Agricultural Extension Service

43rd Minnesota Nutrition Conference Sept.20—21, 1982

Thunderbird Motel
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Bloomington MN 55420

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WITH APPRECIATION

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We are also grateful to Blomfield-Swanson, Inc., and Quali-Tech Products for special assistance in obtaining many of the items.

PROGRAM

Monday, September 20, 1982

a.m. **John Vignes, presiding**

8:30 Registration and Refreshments

9:45 Microbial Protein Synthesis in the Rumen — *Marshall Stern*

10:15 Associative Effects of Feedstuffs — *Fred Ehle*

10:45 Break

11:05 Methionine Hydroxy Analog for Lactating Dairy Cattle — *Rick Lundquist*

11:35 Panel Discussion

12:00 Luncheon — Topic: "Cloning of Cattle" — *Alan G. Hunter*

p.m. **John Foley, presiding**

1:30 Nutrition of Cold Water Fishes — *Gary Rumsey*
Symposium: Vitamin Nutrition of Ruminants

2:00 Vitamin A and Beta-Carotene — *Ted Frye*

2:30 B Vitamins — *E. E. Bartley*

3:00 Break

3:20 Vitamin D — *William G. Olson*

3:50 Vitamin E — *Ted Frye*

4:20 Panel Discussion

4:45 Adjourn

6:00 Social Hour

7:00 Dinner

Purchase tickets from Northwest Feed Manufacturers Association.

Tuesday, September 21, 1982

a.m. **Paul E. Waibel, presiding**

8:30 Egg Shell Quality — *David Roland*

9:10 Nutrition of Capons — *Norman G. Magruder*

9:40 Influence of Temperature on the Lysine Requirement of Turkeys — *Sally L. Noll*

10:10 Break

10:30 Soybeans and Soybean Meal — *Kenneth Lepley*

11:00 Sulfur Amino Acid Nutrition of Poultry: An Update — *David H. Baker*

11:30 Panel Discussion

12:00 Luncheon Topic: The Future of Animal Agriculture — *Ed Schuh*

p.m. **James E. Pettigrew, Jr., presiding**

1:30 Significance of Amino Acid Imbalance in Swine Nutrition — *David H. Baker*

2:00 Antibacterials and Swine Management Interactions — *Ronny L. Moser*

2:30 Break

2:45 Applications of a Computer Model of Swine Growth — *Steven G. Cornelius*

3:15 Panel Discussion

3:45 Adjourn

David H. Baker is a professor of comparative nutrition at the University of Illinois. After receiving his Ph.D. degree at the University of Illinois in 1965, he spent two years as a senior scientist at Eli Lilly and Company, after which he returned to Illinois to become a faculty member in the Department of Animal Science. Baker has received the ASAS Young Scientist Research Award (1971), the AFMA Nutrition Research Award (1973), The Merck Award in Poultry Research (1977), the Paul A. Funk Award (1977) for meritorious contributions to agricultural science and the H.H. Mitchell Award (1979) for excellence in graduate teaching and research. He has also served on the editorial boards of the Journal of Animal Science, Poultry Science, The Journal of Nutrition, and Nutrition Reviews.

Erle Bartley, is professor of the Department of Animal Sciences at Kansas State University. He received his Ph.D. degree in Dairy Cattle Nutrition at Iowa State University. Dr. Bartley has thirty seven years of research experience. The awards he received include Morrison Award (1981), National Award for Agricultural Excellence (1981), American Feed Manufacturers Award (1957) and Borden Award (1975). He is also a fellow of the American Society of Animal Science, 1978. His area of interest is rumen metabolism. Dr. Bartley has published approximately 100 journal articles on action of antibiotics; causes of grain and legume bloat; microbial protein synthesis in the rumen; ammonia toxicity; role of rumen bacterial endotoxin and their relation to the sudden death syndrome in cattle; effect of niacin and polyether antibiotics on rumen metabolism, etc.

