the four new chapters on "Social and Economic Aspects of Aging."[257] This was what Cowdry had had in mind, at least vaguely, since he had worked on the first edition of the book. He had invited Dewey, Wissler, and Dublin to write about social implication of aging for the first and second editions.

However, gerontology had already grown beyond the domain of *Problems of Ageing.* While gerontology handbooks continued to be published, the people Cowdry recruited for his book and the Club formed the first professional society of American researchers on aging—the Gerontological Society in 1945. Moreover, as will be discussed in the next chapter, the Gerontology Unit within the NIH became a permanent institution, and Shock, who contributed to the third edition of *Problems of Ageing*,[258] directed the Unit from 1941 until it became the National Institute on Aging in 1974. Research programs for gerontology were created in other academic institutions in the United States, such as Washington University, the University of Chicago, the University of Michigan, and Duke University.

Conclusion

Although Cowdry was not directly involved in all these events, he made substantial contributions to them in many ways. He was a leader of the St. Louis group of gerontology and served the Club for Research on Ageing as president from 1946 to 1948. He was also elected president of the Gerontological Society in 1953 and the International Association of Gerontology in 1951. The origins of these new multidisciplinary organizations he directed can be traced back to what he learned at Chicago and Woods

[257] Albert I. Lansing (ed.), *Cowdry's Problems of Ageing: Biological and Medical Aspects* (Baltimore: Williams and Wilkins, 1952), p. xiv.
[258] Nathan W. Shock, "Ageing of Homeostatic Mechanisms," *Cowdry's Problems of Ageing,* pp. 415-446; "Age Changes in Renal Function," *Cowdry's Problems of Ageing*, pp. 614-630.

Hole—the ideal of scientific cooperation and the hope that biologists could offer answers to social problems. Cowdry, with this ideal and hope along with his expertise in textbook editing, began to construct gerontology as a response to the problems of aging that emerged during the deeply troubling period of the Great Depression. His efforts contributed to the birth of the multidisciplinary field in the United States which aimed at offering its scientific expertise on the problems of senescence.

It is useful to compare this story with its counterpart in Great Britain. Basically, it seems certain that the factors unique in America—such as the biosocial vision widely shared among biologists, the ideal of cooperative research at Chicago, and Cowdry's textbook editorship—did not necessarily mean that the American gerontologists were in a better state than the Britons. Admittedly, there was a larger group of scholars who were involved in aging research in America. Yet it should also be pointed out that Korenchevsky failed to include a number of scholars who could have joined the British Society for Research on Ageing, especially non-biomedical scientists such as botanists and social scientists. Furthermore, he was not successful in encouraging even those who did join him to start more systematic aging research and collaboration, such as E. C. Dodds, Robert Robinson, G. Roy Cameron, and Peter Medawar. This shows that Cowdry's success at least partially stemmed from contingent factors. In contrast to Korenchevsky, Cowdry was a scientist with a stable position and good reputation. He also shared his biosocial vision with his colleagues and had experience in textbook editing. These *happened* to be quite effective in organizing gerontology in America.

Cowdry's views on the body politic and the body anatomic also indicate his peculiarities, which might also be related to the way gerontology was received and constructed in the United States. His emphasis on local distinctiveness of cellular community and his caution against the body's centralized control seem to reflect what actually happened in the country about aging and gerontology. Cowdry liked to approach the problem of aging through the cooperation of individual elite scientists. Even though he never mentioned explicitly, he might have wanted to describe the British government's responses toward old age as "totalitarian." Does this, along with the ideal of the "civilized old age" McCay's research encouraged, explain a meaningful difference

between American and British gerontology? Indeed, a major American way of dealing with old age was scientific research, which, interestingly, was initiated by Cowdry who preferred individualism and localism in both the body politic and body anatomic. In contrast, a typical British way was to contruct a government-sponsored mechanism for offering assistance to the aged rather than scientific research. Does this difference reveal the distinct paths America and Britain took in terms of welfare policies, social systems, and scientific research after World War II?

While answering these questions could enable us to learn more about the place of gerontology in the two countries, this will be done by historical research in the future. The next chapter aims at an analysis of the developments of gerontology after the publication of Cowdry's *Problems of Ageing*, including the development of the Club for Research on Ageing, the Gerontological Society, and the Unit on Gerontology within the NIH. This chapter will reveal the differences and similarities between American and British gerontology in a more concrete way.

Chapter 6

"The Limitations on the Physiological Level of Older People Are More Apparent Than Real"—Institution, Funding, and the Development of the American Science of Aging

Although Edmund Vincent Cowdry's *Problems of Ageing* (1939) initiated a series of events leading to the establishment of gerontology as a scientific field in the United States, the development of gerontology did not follow a smooth and even path toward expansion and professional maturity. To establish gerontology as an active and respectable field, gerontologists had to find the adequate sources of funding and to justify the pursuit of aging research among the lay and professional public. They also needed to control the quality of their academic activities and to promote what had emerged as an important agenda, multidisciplinarity, in terms of its institutional organization and actual research. Yet these tasks were quite challenging for early gerontologists. Adequate sources of funding were hard to find especially during World War II, and justifying gerontology as an important scientific field among both professional scientists and the general public was never easy. Nor did the efforts at making gerontology a multidisciplinary scientific field with strong internal organization and high-quality academic productions always lead to a successful outcome.

This chapter discusses gerontologists' efforts to build their new scientific field along with the problems and frustrations they faced. I argue that in spite of a number of challenges and difficulties they were eventually successful in constructing and stabilizing their field in terms of funding, influence, and organization. Gerontology has been far from a tightly organized field and has not become a part of standard undergraduate curricula in many colleges. Moreover, the size of the Gerontological Society has been smaller than that of other major scientific organizations, and some gerontologists, due to

the absence of paradigmatic theories and methodologies shared by every scholar in the field, have questioned the identity of the science of aging as a legitimate academic area.[1] Nevertheless, the growth of the science of aging in the United States was unparalleled in other countries and other periods in human history. American gerontologists found adequate, if not abundant, financial resources in the extramural grants program of the National Institutes of Health (NIH) and other private patrons, although their attempts at fundraising through the Committee on the Biological Processes on Ageing (CBPA) were not successful. The scientists of aging also succeeded in launching the *Journal of Gerontology* which, in turn, led them to establish the Gerontological Society. The birth of many local societies for the study of aging and the efforts of the Gerontological Society to incorporate them are another important phenomenon which indicated that the influence of gerontology was broadening at the grass roots level.

Perhaps the most important event in this growth was the establishment of the Unit on Gerontology within the NIH in 1940, which developed into the largest research facility and funding agency in gerontology, the National Institute on Ageing. The success of its longest-running research project, the Baltimore Longitudinal Study of Aging (BLSA), shows how firmly gerontology had become established within the federal biomedical research organization as well as among lay people, many of whom would keep volunteering for the BLSA as research subjects across their lifespan.

During this period of gerontology's efforts to thrive and grow, multidisciplinarity was one of its most important agendas that gerontologists continued to emphasize and promote, although it sometimes became a complicating factor that frustrated their efforts to secure research funding. Aging was a complex problem that included the elderly's health, employment, and social and psychological well-being as well as various research problems on humans and other species. Hence, while most of early gerontologists came from biology and medicine, they thought that senescence should be approached through

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[1] W. Andrew Achenbaum, *Crossing Frontiers: Gerontology Emerges as a Science* (Cambridge: Cambridge University Press, 1995), pp. 135, 152-156; Stephen Katz, *Disciplining Old Age: The Formation of Gerontological Knowledge* (Charlottesville: University Press of Virginia, 1996), pp. 105-107, 112; Ira S. Hirschfield and David A. Peterson, "The Professionalization of Gerontology," *The Gerontologist* 22 (1982), pp. 215-220; R. D. Bramwell, "Gerontology as a Discipline," *Educational Gerontology* 11 (1985), pp. 201-205.

diverse viewpoints and expertise in biological, social, psychological, and medical sciences. These disciplines needed to be represented in gerontology organizations—including the CBPA, the Club for Research on Ageing, the Gerontological Society, the Gerontology Study Section (GSS), and the National Institute on Aging. The multidisciplinarity, however, could become a factor hampering available funding opportunities. For many potential donors of research funds, multidisciplinary approach meant that a clear focus or direction was absent in the proposed projects. To the NIH officials who decided to disband the GSS, multidisciplinarity meant the lack of expertise in a specific field that was essential for the fair and accurate evaluation of research proposals. In this sense, this chapter shows that multidisciplinarity, as sociologist Stephen Katz has pointed out, was both a strength and weakness of gerontology, since it could broaden the domain to which gerontology could be applied and, at the same time, threaten its existence as a scientific field.[2]

While describing these two aspects of gerontology's multidisciplinarity, this chapter will examine another important issue in this dissertation—how the social conditions surrounding gerontology in America differed from those in Britain. Admittedly, gerontologists on both sides of the Atlantic complained about finding adequate financial resources and institutional supports. Yet American gerontologists found a larger number of patrons of their research, including the NIH. I claim that these patrons' support of aging research was sustained instead of fulfilling more immediate needs of the elderly through pensions and healthcare as Britons did on the other side of the Atlantic. The ideology of the primacy of science, plus the difficulty of initiating the universal healthcare program, contributed to the relatively ample support American gerontological investigators enjoyed, whereas Britons chose to concentrate more on constructing the social system of caring for the aged which was not quite successful in the United States. Of course, this was not the only reason for the growth of gerontology in America, and I will explain other important factors, such as the efforts of gerontologists for survival within the academia and research institutions.

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[2] Katz, *Disciplining Old Age*, pp. 104-111.

This chapter has six sections. The first section will show how the scientists of aging interacted with the lay public and professional scientists who were increasingly becoming interested in aging after the publication of Cowdry's handbook. The second section will trace the growth of the Club for Research on Ageing and analyze its functions as a means to determine the major research problems and to promote multidisciplinary interactions among the disciplines constituting the field. The product of the Club's continuous evolution throughout the 1940s was the founding of the Gerontological Society and the *Journal of Gerontology*, which are the subjects of the third section. I will describe the Society's struggles to achieve its aims—having a strong internal organization as a multidisciplinary scientific field as well as a broader influence among professional scholars and lay Americans. In the same section, I also discuss The *Journal*'s efforts to survive its early years when it was financially insecure and its identity as a multidisciplinary academic forum was questioned. The fourth and fifth sections deal with the funding for aging research, which was probably one of the most problematic issues for early gerontologists. Through the example of the CBPA and the GSS, I will show gerontologists' efforts to find adequate and stable sources of support. The final section describes the growth of intramural gerontological research within the NIH, which eventually developed into the most influential national agency in the field of aging, the National Institute on Aging.

"My Own Praxis Has Had Something to Do with My Advanced Years": American Gerontologists and their Audience

Cowdry's *Problems of Ageing* was favorably received by a number of journals and scholars in diverse scientific and medical fields. *Yale Journal of Biology and Medicine* wrote that the book was "not only highly informative but also stimulating," and the *Medical Press and Circular* praised the "skill and care the work has been planned originally."[3] The *British Medical Journal* also reported that the book was a "storehouse of knowledge and must long be a source of reference," and *Archives of Pathology* stated

[3] H. S. Burr, "Problems of Ageing," *Yale Journal of Biology and Medicine* 11 (1939), pp. 690-691; "Problems of Ageing," *The Medical Press and Circular* 202 (1939), p. 106.

that "there can be nothing but praise" for the book.[4] While *The American Journal of Physical Anthropology* was more critical of the book's "fragmentation" due to the different orientation of each contributor, it still admitted that the book was "of decided value or interest."[5] Similarly, *Survey Midmonthly* praised the "signal success" of Cowdry's book, while *Time* emphasized that *Problems of Ageing* clearly revealed the significance of aging as one of the most important problems in civilized countries.[6] These favorable reviews stimulated readers to contact the contributors to the book for further information. For example, Lawrence Jones of the department of botany at the University of Minnesota became interested in MacNider's research and asked him to send a reprint of his chapter in *Problems of Ageing*.[7] Belle Boone Beard, a professor of sociology at Vanderbilt University, wrote a letter to Cowdry, asking why sociology was not represented in *Problems of Ageing*.[8] After introducing his colleague at the University of Oregon who was studying social aspects of aging, Beard asked Cowdry to send him any information about future publication plans on old age. Similarly, Walter Pitkin, a professor of journalism at Columbia University, contacted Cowdry to ask a few questions on the sociology of senescence.[9] He wanted to obtain more information about elderly people's adjustment to the life after retirement, especially after the establishment of the Social Security Act. Cowdry, pleased with this inquiry, suggested that Pitkin contact Louis Dublin, a statistician and a contributor to *Problems of Ageing*, who knew more about the current state of retirement and pension.[10]

Cowdry targeted his book to the general public as well as to these scholarly readers. By recommending that the contributors eliminate unnecessary tables and graphs and to "describe complicated matters simply" as far as they could, he tried to make the

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[4] Humphry Rolleston, "Problems of Ageing," *British Medical Journal* 2 (1939), p. 399; "Problems of Ageing," *Archives of Pathology* 28 (1939), pp. 280-281.
[5] A. H., "Problems of Ageing," *The American Journal of Physical Anthropology* 25 (1939), p. 136.
[6] Ernst P. Boas, "On Growing Older," *Survey Midmonthly: Journal of Social Work* 75 (August 1939), pp. 262-263; "For Old Folks," *Time* 33:2 (June 12, 1939), pp. 38, 40.
[7] Jones to MacNider, 24 October 1939, Box 12, Folder 492, WDM.
[8] Beard to Cowdry, 16 April 1946, Box 41, Folder 6, EVC.
[9] Pitkin to Cowdry, 13 December 1945, Box 41, Folder 5, EVC.
[10] Cowdry to Pitkin, 18 December 1945, Box 41, Folder 5, EVC.

book accessible to both lay and professional readers.[11] These editorial efforts stemmed from his view that aging was a social, as well as biological, problem, and that the book should be read by lay people who were facing the problem of their own old age in their social world. Indeed, Cowdry and other contributors who shared this view wrote a number of popular articles as well, which aimed at reaching a wider audience in the country.[12]

Many lay people who read these writings became interested in gerontology and contacted Cowdry and other scientists of aging. For gerontologists, however, some of these readers were overly enthusiastic and were seriously misunderstanding what they were doing. For example, H. A. McIlvaine, president of the Continental Electric Company in Illinois, asserted to Cowdry that he knew "a method of curing exopthalmic goitre by gland extracts and chemicals which rejuvenates all the glands and is much more satisfactory than surgical cure."[13] He then asked Cowdry the price of *Problems of Ageing* and the means to purchase it, expressing a hope to "exchange information with [Cowdry]…at any time."[14] C. E. Adelhelm, a chemical engineer in Denver, Colorado, also believed that gerontology was essentially a project for rejuvenation. Citing Serge Voronoff's and Alexis Carrel's arguments, Adelhelm suggested that replacing aged organisms' blood serum with pure Ringer's solution could rejuvenate them. To initiate an experimental study along this line, he promised to offer "one hundred dollars to begin with and about fifty dollars monthly thereafter."[15] As historian David Hamilton has pointed out, however, rejuvenation of the whole body as advocated by Voronoff and others had been thoroughly rejected by most professional scientists in the 1930s.[16]

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[11] Cowdry to C. M. McCay, 15 December 1937, Box 32, Folder 21, EVC; Cowdry to A. J. Carlson, 23 March 1938, Box 25, Folder 23, EVC.

[12] For example, see E. V. Cowdry, "We Grow Old," *The Scientific Monthly* 50 (1940), pp. 51-58; E. V. Cowdry, "Factors in Aging," *The Scientific Monthly* 56 (1943), pp. 370-374; Edward J. Stieglitz, "Aiding Aging," *Technology Review* 43 (1941), pp. 358-359, 382, 384, 386, 388, 390.

[13] McIlvaine to Cowdry, 21 March 1940, Box 41, Folder 5, EVC.

[14] McIlvaine to Cowdry, 21 March 1940, Box 41, Folder 5, EVC.

[15] Adelhelm to Cowdry, 21 September 1944, Box 41, Folder 1, EVC.

[16] David Hamilton, *The Monkey Gland Affair* (London: Chatto and Windus, 1986), pp. 120-142. The case of Carrel is more complicated. See chapter one in this dissertation. Also see, Laura Davidow Hirshbein, "The Glandular Solution: Sex, Masculinity, and Aging in the 1920s," *Journal of the History of Sexuality* 9 (2000), pp. 277-304.

Indeed, while apparently welcoming these people's interest in aging, Cowdry and other gerontologists did not really attempt to collaborate with them or to support their plans.[17]

There were other lay people who were not only uninterested in gerontological research but also explicitly hostile to it. In particular, antivivisectionists vehemently opposed biomedical research on aging, because, they thought, it relied upon cruel animal experimentation. Indeed, James Cruikshank, an antivivisectionist in New York, sent a letter to William MacNider with a clipping from a magazine which reported that "repeated doses of poison of varying strength administered to large groups of young animals result in a condition very similar to that occurring in aging animals through normal tissue changes."[18] He warned to MacNider that "the brutality of you vivisectors is beyond belief" and "the public, through Radio and newspapers, are being enlightened to what is really happening behind your locked doors."[19] While there is no record that MacNider or other gerontologists' research was actually hampered by these people's activities, their warnings certainly show that gerontology was gradually becoming an important issue even among those who did not support it.[20] Like other biomedical research programs which were developing during the first half of the twentieth century, biogerontological investigations began to draw the attention of antivivisectionists.

In general, however, most letters that the gerontologists received from the general public were very favorable to their research activities and writings. Many middle-aged and elderly people wrote to the scientists of aging to inquire about the means to regain their vigor, to report the way through which they achieved a healthy old age, or to express their willingness to volunteer for gerontological investigations as research subjects.[21] For

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[17] See Cowdry to Adelhelm, 30 September 1944, Box 41, Folder 1, EVC; Adelhelm to Cowdry, 4 December 1944, Box 41, Folder 1, EVC; Cowdry to Adelhelm, 29 November 1944, Box 41, Folder 1. EVC. Adelhelm asked Cowdry to read his manuscript of a scientific monograph. While praising that Adelhelm's "point of view is an interesting one and [he has] certainly been most diligent in expressing it," Cowdry explicitly wrote that "what the reader from Missouri requires is, of course, evidence." Moreover, although Adelhelm contacted MacNider in accordance with Cowdry's suggestion, MacNider did not respond to him.

[18] An unattributed magazine clipping, Box 11, Folder 414, WDM.

[19] James H. Cruikshank to MacNider, 19 November 1937, Box 11, Folder 414, WDM.

[20] But MacNider was deeply worried about antivivisectionists' potential threat toward biomedical research on aging. See MacNider to Robert Coker, 6 January 1939, Box 12, Folder 467, WDM.

[21] Some people whose aged parents were suffering from chronic diseases also contacted gerontologists to get the information for the treatment of the ailments. See, for example, Olga Frank to Edward Stieglitz, 4

example, Everett Carpenter, a middle-aged arteriosclerosis patient in California, wrote the following letter in 1940.

> Dr. Cowdry I am forty seven years of age and have a family but I am unable to be employed by any one not even the W.P.A. will [accept] me, because of my condition. My wife works on the W.P.A. Sewing Project. I have not money to offer you but if you could offer any advice that would benefit my condition I would surely appreciate it from the bottom of my heart.[22]

Although Cowdry was not a physician and there was yet no specific remedy that he could recommend, he nevertheless tried to help Carpenter by suggesting a better diet and exercise and any assistance available from local doctors.[23] On the other hand, Mary Bulkley, another person living in California, wrote a positive letter, explaining how she remained vigorous and active even after passing her eighty-ninth birthday. Declaring that "my own praxis has had something to do with my advanced years," she described various activities that she practiced throughout her entire life, such as being temperate in diet, memorizing many poems, and pursuing various different handicrafts.[24] Nancy Emery, an elderly nurse working in a home for the aged, also composed a hopeful letter that praised "the excellent, but neglected, opportunity for study which might yield rich returns: (a) to individuals given the satisfaction of making a continuing contribution instead of facing merely increasing helplessness and dependence; (b) to family life so often hampered, if not disturbed, by unadjusting old people; and (c) to our entire civilization, which has

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December 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee 1940-1942, NWS; Raymond Magill to Stieglitz, 24 November 1942, Box 11, Folder E. J. Stieglitz National Advisory Committee 1940-1942, NWS. A woman in New Mexico also wrote to Stieglitz to ask him to send her the information on the publications on aging which might be necessary for her to complete her "non-technical book designed to encourage old people to make the most of their advancing years." See W. L. Fleck to Stieglitz, 31 January 1941, Box 11, Folder E. J. Stieglitz National Advisory Committee 1940-1942, NWS.

[22] Carpenter to Cowdry, 3 November 1940, Box 41, Folder 5, EVC. He wrote that he came to know Cowdry's name through an article in *Los Angeles Examiner* that reported Cowdry's "statement…to the effect that Arterio Sclerosis could be checked."

[23] Cowdry to Carpenter, 9 November 1940, Box 41, Folder 5, EVC.

[24] Bulkley to Cowdry, 21 November 1945, Box 41, Folder 5, EVC.

lengthened the physical, but not the mental, span of life."[25] She then offered to "serve as a human guinea pig" for gerontological research during her old age and after her death.[26] MacNider was very pleased with this correspondence, which was indeed "one of the most thoughtful letters that [he had] ever received from an individual other than a medical person who is interested in doing something for ageing and aged people."[27] MacNider forwarded her letter to Cowdry who was then chairman of the Club for Research on Ageing, and recommended that she subscribe to nontechnical supplements of the *Journal of Gerontology*.

Besides responding to the general public through these letters, gerontologists tried to reach out and to help aged people more directly through the institutions they created. One of them was the "Old Age Counseling Center" directed by George Lawton, a renowned psychologist and a member of the Club for Research on Ageing. According to Lawton, this institute, established in 1943, aimed at helping elderly people adjust successfully to their old age in terms of physical and mental health. A number of eminent gerontologists—including Cowdry, Edward Stieglitz, Lawrence Frank, Anton J. Carlson, and Frederic Zeman—were invited to serve as its advisory council members. They were expected to "be consulted on an individual basis either by mail or personally" about the issue concerning the elderly's social, medical, and psychological well-being.[28] Lawton reported that the Center was quite successful, although he was often troubled by "irrelevant requests" on pensions and government services on old age.[29] Reflecting the growing interest in welfare among the increasing number of older people, the "requests for old age [counseling] have increased 500% in the last year" and a new plan was made for increasing the space and personnel of the Center.[30]

To give adequate advice to their elderly customers, however, the scientists needed to become genuine "experts" in the field of aging. Although it was true that each scientist

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[25] Emery to MacNider, 12 August 1946, Box 42, Folder 7, EVC.
[26] Emery to MacNider, 12 August 1946, Box 42, Folder 7, EVC.
[27] MacNider to Cowdry, MacNider to Emery, 20 August 1946, Box 42, Folder 7, EVC.
[28] Lawton to Cowdry, 20 October 1941, Box 42, Folder 21, EVC; Lawton to Cowdry, 6 August 1945, Box 41, Folder 5, EVC.
[29] Lawton to Cowdry, 11 November 1946, Box 42, Folder 5, EVC.
[30] Lawton to Cowdry, 11 November 1946, Box 42, Folder 5, EVC.

was a specialist on some specific issues concerning senescence, their role as gerontological experts, such as that required as members of the Center's advisory council, demanded that they be knowledgeable on broader and more varied aspects of aging. The next section will discuss how the early scientists of senescence educated themselves in these complex multidimensional problems of aging in their new field. I will describe this self-education process by analyzing gerontologists' participation in their earliest professional organization, the Club for Research on Ageing.

"An Experiment in Communication": The Club for Research on Ageing, 1940-1953

The Club for Research on Ageing in the United States was a small, informal, and elitist organization that stood at the center of gerontologists' various academic and institutional activities. Starting as the "American branch" of the International Club for Research on Ageing Vladimir Korenchevsky built during the late 1930s, the Club of the United States grew as an active and unique organization through which gerontologists could discuss their recent research topics as well as diverse institutional and organizational matters. While the activity of the British Club virtually stopped during World War II, its American counterpart was able to meet continuously through the support of the Josiah Macy, Jr. Foundation that paid the cost for traveling and lodging for the members. The American Club's meetings continued until 1953, even after the British Club was transformed into the British Society for Research on Ageing and the Americans formed the Gerontological Society after the War.[31] Geneticist Oscar Riddle recollected the significance of these meetings in terms of "one very worthwhile thing" the Club achieved, which was "educating itself" on the current research topics on aging and "supplying a nucleus" of the new science of senescence.[32] Indeed, as a "nucleus" of a fledgling research field, the Club remained a tiny and closed organization of serious

[31] After the British Club for Research on Ageing became the Society for Research on Ageing, the American Club was formally renamed the "Conferences on Problems of Aging." It was also voted to drop "e" in "ageing." See Notes on Business Meeting of Club for Research on Ageing, 25 April 1940, Box 30, Folder Club for Aging 1949, Feb, NWS.

[32] Notes on Business Meeting of Club for Research on Ageing, p. 5, 25 April 1949, Box 30, Folder Club for Aging 1949, April, NWS.

researchers of aging, who granted new memberships only to the leading scholars in the nation through their nomination and majority vote.[33]

It was necessary to maintain the Club as a closed elitist organization, because gerontologists still had a precarious status in the academia and often worried about the possibility that they could be considered another group of rejuvenation theorists like Serge Voronoff (1866-1951) and Eugen Steinach (1861-1944) whose past works had been denounced as fraudulent.[34] Hence, the attendance of leading senior scientists in the country, such as physiologist Walter Cannon, in the Club was considered very important, since they could enhance the status of the science of aging as a legitimate academic field and contribute to its dissociation from the questionable works on rejuvenation. In the case of Cannon, his participation in gerontology meetings and conferences was highly valued, and he was often asked to introduce the topics that would be dealt with during the meeting.[35] Even if he could not attend, he was encouraged to send his views by mail, which could be read and discussed during the meeting.[36] The fact that several younger members of the Club—including Joseph Aub, Roy Hoskins, and A. Baird Hastings—were Cannon's colleagues or students at Harvard Medical School also shows the character of the Club as a closed and elitist organization.[37]

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[33] For example, see Cohn to MacNider, 24 June 1941, 7 February 1942, Box 9, Folder 6, AEC.

[34] Vladimir Korenchevsky explicitly emphasized that gerontologists did not aim at rejuvenation. See Korenchevsky to Cannon, 28 July 1939, Box 82, Folder 1107, WBC. From the lay public, Cowdry and other gerontologists received inquires about rejuvenation, which never pleased them. See C. E. Adelheim to Cowdry, 21 September 1944, Cowdry to Adelheim, 30 September 1944, Box 41, Folder 1, EVC; H. A. McIlvaine to Cowdry, 21 March 1940, Box 41, Folder 4, EVC. Historian David Hamilton has described that Roy Hoskins and F. A. E. Crew—who would become gerontologists after the 1930s—were particularly critical to Voronoff. See Hamilton, *The Monkey Gland Affair*, pp. 105-107, 124-125.

[35] Meeting of the Club for Research on Ageing, p. 4, 11-12 February 1940, Box 41, Folder 2, EVC; Club for Research on Ageing Minutes of Meeting, p. 2, 21 and 22 March 1941, Box 41, Folder 2, EVC; Club for Research on Ageing, Secretary's Report, pp. 1-2, 28 February-1 March 1942, Box 30, Folder Club for Aging 1942, March, NWS.

[36] See, for example, Lawrence K. Frank to Cannon, 14 October 1941; Cannon to Frank, 27 October 1941, Box 94, Folder 1296, WBC; "Conference on Nutritional Requirements for the Ageing Population," p. 2, 1-2 November 1941, Box 1, Folder Nutritional Requirements Conference, GS.

[37] Aub was a personal physician of Cannon and studied traumatic shock with him. Hoskins was Cannon's student at Harvard Medical School. Frank Fremont-Smith of the Macy Foundation also studied under Cannon. See Cannon to Kast, 13 November 1933, Box 94, Folder 1290, WBC; Cannon to Kast, 6 October 1933, Box 94, Folder 1290, WBC; Cannon to Dave H. Morris, 1 October 1941, Box 94, Folder 1296, WBC.

During its annual meetings, the Club members discussed various biological and medical topics on aging, particularly those concerning their own research problems. The discussion subjects included the lifespan of cancer and plant cells studied by Cowdry and Crocker and the relation of nutrition to longevity investigated by Clive McCay as well as the aging of homeostatic mechanisms studied by Walter Cannon and William MacNider.[38] The Club also discussed the topics created by Alexis Carrel's research, such as the meaning of cultured tissues' immortality for senescence and the use of the culture technology for furthering the understanding of old age.[39] The Club did not neglect the research projects on aging pursued in wild nature rather than in the lab. Indeed, George Wislocki and Joseph Aub's study of the anatomical and chemical changes of the deer's antler according to its age[40] was intensively discussed along with Raymond Pearl's and Alfred Cohn's laboratory or clinical investigations on the genetic, racial, and sexual factors influencing longevity and senescence.[41]

Although most of these members of the Club came from biology and medicine, the social problems of old age was never ignored during their discussions. As Edward Stieglitz stated in 1941, the actual "limitations of the old man" needed to be precisely measured through more thorough biological research on aging "before we tackle the social and economic problems."[42] As I have written in chapter five, such a necessity arose amid the Great Depression, when many older individuals suffered from age discrimination and social displacement. Although this situation was dramatically reversed with the outbreak of World War II due to the wartime labor shortage, gerontologists still had something to argue. If industrialists, faced with labor shortage, hoped to employ

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[38] The Club for Research on Ageing Minutes of Meeting, pp. 4. 11-15, 18, 21-22 March 1941, Box 41, Folder 2, EVC.

[39] The Club for Research on Ageing Minutes of Meeting Part I, pp. 1-5, 13-14 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS. Based on his own research, Henry Simms criticized Carrel's argument that older animal's plasma was less favorable for tissue growth. See Henry S. Simms, Abstract of Presentation at the Meeting of the Club for Research on Aging, 13 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS.

[40] The Club for Research on Ageing Minutes of Meeting, pp. 3-10, 5-6 February 1944, Box 41, Folder 8, EVC.

[41] The Club for Research on Ageing Minutes of Meeting, p. 11, 21-22 March 1941, Box 41, Folder 2, EVC; Club for Research on Ageing Minutes of Meeting, p. 7, 28 February-1 March 1942, Box 30, Folder Club for Aging 1942, March, NWS.

[42] The Club for Research on Ageing Minutes of Meeting, p. 15, 22-22 March 1941, Box 41, Folder 2, EVC.

older workers that they had not used before the War, it was necessary to know their work capacity precisely to deploy them in their most appropriate positions.[43] Research on aging was also indispensable for checking the health status of many aged military leaders, who could be subject to the sudden physical frailty and chronic diseases.[44] Gerontological knowledge was demanded for the proper preparation of the postwar period as well. MacNider argued that since many young people would return to the country with crippled bodies and some of them would not return at all, the physiology and psychology of elderly people who would replace them needed to be understood more systematically for an effective use of manpower.[45] Indeed, during the 1943 meeting, psychologists Walter Miles and George Lawton were requested by MacNider to discuss their current research on elderly people's work performance and the need for longitudinal studies of the changes of mental and bodily functions with aging.[46]

Among these social problems on old age, retirement was a highly significant issue during the Club meetings, since the Social Security coverage and private pension systems were expected to expand during the postwar period.[47] Hence, Henry James and George Johnson from the Teacher's Insurance and Annuity Association were invited to present their views on the current state of old age pension systems in the country and the need for adult education for a more fruitful old age.[48] On this issue, most members of the Club did not like the idea of complete retirement at a fixed age. Instead, they suggested that the elderly needed to work continuously even after their official retirement, although it might

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[43] MacNider to Benjamin Horning, 4 October 1943, Box 16, Folder 649, WDM; Robert Griggs to MacNider, 16 September 1943, Box 16, Folder 647, WDM; Edward J. Stieglitz, "Geriatrics in Wartime," *Medical Annals of the District of Columbia* 12 (1943), pp. 19-23.

[44] Stieglitz to Lewis Thompson, 21 June 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder 0110, NARA.

[45] MacNider to McCay, 20 October 1942, Box 30, Folder Club for Aging Misc. I, NWS.

[46] The Club for Research on Ageing Minutes of Meeting Part II, pp. 4-10, 13-14 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS; George Lawton, "Abstract: Research Problems and Suggestions in the Psychology of Old Age," Box 30, Folder Club for Aging 1943, Feb, NWS.

[47] W. Andrew Achenbaum, *Social Security: Visions and Revisions*, *A Twentieth Century Fund Study* (Cambridge: Cambridge University Press, 1986), pp. 38-60. Also see Edward J. Stieglitz, "Psychiatric Problems of Senescents in Industry: Abstracts of Presentation at the Meeting of the Club for Research on Aging," p. 1, 13 February 1943, Box 41, Folder 8, EVC.

[48] The Club for Research on Ageing Minutes of Meeting, p. 18, 5-6, February 1944, Box 41, Folder 8, EVC; The Club for Research on Ageing Minutes of Meeting, pp 63-78, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS.

not be possible for them to work as vigorously as before.[49] Moreover, Stieglitz, Alfred Cohn, and other gerontologists argued, it was necessary to measure the "physiological age," which could replace the chronological age as the basis of determining the proper time of retirement.[50] It was also suggested that if retirement was unavoidable, it had to be planned carefully for a happy later life.[51] The Club decided that a committee for advising the Teacher's Insurance and Annuity Association should be appointed and that the *Journal of Gerontology* must deal with this issue thoroughly.[52]

The Club members' interest in these issues led them to invite to their 1950 meeting several scholars—such as Robert J. Havighurst of the University of Chicago, Wilma Donahue of the University of Michigan, and Leland C. DeVinney, an assistant director for social sciences of the Rockefeller Foundation—who would later contribute to the development of social gerontology. These people were able to initiate discussions of aging from the perspectives derived from their expertise. For example, Havighurst presented his research on the "interpersonal relations" with regard to the progress of senescence. He discussed how various sorts of interpersonal relations could be categorized, how they changed with aging, and what their relationship was with a person's happiness, morality, health, and personal adjustment in old age.[53] Biological

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[49] The Club for Research on Ageing Minutes of Meeting Part II, p. 8, 13-14 February 1943, Box 30, Folder Club for Aging 1943, Feb, NWS; The Club for Research on Ageing Minutes of Meeting, pp. 82-86, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS; Edward J. Stieglitz, "Psychiatric Problems of Senescents in Industry: Abstracts of Presentation at the Meeting of the Club for Research on Aging," p. 2, 13 February 1943, Box 41, Folder 8, EVC; Robert J. Havighurst, "Problems of Interpersonal Relations in Later Maturity and Old Age," p. 4, 6-7 February 1950, Box 30, Folder Macy Conference on Aging 1950, NWS.

[50] Edward J. Stieglitz, "Psychiatric Problems of Senescents in Industry: Abstracts of Presentation at the Meeting of the Club for Research on Aging," p. 2, 13 February 1943, Box 41, Folder 8, EVC; The Club for Research on Ageing Minutes of Meeting, p. 82, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS.

[51] Edward J. Stieglitz, "Psychiatric Problems of Senescents in Industry: Abstracts of Presentation at the Meeting of the Club for Research on Aging," p. 3, 13 February 1943, Box 41, Folder 8, EVC; The Club for Research on Ageing Minutes of Meeting, p. 72, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS

[52] On the Committee for Retirement within the Club, see the Club for Research on Ageing Business Minutes of Meeting, p. 1, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS. Roy Hoskins first suggested that the Journal should deal with the issue of retirement. See The Club for Research on Ageing Minutes of Meeting, p. 79, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS.

[53] Robert J. Havighurst, "Problems of Interpersonal Relations in Later Maturity and Old Age," 6-7 February 1950, Box 30, Folder Macy Conference on Aging 1950, NWS.

and medical experts in the Club, along with social scientists, responded to this presentation by talking about how aged people's good interpersonal relations influenced their emotions, which affected their physiological state and health.[54] They also discussed the ways to measure a person's emotions by checking the bodily conditions and relate them to his or her interpersonal relations.

The Club made consistent efforts to promote multidisciplinary interactions through its meetings, although these efforts were not always successful. For instance, after Cowdry was appointed chairman of the Club in 1946, Frank Fremont-Smith of the Macy Foundation pointed out that the Club had not completely achieved its original goal.[55] Whereas the Club initially aimed to focus on informal yet intensive interactions among the scholars from diverse fields, the previous meetings of the Club had too many long formal presentations by single scholars, which left only a short time for actual multidisciplinary discussion. Therefore, Fremont-Smith suggested that presentations should be finished within fifteen minutes and the rest of the time must be devoted to active cross-disciplinary discussions. The presentation also had to be a short yet provocative suggestion of a topic rather than a lengthy report of a finished research project. Fremont-Smith wrote,

> The Foundation's whole conference program is built on the need for improved communication between the members of the various specialties concerned with a single topic. We make a sharp distinction between the making of statements <u>at people</u> and communication <u>with people</u>. The latter implies and requires a two-way process, an active participation. Our whole program….is conceived as an experiment in communication.[56]

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[54] Nathan W. Shock, (ed.), *Conference on Problems of Aging, Transactions of the Thirteenth Conference, February 5-6, 1950 New York, N.Y.* (New York: Josiah Macy, Jr. Foundation, 1951), pp. 80-89.
[55] "Part of Letter from F. Fremont-Smith to E. V. Cowdry," 3 January 1947, Box 41, Folder 8, EVC; "Extract from a Letter November 21, 1947 from Dr. Frank Fremont-Smith to Dr. E. V. Cowdry," Box 41, Folder 8, EVC.
[56] "Extract from a Letter November 21, 1947 from Dr. Frank Fremont-Smith to Dr. E. V. Cowdry," Box 41, Folder 8, EVC.

In retrospect, this policy of the Foundation was in complete agreement with E. V. Cowdry's original plan of editing *Problems of Ageing* (1939) and organizing the first conference on aging at Woods Hole, Massachusetts. Indeed, during the final meeting of the Club in 1953, Cowdry recollected the reason why the Club had been organized as a multidisciplinary discussion forum. (See Figure 6.1.) While "all living things age," Cowdry said, "at the human level….the study of aging becomes more complicated because of the entry of social and economic factors."[57] These factors should be "added to the hereditary, environmental, chemical, and physical factors that operate in a great many levels of animals and plants," and for this reason, "the appraisal and the approach must….be multidisciplinary."[58] Clive McCay expressed his deep gratitude for this multidisciplinary meeting. He said, "I come from agriculture. I would not have known, I believe, any other person in this room….if it were not for the Macy Foundation."[59] As I have written in chapter two, the members of the Club greatly contributed to the expansion of McCay' research program on nutrition and longevity as a cross-disciplinary project.

[57] Minutes of the Business Meeting, Fifteenth Conference on Problems of Aging, p. 2, 22 January 1953, Box 30, Folder Macy Conference on Aging 1953 [final], NWS.
[58] Minutes of the Business Meeting, Fifteenth Conference on Problems of Aging, p. 2, 22 January 1953, Box 30, Folder Macy Conference on Aging 1953 [final], NWS.
[59] Minutes of the Business Meeting, Fifteenth Conference on Problems of Aging, p. 6, 22 January 1953, Box 30, Folder Macy Conference on Aging 1953 [final], NWS.

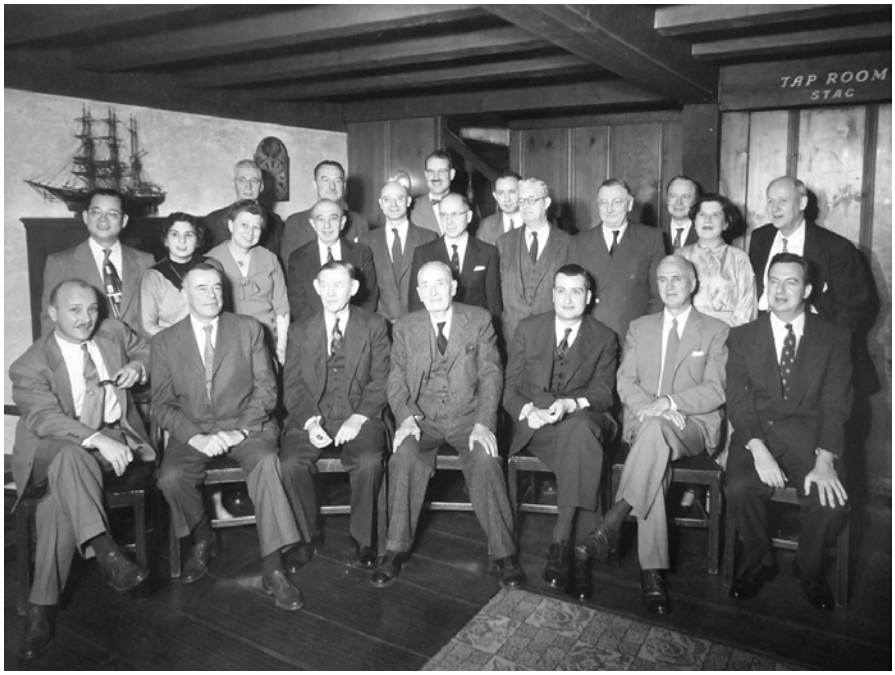

Figure 6.1. *The Final Meeting of the Macy Conference on Problems of Aging. January, 1953. Box 30, The Clive Maine McCay Papers, Cornell University Archive.*

Another significant function of the Club meetings was to provide a forum for discussing various organizational and institutional issues in the presence of the people who were directly responsible for them. Representatives of many new gerontology organizations attended the meeting as members or guests, reporting their recent activities and discussing their concerns with other members of the Club. In fact, there were substantial overlaps in membership between the Club and the other gerontology-related organizations and institutions, such as the Committee on Biological Processes of Ageing, the Gerontology Study Section, and the Gerontology Unit within the NIH.[60] The issues

[60] The charter members of the Committee on Cellular Physiology, which became the Committee on the Biological Processes of Aging in 1938, were E. V. Cowdry (chairman), A. J. Carlson, William Crocker, H. S. Jennings, E. B. Krumbhaar, I. F. Lewis, Warren Lewis, C. M. McCay, and William MacNider. Except the I. F. and Warren Lewis, all of the members of the Committee were members of the Club as well. See Robert Coker to Cowdry, 15 May 1937, Box 25, Folder 39, EVC. Edward Stieglitz, the first chief of the Gerontology Unit, was a member of the Club since 1940, and Nathan Shock, who directed the Unit after Stieglitz' resignation, attended the Club meeting from 1942. The Gerontology Study Section was consisted of Shock, McCay, MacNider, Fremont-Smith, Ephraim Shorr, Henry Simms, Oscar Kaplan, Robert Moore, Roy Hoskins, and G. B. Taylor. Among them, only Taylor and Kaplan were not members of the Club. See C. J. Van Slyke to Ephraim Shorr, 17 December 1947, Record Group 443, Records of the National

concerning the institutions for psychological and social scientific research—such as Havighurst's Committee on Human Development at the University of Chicago, Donahue's Institute of Gerontology at the University of Michigan, and DeVinney's social science division of the Rockefeller Foundation—were also presented and discussed during the Club's meetings.[61]

Yet perhaps the most important matter that the Club addressed during its gatherings was the issue about the *Journal of Gerontology* and the Gerontological Society, which considerably extended the scope of the science of aging. The next section will discuss how the Club decided to publish this novel multidisciplinary journal and to create the newer and larger scientific organization. I will also describe how the Society adapted itself to the new problems and challenges during the 1940s and 1950s by changing its organization and structure.