Steven G. Cornelious joined the Department of Animal Science of the University of Minnesota in July 1978 from the U.S. Meat Animal Research Center at Clay, Nebraska. He received his B.S. (animal science) and Ph.D. (nonruminant nutrition) degrees from the University of Illinois. His dissertation was "Investigations concerning the Absorption, Distribution, and Metabolism of Iron in Neonatal Piglets." His general research interests include: factors influencing composition and the utilization of nutrients for various physiological functions including fat and lean tissue synthesis, maintenance, pregnancy and lactation, regulation of food intake, nutrition of the fetus and neonate, and nutrition of the male.

Fred Ehle is a USDA research scientist associated with the U.S. Dairy Forage Research Center. Ehle holds an adjunct appointment as assistant professor in the Department of Animal Science at the University of Minnesota. A native of New York, he received his graduate degrees from Cornell University. Ehle was a research associate in the Department of Dairy Science at the University of Illinois prior to joining the faculty of the University of Minnesota. His applied and basic research efforts have emphasized the interaction of forage characteristics and ruminant nutrition.

Ted Frye is a technical service nutritionist in the Department of Agriculture and Animal Health, Roche Chemical Division, Hoffmann-La Roche. A native of Virginia, he earned his B.S. degree in Agricultural Education at Virginia State University in 1966, and the M.S. and Ph.D. degrees in Ruminant Nutrition from Virginia Polytechnic Institute and State University in 1976. His area of responsibility is the provision of product data and technical support for vitamin products to the livestock industry.

Alan G. Hunter, is a Professor of animal science at the University of Minnesota. He joined the staff in 1963. A native of Pawtucket, Rhode Island, he received his B.S. degree in 1955 and M.S. degree in 1958 from the University of Rhode Island. He earned his Ph.D. degree in 1963 at Michigan State University. Hunter was a research assistant at the University of Rhode Island from 1955-1958 and an instructor at Michigan State University from 1958-1963. His specialization is reproductive physiology and biochemistry and his is the author of 80 articles on reproduction. He has taught courses on the literature of animal science, endocrinology, lactation reproduction, fertilization, gestation and immunoreproduction. He currently heads a research team that is attempting to clone dairy cattle at the University of Minnesota.

Ken Lepley, is the Technical Director in Animal Nutrition at the American Soybean Association. He is a native of South Dakota and lived in Iowa for many years. He received his B.S. degree in Animal Husbandry and M.S. degree in Animal Nutrition at Iowa State University. Lepley was Director of Research and Nutrition for Farmers Regional Cooperative and Director of Nutrition for Land O'Lakes at Fort Dodge, Iowa. In 1974 he joined the American Soybean Association and moved to Vienna, Austria to help open a new office and start market development activities in the seven East European countries. Upon his return to the U.S., he was assigned to his present position and presently is responsible for supplying animal nutrition support to ASA's ten overseas offices.

Rick Lundquist is an assistant scientist in the Department of Animal Science at the University of Minnesota. Born and raised in northern Minnesota, he earned a B.S. degree in animal science from Minnesota in 1977 and is currently completing the requirements for a Ph.D. degree in dairy cattle nutrition. Lundquist is the supervisor of the dairy nutrition laboratories and he teaches courses in ruminant nutrition and feedstuffs analyses. His research interests include protein nutrition in dairy cows, methionine hydroxy analog, and nutrition of the calf.

Norman D. Magruder, is Poultry Research and Marketing Director, Cargill, Inc. He received his B.S. (1949) and M.S. (1951) degrees from West Virginia University and his Ph.D. (1953) degree from Pennsylvania State University. He was assistant professor in the Animal Nutrition Department at Pennsylvania State University for 4 years and worked in Animal and Poultry Research for Chas. Pfizer and Company for 3 years. Since 1960 he has been associated with Nutrena and Cargill. Major areas of poultry research include environmental nutrition studies with layers, cage density and management of layers, nutritional studies involving amino acid levels, calcium and phosphorus ratios, energy levels and mycotoxins. He is a member of many scientific associations including Poultry Science, World Poultry Science and The New York Academy.