The *Journal of Gerontology* and the Gerontological Society: Broadening the Scope of the Science of Aging, 1941-1958

In 1941, two members of the Club, Henry Simms and Lawrence Frank, discussed the possibility of establishing a professional society for researchers of aging. According to Simms, the society would be beneficial to the scientists of senescence, since it could help them "exchange ideas," "[arouse] interest in the aging problem," and find possible sources of funding.[62] Mailing the "abstracts of scientific papers on aging….at intervals" was also necessary to keep members of the prospective society "informed of progress in the field."[63] Yet the establishment of the society was not seriously considered or urgently demanded at that time. Frank was worried about "the general reaction of professional men toward the organization of a new society, with dues and officers and meetings."[64] While Frank, anticipating such a reaction, suggested that "it might be wise to plan a

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Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence I, NARA.

[61] On Donahue and Havighurst, see Achenbaum, *Crossing Frontiers*, pp. 104-106, 159-165.

[62] Simms to Frank, 28 October 1941, Box 5, Folder History of the field of aging, NWS.

[63] Simms to Frank, 28 October 1941, Box 5, Folder History of the field of aging, NWS.

[64] Frank to Simms, 29 October 1941, Box 5, Folder History of the field of aging, NWS.

society" which had "no dues, or officers" and "no formal meetings planned," no concrete action for establishing such a society was attempted at that time.[65]

However, several problems gradually emerged concerning the function and identity of the Club which was maintained as a small and closed community of elite scientists. The first was the possibility that the Club's identity as an active discussion forum might not be maintained as efficiently as before, as its size expanded.[66] Simultaneously, it was necessary for the Club to enhance its publicity among the scientific and general public to justify gerontology as a legitimate study field and to acquire research funds.[67] Indeed, an increasing number of scholars and lay people read the Club members' publications and had become interested in gerontology. If the Club could not include these people in their community and would not expand beyond a certain size limit, what, then, should be done to encourage them to keep supporting gerontology and to educate others on the importance of aging as a multidimensional problem? The Club members also worried about the need for each member to keep abreast of other members' activities. In fact, the meeting was held only once a year, and between these meetings, MacNider sent every Club member a letter containing a summary of current research activities of the members.[68] But such letters were certainly not enough, and gerontologists needed to get a more up-to-date and detailed information on the academic works of their community as a whole. For this reason, Henry Simms's previous suggestion for the regular mailing of the abstracts of current papers in aging research was brought forth again.[69]

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[65] Frank to Simms, 29 October 1941, Box 5, Folder History of the field of aging, NWS.

[66] The Minutes of the Business Session of the Club for Research on Ageing, pp. 6-7, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS; Notes on Business Meeting of Club for Research on Ageing, p. 5, 25 April 1949, Box 30, Folder Club for Aging 1949, April, NWS.

[67] The Club for Research on Ageing Minutes of Meeting, p. 18, 11-12 January 1940, Box 41, Folder 2, EVC; The Club for Research on Ageing Minutes of Meeting, p. 11, 21-22 March 1941, Box 41, Folder 2, EVC; The Club for Research on Ageing Minutes of Meeting, pp. 15-17, Box 30, Folder Club for Aging 1942, March, NWS; The Club for Research on Ageing Minutes of Meeting Part II, pp. 2-3, 13-14 February 1943, Box 41, Folder 8, EVC.

[68] MacNider to Cohn, 1 June 1942, Box 19, Folder 6, AEC. MacNider's role was similar to that of Henry Oldenberg (1618-1677), the early secretary of the Royal Society. Oldenberg's correspondence networks evolved into the first scientific journal, *Philosophical Transactions*.

[69] MacNider to the Members of the Club for Research on Ageing, 8 November 1943, Box 19, Folder 7, AEC.

The Club decided to respond to these problems by establishing its own journal. The journal was a way to circulate the most recent scientific knowledge on aging among the general audience as well as professional scientists. MacNider wrote to Cowdry before the 1944 meeting that a plan to publish a gerontology journal should be considered, since nobody except the members could see "the important material which has come out at our various meetings."[70] With this initiative of MacNider, the members of the Club discussed during their 1944 meeting the potential merits of the prospective journal—such as its possible role in enhancing publicity among the public as well as its function as a "repository of references" as was proposed by Simms.[71] The latter was especially important, because scientific articles on aging tended to be found in so many different journals and some of them could not be identified as having a relevance to senescence if they were "judged by their titles."[72] Since most members of the Club agreed with these reasons, the plan was seconded and passed during the 1944 meeting and the journal committee was created with Roy Hoskins as chairman. This committee appointed Robert Moore as the first editor-in-chief, and decided that the journal should "have a broad appeal—to deal with the problems of ageing from a variety of viewpoints" and that "articles [should] be accepted for publication irrespective of country of origin."[73] The committee also made a contract with the renowned medical publisher Charles C. Thomas who promised to take care of the cost of printing and distribution, provided that there were enough subscriptions to cover the cost. The initial cost for the journal editing came from a grant awarded by the Macy Foundation.[74]

The establishment of the Gerontological Society, Inc. was another way to solve the problems suggested during the Club meetings and a means to respond to the financial

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[70] MacNider to Cowdry, 28 January 1944, Box 41, Folder 3, EVC.
[71] The Minutes of the Business Session of the Club for Research on Ageing, pp. 2-3, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS.
[72] The Minutes of the Business Session of the Club for Research on Ageing, p. 1, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS.
[73] Report of "Committee on Journal" to the Club for Research on Ageing, undated, Box 41, Folder 3, EVC; Hoskins to the Members of the "Committee on Journal" of the Club for Research on Ageing, 10 June 1944, Box 41, Folder 9, EVC.
[74] Gerontological Society, Inc. Financial Statement, 15 February 1946, Box 30, Folder Club for Aging 1946, Feb, NWS. The initial Macy grant was $4,000.

uncertainty surrounding the new Journal. When it was reported in 1944 that an increasing number of people in the country were becoming interested in aging, it was proposed that the Club "might encourage the organization of local sectional groups."[75] On this issue, Stieglitz suggested that it might be feasible to "have an inner group, that is, the Club for Research on Ageing, and an outer group, that is, an association, made up of many smaller sectional groups."[76] An organization larger than the Club was necessary for another purpose, especially in relation to the journal. It was suggested in 1945 that "a separate corporation" was necessary, because, without it, "the members of the club would be individually and collectively responsible for all debts of the Journal."[77] For these reasons, the Club decided in 1945 that five members residing in New York City—Simms, Frank, Jean Oliver, Oscar Riddle, and Earl T. Engle—should "incorporate" the Geontological Society "under the laws of the State of New York."[78] The "Certificate of Incorporation of the Gerontological Society, Inc., pursuant to the Membership Corporations Law" was signed by these members and submitted to the Division of Corporations of New York State on May 18, 1945, with the fee of $40.[79] The Society was designated as an organization

> ….to promote the scientific study of aging, in order to advance public health and mental hygiene, the science and art of medicine, and the cure of diseases: to foster the growth and diffusion of knowledge relating to problems of aging and of the sciences contributing to an understanding thereof; to afford a common meeting ground for representation of the various scientific fields interested in such

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[75] The Minutes of the Business Session of the Club for Research on Ageing, p. 7, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS.
[76] The Minutes of the Business Session of the Club for Research on Ageing, p. 7, 5-6 February 1944, Box 30, Folder Club for Aging 1944, Feb, NWS.
[77] The Club for Research on Ageing Minutes of Meeting, p. 89, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS.
[78] The Club for Research on Ageing Minutes of Meeting, p. 89, 9-10 February 1945, Box 30, Folder Club for Aging 1945, Feb, NWS. It is not certain why the Club chose the New York residents as the initial incorporators, and why the Society had to be incorporated following the laws of New York State.
[79] "Certificate of Incorporation of the Gerontological Society, Inc.," Box 35, Folder GSA, Certificate of Incorporation, NWS; Financial Statement of the Gerontological Society, Inc., 15 February 1946, Box 30, Folder Club for Aging 1946, Feb, NWS; Minutes of the First Meeting of Gerontological Society, Inc., 15 June 1945, Box 42, Folder 1, EVC.

> problems and those responsible for care and treatment of the aged; all without profit to the corporation or its members.[80]

The first meeting of the Society was held approximately one month later, on June 15, 1945 at New York City. On that day, the Society elected William MacNider as its first president and created its constitution and by-laws.

As historian W. Andrew Achenbaum has put it, the Gerontological Society had to carry out several significant tasks in its early years, many of which are still important today.[81] First of all, the Society had to maintain its "multidisciplinary collegiality" among the members who came from various distinct specialties. It was also necessary for gerontology to be recognized as a legitimate and important scientific field in the academia of the United States. But a more challenging task for the Society was to control the quality of its academic productions, and, at the same time, to disseminate the knowledge on aging to larger audience who thereby might contribute to increasing the Society's membership and research funds as well as widening its broader influence. Achenbaum has argued that the Society has been quite successful in dealing with these tasks, especially its "multidisciplinary collegiality" in a "savvy" manner.[82] This explains how the Society has been able to survive since its establishment in 1945.

What, then, were the actual ways that the Society adopted to accomplish these tasks? The *Journal of Gerontology* was one of these means and was especially significant for the Society's task of promoting gerontology's multidisciplinary structure. To this end, Robert Moore and the editorial committee decided to include in their journal "all of the natural and social sciences, which have something to contribute to the care of the ageing individual."[83] This meant that the *Journal* should accept papers from "sociology, economics, and social work" as well as those from biology and medicine.[84] Table 6.1 is a rough categorical summary of the articles published in the *Journal* from 1946 to 1953

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[80] "Certificate of Incorporation of the Gerontological Society, Inc.," Box 35, Folder GSA, Certificate of Incorporation, NWS.
[81] See Achenbaum, *Crossing Frontiers*, pp. 125-129, 134-136, 141-143.
[82] See Achenbaum, *Crossing Frontiers*, p. 129.
[83] Moore to Joseph H. Willits, 28 March 1945, Box 289, Folder 1973, Series 200, Record Group 2, RF.
[84] Moore to Joseph H. Willits, 28 March 1945, Box 289, Folder 1973, Series 200, Record Group 2, RF.

by research fields, which shows that the *Journal* consistently published articles in the social sciences, social work, and psychology, despite the fact that the relative number in these fields was smaller than that of the papers in biology and medicine.[85] While not specified in the table, the range of researchers' disciplines who published articles in biology and medicine was also quite broad, including medical science, botany, pharmacology, and animal husbandry.

	1946	1947	1948	1949	1950	1951	1952	1953
Biology / Medicine (using nonhuman subjects)	13	9	4	4	4	6	10	6
Biology / Medicine (using human subjects)	10	10	14	20	20	13	21	28
Social science / Social works	8	2	5	5	6	10	11	10
Psychology (including psychiatry)	0	2	1	4	2	2	2	5

Table 6.1. *Articles Published in the Journal of Gerontology from 1946 to 1953 by Research Fields.*

Each article in the *Journal* was accompanied by a short biographical sketch which helped readers become familiar with the author, if the latter's academic background was different from that of the former.[86] The articles also had "an abstract comment written in

[85] A reason why this summary is "rough" is that the categoriazation of the articles is not easy. Most of all, it is often impossible to classify an article definitely in either "biology" or "medicine," since many articles belonged to both. Moreover, the "biology" articles were published by researchers in many different research fields. But assigning an article to a research field according to the authors' institutional affiliation or training does not always reveal the actual content of the article. For example, the articles by Clive McCay in Cornell's animal husbandry department did not have direct relation to animal husbandry. Hence I have used the simpler category of "biology and medicine using human subjects" and "biology and medicine using nonhuman subjects." Furthermore, the boundary between psychology and the social sciences is also often highly blurred, and psychiatric articles are basically "medical" as well. Other papers were written through interdisciplinary groups of investigators from diverse scientific disciplines.

[86] Moore to Joseph H. Willits, 28 March 1945, Box 289, Folder 1973, Series 200, Record Group 2, RF.

nontechnical language so that those in one field may understand clearly the work in other fields."[87]

The *Journal* dealt with other issues that reflected gerontologists' desire to achieve both professional development and wider influence. While the *Journal* had to be the official publication for professional researchers, it should also include portions for the general readers as well. As an attempt to meet its role for professional readers, the *Journal*, following Simms' former suggestion, created the cumulative index of literature, which could be used by scholars in finding recent publications on particular research problems in gerontology. Experts' reviews on major problems in gerontology was another section in the *Journal* that the scientists of aging could read to update their knowledge of the current state of specific research topics, such as the "influence of age on the regeneration and repair of tissue," "ageing in plants," "mental hygiene in the aged," and "changing concepts in the social and economic care of the aged."[88] The editorial committee of the *Journal* also created the original article section that was filled by submitted papers which passed quite a stringent review process.[89] Interestingly, some of these papers, in an easier language, appeared again in the *Journal*'s "non-technical supplements," which were made to carry out another important function of the *Journal*—the wider dissemination of gerontological knowledge among lay readers. The section on "legal aspects" that reviewed the current legal trends on retirement and pension was another portion of the *Journal* accessible to nonprofessional readers.

The Society's membership categories were also organized by its intention to promote both the professionalism of its core members and openness to lay people. It had four membership categories—"active," "honorary," "senior," and "sustaining" members. While the "active members" were elected among those who were "in the immediately preceding three years….engaged in the study or practice of any phase of gerontology," other people who were not currently pursuing gerontological research could also obtain

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[87] Moore to Joseph H. Willits, 28 March 1945, Box 289, Folder 1973, Series 200, Record Group 2, RF.
[88] Moore to Joseph H. Willits, 28 March 1945, Box 289, Folder 1973, Series 200, Record Group 2, RF.
[89] Indeed, among the eight manuscripts submitted to the editorial office by the end of January, 1946, only four were accepted for publication. See The Gerontological Society, Inc. Report of the Editor of the Journal of Gerontology, p. 3, 15 February 1946, Box 30, Folder Club for Aging 1946, Feb, NWS.

other sorts of memberships.[90] The "honorary members" were those who had "rendered distinguished service to the science of gerontology or geriatrics" in the past, and the "sustaining members" were elected among the people who were interested in aging but were not currently doing any active research.[91] "Senior members" were the scholars who had performed an active service to the Society or the Club but were now over sixty-five and retired. This organization of membership was made in order to maintain the unique ecology within the Society—whereas only dedicated professional researchers could be elected as active members and had the right to vote during the Society's meetings, other people who were sympathetic to aging research or had been engaged in it in the past could also join the Society and attend its meetings as honorary, senior, or sustaining members.[92] Even though these non-active members also had to be formally "elected," the chance of non-election was probably small, since the Council of the Society acutely felt the need to increase the number of its member. In fact, the Society made deliberate and sustained efforts for membership expansion—it circulated the "information sheet" and "letters of invitation" among the people who might be favorable to aging research, and sent free copies of the *Journal* to various clinics, public libraries, and homes for the aged.[93] Because of these efforts, the Society came to have six hundred members by 1952, while it had only sixty in 1946.[94]

The Society's "Council" and sections categories were created to promote the balance between academic disciplines constituting gerontology as well as cooperation among them. The Society had three sections—clinical medicine, biological science, and social science. The Society's "Council," which managed these sections, was initially constituted with the members of the Club's Executive Committee in 1945 and was

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[90] "By-laws of Gerontological Society, Inc. As Adopted June 15, 1945, and Revised Feb. 18, 1946," *Journal of Gerontology* 1 (1946), p. 266.
[91] "By-laws of Gerontological Society, Inc. As Adopted June 15, 1945, and Revised Feb. 18, 1946," p. 266.
[92] Although the senior members could not vote, they could choose not to be a senior member, if they wanted. See "By-laws of Gerontological Society, Inc. As Adopted June 15, 1945, and Revised Feb. 18, 1946," p. 266.
[93] Shock to Cowdry, 20 October 1948, Box 30, Folder Club for Aging 1949, April, NWS; "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," *Journal of Gerontology* 2 (1947), p. 165.
[94] See Roy Hoskins to Cowdry, 18 October 1946, Box 42, Folder 1, EVC; "Minutes of the Sixth Annual Meeting of the Council of the Gerontological Society, Inc.," *Journal of Gerontology* 7 (1952), p. 121.

maintained thereafter as the body consisting of president and president-elect of the Society, the secretary, the *Journal* editor, and other active members.[95] The Council constituted the core of the Society which set its boundary of professionalism and supported its main agenda, multidisciplinarity. It was decided in 1945 that "at least two of the elected members [of the Council] shall represent each of the following fields: Clinical Medicine, Biological Science, and the Social Science."[96] The Council's official meetings were thus expected to be gatherings of the representatives of the distinct disciplines who would cooperatively promote the Society's professional growth and outward influence.

Yet the Society's early years were not free from problems. First of all, its official publication, the *Journal of Gerontology*, did not have enough subscriptions to become financially independent. Although Robert Moore hoped in 1946 that the *Journal* would soon become self-supporting, it had only nine-hundred and sixty subscriptions by 1947, while it needed more than two thousand subscriptions to survive without external support.[97] Moreover, the *Journal*'s "non-technical supplements" had fewer than one hundred subscriptions in 1946, and this led both the publisher and gerontologists to doubt their viability. For these reasons, Charles Thomas wanted to be completely released from the contract with the Society.[98] Therefore, on February 2, 1947, the Council had to decide if the *Journal* should be continued or not. If the *Journal* were to be continued, the Council had to make a decision about whether the "present editorial policy of a multidiscipline JOURNAL be pursued, or should the *Journal* be oriented to" biology and medicine.[99] The question of its non-technical supplements was another topic that had to be discussed during the Council meeting.

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[95] By-laws of Gerontological Society, Inc., p. 2, 15 June 1945, Box 42, Folder 1, EVC.
[96] By-laws of Gerontological Society, Inc., p. 2, 15 June 1945, Box 42, Folder 1, EVC. The importance of social science section seemed to be downplayed in the by-laws revised in 1946, since its name was changed into "the General Section." See "By-laws of Gerontological Society, Inc. As Adopted June 15, 1945, and Revised Feb. 18, 1946," pp. 266-267.
[97] Gerontological Society, Inc. Report of the Editor of the Journal of Gerontology, p. 3, 15 February 1946, Box 30, Folder Club for Aging 1946, Feb, NWS; Editor's Report for 1948, p. 3, Box 42, Folder 1, EVC; "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," p. 165.
[98] Cowdry to Fremont-Smith, 7 February 1947, Box 41, Folder 8, EVC; "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," *Journal of Gerontology* 2 (1947), p. 166.
[99] "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," p. 166.

The Council's decision clearly reflected the priorities of the Society at that time. Basically, discontinuing the *Journal* was not an option. The existence of their own official publication was too important for gerontologists to give up. What was actually at stake was to decide whether it should be continued as a multidisciplinary publication or not. On this issue, Stieglitz and Engle suggested that "the Council approve the principle of continued publication of a multidiscipline JOURNAL having the same general editorial policy," and other members unanimously decided to follow their suggestion.[100] Since multidisciplinarity was critically important as one of gerontologists' core agendas, it could never be abandoned as a guiding principle of the *Journal*. The Society's goal of interacting with the general public was not given up, either. Although the publication of the *Journal*'s non-technical supplements was never resumed after the Council's 1947 decision to suspend them "temporarily," *The Gerontologist* was launched in 1961 as a new journal of the Society for the general public.[101] Fortunately, the Macy Foundation helped the Society to survive these deep financial woes again. It offered $5,000 in 1948 and $3,000 in 1949 to support the publication of the *Journal*.[102] The portion of the subscription fee within the membership due was also increased from $6 to $8 in 1949, although the total dues of $10 did not change.[103] These additional funds, along with the income from advertisements and other small philanthropies' grants, enabled the Society to pay the editorial and publication cost of the *Journal*.[104]

The Society's efforts to support its agendas—maintaining itself as a multidisciplinary organization, enhancing professionalism, and promoting its communication with lay people—continued and was intensified through the revision of its by-laws in 1952. In the early summer of that year, Shock suggested that it was

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[100] "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," p. 166.
[101] "Minutes of the Second Annual Meeting of the Council of the Gerontological Society, Inc.," p. 167; Achenbaum, *Crossing Frontiers*, p. 141.
[102] Majorie Adler, "History of Gerontological Society, Inc.," *Journal of Gerontology* 13 (1958), p. 97; Gerontological Society, Inc. Editorial Account: For the Year Ended December 31, 1949, Box 42, Folder 1, EVC.
[103] Adler, "History of Gerontological Society," p. 97.
[104] Adler, "History of Gerontological Society," p. 100; Gerontological Society, Inc. Journal Account: For the Year Ended December 31, 1949, Box 42, Folder 1, EVC; Gerontological Society, Inc. Membership Account: For the Year Ended December 31, 1949, Box 42, Folder 1, EVC.

necessary to "give professional groups other than biology and medicine proper recognition in the Society." It was also important to "protect the professional interest of present members of the Society and at the same time to broaden the base of membership."[105] Since other members agreed with this suggestion, the by-laws were revised in 1952,[106] introducing a more complex organization within the Society. The new by-laws established three divisions—the general member division, affiliate organization division, and professional division—among which the professional division were further subdivided into four sections, including biological science, psychological and social science, clinical medicine, and social work and administration sections. Through this change, the Society's scope of multidisciplinarity was broadened by including a new specialty, "social work and administration," within its domain. At the same time, the Society made a distinction between "fellows," who were currently pursuing active research in gerontology or geriatrics, and "members," who were just "sympathetic with the purposes of the Society." The fact that these "members" in the new by-laws, unlike "sustaining members" in the previous ones, had the right to vote revealed that the Society explicitly aimed to represent the voices of lay people in their decision-making, while trying to maintain a special status of those who were actively engaged in gerontological research.[107]

The aborted suggestion for amalgamation or making a federation between the Gerontological Society and the American Geriatrics Society (AGS) also revealed the Gerontological Society's continued efforts at promoting its multidisciplinary collegiality. Indeed, the AGS was perhaps the most important organization among the groups affiliated with the Gerontological Society in terms of its professionalism and influence. However, the Gerontological Society took special care not to lose its multidisciplinary character due to an overly close relationship with the AGS. Although "the formation of a federation" between these two societies and even "amalgamation" was proposed in 1951

[105] Shock to "Dear," undated, Box 35, Folder GSA, Bylaws 1952 Revision, NWS. This is a circular letter sent from Shock, the Society's secretary. Although this letter is undated, it was probably written during the early summer of 1952, since Shock asks the members to respond to his letter by August 11, 1952.
[106] "By-laws of the Gerontological Society, Inc. Adopted September 6, 1952," *Journal of Gerontology* 8 (1953), pp. 107-111.
[107] "By-laws of the Gerontological Society, Inc. Adopted September 6, 1952," p. 108.

during the meeting of the affiliation committees of both societies, leading fellows of the Gerontological Society did not seriously consider such proposals, because it could seriously harm one of the most important characters of the Society, multidisciplinarity.[108] Indeed, when it was suggested that "only the members of the medical profession" would be allowed to vote in the proposed federation, Shock, who was then secretary of the Society, was "frankly disturbed."[109] According to him, "the strength of the Gerontological Society lies in the fact that it is willing to accept workers in the field on equal basis without respect to their professional affiliations or degrees." Therefore, it was "a grave error" to relegate "all professional people from the social sciences to a second-class membership."[110] For Shock, it was more desirable to "attempt to expand [the Society's] own membership among [the AGS]" than to try to make a federation between the two societies.[111] After this problematic encounter, the merger or federation formation between the two societies was not seriously considered, although the idea of uniting gerontology and geriatrics was continuously brought forth as a discussion topic.[112]

But the Gerontological Society tried to encourage and maintain good relations with aging-related societies other than the AGS, especially regional gerontology groups. In fact, many local gerontological societies were established during the 1950s, such as "The Greater Washington Area Gerontological Group," "The Connecticut Society of Gerontology," "Western Gerontological Society," and "Georgia Gerontological Society."[113] To make a formal rule concerning its relation with these new organizations, the Gerontological Society's 1952 revised by-laws introduced another novel membership

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[108] Report of the Affiliation Committees of the American Geriatrics Society and the Gerontological Society, Inc., Box 35, Folder GSA, Relation to AGS, NWS. Also see "Minutes of the Fourth Business Meeting of the Gerontological Society, Inc.," *Journal of Gerontology* 7 (1952), p. 123.
[109] Shock to Moore, 12 June 1951, Box 35, Folder GSA, Relation to AGS, NWS.
[110] Shock to Moore, 12 June 1951, Box 35, Folder GSA, Relation to AGS, NWS.
[111] Shock to Moore, 28 June 1951, Box 35, Folder GSA, Relation to AGS, NWS.
[112] Achenbaum, *Crossing Frontiers*, p. 151.
[113] Minutes of the Second Meeting of the Greater Washington Area Gerontological Group, 14 May 1953, Box 35, Folder GSA-affiliate Groups, NWS; Report of the 1st Annual Meeting of the Connecticut Society of Gerontology, 20 November 1953, Box 35, Folder GSA-affiliate Groups, NWS; Program: Second Annual Meeting of the Western Gerontological Society, 20-21 April 1956, Box 35, Folder GSA-affiliate Groups, NWS; Louis Newmark to Shock, 15 May 1956, Box 35, Folder GSA-affiliate Groups, NWS.

category, "affiliates," who were the members of the Society's affiliate organizations.[114] The making of this new category reflected the fact that the Society welcomed these grass-root organizations and tried to make more detailed rules and plans concerning its relations with them.[115] The establishment of the new category of the Society's meetings, namely, the "regional meeting," was one of these plans to incorporate the new local groups within the Society's domain.[116]

However, even while many local gerontology groups were established and the importance of gerontology as the science dealing with aging became widely recognized, the problem of finding a stable source of funding for gerontology remained a difficult issue. Indeed, fundraising was perhaps the most troublesome task that the early American gerontologists faced, despite their success in building a national organization and launching its official journal. The following two sections will discuss this problem in detail, beginning with the Committee on the Biological Processes of Ageing (CBPA) within the National Research Council (NRC).

The Committee on the Biological Processes of Ageing and Gerontologists' Struggles for Money, 1937-1946

The troubled history of the CBPA illustrates the gerontologists' struggles for existence in these difficult times particularly well. The CBPA, which aimed at becoming a funding agency for the science of aging controlled by gerontologists themselves, was not successful in securing a sizable amount of funds and was abolished after several years of aborted fundraising activities. An analysis of their work shows how difficult it was to persuade private philanthropies and affluent individuals of the necessity of aging research

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[114] "By-laws of the Gerontological Society, Inc. Adopted September 6, 1952," p. 107.

[115] See, for example, "Requirement for Designation as a Member Society in the Gerontological Society, Inc.," 30 November 1955, Box 35, Folder GSA-affiliate Groups, NWS; Shock to Adler, 26 February 1958, Box 35, Folder GSA-affiliate Groups, NWS; "Status of Affiliate Organizations," 25 September 1957, Box 35, Folder GSA-affiliate Groups, NWS; Report of the Committee on Affiliate and Associate Groups-1957, Box 35, Folder GSA-affiliate Groups, NWS.

[116] "By-laws of the Gerontological Society, Inc. Adopted September 6, 1952," p. 109. However, many regional societies did not want to become a local "chapter" of the Gerontological Society, although they wanted a healthy and mutually supporting relation with the Society. See "Status of Affiliate Organizations," 25 September 1957, Box 35, Folder GSA-affiliate Groups, NWS.

at that time and how important the skill and talent of fundraisers themselves were in securing money. This section also shows why the multidisciplinary subjects the CBPA proposed to investigate were not attractive to philanthropies at that time, although some multidisciplinary projects did get support. For instance, the Rockefeller Foundation regarded the multidisciplinary character of Clive McCay's project on caloric restriction as its strength and funded McCay during the 1930s and 1940s. But this project, unlike the CBPA's projects, had interesting past results as well as a clear focus for future research. (See Chapter 2.) The multidisciplinary nature of the CBPA's research proposals, which lacked such a focus and previous achievements, was considered its weakness rather than strength, and thus could not have as much appeal to the foundations as that of McCay. In fact, the early gerontologists were too excited about the future and possibilities of multidisciplinary research to appreciate what the prospective patrons actually wanted in projects that aimed to garner cooperation across disciplinary boundaries.

But the scholars who established in 1937 the precursor of the CBPA, the Committee on Cellular Physiology (CCP), did not anticipate these problems. Edmund Vincent Cowdry, Ludwig Kast, and the "Borderland Committee" within the NRC were very optimistic about the possibilities and importance of aging research. They formed the CCP as a national funding agency for the study of senescence that could "[bring] together at least three divisions of the Council, namely, those of the Medical Sciences, Biology and Agriculture, and Anthropology and Psychology."[117] While the name of the CCP apparently indicated that its main field would be cytology or physiology, Cowdry, who was then chairman of the CCP, along with other members like Anton Carlson and William MacNider, soon determined that the direction of its primary activity should be scientific and medical research on aging.[118] The first meeting of the CCP was also held along with the event Cowdry organized, the Woods Hole Conference of Aging. Many of the founding members of the CCP came from the participants of the conference, including Carlson, MacNider, McCay, H. S. Jennings, William Crocker, E. B. Krumbhaar,

[117] E. V. Cowdry to W. MacNider, 29 March 1937, Box 10, Folder 381, WDM.
[118] Cowdry to Coker, 11 March 1937, Box 25, Folder 39, EVC; Cowdry to Coker, 29 June 1937, Box 25, Folder 39, EVC; MacNider to Cowdry, 9 July 1937, Box 31, Folder 8, EVC; MacNider to Cowdry, 11 March 1938, Box 31, Folder 8, EVC.

and W. S. Hunter.[119] At the second meeting held on February 6th, 1938 at Washington, DC, the CCP formally changed its name to the CBPA.[120] In the chairman's report, Cowdry argued that aging had become a serious national problem as the size of the elderly population increased and the social displacement of aged people made their later years unhappy and even miserable. Therefore, it was necessary to initiate a long-term study of aging using "chemical, histological, physiological, pathological, psychological and many other techniques" to "reveal the processes of ageing, how we can retard them and make the inevitable decline easier to bear."[121]

Despite such an ambitious beginning, the state of the CBPA was not very hopeful, since it was not financially supported by its sponsoring agency, the NRC, which ceased to be a government-funded research institute at the end of World War I. During the 1920s and 1930s, the NRC was merely an organization for awarding research grants and fellowships which were supplied by private philanthropies such as the Rockefeller Foundation and the Carnegie Corporation. It was the responsibility of NRC chairmen to secure money that would be used as research grants. Admittedly, some committees, such as the Committee for Research on the Problems of Sex, were quite successful in obtaining a large amount of money from the Rockefeller Foundation.

But the fundraising activity of the CBPA under Cowdry's chairmanship did not turn out very well. First of all, the initial response of Robert E. Coker—president of the NRC Division of Biology and Agriculture and a member of the Club for Research on Ageing—toward Cowdry's proposal was negative. While Coker suggested that Cowdry should submit a concise proposal based on the minutes of their meeting after a thorough discussion with other members, Cowdry submitted the minutes themselves without change and thereby deeply disappointed Coker.[122] Coker thought that the minutes

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[119] R. E. Coker to MacNider, 11 June 1937, Box 10, Folder 394, WDM.
[120] MacNider to Cowdry, 11 March 1938, Box 31, Folder 8, EVC.
[121] E. V. Cowdry, "Report of Committee on Biological Processes of Ageing," pp. 3-4, 19 March 1938, Box 11, Folder 430, WDM.
[122] Coker to Cowdry, 4 June 1938, 23 June 1938, Box 25, Folder 39, EVC. Cowdry thought that "the generous support has not come for specific projects outlined concisely in the way which [Coker] suggested" and the projects should be "flexible and permit shift of emphasis." See Cowdry to Coker, 11 June 1938, 20 October 1938, Box 25, Folder 39, EVC.

contained only a "nebulous idea" and were too long to be read by foundation officers.[123] Coker thus recommended that Cowdry submit a new proposal with a clearer main concept and some "practicable prospectus."[124] Yet Cowdry replied that writing such a proposal was extremely difficult "because the scope is so wide embracing the ageing of all forms of life."[125] For him, "the strength of our Committee resides in the fact that it is representative of many highly specialized lines of investigation all of which have definite bearing on the biological processes of ageing and in any one of which an important project could be rationalized."[126] Cowdry regarded what Coker saw as gerontology's weakness—multidisciplinarity—as its strength. Since Coker did not still agree with Cowdry's view, he, as a chairman of the Division to which the CBPA belonged, authorized a new subcommittee consisting of McCay, MacNider, and Carlson to "draw up a program for which we might ask early support."[127] But even this subcommittee's proposal, while being more focused and concrete, did not differ substantially from Cowdry's former plan in that it still listed various independent lines of research—including histological, pathological, biochemical, and endocrinological studies of various problems regarding aging, such as the rate of oxidation and basic metabolism, research on hydrogen ion concentration, and the investigation of the changing effect of intoxicants with aging.[128] The final proposal submitted to the Rockefeller Foundation emphasized more explicitly that the study of aging had to be done "along a diversity of lines" of research, including pathology, clinical medicine, botany, protozoology, cytology, physiology, and oncology.[129] How was this proposal received by the Foundation's officers? While the Rockefeller Foundation did prefer cooperative projects involving two or more disciplines, the proposal had to have a clear focus in terms of subjects, future

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[123] Coker to Cowdry, 2 November 1938, Box 12, Folder 457, WDM.
[124] Coker to Cowdry, 2 November 1938, Box 12, Folder 457, WDM.
[125] Cowdry to Coker, 4 November 1938, Box 12, Folder 457, WDM.
[126] Cowdry to Coker, 4 November 1938, Box 12, Folder 457, WDM.
[127] Coker to Cowdry, 15 November 1938, Box 12, Folder 459, WDM.
[128] "Report of the Sub-Committee on the Biological Processes of Ageing," 20 January 1939, Box 12, Folder 470, WDM. It contained a budget justification and a statement that the committee would focus on mammals' aging.
[129] "Proposal for Studies Relating to Biological Processes of Aging," pp. 2, 8-9, Box 170, Folder 1236, Series 200, Record Group 2, RF.

prospects, and past achievements. The CBPA's proposal did not have such a focus and was not seriously considered, probably for this reason. Indeed, Frank Blair Hanson, associate director of the Rockefeller's Natural Science Division, talked about the proposal with Coker and thought that "the whole thing is still in the 'study' stage."[130] Coker himself also wrote that "we are not yet prepared to make requests for support from any source."[131] Although the proposal was eventually submitted to the Foundation, no action was taken about it.[132]

The CBPA still failed to make any progress in winning research funds, even after it changed its chairman from Cowdry to Carlson during the summer of 1940. Although the CBPA held its third meeting on November 26 and 27 of 1940 at Washington, D.C., along with the newly formed Advisory Committee on Gerontology within the U.S. Public Health Service, no definite fundraising activity was resumed after the meeting.[133] Carlson himself said in 1942 that he "had every intention of carrying on an intensive campaign for the collection of funds," although the "national emergency has interfered with this program."[134] What Carlson did was only to reconfirm the conclusion reached by the previous CBPA under Cowdry's chairmanship—the necessity of long-term funding for aging research which might have to follow the successful precedent of the NRC Committee for Research on the Problems of Sex.[135] Two and a half years after this meeting, Coker finally wrote that the CBPA "was about to be abolished for inactivity but a protest saved the day, assuming that [MacNider] would accept the responsibility" of the chairmanship.[136] MacNider gladly accepted this offer and the CBPA was reconstituted

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[130] Frank Blair Hanson Diary, 15 May 1939, Box 170, Folder 1236, Series 200, Record Group 2, RF.
[131] Coker to Hanson, 29 September 1939, Box 170, Folder 1236, Series 200, Record Group 2, RF.
[132] Frank Blair Hanson Diary, 12-13 October 1943, Box 243, Folder 1680, Series 200, Record Group 2, RF.
[133] Carlson to Gentlemen, 16 November 1940, Box 14, Folder 536, EVC; Report of the Meeting of the Committee on Biological Processes of Aging, 27 November 1940, Box 30, Folder Club for Aging Misc. I, NWS. During the meeting, it was suggested that further public relations efforts were needed, especially toward industrial leaders. But no definite fundraising activity or it plan was discussed.
[134] Club for Research on Ageing, Secretary's Report, 28 February-1 March 1942, Box 30, Folder Club for Aging 1942, March, NWS.
[135] Carlson to Gentlemen, 10 October 1940, Box 25, Folder 23, EVC.
[136] Coker to MacNider, 13 July 1943, Box 16, Folder 639, WDM.

with the new members, most of who were drawn from the Club for Research on Ageing, such as Cowdry, Walter Cannon, Baird Hastings, Robert Moore, and F. L. Hisaw.[137]

MacNider was very resolute in his determination to revive the CBPA as an active funding agency. He wrote to his old friend Frank Fremont-Smith, "As you likely know, the Committee has been a flat failure on two different occasions, and I am bent and determined to use all of my energies to keep this from happening again."[138] Indeed, he tried to employ every means that he could use to contact wealthy philanthropies and individuals and to appropriate research funds for the CBPA. He asked Lord Nuffield in England to write letters of introduction to two affluent Americans, Hugh L. Adams and Henry Ford.[139] MacNider also requested the NRC president Ross Harrison to introduce the prospective activity of the CBPA to three influential people, Henry Ford's secretary E. G. Liebold, Warren Weaver of the Rockefeller Foundation, and Benjamin Horning of the W. K. Kellogg Foundation.[140] MacNider also used his friends' network to contact the International Cancer Research Foundation in Philadelphia, which, he thought, might be interested in aging research.[141] Since these early efforts seemed successful, MacNider was able to contact them in person or through correspondence.

However, the CBPA was not able to get any money from these prospective donors. As early as in July, 1943, Hugh Adams sent MacNider "a very pleasant note," which nevertheless clearly indicated that he was not interested in funding gerontology.[142] MacNider's contact with the Kellogg Foundation led to a similar result. Benjamin Horning wrote that aging research was "not a field which the W. K. Kellogg Foundation

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[137] MacNider to Cowdry, 10 September 1943, Box 42, Folder 7, EVC; "Committee on the Biological Processes of Ageing of the National Research Council," undated, Box 42, Folder 7, EVC.
[138] MacNider to Fremont-Smith, 15 November 1943, Box 17, Folder 654, WDM.
[139] MacNider to Nuffield, 19 January 1943, Box 16, Folder 625, WDM; Nuffield to MacNider, 3 March 1943, Box 16, Folder 629, WDM. When these letters were written, MacNider was a member of the CBPA under Carlson's chairmanship. Although the contents of these letters are not known, they probably would be about the support of the aging research of the CBPA, since MacNider subsequently contacted the recipient of the letters on this issue.
[140] MacNider to Harrison, 13 September 1943, Box 16, Folder 647, WDM.
[141] See MacNider to Korenchevsky, 30 July 1943, Box 16, Folder 641, WDM; MacNider to Robert F. Griggs, 15 October 1943, Box 16, Folder 650, WDM.
[142] MacNider to Korenchevsky, 30 July 1943, Box 16, Folder 641, WDM.

is in a position to support financially at the present time."[143] The Foundation was funding only those whose research related to the childhood welfare and war efforts. After receiving this reply, MacNider wrote a long letter explaining that the CBPA was not only concerned about old age but with the whole lifespan which might include childhood as well, and that aging research would eventually help war efforts and postwar reconstruction because younger people's death and injury during the war would necessitate the use of aged manpower.[144] Even though MacNider succeeded in prompting Horning to rethink the issue and to meet with MacNider in person, Horning eventually rejected the application, since the Foundation could not change its policy of supporting only the projects which were *directly* related to childhood problems.[145] The trip of Frank Hanson, a Rockefeller officer, to Chapel Hill where MacNider resided did not bring about a different result, either. Whereas Hanson admitted that the CBPA had "a number of excellent and energetic people," he found that it had not yet conducted a thorough survey of the field and had no clear "plan of attack upon the problem of ageing."[146] The only suggestion Hanson gave to MacNider was to withdraw the previous proposal submitted to the Foundation during Cowdry's chairmanship and to submit a new application based on a more extensive study of the current problems in aging research.

MacNider's own trip to Dearbon and Philadelphia to secure funds was not very satisfactory, either. Indeed, even before the trip, MacNider worried that he might be "the worst possible person to go on such a venture and that it [would] more than likely be a complete failure."[147] Unfortunately, the outcome of his "first adventure in salesmanship" was not different from his expectation.[148] At Dearbon, he met with E. G. Liebold, who, according to MacNider, was "a rather difficult type of big business individual, who was, to an extent, obsessed with the idea that all the money, except some very small amount, that the Ford interests are making is being taken away through federal taxation."[149] For

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143 Horning to MacNider, 24 September 1943, Box 16, Folder 648, WDM.
144 MacNider to Horning, 4 October 1943, Box 16, Folder 649, WDM.
145 Horning to MacNider, 11 December 1943, Box 17, Folder 657, WDM.
146 Frank Blair Hanson Diary, 12-13 October 1943, Box 243, Folder 1680, Series 200, Record Group 2, RF.
147 MacNider to Griggs, 5 August 1943, Box 16, Folder 642, WDM.
148 MacNider to Griggs, 15 October 1943, Box 16, Folder 650, WDM.
149 MacNider to Griggs, 15 October 1943, Box 16, Folder 650, WDM.

MacNider, such an obsession of Liebold was a strong "barrier" which could not be broken easily. MacNider failed to persuade Liebold of the necessity of aging research mediated by the NRC, a federal agency. Although MacNider, after this disappointing encounter with Liebold, met with Mildred Schramm, a secretary of the Cancer Research Foundation in Philadelphia, the result of the meeting was also unsatisfactory. Schramm "was pretty clear in indicating that any [prospective] project would have to concern itself….with the experimental interest in cancer."[150] Although it was possible that the Foundation might offer grants to study the relationship between aging and cancer, the CBPA's goal was more ambitious.[151] The CBPA hoped to secure a lump-sum grant, which could be freely used in accordance with the decision of its members rather than a grant whose usage was already determined for a narrowly limited project.