Ronny Moser is a native of Oklahoma where he received his B.S. degree in animal science from Oklahoma State University in 1976. He earned a M.S. in animal science and Ph.D. in swine nutrition at the University of Nebraska. In 1980 Moser was appointed assistant professor in the Department of Animal Science at the University of Minnesota where he teaches courses in swine production, applied swine nutrition, and swine practicums. His research interests are in the effect of diet fed during lactation and post-weaning on the productive performance of sows and in the diet and management interaction in swine.

Sally Noll, a native of Wisconsin, is an Assistant Scientist at the University of Minnesota working in poultry nutrition. She received her B.S. and M.S. degrees from the University of Minnesota and is currently seeking the Ph.D. degree. Her thesis research has emphasized lysine availability and nutritional requirement studies involving turkeys. Prior to the scientist position she was a Research Assistant at the University and assisted in teaching a poultry nutrition course.

William Olson, is an Associate Professor specializing in nutrition in the department of Large Animal Clinical Sciences. Prior to this he was the manager of a private clinical research farm for four years. He earned the D.V.M. degree in 1966 at the University of Minnesota, the M.S. degree in Veterinary Science in 1969 at the University of Wisconsin, and a Ph.D. degree in Dairy Science at the University of Wisconsin in 1972. His teaching responsibilities include metabolic diseases and nutrition. Research interests include vitamin and mineral metabolism and metabolic profile testing. His clinical teaching is accomplished by reviewing with students nutrition and management assessments of metabolic disease and production problems on dairy farms.

David A. Roland, Sr., is an Alumni Professor at the Auburn University. He graduated from the University of Georgia in 1970 with a Ph.D. in Nutritional Biochemistry; he served on the staff of the Poultry Science Department at the University of Florida for 6 years. Dr. Roland's primary interests have been involved with each of the various types of shell quality problems and other related nutrition and management problems. During the past 10 years he has authored or co-authored over 200 publications and presented over 100 presentations of his research throughout the United States and several foreign countries. He is a member of numerous professional and honor societies. He has also received several awards for excellence in research, including the Poultry Science Association Research Award, American Egg Board Egg Science Award, and the Alumni Foundation Distinguished Professor Award. He has served as a technical consultant to firms or individuals in 24 states and 9 countries.

Gary Rumsey, is Director of the Tunison Laboratory with research facilities in New York and Idaho. The Tunison Laboratory, which has been involved in fish nutrition research for 50 years, operates under the auspices of the Fish and Wildlife Service (USDI) and a cooperative agreement with Cornell. He earned his B.S. and M.S. degrees in biochemistry from Penn State University and a Ph.D. in nutrition from Cornell. He is an adjunct Professor in Cornell's Department of Poultry and Avian Science and graduate Field of Nutrition. Prior to this, Rumsey spent 11 years in various positions with the U.S. feed industry. He served for 6 years on the National Research Council's Committee on Animal Nutrition and chaired the Subcommittee on Fish Nutrition which authored the recent publication on Nutritional Requirements of Coldwater Fish. He presently serves on the advisory board of the Aquavet Program of the respective Veterinary Colleges of Cornell and Pennsylvania. His research interests stem primarily around nutritional energetics and nitrogen metabolism in ectothermic animals.

G. Edward Schuh, is Professor and Head of the Department of Agricultural and Applied Economics at the University of Minnesota. Prior to assuming this position July 1, 1979, he served as Deputy Under Secretary for International Affairs and Commodity Programs at the U.S. Department of Agriculture since February 1978. Dr. Schuh occupied previous academic positions in Agricultural Economics at Purdue University from 1959-1978. He is a native of Indiana and holds a B.S. degree from Purdue, an M.S. degree from Michigan State, and an M.A. and Ph.D. degrees from the University of Chicago. He served as Senior Staff Economist on President Ford's Council of Economic Advisors during 1974-75, was Program Advisor to the Ford Foundation in Brazil from 1966-72, and Director of the Center for Public Policy and Public Administration at Purdue University from 1977-78. He is Director of the National Bureau of Economic Research and of the Economics Institute at the University of Colorado, and was Director of the American Agricultural Economics Association from 1977-80. As of July 1980, he is President-Elect of that Association. Dr. Schuh has extensive International Teaching, Consulting, and Advising experience in Latin America and India, and with the U.S. government. His particular areas of subject matter interest include Agricultural and Food Policy, Economic Development, and International Trade. He is the author or co-author of three books and has over 80 technical and scientific papers to his credit.