These repeated failures deeply frustrated gerontologists. With regret, Cowdry wrote in 1945 that "we all receive commendation on our work from foundations and many leading people but when it comes to getting support, these foundations and leading people give us the cold shoulder."[152] Although MacNider was eventually able to raise $5,000 in 1946 from the Markle Foundation and $3,600 from the International Cancer Research Foundation, the amounts of these grants were far smaller than what the CBPA had originally anticipated and was used only for Cowdry's project on aging and skin cancer rather than for multidisciplinary research which would be directed by gerontologists in the CBPA.[153] Indeed, Cowdry recollected in 1948 that this was the only successful fundraising activity of the CBPA amid "these years of frustration."[154] After such disappointing attempts, the CBPA was ultimately disbanded.[155]

In general, however, gerontologists' past achievements and future prospects in the academic funding market were not so poor. From the early twentieth century, they were supported by various private and public agencies and philanthropies. The only difference

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[150] MacNider to Griggs, 15 October 1943, Box 16, Folder 650, WDM.
[151] MacNider to Griggs, 21 October 1943, Box 16, Folder 651, WDM.
[152] Cowdry to MacNider, 29 June 1945, Box 49, Folder 103, EVC.
[153] Cowdry to MacNider, 15 March 1946, Box 42, Folder 7, EVC; MacNider to Cowdry, 11 March 1946, Box 42, Folder 7, EVC.
[154] Cowdry to Fremont-Smith, 17 February 1948, Box 42, Folder 21, EVC.
[155] Although it is not clear when the CBPA was disbanded, a letter to Cowdry written in 1950 indicates that it was disbanded during the late 1940s. See Lee Laird to Cowdry, 3 May 1950, Box 42, Folder 5, EVC.

between these successful instances and that of the CBPA was that the former was the case of applications by individual scientists of aging who hoped to get funding for their specific projects, while the latter was an attempt to obtain a lump-sum grant that could be spent in accordance with gerontologists' own decision. But the Gerontology Study Section within the National Institutes of Health would realize the CBPA's failed hopes. With a substantial amount of budget appropriated by the United States Congress, it was able to award quite a large number of grants to various researchers of aging in North America according to gerontologists' own decisions. The next section will discuss these cases of gerontologists' funding, beginning with the roles of various private foundations. The birth, demise, and legacy of the Gerontology Study Section will also be discussed in detail with respect to the contemporary state and agenda of gerontology.

Funding Gerontology: Private Philanthropies and the Federal Government as Patrons of Aging Research

The Macy Foundation was perhaps the most important contributor to the birth and early development of gerontology in the United States. As I have described in chapter five, the Foundation financially sponsored the publication of Cowdry's *Problems of Ageing* and the first scientific conference on aging at Woods Hole, Massachusetts in 1937. After that, the Macy officers kept supporting gerontology, by offering the grants for establishing the Unit on Gerontology within the NIH in 1940, helping the publication of the *Journal of Gerontology*, and sponsoring the meeting of the Club for Research on Ageing from 1940 to 1953. The Foundation's contribution to gerontology was not limited to these organizational and institutional domains. As a part of the "Life Cycle" program of the Foundation, it helped actual biological and medical studies of aging by various scientists.[156] Henry Simms and his colleagues' research at Columbia University on arteriosclerosis and fat deposition was one of the projects supported by the "Life Cycle" program, and John Saxton's pathological research using McCay's rats at Cornell Medical

[156] Josiah Macy, Jr. Foundation, *The Josiah Macy, Jr. Foundation, 1930-1955: A Review of Activities* (New York: 1955), pp. 71-87. The Macy Foundation's Life Cycle Program included the studies of pregnancy, early development, and childhood as well as old age.

College was also funded by the Foundation.[157] In addition, MacNider's investigation of the age-changes of cellular resistance against toxins and Cowdry and his associates' research on the aging of skin and endocrine glands were supported by the Foundation.[158] The Macy Foundation funded clinical research as well, especially the study of the relation of aging to the vitamin supply, which was conducted at Elgin State Hospital in Illinois from 1943 under the sponsorship of the NRC Committee on the Nutritional Aspects of Ageing.[159]

The Macy Foundation's support for gerontology began from its officers' ideas and thoughts, especially those of executive secretary, Lawrence Frank. While it was Ludwig Kast who initiated the Foundation's gerontology programs, it was Frank who subsequently became a major driving force of the Foundation's early support program for aging research. Indeed, Frank's interest in aging stemmed from his earlier work at the Laura Spellman Rockefeller Memorial and the General Education Board for the study of human growth phases, including infancy, childhood, and adolescence.[160] To understand

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[157] Josiah Macy, Jr. Foundation, *The Josiah Macy, Jr. Foundation, 1930-1955*, pp. 82-87; *Twentieth Anniversary Review of the Josiah Macy, Jr. Foundation* (New York: 1950), pp. 36-37; Lawrence Frank to Clive McCay, 14 October 1937, Box 6, Folder Clive, McCay [comments listed], NWS; McCay to Frank, 15 October 1937, Box 6, Folder Clive, McCay [comments listed], NWS; Maynard to Weaver, 2 December 1937, Series 200D, Record Group 1.1, Box 136, Folder 1688, RF. For a brief review of Simms and his colleagues' research at Columbia University, see Program for Research on Aging Conducted at the College of Physicians and Surgeons, Columbia University, Box 30, Folder Club for Aging Misc. I, NWS.

[158] Josiah Macy, Jr. Foundation, *Twentieth Anniversary Review*, pp. 36-37; William deB. MacNider, "A Study of the Acquired Resistance of Fixed Tissue Cells Morphologically Altered through Processes of Repair. III. The Resistance to Chloroform of a Naturally Acquired Atypical Type of Liver Epithelium Occurring in Senile Animals," *Journal of Pharmacology and Experimental Therapeutics* 56 (1936), pp. 383-387.

[159] Josiah Macy, Jr. Foundation, *Twentieth Anniversary Review*, p. 37; William MacNider to Walter Cannon, 30 November 1942, Box 16, Folder 618, WDM; MacNider to Vladimir Korenchevsky, 4 January 1943, Box 16, Folder 624, WDM; MacNider to Korenchevsky, 4 August 1942, Box 15, Folder 607, WDM; Korenchevsky to Ross Harrison, 27 July 1942, Box 82, Folder 1117, WBC. Korenchevsky initiated a similar project in London before Americans. He encouraged the American gerontologists to implement a similar clinical research project.

[160] Frank has been primarily known as a social scientist who played a major role in the rise of the child development movement in the United States, which adopted a multidisciplinary approach and holistic perspective. See, for example, "Lawrence K. Frank," *Child Development* 40 (1969), pp. 347-353; Dennis Bryson, "Lawrence K. Frank, Knowledge, and the Production of the 'Social,'" *Poetics Today* 19 (1998), pp. 401-421. I do not wholly accept Dennis Bryson's argument that Frank had a technocratic vision, which aimed to foster "'socially adjusted and cooperative personalities' through various biological, social, and psychological approaches," thereby obscuring underlying political tensions concerning class, race, and gender. By promoting age as a category as significant as race, class, and gender, Frank uncovered another political tension involving age.

the complexities of human development and to produce a mentally and physically sound human well-adapted to modern society, he argued that various scientific experts needed to cooperate to obtain knowledge of human development and to apply it to the actual family life and schooling.[161] After he moved to the Macy Foundation, he came to include in his view on human development old age which he considered the final phase of human's maturation rather than the period of decrepitude and obsolescence.[162] To understand this phase properly and to remedy the serious social problems such as age discrimination, Frank argued that multidisciplinary research, whose importance he had already emphasized for the study of development, was demanded.[163] This was the basis upon which the Macy Foundation began its "Life Cycle" program to support multiple disciplines' cooperative approaches to pregnancy, personality development in children, and aging.

The Macy Foundation's support for gerontology was based upon its thorough appreciation of the state of the field, which can be revealed through Frank's review article published in the first volume of the *Journal of Gerontology*.[164] Frank discussed the current issues in the science of aging—the different rates of aging in distinct parts of the body, the importance of experimental approaches, the distinction between physiological and chronological age, and the works of Minot, Pearl, and Cannon. He also described how old age emerged as a social and political problem during the twentieth century and how this problem could be solved through the application of gerontological knowledge.

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[161] "Autobiographical Interview," 1966-1967, p. 11, Box 12, LKF; Frank to Cannon, 18 December 1928, Box 18, Folder Cannon, Walter B, LKF.

[162] Lawrence K. Frank, "Live Long and Like It," *Crozer Quarterly* 24 (1947), pp. 316-325.

[163] Lawrence K. Frank, "Gerontology," *Journal of Gerontology* 1 (1946), p. 2; "Comments on the Problems of Ageing: Presented to the Meeting of the Club for Research on Ageing," 12-13 January 1940, Box 11, Folder Admin. files E. J. Stieglitz 1939-41, I, NWS; Frank, "Live Long and Like It," pp. 316, 323; "The Older Person in the Changing Social Scene," p. 4, 4 December 1940, Box 21, Folder Mental Age in Old Age, LKF.

[164] Frank, "Gerontology," pp. 1-11. Moreover, Frank had known most major gerontologists in person, including Robert Havighurst, Walter Cannon, Alexis Carrel, Thomas Wingate Todd, and Earl T. Engle. See "Lawrence K. Frank," p. 348; Milton J. E. Senn, "A Tribute to Lawrence K. Frank," undated, Box 23, Folder Obituary, LKF. Frank also was deeply impressed by Cannon's concept of homeostasis. See Frank to Cannon, 23 September 1929, Box 18, Folder Cannon, Walter B, LKF. For Frank's relation to Carrel, Todd, and Engle, see Frank to Carrel, 14 October 1937, Box 18, Folder Carrel, Alexis, LKF.

While it had no officer like Frank who was seriously committed to supporting gerontology, the Rockefeller Foundation also played an important role in the development of the science of aging. As I have discussed in chapter one, the Foundation supported during the 1920s Raymond Pearl's Institute for Biological Research at the Johns Hopkins University where various environmental, nutritional, and hereditary factors concerning longevity and senescence were studied. In chapter two, I have also described how Warren Weaver's Natural Science Division supported McCay's research on the relation of nutritional factors to aging and lifespan from 1936. In the 1940s, the Foundation supported another biomedical project directed by a German émigré Franz J. Kallmann, who studied the genetic factors in the incidence of mental diseases occurring in old age.[165] Interestingly, unlike the Macy Foundation, the Rockefeller Foundation supported social gerontology as well during the 1950s. The Social Science Program under Norman Buchanan and Leland DeVinney offered $20,500 for the research on the continuing employment of elderly workers by Robert Havighurst and his colleagues at the University of Chicago.[166] The University of California's research project on the influence of aging population upon the American society was another social science program supported by the Rockefeller Foundation.[167]

Several smaller foundations and institutions joined the Rockefeller and Macy Foundations in this continuing support of the science of aging. While their size was smaller, they made important contributions to the growth of gerontology as a scientific field. First, the Mary R. Markle Foundation supported research on aging and cancer in relation to hormone functions through the NRC Committee on Research in Endocrinology chaired by Walter Cannon. During the 1950s, the Forest Park Foundation also donated $16,500 to the *Journal of Gerontology* for the improvement of its publications, and the Glendorn Foundation granted $7,000 for "the conference on the chemistry of blood vessels with special reference to age changes," which was later

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[165] From 1945 to 1951, the Foundation awarded $55,500 to Kallmann's work at Columbia University. See Minutes of Meeting of the Executive Committee of the Rockefeller Foundation, 19 January 1945, 13 June 1947, Record Group 16, RF.

[166] DeVinney to Buchanan, 5 October 1955, Series 910, Record Group 3.1, Box 3, Folder 22, RF.

[167] DeVinney to Buchanan, 5 October 1955, Series 910, Record Group 3.1, Box 3, Folder 22, RF.

changed into the honoraria for the contributors to Albert Lansing's book on the same topic.[168] Individual researchers of aging also received small grants from various agencies for their specific projects. For example, William MacNider studied the changing response of aging animals to uranium nitrate through a grant from the American Medical Association. He was also awarded grants by the Ella Sachs Plotz Foundation and the New York Academy of Medicine for his research on how aging influenced the chemical alterations of blood during pregnancy and the resistance to alcohol and chloroform.[169]

These grants for gerontology illustrate the financial state of American science in general during the first half of the twentieth century in which gerontologists were situated. While the nineteenth century was the era of "genteel poverty" for American scientists, more systematic and quite abundant funding was started during the early twentieth century following the initiatives of the Rockefeller and Carnegie philanthropies.[170] While their enthusiasm for science during the Progressive Era was certainly a factor in directing their policy in general, the actual funding decisions—who should be funded for which projects, how much should be granted for how many years, and other issues—depended upon many contingent factors which often changed according to standpoints of directors, composition of executive boards, and general economic conditions of society. The fact that the early leading private philanthropies were mostly American institutions reveal that the scientists of aging in the United States were in a favorable situation in getting financial resources, especially if compared with British gerontologists. While aging itself was a relatively new subject, gerontologists could find a niche in the established funding system for various fields, such as medical research, nutrition, and physiology.

But the major sponsor of scientific research in the United States after World War II was the federal government rather than private philanthropies, and gerontology was no

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[168] Adler, "History of Gerontological Society," p. 100.
[169] William deB. MacNider, "Concerning the Naturally Acquired Resistance of the Livers of Certain Senile Dogs to Alcohol and Chloroform," *Proceedings of the Society for Experimental Biology and Medicine* 30 (1932), p. 237.
[170] Robert E. Kohler, *Partners in Science: Foundations and Natural Scientists, 1900-1945* (Chicago: University of Chicago Press, 1991), p. 7.

exception to this changed situation.[171] Indeed, the federal government supported both biomedical and social scientific research on aging and longevity at various laboratories and institutions.[172] At the Oak Ridge laboratories of the Atomic Energy Commission, the effects of radiation upon mice' longevity began to be investigated. The Bureau of Labor Statistics and the Office of Employment Security also conducted research on the relationship between the aging of workers and their prospect for getting secure employment. However, it was through the NIH's Division of Research Grants and Fellowships that the largest amount of federal money was spent for the study of aging. The emergence of the Unit on Gerontology—an intramural institute which also spent federal money for aging research—will be discussed in the next section.

In 1945, the Surgeon General authorized the Research Grants Program of the NIH "to provide absolute autonomy to the research worker," who would thereby make decisions on their own funding through their peer-review process without being directly controlled by the patron, the federal government.[173] Indeed, as historian Daniel Kevles has shown, the science policy in postwar America promoted by Vannevar Bush and others placed the authority and autonomy of scientists ahead of all other issues, including the democratic control of science and scientists' social responsibility.[174] The organizing principles for the NIH's Research Grants Program mandated that "there will be no attempt on the part of the Government to control, direct, or regiment the investigator," and the investigators themselves would have the full authority to choose the grantees and the amount of money that would be awarded to them.[175] To implement this plan, the NIH invited eminent physicians and biomedical scientists from various fields to form the

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[171] See Daniel J. Kevles, "The National Science Foundation and the Debate over Postwar Research Policy, 1942-1945," *Isis* 68 (1977), pp. 5-26; Kohler, *Partners in Science*, pp. 404-406.

[172] Nathan W. Shock and Annabel Wehrwein, "Government-Conducted Research in Gerontology," *Journal of Gerontology* 6 (1951), pp. 68-70.

[173] Gerontology Study Section Minutes of Meeting, 20 November 1946, p. 1, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA. For a historical review of the NIH's grant program, see Richard Mandel, *A Half Century of Peer Review, 1946-1996* (Bethesda, The National Institutes of Health, 1996).

[174] Kevles, "The National Science Foundation," pp. 5-26.

[175] Gerontology Study Section Minutes of Meeting, 20 November 1946, p. 1, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

"Study Sections" that were authorized to make decisions on funding in their area of specialty, including physiology, surgery, bacteriology, mental hygiene, and dentistry. After being recommended by a Study Section, applications were sent to the National Advisory Councils—such as the National Advisory Health Council, the National Advisory Cancer Council, or the National Advisory Mental Health Council—which would "ordinarily accept recommendation made by the Study Sections," although they could, in principle, turn down applications or request some modifications of the amount of money.[176]

At that time, the Gerontology Study Section (GSS) was created along with other Study Sections as a committee for funding medical and biological research on aging. There were several reasons why the NIH established the GSS to support aging research. First of all, the Unit on Gerontology had already been created within the NIH's Division of Chemotherapy in 1940, and its facility, funding, and scale of research became substantially expanded after the end of the War. Through the activity of this Unit, the problems of aging emerged as an important issue among the federal biomedical scientists during the postwar era. As one of these federal scientists, Cassius J. van Slyke, chief of the Division of Research Grants and Fellowships and director of the National Heart Institute (NHI), played a particularly important role in promoting research on aging. As chief of the Division, he was involved in assigning money to each Study Section including the GSS, and as the first director of the NHI he understood that patients' age was a crucial factor in the incidence of heart diseases. It is thus not surprising that he was appointed president of the Gerontological Society in 1950 and sponsored the growth of the intramural gerontology program, which was transferred from the Division of Physiology to the NHI in 1948. There were other people in the NIH who were sympathetic to research on aging. Most importantly, Lewis R. Thompson, who directed the NIH from 1937 to 1942, attended the meetings of the Club for Research on Ageing, and was involved in creating the Gerontology Unit and the National Advisory Committee

[176] Gerontology Study Section Minutes of Meeting, 20 November 1946, p. 1, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

on Gerontology.[177] William H. Sebrell, Jr., director of the NIH from 1950 to 1955, was also interested in aging as a scientific research topic since he was chief of the Division of Physiology to which the Gerontology Unit had initially belonged. Sebrell wrote several articles with Shock, and actively participated in the first meeting of the National Advisory Committee on Gerontology.[178]

But perhaps these people's interest in gerontology was less important in the establishment of the GSS than that of Rolla E. Dyer, who directed the NIH from 1942 to 1950 and was directly responsible for the creation of the Division of Research Grants and Fellowships in 1946. On January 6 of that year, he received a detailed report on the current state and the significance of the science of aging.[179] Although Dyer's response to this report is not known, we can assume that it was positive, because he eventually endorsed the creation of the GSS and invited a number of leading gerontologists as its panel members, including Henry Simms, William MacNider, Clive McCay, Nathan Shock, Ephraim Shorr, and Robert Moore.

As core members of the Club for Research on Ageing and the Gerontological Society, these scientists of the GSS tried to promote gerontology as a multidisciplinary scientific field. During their first meeting, it was argued that "aging is a complicated problem about which very little is known at the present time and that the problem should

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[177] Thompson wrote that he "[recognized the] importance" of gerontology, although he could not "lay any claim to know very much about the subject." See Thompson to Stieglitz, 18 November 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder Meeting November 25 & 26, NARA. A "fellowship" program for gerontologists was planned and discussed during the first meeting of the National Advisory Committee on Gerontology on November 25-26, 1940. See "Agenda for the First Meeting of the National Advisory Committee on Gerontology, 20 November 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder Meeting November 25 & 26, NARA; Minutes of the Meeting, 25-26 November 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee First Meeting November 25-26, NWS. Thompson actively participated in the first meeting of the National Advisory Committee on Gerontology.

[178] See, for example, N. W. Shock and W. H. Sebrell, "The Effect of Different Concentrations of Nicotinic Acid Amide on the Work Output of Perfused Frog Muscles," *American Journal of Physiology* 146 (1946), pp. 52-56. For Sebrell's participation in the first meeting of the National Advisory Committee on Gerontology, see Minutes of the Meeting, 25-26 November 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee First Meeting November 25-26, NWS.

[179] Justin J. Foley to Rolla E. Dyer, 6 January 1946, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder Gerontology, NARA.

be attacked from as many points of view as possible."[180] After this meeting, the GSS began to provide substantial financial assistance to many scientists of aging in the United States and Canada—including the panel members themselves—who worked in various fields such as endocrinology, neurology, psychology, physiology, biochemistry, and animal husbandry.[181] (See Table 6.2.) While social scientists were absent among the awardees of the grants, Henry Simms, as chairman of the GSS, stated in 1949 that "although the National Institutes of Health and Public Health Service are concerned primarily with medical fields, some sociological problems do border on the gerontological field and might very well be considered by this group."[182] Even though the GSS itself could not implement this statement of Simms, the NIH began to support social gerontologists from the 1950s through the National Institute of Mental Health.[183]

[180] Gerontology Study Section Minutes of Meeting, 20 November 1946, p. 4, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

[181] The GSS solicited funding applications to the members of the Club for Research on Ageing. See "To the Members of the Club for Research on Ageing," 7 January 1947, Box 41, Folder 10, EVC.

[182] Gerontology Study Section Minutes of Meeting, 9 May 1949, p. 2, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

[183] Before the establishment of the National Institute on Ageing, most social gerontology grants were awarded by the National Institute of Mental Health. In 1958, fifteen research projects in social gerontology were supported by the NIH. See "NIH Research and Training Grants Active on January 31, 1958," Box 12, Folder Annual Reports 1957, NWS.

Date	Applicants' Name	Discipline	Awarded
Nov 20 46	H.P. Smith and Henry Simms	Physiology	19,737
Feb 21 47	Robert Loeb and George Perera	Internal Medicine	19,737
	Henry Simms	Physiology	9,222
	Hans Selye	Physiology	27,540
	Joseph King and Maurice Visscher	Nutrition	16,753
	Clive McCay and H. S. Liddell	Nutrition and Animal Husbandry	29,109
	Gregory Pincus	Endocrinology	9,217
May 9 47	Mildred Trotter	Physiology	4,590
	Albert Lansing	Physiology	9,350
	Gregory Pincus	Clinical Psychology	21,251
	Ephraim Shorr	Physiology	36,866
Sep 29 47	Irvine Page	Physiology(Arteriosclerosis)	21,310
Feb 2 48	Henry Simms	Physiology	28,944
	Joseph King and Maurice Visscher	Nutrition	19,701
	Clive McCay and H. S. Liddell	Nutrition and Animal Husbandry	23,868
	Hans Selye	Physiology	27,540
	Albert Lansing	Physiology	12,258
May10 48	Max Goldzieher and William Rawls	Dermatology and Endocrinology	9,900
	Gregory Pincus	Clinical Psychology	23,684
	Ephraim Shorr	Physiology	36,720
	Ephraim Shorr	Physiology	7,414
	Nathan Shock	Physiology	15,000
	Anita Zorzoli	Biochemistry and Pathology	5,468
	Robert Newburger	Physiology	5,000
Sep 12 48	Irvine Page	Physiology(Arteriosclerosis)	23,750
	Irving Wagman	Neurology	10,600
Jan 7 49	James Hamilton	Endocrinology	11,718
	Walter Bauer and Jerome Gross	Pathology	45,674
	Paul Holbrook and A. R. Kemmerer	Internal Medicine	10,000
	Henry Simms	Physiology	31,104
	Hans Selye	Physiology	27,540
	Albert Lansing	Physiology	11,826
	Joseph King and Maurice Visscher	Nutrition	19,701
	Clive McCay	Nutrition and Animal Husbandry	23,868
	Oliver Duggins	Physiology	2,214
	Gregory Pincus	Endocrinology	10,216
	Christianna Smith	Cytology and Physiology	3,000
	John Lawrence and others	Clinical Psychology/Physiology	36,007
	Philip Hench	Conference on Rheumatic Diseases	15,000
May 9 49	Ephraim Shorr	Physiology	36,455
	Gregory Pincus	Clinical Psychology	23,965
	Max Goldzieher and William Rawls	Endocrinology	9,940
	Albert Lansing	Physiology	3,078
	Roland Davison	Internal Medicine	9,140
Total			804,975

Table 6.2. *The Name and Discipline of the Applicants Recommended by the Gerontology Study Section. From "Medical Research Projects: Gerontology," Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence II, The National Archives and Records Administration, College Park, Maryland. This Table Includes the Applications Recommended during the Final Meeting of the GSS.*

Obviously, the GSS was successful in realizing, at least partially, the ideal of the multidisciplinary approach to aging because its annual budget was quite large—it was $350,000 in 1948 and was expected to increase in later years.[184] The total amount of grant money actually awarded to scientists of aging through the GSS's recommendation was also large from the standard of the period, exceeding $800,000 in 1949.[185] The presence of such a sizeable lump-sum grant that could be spent according to the will and decision of gerontologists made the GSS completely different from its frustrated elder brother, the CBPA, which had to find it own sources of money through its chairmen's and members' fundraising efforts. While both aimed at becoming a funding agency that could promote multidisciplinary approach to aging by controlling the use of its budget, they differed in a crucial aspect—the CBPA had to find money from reluctant private philanthropies with fewer resources while the GSS's money was already guaranteed by the Congressional decision and the federal law. The same ambitious plan for funding multidisciplinary projects brought about very different results in distinct institutional settings.

However, the term of GSS's successful activities was not long. Ironically, the multidisciplinary approach to aging that the GSS hoped to encourage became a major cause for its disbanding in 1949. Whereas Eleanor Darby, executive secretary of the GSS, wrote that the NIH decided to disband the GSS because its "project load is just too small to justify the maintenance of a separate Study Section for that field," the issue of the

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[184] Gerontology Study Section Minutes of Meeting, 10 May 1948, p. 2, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

[185] See "Medical Research Projects: Gerontology," Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence II, NARA. While this chart shows that the total amount of grants was $725,739, it did not yet include the grants approved during the GSS's final meeting on May 9, 1949.

small project load concealed a more complex problem of the GSS concerning its multidisciplinary character.[186] It was true that the number of proposals that the GSS had to review was not large. The total number of its grants recommended by the GSS was less than twenty by August, 1948, while the Antibiotics Study Section recommended thirty-six and the Tuberculosis Study Section eighty-three.[187] However, these two Study Sections were disbanded along with the GSS in 1949, even though others with even smaller project loads, such as Gastroenterology and Public Health Study Sections, survived at that time.[188] What, then, was the real cause that brought about the demise of the GSS in 1949? To answer this question, it is important to note that the "project load," the number of applications assigned to each Study Section for review, was determined by the Project Review Officer within the Division of Research Grants and Fellowships.[189] This meant that applications were not directly submitted to each Study Section. Rather, the Officer within the Division had the authority to decide which Study Section should review each application. This system, even if the Officer worked in a completely fair and objective manner, was definitely unfavorable to the survival of the GSS that dealt with multidisciplinary projects. It was possible that a project that could have been assigned to the GSS was sent instead to other Sections whose field was a part of the multidisciplinary science of gerontology. For instance, most projects for studying cardiovascular diseases

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[186] Eleanor M. K. Darby to Members of Gerontology Study Section, 8 July 1949, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence II, NARA.

[187] David E. Price, "Research Grants and Research Fellowships Awarded through the National Institutes of Health by the Public Health Service," Record Group 443, Records of NIH, NIH 1930-48, Individual Institutes (Organization Files) Research Grants, Box 142, Folder 0745, NARA.

[188] By 1948, the Gastroenterology Study Section recommended only seven projects and the Public Health Study Section approved only eighteen. See Price to Simms, 30 June 1949, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence III, NARA; David E. Price, "Research Grants and Research Fellowships Awarded through the National Institutes of Health by the Public Health Service," Record Group 443, Records of NIH, NIH 1930-48, Individual Institutes (Organization Files) Research Grants, Box 142, Folder 0745, NARA.

[189] Gerontology Study Section Minutes of Meeting, 7 January 1949, p. 1, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

occurring in old age went to the Cardiovascular Study Section rather than the GSS.[190] In this sense, Darby wrote,

> Projects with implications for aging utilize a wide variety of techniques, from the culture of microorganisms to psychiatric survey procedures. These technical considerations made it necessary to assign a number of possible gerontological projects to other sections for review, leaving only a small ill-defined group to be classified as "Gerontology."[191]

Under this procedure, the project load of the GSS could not be high. Moreover, this situation led some people within the NIH to believe that the GSS did not have enough experts to review proposals belonging to a specific discipline. Darby recollected in 1965 that "the multidisciplinary group of scientists chosen to review such proposals provided very thin technical coverage of any one portion of the field—so thin, indeed, that the scientific merit review of a proposal assigned to the Gerontology Study Section was apt to depend much too heavily on the views of one or at the most two members, the rest being insufficiently conversant with the discipline under discussion."[192] The GSS did not only have too small work load but also had insufficient number of experts as a reviewing body. These were the crucial reasons for disbanding the GSS.

In retrospect, however, Darby's recollection was based on a view with which gerontologists themselves hardly agreed. While Darby and her NIH superiors, who had not been professionally associated with gerontologists, thought that gerontology's lack of disciplinary rigor could be a reason for disbanding the GSS, gerontologists themselves

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[190] Indeed, it was discussed that the projects on arteriosclerosis and hypertension were assigned to the Cardiovascular Study Section, even though these diseases were related to aging. See Gerontology Study Section Minutes of Meeting, 7 January 1949, p. 1, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Minutes of Meetings, NARA.

[191] Darby to Charles V. Kidd, 9 December 1949, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence III, NARA.

[192] Darby to Laurence Pilgeram, 18 October 1966, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Misc Items 655, NARA.

thought that cooperation and cross-disciplinary discussions were crucial for their research.[193] In fact, most gerontological organizations—including the Club for Research on Ageing and the Gerontological Society—were constructed according to this idea, and the GSS was also expected to realize the ideal of multidisciplinary cooperation through systematic funding. If Darby and other NIH officials thought that the GSS could not properly review the actual merit of an application due to the lack of sufficient number of experts in the field from which the application came, gerontologists could say that they, from a larger perspective based on multidisciplinary thinking, were able to see other important aspects of the application that the reviewers with narrow expertise could not recognize.[194] Unfortunately, this view of gerontologists was too ambitious and beyond the usual way of viewing scientific research at that time.

However, their agenda for multidisciplinary interaction, along with its actual usefulness in grant review process, was not completely ignored, since most members of the GSS were asked again in September, 1949 to form the Advisory Committee on Gerontology. This Committee conducted preliminary reviews of the applications related to aging, although it did not have the authority to recommend them directly to the National Advisory Councils. An application that was favorably evaluated by the Committee was sent to its "appropriate" Study Section for another round of review, and if the result of this review was also positive, the applications would be sent to the National Advisory Councils for the final approval for funding.[195] Through this process, the result of the Committee's "appraisals of individual projects as to scientific merit and

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[193] It is possible that David Price, who was appointed chief of the Division of Grants and Fellowships in 1948 after C. J. van Slyke became director of the NHI, held this view. However, multidisciplinary research was highly encouraged in several intramural research institutes within the NIH built according to disease categories. See Buhm Soon Park, "Disease Categories and Scientific Disciplines: Reorganizing the NIH Intramural Program, 1945-1960," in Caroline Hannaway (ed.), *Biomedicine in the Twentieth Century: Practices, Policies, and Politics* (Amsterdam: IOS, 2008), p. 43.

[194] This standpoint can be found in the view of James Birren of the Gerontology Research Center. See James E. Birren to John F. Sherman, 1 February 1965, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Misc Items 655, NARA. Also see Minutes of Meeting on Conference on Aging, p. 2, undated but probably 11 October 1950, Box 5, Folder USPHS, Conference on Aging, NWS.

[195] The Minutes of Meeting of the Advisory Committee on Gerontology, 9 December 1949, p. 2, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence III, NARA.

importance to the field of gerontology" could be "given due consideration by the study sections."[196]

In retrospect, this two-tiered review system, which would continue until 1973, represented the growth as well as the limit of the multidisciplinary approach of gerontologists in postwar America. On the one hand, the multidisciplinary science of gerontology was a field that was chosen as a beneficiary of the increasing federal budget for scientific research after World War II. It succeeded in being regarded as an area that the federal government committed to support. On the other hand, this benefit for gerontology was definitely circumscribed due to its multidisciplinary nature. Since gerontology was not a unified field, scientists of aging could not persuade the federal government to have their own review panel that had the full authority to determine their own support. Nevertheless, they maintained a certain degree of control of their funding through their Advisory Committee. Although gerontologists could not realize their original high expectation, they were able to survive as an academic field amid the rough sea of postwar federal funding.

In fact, even this half-success was very important for the later development of gerontology, because it laid the steps toward the establishment of the National Institute on Aging (NIA) in 1974, which would completely change the situation in favor of gerontologists' original agenda. Although its budget was still smaller than other richer institutes within the NIH, the NIA certainly transformed the research landscape of American gerontology after the 1970s. The next section will be devoted to explaining how gerontologists within the NIH, especially Nathan W. Shock in the Gerontology Unit, succeeded in preparing the pathway that led to the construction of the NIA. To do so, we have to go back to the late 1930s and to discuss the state of the Baltimore City Hospitals where the laboratories of the Unit were located. The role of the Macy Foundation in initiating the aging research there will also be described.

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[196] The Minutes of Meeting of the Advisory Committee on Gerontology, 25 March 1950, p. 2, Record Group 443, Records of the National Institutes of Health, Division of Research Grants, Records of the Gerontology Study Section, 1946-1950, Box 1 of 1, Folder Correspondence III, NARA.

The Growth of the Science of Aging as an Intramural Research Project within the National Institutes of Health, 1940-1958

The Baltimore City Hospitals (BCH) had a long tradition of caring for the sick and the poor within the town. From the late eighteenth century, the city of Baltimore had maintained its asylum to abate the "public nuisance" of indigent, vagrant, alcoholic, psychotic, and aged people.[197] While the hospital reform movement and the rise of the new public health inspired by germ theories did make a considerable change in the asylum which was renamed as the Baltimore City Hospitals in 1925, the role of taking care of the city's poor and ill had to be continued within their wards.[198] Whereas most private hospitals in the United States were transforming themselves with modern facilities focusing on more curable and acute cases, the BCH, like many other public hospitals, kept their traditional function of providing beds for the city's indigent and sick people, many of whom were aged and chronically ill.[199] In 1940, according to John T. King, chief of the medical service, the BCH had "350 chronic patients in addition to a large Alms House where the indigent and ambulatory old people are maintained."[200] In other words, borrowing historian Charles Rosenberg's phrase, the BCH was a public hospital where the "intractable burden of age, dependence, and chronic illness"—which could not be eliminated despite the success of modern medicine—had to be dealt with.[201]

However, as the focus of modern medical science was changing from infectious to chronic diseases, some people began to regard these aged patients at the BCH not as a "burden" but as a useful "clinical material" for the biomedical study of chronic diseases

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[197] Douglas Carroll, "The First Almhouse: Abating a Public Nuisance (1773-1822)," *Maryland State Medical Journal* 15:1 (1966), p. 87.

[198] Douglas Carroll, "The Bayview Asylum: Clinical Medicine (1911-1934)," *Maryland State Medical Journal* 15:8 (1966), pp. 69-71.

[199] Charles E. Rosenberg, *The Care of Strangers: The Rise of America's Hospital System* (Baltimore: Johns Hopkins University Press, 1987), pp. 322-327. Also see Morris J. Vogel, *The Invention of the Modern Hospital: Boston, 1870-1930* (Chicago: University of Chicago Press, 1980), pp. 72-75. Rosemary Stevens, *In Sickness and in Wealth: American Hospitals in the Twentieth Century* (New York: Basic Books, 1989), pp. 9-10, 28-29, 58-63, 142-143.

[200] King to Stieglitz, 23 May 1940, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS. For a more detailed report on the "clinical material" of the hospital, see "Clinical Material in Four Chronic Medical Wards at Baltimore City Hospitals as of March 20, 1941," Box 11, Folder E. J. Stieglitz Research Proposals, NWS.

[201] Rosenberg, *The Care of Strangers*, p. 326,

and senescence. An initial step in this alteration was taken by J. Murray Steele, a cardiologist who worked with Alfred Cohn at the Rockefeller Institute Hospital. Since his tenure as an associate member of the Institute was about to be terminated, he had to find another place of work where he could pursue clinical research on senescence and chronic diseases. For this purpose, he visited Baltimore on February, 1939, and discussed his research plan to use patients at the BCH with Thomas J. S. Waxter, director of Baltimore's Department of Public Welfare, and Alan Chesney, dean of the Johns Hopkins Medical School.[202] Steele also corresponded with John King, who, impressed by Steele's plan, wrote the following letter to Waxter. The clinical research using BCH patients would be an excellent opportunity for the people associated with the BCH's affairs as well as for the Baltimore city government officials, because

> ….it stimulates further investigation, and creates an alert progressive atmosphere; another is that the best type of men are attracted to the hospital for house staff appointments; and third, an institution that carries out investigative work successfully becomes a real city institution in the best sense. Boston City Hospital, it seems to me, now ranks as one of the cultural centers of Boston, comparable to the Conservatory of Music and the Harvard Medical School. In other words, research work of the type proposed is one of those things which, if properly handled, reacts to everyone's benefit, including that of the patients.[203]

Probably due to the lack of funds, however, Steele's plan could not be realized, even though Waxter, Chesney, and the Advisory Committee of the BCH were very favorable to Steele's idea.[204]

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[202] Steele to King, 17 February 1939, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS. In 1939, Steele went to Goldwater Memorial Hospital in New York. See J. Murray Steele, Application for Research Grant: Multidisciplinary Study of Aging, 16 January 1958, Box 11, Folder Aging Projects for Review, NWS.

[203] King to Waxter, 23 February 1939, Box 11, Folder Administrative files—E. J. Stieglitz 1941, I, NWS.

[204] See King to Waxter, 23 February 1939, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS; King to Steele, 6 March 1939, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS; King to Steele, 7 June 1940, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS.

But Steele was not alone in planning aging research at the BCH. On March, 1940, Lawrence Frank of the Macy Foundation met with NIH director Lewis Thompson in Washington, D.C. to discuss the issue of starting aging research at the NIH. Two months later, the Foundation awarded the NIH a short-term grant for establishing the Unit on Gerontology within the NIH and hiring a renowned medical researcher Edward Stieglitz as chief of the Unit.[205] Stieglitz, who specialized in chronic diseases in the urinal system and blood vessels, immediately recognized the potential usefulness of the BCH which was quite close to the NIH's Bethesda campus.[206] He thus contacted John King about "the potentiality of using certain portions of the vast clinical material….at the City Hospital and the Alms House."[207] King gladly reported this new opportunity to all the concerned people and committees, including Waxter, the Advisory Committee of the BCH, and the deans of the Johns Hopkins and the University of Maryland Medical Schools. All of them responded to Stieglitz' proposal very favorably.[208] Through their approval, Stieglitz was officially appointed a medical staff member of the BCH on October 1940 and was allowed to use aged patients there for his clinical investigation of senescence.[209] (See Figure 6.2.) The association between the Unit and the BCH began from the fall of 1940.

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[205] Frank to Thompson, 11 March 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder 0110, NARA; Thompson to Frank, 12 March 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder 0110, NARA; Thompson to Karl Meyer, 1 May 1940, Record Group 443, Records of NIH 1930-1948, Individual Institutes (Org. File) Chemistry—Phys Biology National Heart Institute, Box 135, Folder 0110, NARA.

[206] Stieglitz finished his bachelor's and master's degrees at the University of Chicago in 1918 and 1919. He then went to Rush Medical College and earned an M.D. degree in 1922. He then taught and did research at Rush Medical College on the chronic diseases involving the kidney, renal function, and hypertension. See "Edward J. Stieglitz, M.D.," Box 82, Folder 1109, WBC.

[207] Stieglitz to King, 21 May 1940, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS.

[208] King to Stieglitz, 8 June 1940, Box 11, Folder Administrative files—E. J. Stieglitz 1941 I, NWS; King to Stieglitz, 16 September 1949, Box 11, Folder Admin. files E. J. Stieglitz 1939-1941. I, NWS.

[209] King to Stiegliz, 4 October, 1940, Box 11, Folder Admin. files E. J. Stieglitz 1939-1941. I, NWS.

Figure 6.2. *Baltimore City Hospital Staff, Department of Medicine, 1941. Edward J. Stieglitz is sitting in the front row, on the sixth from the left. John T. King is the fourth in the same row. Picture 1-2, Box 44, Folder Photographs, Professional 1937-1958, The Nathan W. Shock Papers, Bentley Historical Library, University of Michigan.*

This association did not occur by chance. It was a product of the NIH's ongoing change of its research priorities, which was in harmony with the agenda of the Club for Research on Ageing and the Macy Foundation. While the precursor of the NIH, the Hygienic Laboratory, focused on short-term bacteriological investigations during the late nineteenth and early twentieth centuries, it gradually expanded the scope of its research, which eventually came to put more stress on the study of chronic diseases occurring in more aged patients.[210] Surgeon General Thomas Parran made this point clear in his opening remarks for the Conference on Mental Health in Later Maturity in 1941. "Because of the effective control of the communicable and infective diseases of infancy and youth has permitted the survival of many formerly dying young," he argued, "the

[210] Victoria A. Harden, *Inventing the NIH: Federal Biomedical Research Policy, 1887-1937* (Baltimore: Johns Hopkins University Press, 1986), p. 55; Buhm Soon Park, "The Development of the Intramural Research Program at the National Institutes of Health after World War II," *Perspectives in Biology and Medicine* 46 (2003), p. 386; G. Burroughs Mider, "The Federal Impact on Biomedical Research," in John Z. Bowers and Elizabeth F. Purcell (eds.), *Advances in American Medicine: Essays at the Bicentennial*, Vol. 2 (New York: The Josiah Macy, Jr. Foundation, 1976), pp. 837, 853.

disorders of later maturity take on greatly increased significance."[211] In particular, "the prolonged disability from cardiovascular-renal disease, arthritis, diabetes mellitus, cancer, and the mental disorders which not infrequently beset those past the fourth decade represent a gigantic medical and socio-economical problem" that urgently demanded proper treatments by biomedical research.[212] Indeed, this was the reason NIH director Thompson attended the first meeting of the Club for Research on Ageing in 1941 where he discussed the problems of senescence and chronic diseases with other attendees. Other participants in the meeting included those who were directly involved in establishing the Unit, Edward Stieglitz and Lawrence Frank.[213] The Unit on Gerontology, born and developed through these people's efforts, was a product of the consensus reached among the NIH, the Macy Foundation, and the Club for Research on Ageing.

But the Unit during Stieglitz' short tenure was quite limited in its activity due to its insufficient funds. The Macy grant was far from being adequate for initiating systematic research on aging, and the U. S. Public Health Service could not appropriate budgets for biomedical projects which were not directly related to war efforts.[214] In such circumstances, the only possible source of the Unit's research expense seemed to be the CBPA, which had just begun a new fundraising activity under its second chairman, Anton Carlson.[215] As I have written in this chapter, however, the CBPA failed to raise any money, and Stieglitz could not carry out his comprehensive research plans which included both clinical and experimental studies of renal function, hypertension, blood

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[211] "Opening Remarks of Surgeon General Thomas Parran," p. 2, 23 May 1941, Box 11, Folder E. J. Stieglitz Conference on Mental Health 1941, NWS. For Parran's other remarks on aging and chronic disease, see U. S. Congress, House, Subcommittee of the Committee on Appropriations, *Department of Labor-Federal Security Agency Appropriation Bill for 1948*, 80th Cong., 1st sess., 1947 (Washington, U.S. Government Printing Office, 1947), 275-277.

[212] "Opening Remarks of Surgeon General Thomas Parran," p. 2, 23 May 1941, Box 11, Folder E. J. Stieglitz Conference on Mental Health 1941, NWS.

[213] The Club for Research on Ageing Minutes of Meeting, p. 20, 11-12 January 1940, Box 41, Folder 2, EVC.

[214] The Minutes of the First Meeting of the National Advisory Committee on Gerontology, pp. 12-13, 26 November 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee First Meeting, Nov. 25-26, NWS.

[215] Minutes of the First Meeting of the National Advisory Committee on Gerontology, pp. 14-15, 25-30, 26 November 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee First Meeting, Nov. 25-26, NWS.

nitrite concentration, and cytoplasmic hydrogen ion concentration.[216] The only research whose result Stieglitz was able to publish was the clinical investigation of uric acid clearance rate using the patients at the BCH.[217] Marvin Yiengst, a lab technician hired by the small fund provided by the Public Health Service, was the sole staff member who assisted this work.[218]

Yet Stieglitz did contribute to the development of the Unit and gerontology in at least three aspects. First, he organized the meeting of the National Advisory Committee on Gerontology in 1940 and the Conference on Mental Health in Later Maturity in 1941.[219] While the former was a closed meeting in which the participants talked about the policies concerning the Unit's future development, the latter was a conference open to the general public where broader issues including psychological and social problems of aging population were discussed.[220] Second, Stieglitz conducted a comprehensive survey of the current state of gerontological research in the country. This survey was expected to help professional scientists of aging update their knowledge of the state of the field and recognize the major research topics in each research area.[221] Third, he interacted with the general public who hoped to get some help from the federal government's involvement in aging research. It is remarkable that he sincerely tried to reply to most inquiries from lay people, even if they raised issues irrelevant to current or future activity of the Unit.[222] In

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[216] See "Proposed Investigations by Unit on Gerontology," Box 11, Folder E. J. Stieglitz Research Proposals, NWS.