Marshall Stern is an assistant professor of animal science at the University of Minnesota. A native of New York, he received the B.S. degree from Cornell University, the M.S. degree from the University of Rhode Island, and the Ph.D. in animal nutrition from the University of Maine in 1977. His dissertation was entitled "Microbial Protein Synthesis in Continuous Culture of Rumen Contents as Affected by Levels of Urea, Nonstructural Carbohydrate and Soluble Protein." Following post doctorate study at the University of Wisconsin, he joined the faculty of the University of Minnesota in 1981. His research interests include protein and carbohydrate metabolism in the digestive tract of ruminants, dietary effects upon fermentation and microbial populations in the rumen, the role of protozoa in ruminants, the development of methods for measuring ruminal protein degradation, and studying digestive physiology in the gastrointestinal tract.

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MICROBIAL PROTEIN SYNTHESIS IN THE RUMEN

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INTRODUCTION

The rumen is a site where digestion occurs through the action of microbes that live in symbiotic association with the animal. Ingested dietary protein is extensively degraded in the rumen to amino acids and deaminated to ammonia, and both are used as a source of nitrogenous nutrients for the synthesis of rumen bacterial and protozoal protein (figure 1). Microbial protein synthesis in the rumen requires specific nutrients such as sulfur, branched-chain fatty acids and trace nutrients, however under most dietary circumstances these substances are not limiting. Nutrient supply to the microbes is therefore considered largely in terms of the ruminal availability of nitrogen and of digestible organic matter (mainly carbohydrate) that can be fermented in the rumen to provide a carbon skeleton and energy in the form of adenosine triphosphate for microbial protein synthesis.

CONTRIBUTION OF MICROBIAL PROTEIN TO THE HOST

Under most feeding practices, microbial protein comprises a substantial part of the protein entering the small intestine, where enzymatic digestion releases amino acids that are absorbed to furnish the animals needs. In general the amino acid composition of duodenal digesta usually reflects that of microbial protein except on diets where significant amounts of dietary protein have avoided degradation. The true digestibility of microbial amino acid-nitrogen was recently determined by Storm and Ørskov (1982) to be 84.7% in lambs, while the efficiency of utilization of absorbed AA-N was 80.1%. Since rumen microbes are an important source of high quality protein for the ruminant and since microbial growth rates can affect amino acid availability to the animal, it is important to maximize microbial protein synthesis in the rumen.

Table 1 shows the theoretical contribution of microbial protein at various levels of efficiency to the total protein requirement of a 650 kg lactating dairy cow producing 25, 35 and 45 kg milk daily with 3.5% milkfat. At these three levels of milk yield, microbial protein would contribute 42 to 56% of the total protein required by the animal when microbial synthesis in the rumen is 15 g CP/100 g organic matter apparently digested (OMD). Stern and Hoover (1979) reviewed the literature and found that about 16.9 g CP are synthesized per 100 g OMD in the rumen, with values ranging from 6.3 to 30.7 g (n=64). Therefore, values for microbial protein synthesis used in table 1 are realistic and the calculated contribution of microbial protein clearly depicts the importance of optimizing microbial growth. For example, when milk yield is 45 kg/d, contribution of microbial protein to total protein required by the cow would increase from 42-71% with an increase in microbial protein synthesis from 15 to 25 g CP/100 g OMD. In addition, figure 2 illustrates the effect that different amounts of metabolizable energy (ME) and subsequent increase in efficiency of microbial growth, would have on the contribution of microbial protein to milk yield in ewes (Ørskov and Robinson, 1981).