[217] Edward J. Stieglitz, "Studies in Uric Acid Clearance," *Public Health Reports* 57 (1942), pp. 1306-1310.

[218] Progress Report, p. 1, October 1943, Box 12, Folder Annual Report 1943, NWS.

[219] Stieglitz to Thompson, 16 May 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee Members, NWS; Stieglitz to Sebrell, 20 May 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee Members, NWS; Moulton to Stieglitz, 21 May 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee Members, NWS.

[220] The "admission ticket" to the Conference on Mental Health was sold at $2.25. See Conference on Mental Health in Later Maturity," 23-24 May 1941, Box 11, Folder E. J. Stieglitz Conference on Mental Health 1941, NWS; Stieglitz to Frank, 27 February 1941, Box 11, Folder E. J. Stieglitz Conference on Mental Health 1941, NWS. About the meeting of the National Advisory Committee on Gerontology, see The Minutes of the First Meeting of the National Advisory Committee on Gerontology, 26 November 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee First Meeting, Nov. 25-26, NWS.

[221] Edward J. Stieglitz, *Report of a Survey of Active Studies in Gerontology* (Bethesda, Maryland: The National Institute of Health, 1942).

[222] Many lay people requested some information about their own or their parents' health in old age, about which Stieglitz could give no other advice than recommending the help available from local doctors. See, for example, Olga Frank to Stieglitz, 4 December 1940, Box 11, Folder E. J. Stieglitz National Advisory Committee 1940-1942, NWS; Raymond Magill to Stieglitz, 24 November 1942, Box 11, Folder E. J.

retrospect, Stieglitz, through these works, contributed to promoting the Gerontological Society's aims. He helped gerontologists obtain broader publicity, keep abreast of the current state of their field, and get recognition as the experts in the field of aging.

On the summer of 1941, however, Stieglitz had to resign, and the Unit's chief position was given to Nathan W. Shock, who was then an assistant professor of physiology at the University of California, Berkeley.[223] (See Figure 6.3.) While no official record remains concerning this change of directorship, Lawrence Frank, in his personal letter to McCay, stated that Stieglitz had to leave the NIH "because of a development in his own personal family life which led the Surgeon General to request his resignation."[224]

Figure 6.3. *Nathan W. Shock in 1954. Picture 10-11: Box 44, Folder Photographs, Professional 1937-1958, The Nathan W. Shock Papers, Bentley Historical Library, University of Michigan.*

Stieglitz National Advisory Committee 1940-1942, NWS. It is noticeable that Stieglitz did not neglect to reply even to a letter from a person who advocated "electrobiology" and its implication for aging. See Stieglitz to Bela Gati, 23 January 1941, Box 11, Folder National Advisory Committee 1940-1942, NWS; Gati to Stieglitz, 27 December 1940, Box 11, Folder National Advisory Committee 1940-1942, NWS.

[223] For a brief biographical sketch of Shock, see "Nathan W. Shock," in W. Andrew Achenbaum and Daniel M. Albert (eds.), *Profiles in Gerontology: A Biographical Dictionary* (Westport, Conn.: Greenwood, 1995), pp. 321-326.

[224] Frank to McCay, 26 September 1953, Box 30, Folder Macy Foundation Contribution, NWS. Although Stieglitz returned to his private medical practice in Washington, D.C., he remained an important member in the gerontology community.

The reason why Shock was chosen as the new chief of the Unit is not clearly known, either. Yet it is certain that he was recommended by two members in the Club for Research on Ageing, Lawrence Frank and Baird Hastings whose opinions were considered important within the NIH.[225] Frank as executive secretary of the Macy Foundation provided a grant for initiating the Unit, and Hastings was a close colleague of NIH director Lewis Thompson in the U. S. Public Health Service.[226] Indeed, Hastings also knew Chemotherapy Division chief William Sebrell, who asked Hastings to recommend an experimental scientist who could fill the position of "physiologist" within the NIH.[227] Hastings, as a mentor of Shock, recommended him without hesitation.

Shock's previous career and his past interactions with Frank and Hastings provide further clues on how he came to take the directorship of the Unit. Born in Lafayette, Indiana in 1908, he earned his bachelor's and master's degree at a school in his hometown, Purdue University. He then went to the University Chicago where he finished his Ph.D. work in physiology and psychology in 1930 under the supervision of Hastings, Anton Carlson, and L. L Thurstone. It is noticeable that Hastings and Carlson became active members of the Club after 1940, and, that upon finishing his dissertation, Shock himself began to pursue research on how children's various physiological parameters changed in accordance with their *age*. As a research associate at Chicago and an assistant professor of physiology at the University of California, he studied and taught the "age changes" in cardiovascular, respiratory, and nervous functions,[228] and used a longitudinal method to trace the actual physiological changes of boys and girls for an

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[225] Sebrell to Shock, 23 May 1941, Box 1, Folder Career Appointments, NWS.

[226] Hastings to Shock, 5 June 1941, Box 1, Folder Career Appointments, NWS. Also see "Dr. Albert Baird Hastings," Box 1, Folder Personal Data—Biographical, ABH.

[227] Hastings to Shock, 5 June 1941, Box 1, Folder Career Appointments, NWS. Shock himself tells a similar story. See Nathan W. Shock, "Historical Perspectives on Aging," in Steven M. Horvath and Mohamed K. Yousef (eds.), *Environmental Physiology: Aging, Heat and Altitude* (New York: Elsevier, 1981), p. 386.

[228] See, for example, "Outline of Course: Physiology #102, Physiology of Growth and Development of the Child," Box 2, Folder U of C Physiology of Growth, NWS; "Inventory of Procedures in Study of Adolescence," 1 January 1938, Box 2, Folder U of C Adolescence studies, NWS. He went to California in 1933.

extended period.[229] Although he did not yet deal with age changes in later periods of life at that time, it is important that he was funded by the Macy Foundation's "Life Cycle" program initiated by Lawrence Frank, which aimed at promoting research on all phases of life, including old age.[230] Shock, by accepting the job offer from the NIH, was closely following the career of Lawrence Frank, whose interest in age changes in childhood and adolescence was extended into senescence after he began to work for the Macy Foundation.[231]

Shock's early research at the Unit clearly shows how this extension was made possible. First, to measure the physiological changes in the elderly patients' body, he continued to employ some of the laboratory apparatus he had used in California for adolescents.[232] Second, he used the method he had adopted in his physiological research with Hastings at Chicago. As he had done during the early 1930s, he deliberately disturbed the acid-base equilibrium in blood by chemical means, and examined how long it took for this disturbed blood to return to its normal state.[233] In this study, he found that that aged people's blood stayed at an altered chemical balance for a longer period than that of younger subjects.

After he changed the age of his experimental subjects, however, Shock became aware of many new academic and social issues concerning aging along with the new scholarly colleagues that he had not known in the past. He participated in various meetings and organizations on aging—including the Conference on Nutritional Requirements for the Ageing Population, the Club for Research on Ageing, and the Gerontological Society. In 1960, he served the Gerontological Society as president. Through his interaction with his gerontologist colleagues, he joined the discussion of the

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[229] This study continued even after he moved to Bethesda in 1941. See N. W. Shock, "The Physiological Changes Taking Place during Adolescence: A Program of Research," p. 5, September 1938, Box 1, Folder Research Proposal, NWS; Harold Jones to H. H. Remmers, 5 April 1954, Box 1, Folder Career Appointments, NWS.

[230] Frank to Robert Underhill, 10 January 1940, Box 2, Folder U of C correspondence K-Z, NWS.

[231] See chapter one. Also see Frank, "Gerontology," p. 2; "Comments on the Problems of Ageing: Presented to the Meeting of the Club for Research on Ageing," 12-13 January 1940, Box 11, Folder Admin. files E. J. Stieglitz 1939-41, I, NWS.

[232] Progress Report, p. 2, October 1943, Box 12, Folder Annual Report 1943, NWS.

[233] Shock to "Tommy," 30 May 1944, Box 7, Folder Correspondence 1934-1974, NWS.

difference between physiological and chronological age as well as the meaning of this difference for the use of aged workers in industries.[234] Since the two was not always the same, it was necessary to measure the former precisely using various physiological techniques and to use this information as a basis for gainful employment of elderly people.[235] Shock also thought that these tests might also be necessary to solve the problem of manpower shortage during World War II, as Stieglitz and others had already pointed out. Indeed, he tried to redirect the research orientation of the Unit toward the study of physical fitness of aged and young workers when the Unit was ordered to assist the war efforts.[236]

But Shock had to implement this research using only young people, suspending his project on old age until the end of the War. Gerontology was considered too far from the war effort by the federal government. Nevertheless, he was able to restart his research on aging after the end of the War with substantially increased budget, space, and personnel. The Unit on Gerontology was promoted to a "Section" within the Division of Physiology in 1946, and its total budget, which was $42,000 in that year, increased more than four fold in 1947, reaching $188,000.[237] The space allotted for Shock's research in the Baltimore City Hospitals was also considerably expanded. Whereas his Unit occupied only 1,500 square feet as its laboratory space from 1942 to 1945, it was allowed to use 11,000 in 1947, which included the room for patient beds and utilities as well as the laboratory space.[238] This enlarged space and increased budget accompanied the addition of new personnel. Although Shock had lost his sole staff member Marvin Yiengst due to

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[234] "Conference on Nutritional Requirements for the Ageing Population," pp. 10-11, 1-2 November 1941, Box 1, Folder Nutritional Requirements Conference 1942, GS.

[235] Report of Unit on Gerontology: Oct. 1941 to Oct. 1943, p. 1, Box 12, Folder Annual Report 1943, NWS; Nathan W. Shock, "Older People and Their Potentialities for Gainful Employment," *Journal of Gerontology* 2 (1947), pp. 93-102. He was interested in ergonomics of aged people since 1942. See Shock to Herbert Conrad, 27 July 1942, Box 7, Folder Correspondence 1934-1974, NWS; Shock to MacNider, 1 October 1942, Box 30, Folder Club for Aging 1942, March, NWS.

[236] Report of Unit on Gerontology: Oct. 1941 to Oct. 1943, p. 4, Box 12, Folder Annual Report 1943, NWS; Shock to Hastings, undated yet probably 1943, Box 25, Folder Shock 1942-71, ABH.

[237] See Sebrell to Shock, 24 July 1946, 23 June 1947, 6 November 1947, 15 March 1948, Box 13, Folder 1941-49 (incomplete), NWS. I took out the amount smaller than $1,000.

[238] "Space Used by Section on Gerontology," 10 December 1947, Box 13, Folder 1941-1949 (incomplete), NWS. Actually even initial space of 1,500 square feet was an increase from what Stieglitz occupied in the BCH by 1941. See Shock to Olmsted, 15 April 1942, Box 7, Folder Correspondence 1934-1974, NWS.

military conscription during World War II, Yiengst came back to the lab after the War along with three professional staff members and four technical assistants.[239] This expansion was accelerated when the National Heart Institute was established in 1948 and the Section was incorporated into it.[240] The budget and personnel of the Gerontology Section kept increasing along with that of its host institution.

This postwar growth of gerontology in the NIH was related to a specific way of promoting healthcare which the United States federal government preferred. Indeed, the NHI was one of the several new research institutes established within the NIH as a part of the postwar expansion of federal biomedical programs, and this expansion has been known as an alternative to the national health insurance plans that could not be introduced due to the vehement opposition by the American Medical Association and others.[241] As historian Victoria Harden has put it, medical research funded by the federal government was the "principal way by which congressmen could vote to improve their constituents' health" including "the elderly and the indigent" when the establishment of the national health insurance and other measures proved to be impossible.[242] In retrospect, this situation at least partially explains why gerontology fared much better in America than in Britain. It is noticeable that when Americans began to build new national medical research institutes such as the NHI, Britons established the National Health Service (NHS) as the comprehensive national health insurance agency. The two countries differed in their major means of responding to issues of health—the United States chose medical research while the United Kingdom chose free healthcare—and this difference influenced the two countries' distinct way of dealing with the issue of aging. Whereas

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[239] Shock to Roy and Frieda, 30 May 1944, Box 7, Folder Correspondence 1934-1974, NWS; Sebrell to Shock, 24 July 1946, Box 13, Folder 1941-49 (incomplete), NWS; "Budget," 6 June 1945, p. 6, Box 13, Folder 1941-49 (incomplete), NWS.

[240] Buhm Soon Park has argued that the postwar NIH intramural program, which was realized in the establishments of new institutes during the 1940s and 1950s, had a "dual structure." According to Park, while the institutes were built according to disease categories, each institute had laboratories and research units organized according to disciplinary boundaries, making the institute a multidisciplinary organization. See Park, "Disease Categories and Scientific Disciplines," pp. 28, 43. Yet gerontological research within the NHI did not follow this structure, since it was already multidisciplinary by itself and did not belong to any disease category.

[241] See Stephen P. Strickland, *Politics, Science, and Dread Disease: A Short History of United States Medical Research Policy* (Cambridge, Mass.: Harvard University Press, 1972), pp. 154-156, 213.

[242] Harden, *Inventing the NIH*, p. 182.

Britons decided to promote a better pension system and the NHS-sponsored geriatric care, Americans supported more basic research on aging supported through the NIH. Shock and his fellow gerontologists certainly benefited from this choice of their government and Congress.

The place of the Social Security Act in this American system of caring for the aged is interesting. As I have pointed out in chapter five, the Act reflected Americans' deep concerns about the plight of the elderly during the 1930s and influenced the people who would build gerontology. While the Act was more comprehensive than in its scope its British counterpart, the Old Age Pensions Act of 1908, it and its later revisions still did not include any plans for national health insurance. Instead, the Social Security Act of 1935 had Title VI which enabled the Public Health Service to spend $2 million each year for health research, which probably meant the research on chronic illness affecting the middle aged or the elderly.[243] The fact that this NIH's budget kept increasing rapidly since the Act was implemented and the gerontology program also benefited from this reveals that in America even this explicitly welfare-oriented measure for the elderly included a part on promoting research which eventually came to include gerontology.

Other factors, including personal, scientific, and institutional matters, contributed to the postwar expansion of gerontology within the NHI. First of all, Shock's struggles to persuade government administrators, particularly those within the U. S. Public Health Service, of the importance of aging research should not be neglected. He wrote to his mentor Hastings that he "spent so much time trying to educate administrators [on] the importance of fundamental research in aging."[244] In fact, through several informal meetings, he tried to persuade federal administrators, some of whom later became strong

[243] While the contents of Title IV did not include any mention of chronic illness or aging, historians have implied that the Title was made for solving chronic disease problems. See Harden, *Inventing the NIH*, p. 173, Mider, "The Federal Impact on Biomedical Research," p. 837.

[244] Shock to Hastings, 11 March 1953, Box 25, Folder Shock 1942-71, ABH. He also said in an interview that he had a particularly hard time in persuading James Shannon, who was then associate director of the NHI, and Robert Berliner, one of Shannon's colleagues. See "NIA Interview Transcript," p. 27-29, Box 1, Folder Biography, NWS.

supporters of gerontology.[245] In particular, as I have written in the previous section, C. J. Van Slyke who was the first director of the NHI became a strong advocate of gerontology. According to Shock's later recollection, Van Slyke was a "permissible character" with whom Shock "got along quite well."[246] Van Slyke was well aware of the importance of age in the incidence of heart diseases and even took the position of president of the Gerontological Society in 1950. Champions of gerontology could also be found outside of the federal institutions. Many of the administrators of the BCH and the Baltimore Department of Public Welfare, including King and Waxter, were very favorable to Shock's research and tried to provide him with as much space as possible in the hospital.[247] The fact that Shock and his colleagues were highly productive in their investigations in this space the BCH provided should also be considered as a factor contributing to the Section's rapid expansion. In fact, his performance in the NIH was always evaluated as "excellent," and his team kept publishing a large number of scientific articles.[248] Moreover, as Shock himself said, the Section' incorporation into the NHI, in which few things were settled at the time, was fortunate for gerontology's survival and proliferation. According to his later recollection on William Sebrell's remark, the Section's "odds were better within the framework of a newly formed institute than in a well-established, ongoing program."[249] This statement can be supported by the financial state of the Section, which, as of the latter half of 1949, had the largest operating budget among all the sections and branches within the NHI.[250] It is also remarkable that various research projects of the Section were not required to justify their relevance to heart

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[245] Minutes of Meeting on Conference on Aging, undated but probably 11 October 1950, Box 5, Folder USPHS, Conference on Aging, NWS; Minutes of the Second Meeting on Gerontology, undated, Box 5, Folder USPHS Conference on Aging, NWS.

[246] See "NIA Interview Transcript," p. 24, Box 1, Folder Biography, NWS.

[247] See "NIA Interview Transcript," pp. 25-26, Box 1, Folder Biography, NWS.

[248] The official evaluation on Shock's performance can be found "Notice of Official Efficiency Rating," Box 7, Folder Correspondence 1934-1974, NWS. See National Heart Institute, Gerontology Branch, *Publications of the Gerontology Branch, National Heart Institute, 1940-1962* (Bethesda, Maryland: The U. S. Department of Health, Education, and Welfare, 1962).

[249] "NIA Interview Transcript," p. 24, Box 1, Folder Biography, NWS.

[250] "National Heart Institute Operating Budget," July-December 1949, Box 13, Folder Budget & Operating Plan Jul-Dec 1949, NWS; "National Heart Institute Operating Budget," January to June 30, 1950, Box 13, Folder Budget & Operating Plan Jan-June, 1950, NWS.

diseases in their official proposals and reports.[251] This implies that research on aging was granted an independent status within the NHI, even though the studies of the influence of aging in the occurrence of heart diseases were always welcomed.

The expansion of the Gerontology Section can be seen in the broadened spectrum of its research programs during the 1950s as well. While Shock's program primarily focused on clinical research on renal and heart functions during its early years, new experimental studies of longevity and metabolic activity using non-human subjects, especially rats and flies, were initiated after the Section was incorporated as a part of the NHI.[252] It is also remarkable that psychological studies of aging, which began to be pursued right after the War, were gradually expanded during the 1950s. Moreover, following the discovery of DNA's structure, molecular biological and biochemical approaches to aging were introduced into the gerontology laboratories within the BCH.[253] Such an expansion of research programs was accompanied by the promotion of the status of gerontology program within the NHI and its further subdivisions which reflected its diversified research projects. In 1956, the Section on Gerontology became the Gerontology Branch that included the units on biochemistry, morphology, biophysics, molecular biology, human physiology, and intermediary metabolism. The budget increase of nearly $200,000 in 1957 expedited this expansion further, since it enabled Shock to establish the unit on cellular physiology and to add new personnel and facilities to the existing units.[254] As of 1958, these units were operated by twenty-two professional staff members along with twenty-six technicians and six administrative and secretarial employees.[255] These gerontology staff represented the multidisciplinary approach to

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[251] Most research proposals did not specify how they were related to heart diseases even though the official project description form had an item on "Significance of Heart Research." See, for example, N. W. Shock, S. P. Baker, G. W. Gaffney, F. A. Silverstone, "Analysis of NIH Program Activities Project Description Sheet: Age Changes in Metabolism and Endocrine Function," undated, Box 12, Folder Annual Report 1955, NWS.

[252] "Adaptive Enzymes and Age," Box 12, Folder Annual Report 1952-56, NWS; N. W. Shock, Gerontology Branch Annual Report—1957, Box 12, Folder Annual Reports 1957, NWS.

[253] See, for example, Charles H. Barrows, "Individual Project Report: Age Changes in Cellular and Tissue Biochemistry," 1956, Box 12, Folder Annual Report 1956, NWS.

[254] "Distribution of New Money ($200,000) for Gerontology Branch – 1958-1959, 15 May 1958, Box 14, Folder Staff 1956-1965, NWS.

[255] "Staff—Division of Research Gerontology," 15 August 1958, Box 14, Folder Staff 1947-1955, NWS.

aging, contributed by physiologists, biochemists, embryologists, psychologists, and physicians.