MEASUREMENT OF MICROBIAL PROTEIN SYNTHESIS

Various methods have been used to estimate the quantity of microbial protein synthesis in vitro and in vivo. The majority of these techniques are based on determination of a single chemical marker believed to characterize the microbial components. Rode (1981) stated that any component of the microbial cell can be used as a suitable marker as long as it meets the following criteria: (1) it must be absent or readily distinguishable from any dietary or endogenous constituents leaving the reticulo-rumen and (2) it must exist in a constant ratio with microbial N under specific experimental conditions. Diaminopimelic acid (DAP), aminoethylphosphonic acid (AEP), ribonucleic acid (RNA), D-alanine, ATP and isotopes (^{35}S, ^{15}N, ^{32}P) incorporated into protein in the rumen have been used as microbial markers. Differences in the amino acid profiles of individual components reaching the duodenum also have been used. Advantages and disadvantages of these various methods will be discussed.

Diaminopimelic acid. DAP is an amino acid which is unique to the murein layer of the prokaryote cell wall (Brock, 1974). Traces of DAP can also be detected in protozoa, as bacteria in vesicles of entodiniomorphs and holotrichs have been observed by electron microscopy (Coleman and Hall, 1969; Stern et al., 1977a,b).

The DAP method involves estimating the ratio of DAP:N in mixed rumen bacteria and the amount of DAP in digesta. From these values the amount of bacterial nitrogen in digesta can be calculated (Hogan and Weston, 1970). A technique for the separation and colorimetric estimation of DAP using an automatic amino acid analyzer was described by Hutton et al. (1971). Ibrahim and Ingalls (1972) used DAP to measure bacterial protein synthesis and AEP, which is found in the lipid traction of protozoa, to estimate protozoal protein synthesis. Czerkawski (1974) described methods for DAP and AEP determination that are relatively fast compared to other methods and require less elaborate equipment. The theory is that DAP and AEP can be used together to estimate total microbial protein synthesis in the rumen, whereas DAP alone measures only bacterial synthesis. The accuracy of the DAP method is dependent on a constant DAP:N ratio among various microbial species, or the maintenance of a constant ratio of microbial species in the rumen. The latter assumption is not consistent with the sequential nature of rumen fermentation and Work and Dewey (1953) have shown the DAP:N ratio to vary among species. The degree of error introduced by these considerations must be weighed against the simplicity of the DAP determinations.

Ribonucleic acid. Smith and McAllan (1970) used the ratio of RNA to total nitrogen in rumen fluid and rumen microbes to estimate the extent of conversion of dietary nitrogen to bacterial and protozoal nitrogen. However, the analytical procedure (McAllan and Smith, 1969) used to determine RNA is tedious. This technique relies on the assumption that nearly all dietary RNA is degraded in the rumen (McAllan and Smith, 1973). Buttery and Cole (1977) expressed doubt as to whether this is entirely true and suggest that microbial protein flow may be overestimated and that this problem may be amplified when large portions of the dietary protein and nucleic acid have been rendered insoluble by exposure to heat or chemical treatment. Evidence by Smith et al. (1978) indicates that the RNA method does slightly overestimate the microbial contribution at the duodenum. Another problem with the RNA method is the variability of the RNA:total N ratio of mixed bacteria due to diet and environment (Smith and McAllan, 1974). A major advantage of RNA is that protozoa are included in the estimate of microbial protein in digesta.

Amino acid profiles. A method for estimating quantities of microbial and dietary proteins in duodenal digesta based upon differences in the amino acid content of the protein reaching the duodenum was reported by Evans et al. (1975). Individual proteins passing to the duodenum were identified by their characteristic amino acid profiles. The method depends upon the computer generation of several profiles which represent mixtures of different proportions of the known profiles of the dietary and endogenous components that may be arriving at the duodenum. The method assumes constant composition for microbial protein and that protein in each dietary component behaves as a single entity. This method is presently limited by a lack of knowledge relative to the differential degradation rates of different proteins present in conventional diets.