Shock and his colleagues' research showed a culmination of the new views on senescence that emerged during the early and mid-twentieth century. As Cowdry, McCay, and others have done, Shock took the local nature of aging for granted and reconfirmed the feasibility of experimental approaches to senescence with more systematic investigations. In a book Shock coauthored with these scholars—the third edition of Cowdry's *Problems of Ageing*—he argued that each apparatus involved in homeostatic regulation showed a distinct pattern and rate of senescence.[256] Therefore, some homeostasis regulation mechanisms still worked efficiently even in extremely old age, while other devices tended to be disrupted more rapidly as the organism underwent senescence. Interestingly, this standpoint on aging enabled Shock to be more rigorous in searching for the *cause* of a particular body part's impaired function with aging. If one portion of an organ did not show any difference with age while others did, the latter could be suspected as the prime cause of the senile symptoms that the organ as a whole showed. In fact, he found a number of cases supporting this standpoint, especially through his investigation of the impairment of the kidney in old age. He argued that the lowered activity of the elderly's kidney was primarily due to the reduced activity of the part responsible for its tubular (secretory) function rather than its glomerular (filteration) function, because only the former became less effective as the organism aged.[257] Shock and his colleagues also found a number of cases showing that many constituents of the body showed little or no change even after it became extremely old, and individual variations in a cohort age group were sometimes much greater than the differences an individual showed as it became older. For an instance, he showed that the water content of the cell did not change much according to age, even though numerous scholars since Aristotle, including some physiologists of the early twentieth century, had believed that

[256] Nathan W. Shock, "Ageing of Homeostatic Mechanism," in Albert I. Lansing (ed.), *Cowdry's Problems of Ageing*, Third Edition (Baltimore: Williams and Wilkins, 1952), pp. 421, 429-31, 436, 438.
[257] Nathan W. Shock, "Kidney Function Tests in Aged Males," *Geriatrics* 1 (1946), pp. 232-9.

aging was basically a process of "drying."[258] Likewise, the reaction time and sensitivity to some types of drugs showed almost no difference between the young and the aged.[259]

These findings on aging were deeply related to larger social and industrial issues on the elderly's employment and work. Since some body parts' functions were hardly altered with aging and individual differences in senescence were greater than commonly thought, it was important to devise ways to measure an individual aged worker's work capacity accurately to deploy the person at the right place in industries and to make more realistic retirement policies.[260] It was also necessary to conduct more systematic intelligence testing and to apply its results to actual workplaces, since it was found that the mental activity that did not require speed showed little age differences.[261] Moreover, despite their initial slowness, the aged laborers tended to maintain their performance for a longer period with fewer accidents than the young.[262] With this study result in mind, Shock wrote to his mentor Hastings that "ten years of research in our laboratory certainly tended to emphasize one of the questions phrased; namely, that the limitations on the physiological level of older people are more apparent than real."[263]

For Shock, however, this assertion had to be qualified in some measure. While the scope and size of Shock and his colleagues' research were expanding rapidly during the 1950s, there were at least two problems that should be dealt with to make their work more meaningful for social and industrial applications. First, their clinical research did not seem to produce reliable data on the actual aging process occurring in individual humans, since these studies still relied upon cross-sectional methodologies. Although the

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[258] Nathan W. Shock, "Physiological Aspects of Mental Disorders in Later Life," in Oscar J. Kaplan (ed.), *Mental Disorders in Later Life* (Stanford: Stanford University Press, 1945), p. 33. Also see, J. A. Murray, "The Chemical Composition of Animal Bodies," *Journal of Agricultural Science* 12 (1922), pp. 103-10; H. H. Donaldson and S. Hatai, "On the Weight of the Parts of the Brain and on the Percentage of Water in Them according to Brain Weight and to Age, in Albino and Wild Norway Rats," *Journal of Comparative Neurology* 53 (1931), pp. 263-307.

[259] Shock, "Physiological Aspects," pp. 44, 48.

[260] Shock, "Older People," p. 101; National Heart Institute Annual Report, Calendar Year 1952, p. 65, Box 12, Folder Annual Report, 1952, NWS; Summary Statement of Research Accomplished—1955 Section on Gerontology, p. 3, Box 12, Folder Annual Report 1955, NWS.

[261] Shock, "Older People," p. 96.

[262] National Heart Institute Annual Report, Calendar Year 1952, p. 66, Box 12, Folder Annual Report, 1952, NWS; Shock, "Older People," pp. 98-99.

[263] Shock to Hastings, 11 March 1953, Box 25, Folder Shock 1942-71, ABH.

conclusion drawn from a comparison between distinct young and old persons was not insignificant, it still contained possible sources of error caused by individual differences. Second, their projects using indigent old people in a public institution, whose physiology was thought to be somewhat different from the "great majority of the aged who lived normal lives in their own communities," might produce results that could not tell much about the "normal" patterns of aging.[264] Even though the elderly research subjects were chosen among those who did not have manifest symptoms of disease, it was still possible, Shock and other thought, that their aging processes had deviated from the "normal" because their poor socio-economic conditions could negatively influence their health and the results of the research.

The Baltimore Longitudinal Study of Aging (BLSA) was devised to solve these two problems. By encouraging healthy and well-off individuals to come to the BCH in a regular interval and have their physiological and psychological parameters—such as their pulmonary, cardiovascular, and neuromuscular functions, as well as the state of their blood, urine, and intelligence—monitored across their lifetime, Shock and others could trace actual aging process in an individualized manner without worrying about using the indigent aged.[265] It is quite remarkable that the BLSA, which began with one volunteer in 1958, gained an increasing number of volunteers who were twenty to ninety years of age. In 2008, more than fourteen hundred volunteers are participating in this longest-running research project on aging.[266]

What, then, made this project so successful? An important factor can be found in the experience of Nathan Shock himself, who had already performed similar research using children in California during and after the 1930s. However, the BLSA could not have been successful without the enthusiastic support of a large number of lay volunteers who believed that gerontological research was worth pursuing and highly important for

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[264] Baltimore City Hospitals Superintendent's Office, p. 2, 25 November 1957, Box 22, Folder Longitudinal Study Administrative Record 1956-1962, NWS.

[265] The BLSA's test item can be found in "Clinical Examinations of Outpatients for Longitudinal Studies," Box 22, Folder Longitudinal Study Administrative Record 1956-1962, NWS. Also see "Longitudinal Studies: THIRD SERIES Testing Schedule," Box 22, Folder Longitudinal Study Administrative Record 1956-1962, NWS.

[266] See http://www.grc.nia.nih.gov/branches/blsa/blsanew.htm

their welfare and healthy life in old age. Beginning with William W. Peter, a retired physician of the U. S. Public Health Service, an increasing number of lay people, from Baltimore and other towns of the country, voluntarily participated in the BLSA as research subjects.[267] These volunteers' long term loyalty to a study program on aging reveals that American gerontology ceased to be a small project limited to the member of the Club for Research on Ageing and other early organizations. American gerontology became a part of public efforts supported by various local communities in the United States.

Conclusion

Admittedly, however, gerontology has not always been seen favorably, even though the number of the BLSA volunteers kept increasing. Some people have doubted the disciplinary state of gerontology, which, for them, is not a scientific discipline but a service field for the welfare of the aged.[268] Moreover, some researchers have even wondered whether it might be better for them to underemphasize the project's relevance for aging in order to have a better chance in their funding applications.[269] In retrospect, this phenomenon may be regarded as a continuation of what the gerontologists in the CBPA experienced during their fundraising activities in the 1940s. Although Shock argued that "the attitude of resignation and futility as well as the belief that the senile changes in old age are inevitable must be actively combated," such negative attitudes toward both aging and aging research still lingered around the scientists of senescence

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[267] Peter played an important role in recruiting early volunteers by persuading local communities in Maryland of the importance of the BLSA. The correspondence concerning this effort of Peter can be found in Box 21, Folder Longitudinal Studies W. W. Peter correspondence 1957-1958 June, NWS.

[268] Hirschfield and Peterson, "The Professionalization of Gerontology," pp. 215-220; Bramwell, "Gerontology as a Discipline," pp. 201-205.

[269] As Martha Hostein and Patrick Fox have pointed out, aging was an undesirable topic for scientists even after the 1960s. Alzheimer's disease began to have a clear pathological identity that could be dealt with through medical sciences only after it began to be thought that its pathogenesis was not directly associated with aging. See Patrick Fox, "From Senility to Alzheimer's Disease: The Rise of the Alzheimer's Disease Movement," *The Milbank Quarterly* 67(1989), pp. 58-102; Martha Holstein, "Aging, Culture, and the Framing of Alzheimer Disease," in Peter J. Whitehouse, Konrad Maurer, and Jesse F. Ballenger (eds.), *Concepts of Alzheimer Disease: Biological, Clinical, and Cultural Perspectives* (Baltimore: Johns Hopkins University Press, 2000), pp. 158-180.

and their lay supporters.[270] Like the science of reproduction Adele Clarke has studied, gerontology might still be considered an "illegitimate science" in some sense.[271]

Yet gerontology in the United States has become a scientific field that has a substantial presence in academia in terms of the number of its practitioners, lay influence, and the amount of research funds, especially in comparison with the science of aging in Britain. Although the Gerontological Society is still smaller than other major scientific organizations, it has continued to increase its membership among both laymen and professional researchers. It has also maintained itself as a multidisciplinary scientific society, while British researchers of aging are divided into two major organizations which represent biomedical and psychosocial sides of gerontology, respectively.[272] This multidisciplinary approach to aging in America has been supported by a number of foundations and agencies, both private and public. Particularly significant is the National Institute on Aging, which has grown from a small laboratory in a public hospital to the largest gerontological research complex and funding agency in the world. In the United States, the science of senescence has emerged as a multidisciplinary scientific field, and American gerontologists' efforts to establish their field has born multiple fruits.

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[270] Shock, "Older People," p. 100.

[271] Adele E. Clarke, *Disciplining Reproduction: Modernity, American Life Sciences, and the "Problems of Sex"* (Berkeley, University of California Press, 1998), pp. 233-258.

[272] The British Society for Research on Aging (BSRA) is the organization for biological and medical scientists, and the British Society of Gerontology (BSG) is for social and psychological investigators.

Epilogue

The Place of Gerontology in Society

This dissertation has traced the origin and development of gerontology in the United States and the United Kingdom. I have shown how the growth of the biological and medical sciences provided new ideas, perspectives, and methodologies which were employed by the scholars who studied aging and tried to build gerontology as a multidisciplinary scientific field. I have also analyzed how the Great Depression and the elderly's social displacement helped lead scientists to organize gerontology, while their subsequent struggles and strategies to survive as a viable multidisciplinary field brought about different results in distinct social and academic environments in America and Britain. Unlike previous historical works arguing that gerontology highlighted the inevitability and pathological nature of senile decline, this dissertation has tried to show gerontologists' broader research interests and the more positive role they tried to play in society.

Yet a question still remains about the role of gerontologists in the postwar society. Have their efforts actually changed people's thinking about old age? Has old age become a less miserable period of life due to gerontologists' works? Has age discrimination been diminished through gerontologists' research data showing that aging does not necessarily mean decline in work capacities? Indeed, a few historians have already pointed out that one of the major aims of gerontologists was to highlight the possibilities and positive aspects of old age in the postwar society, and their work has been successful due to the changed social and political conditions after the War. For example, Richard Calhoun has argued that during the postwar period various experts and interest groups created a more positive image of old age and retirement in America. Gerontologists were certainly among these experts, and their efforts contributed to the substantial weakening of the

traditional stereotype of old age as the most decrepit period of life.[1] I think that Calhoun's argument reveals some truth on the change of Americans' attitude toward old age after the 1940s. It is true that affirmative action has weakened discrimination based on age as well as race or gender. Retirement has also begun to be viewed from a more positive angle through the extension of the Social Security benefits and other private pension plans that could be purchased by those who want more comfortable living after retirement. The expanding "gray market" is also redefining the elderly not as a decrepit individual nearing death but as an active consumer in capitalist society.

But there are other historians who view even this seemingly desirable aspect of gerontology from a critical perspective. For an instance, through his historical study of Alzheimer's disease in the United States, Jesse Ballenger has argued that gerontologists' attempts to promote a more positive view on old age by finding the causes of the elderly's problems including senile dementia from social rather than biological factors had ironic consequences.[2] Even though gerontologists convincingly argued that physiological and mental decline was due not so much to biological aging as to problems in social adjustment, the stigma attached to old age was intensified and the elderly increasingly became a subject of gerontologists' professional scrutiny. According to Ballenger, this occurred because gerontologists made the successful adjustment to old age—which seemed quite possible because senile decrepitude had social, not biological, origins that were more amenable to change—in terms of health and social activity the only business remaining for the elderly. Ballenger has argued that "as the meaning and purpose of old age was reduced to maintaining one's health and activity levels" according to gerontologists' professional advice, "disease and dependency grew even less tolerable, and the prospect of losing one's mind in old age became more frightening than ever."[3] Historian Thomas R. Cole has also pointed to a similar problem. Although Cole has argued that biological and medical researchers of the early twentieth century contributed

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[1] Richard B. Calhoun, *In Search of the New Old: Redefining Old Age in America, 1945-1970* (New York: Elsevier, 1978), pp. 69-72, 77.
[2] Jesse F. Ballenger, *Self, Senility, and Alzheimer's Disease in Modern America: A History* (Baltimore: Johns Hopkins University Press, 2006), pp. 56-75.
[3] Ballenger, *Self, Senility, and Alzheimer's Disease*, pp. 9-10.

to the negative stereotype of old age, he has admitted that gerontologists after the 1960s have tried to dispel ageism and to promote more positive image of old age. To become healthy and active, gerontologists argued, the elderly needed sustained self-maintenance and disciplined life based on gerontologists' scientific advice. However, Cole has argued, even this argument originated from the same modern view of old age that arose after the late Victorian period which emphasized self-maintenance and discipline as a desirable practice in industrial capitalism.[4] Whereas the discourse on senile decrepitude seems to be far removed from the emphasis on healthy and active old age, both are products of the modern ideology which defined human characters that did not correspond with capitalistic mode of production as undesirable. Even if a person tries thorough self-care according to the advice of gerontologists to arrive at a healthy and active old age, this effort, according to Cole, is still based on a deep fear of falling behind others due to senile decrepitude in the competitive and production-oriented modern society. Cole implies that old age has never obtained a purely positive connotation in modern America.

Although this dissertation does not deal very much with the period after the late 1950s and does not aim at providing an exhaustive study of gerotologists' influence upon society, I believe that Ballenger's and Cole's arguments suggest an important issue that demands further historical investigation. In fact, chapter two in this dissertation discusses how Clive McCay's research on caloric restriction and lifespan contributed to popular discourses on health and longevity whose origin can be traced back to the ideal of "civilized old age" described by Cole. Chapter six has also cited a number of lay people's response to gerontological research which, for them, seemed to be opening the way for a long and active life based on a scientific understanding of old age. Certainly, both gerontologists and their lay audience were searching for effective ways to adapt the aging population to modern society. It is possible that these ways, ironically, contributed to further stigmatization of old age.

But I think that Ballenger's and Cole's arguments can lead to a skewed view of the character of gerontology. Science is a complex entity whose place and role in society

[4] Thomas R. Cole, *The Journey of Life: A Cultural History of Aging in America* (Cambridge: Cambridge University Press, 1992), pp. 227-233.

can hardly be characterized in a simple way. Gerontology is particularly complex, because it is a multidisciplinary field including many different research activities and disciplinary norms. In particular, the biological and biomedical researchers who established the field were influenced by the new ideas and approaches developed within their disciplines as well as by their worries about the elderly's social adaptation. For them, the concerns about their academic career and the proper solution of current research problems were at least as important as the condition of the society in which they lived and aged. In fact, this dissertation has shown that gerontology itself was constructed as a scientific field by the coalition of various factors—such as the growth of the elderly population, the plight of the aged during the Depression, and the development of new concepts and approaches through the growth of the biological and biomedical sciences. Early gerontologists were influenced by these multiple factors in relation to which they hoped to conceptualize aging as a complex phenomenon worthy of multidisciplinary scientific investigation. For these scholars, hence, the problem of old age in society was only one—albeit an important one, indeed—among their multilayered concerns. Even if it is true that these gerontologists have contributed to furthering the negative view on old age, I think that the alleged social function attributed to gerontology should not overshadow our historical understanding of gerontology as a broad scientific field.

While describing the creation of this field, this dissertation has tried to challenge some historians' deep-seated distrust of the role of science in society. I have argued that gerontologists during the first half of the twentieth century struggled to show that decline in physiological and mental functions did not necessarily accompany aging. Rather, they studied senescence as a contingent phenomenon at the cell level and attempted to promote more possibilities than limitation of old age through their scientific work. Admittedly, even these efforts of gerontologists might have stayed within the stereotype of old age and furthered the stigma attached to senescence. As historians Robert Kohler and Hans-Jörg Rheinberger have pointed out, however, science often develops in a completely unexpected direction and does not necessarily reflect the state and political

ideology of the larger society in which scientists work.[5] If that is true, we may not always expect a neat correspondence between the general public's view of old age and the nature of senescence professional gerontologists have studied. Gerontologists have produced new knowledge which is not always related to the contemporary political controversies on ageism and retirement. The products of their investigations have often complicated and confused, rather than strengthened, the received views on senescence and the elderly.

The considerable expansion of the science of aging since the late 1950s reveals how diversified these investigations in the multidisciplinary field have become. Most importantly, the National Institute on Aging (NIA) was established in 1974, becoming the largest research organization and funding agency in gerontology in the world. Among the recipients of NIA's research funds, the scientists studying caloric restriction and longevity have recently turned toward the molecular and cellular mechanisms involved in the effect of reduced energy intake, while evolutionary geneticists have studied the genes controlling the rate of senescence in relation to evolutionary pressures. Simultaneously, several new theories of aging have been proposed, such as telomere reduction theory, oxidative damage theory, and the theories based on apoptosis or programmed cell death. In the medical fields, anti-aging treatments, such as botulinum toxin and hormone therapies, have also grown into a big industry targeting middle-aged or elderly people who want to look younger and become healthier.

The increasing number of professional or semi-popular journals also testifies to the substantial growth of gerontology. While the *Journal of Gerontology* was the only periodical for the scientists of aging in 1946, many new journals have been launched

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[5] In his book, *Lords of the Fly*, Kohler has argued the practical imperatives and significance of production in scientific research. According to him, the fly *happened* to be chosen and promoted as a legitimate experimental organism for genetic research because of its usefulness and productivity within a particular laboratory environment. See Robert E. Kohler, *Lords of the Fly: Drosophila Genetics and the Experimental Life* (Chicago: University of Chicago Press, 1994), pp. 1-15, 19-52. Rheinberger has argued that the production of unexpected results and new ways of representing research subjects was the character of productive experimental systems. See Hans-Jörg Rheinberger, *Toward a History of Epistemic Things: Synthesizing Proteins in the Test Tube* (Stanford, California: Stanford University Press, 1997). A similar historical perspective is found in a more recent monograph on the history of using mice as an experimental organism. See Karen A. Rader, *Making Mice: Standardizing Animals for American Biomedical Research, 1900-1955* (Princeton: Princeton University Press, 2004).

thereafter by the Gerontological Society, the British Society for Research on Aging, and other organizations—including *The Gerontologist*, *Gerontology*, *Aging Cell*, *Age and Ageing*, *Experimental Gerontology*, *Mechanisms of Ageing and Development*, *Biogerontology*, *Rejuvenation Research*, and *Gerontology & Geriatrics Education*. The *Journal of Gerontology* itself became the *Journals of Gerontology* in 1995 and was divided into "Series A," dealing with the biological and medical sciences, and "Series B," publishing articles in psychology and the social sciences. Social gerontologists have also lauched a number of new periodicals specialzing in their study area, including *Ageing and Society*, *Journal of Gerontological Social Work*, *Research on Aging*, and *Journal of Aging Studies*. Several journals devoted to geriatrics rather than gerontology also include basic research on aging, such as the *Journal of the American Geriatrics Society*, *Geriatrics*, *Annals of Long-Term Care*, *Clinical Geriatrics*, *Journal of Geriatric Psychiatry and Neurology*, *Geriatric Nursing*, and *Journal of Gerontological Nursing.* Since there are many other journals devoted to the multiple dimensions of aging, including those written in languages other than English, gerontology has truly become a large and multifaceted academic arena, in terms of the number of articles and the diversity of the topics dealt with in them.

Gerontology has been successfully established as a scientific field, while the cultural and social meaning of old age still remains as a controversial and problematic issue. In fact, both the absolute number and the relative proportion of the elderly in the population have increased even in comparison with the 1930s and 1940s when gerontology was born.[6] But some contemporary elderly, especially affluent seniors, do not share the concerns about old age with Cowdry who expressed his deep worries when the field was created. For them, old age has quite a different meaning, because they hardly doubt the prospect of their peaceful retirement with adequate pensions and proper healthcare. More active elderly people do not expect that aging will substantially alter

[6] According to the U.S. Census Bureau, as of 2006, there are 35,505,000 people over 65 in the United States, which is 12.1% of the total population. In 1940, there were 9,019,000 people over 65, and this population constituted 6.9% within the whole. The data can be downloaded from the Bureau's website. For the data of 1940, visit http://www2.census.gov/prod2/decennial/documents/33973538v4p1ch1.pdf
For the date of 2006, visit http://www.census.gov/population/www/socdemo/age/age_2006.html

their life, since they are willing and able to continue their social and vocational life after old age. Yet many people, particularly those who are less affluent and privileged, are still worrying about mandatory retirement, age discrimination, and the severe financial difficulties which concerned Cowdry and his colleagues during the Depression. This concern has become more pressing after the financial crisis of 2008. Moreover, a large number of the elderly eventually suffer from chronic illness and declining mental and physical capacities before they die, even though they have maintained a healthy and active lifestyle for a long time.

For gerontologists, these diverse aspects of aging can be their research subjects or at least influence the direction of their works, just as most early gerontologists were influenced by the social conditions surrounding the elderly during the 1930s and 1940s. But many scientists of aging, especially those in the biological or medical fields, concentrate on the phenomenon of senescence observable through the cell, the genes, and the intracellular organelles rather than on the topics connected to humans' old age in society. Of course, as a number of historians of science have pointed out, even these biological entities may have political dimensions. Yet it is also true that the political problems related to conducting gerontological research might not always be about retirement or social adpatation. Gerontologists can also worry about their career, research grant, and study projects. These multilayered concerns of the scientists of aging reveal the current state of gerontology as an established research field situated within the complex world of funding, administration, and biomolecules as well as the elderly's social adaptation and retirement.

Appreciating this contemporary state of gerontology, we may look back upon Vladimir Korenchevsky's final words to Edward Mellanby in 1944 with a different feeling. As I have written in chapter four, Korenchevsky, after failing to persuade the Medical Research Council of the need to support gerontology, retired from his post at the Lister Institute with a deep regret. To Mellanby, he wrote that "the future will show whether I was right or wrong."[7] He was right.

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[7] "Extract from Prof. V. Korenchevsky's letter to Sir Edward Mellanby," 26 June 1944, FD 1/675 (Korenchevsky's Research, 1942-1953), NAUK.

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