Adenosine triphosphate. Forsberg and Lam (1977) studied the use of ATP as a rumen microbial marker because of the following assumptions: (1) ATP is present in all living cells and absent from dead cells; (2) ATP concentration is similar in all microbes; and (3) extraction and assay of ATP is relatively simple to perform and inexpensive. However, their experiments indicated that there are possible variations in the efficiency of extraction of ATP from rumen contents and they also observed differences in the concentration of ATP in rumen microbes. In contrast, Wolstrup and Jensen (1978) used similar methods to extract ATP from rumen contents and noted that variability was not as great as that found by Forsberg and Lam (1977). Wolstrup and Jensen (1978) indicated that choice of equipment was probably the source of variation. However, even with a suitable technique for analysis of ATP, the labile nature of ATP, especially under acidic conditions (Merck, 1978) similar to that found in the abomasum, make it an unlikely choice for a marker.

^{35}Sulfur. Of the radioisotopes used as tracers to distinguish between microbial and dietary protein, ^{35}S has been used most frequently. Use of ^{35}S as a microbial marker was first suggested by Henderickx (1961). Walker and Nader (1968) used radioactive sodium sulfide ($Na_2{}^{35}S$) to label the sulfide pool of rumen contents in vitro. The basic premise of this method was that all of the sulfur incorporated into microbial protein was first passed through the free H_2S pool. While in vitro studies of Nader and Walker (1970) suggested the error introduced by sulfur incorporation from amino acids was neglible, in vivo studies revealed that considerable amounts of sulfur containing amino acids in microbial protein may arise from sulfur which has not passed through the H_2S pool (Gawthorne and Nader, 1976; McMeniman et al., 1976; Salter et al., 1979).

Walker and Nader (1975) subsequently described a method for the in vivo measurement of rumen microbial protein synthesis which depends upon the incorporation into microbial protein of sulfur derived from ^{35}S-labeled inorganic sulfate infused continuously into the rumen. This method has the advantage of not relying on use of the highly labile sulfide and does not require that virtually all microbial sulfur be derived from hydrogen sulfide. Kennedy et al. (1980) introduced a method using $Na_2{}^{35}SO_4$ infusion based on their observation that sulfide which was not incorporated into microbial protein entered the plasma sulfate pool. This technique would allow estimation of microbial protein synthesis without the need for intestinally cannulated animals.

The predominant method of using ^{35}S as a microbial marker has been to measure differences in the ratio of specific activities of ^{35}S in either the cysteine

(Leibholz, 1972), methionine (Beever et al., 1974) or total sulfur amino acids (Hume, 1974; Mathers and Miller, 1980) of duodenal digesta and a separated microbial fraction. A possible limitation of the ^{35}S method is the error introduced due to the direct incorporation of dietary sulfur amino acids into the microbial fraction. Also, a similar ratio of sulfur amino acids to total protein for microbial and dietary material is assumed. In addition, the method creates problems in handling large quantities of radioactive waste, especially with cattle and is relatively expensive and laborious for routine screening of diets. Although the ^{35}S technique is laborious and may not be completely accurate, it does have definite advantages. Quantitative recovery of the sulfur amino acids is not essential, as the technique is based on a ratio of specific activities. Also, the technique is capable of determining total microbial protein synthesis rather than just bacterial protein synthesis as with the DAP technique.

^{15}Nitrogen. Microbial protein synthesis has also been estimated by quantitating ^{15}N incorporation into microbes from either $(^{15}NH_4)_2SO_4$ (Pilgrim et al., 1970) or $^{15}NH_4Cl$ (Mathison and Milligan, 1971). These methods are based on the incorporation of nitrogen from ammonia and do not account for microbial protein synthesized directly from amino acids or peptides. Besides being costly, the technique utilizing ^{15}N as a marker is quite complicated and as a result has not been extensively used. However, because it deals directly with nitrogen it has proven to be a useful tool in studying the dynamics of nitrogen metabolism in the ruminant (Nolan et al., 1976).

Phosphorus. Bucholtz and Bergen (1973) observed that phosphorus uptake and incorporation into microbial phospholipids was highly related (r=.98) to ruminal protein synthesis and proposed an in vitro method for estimating microbial protein production based on incorporation of ^{33}P into microbial phospholipids. Van Nevel and Demeyer (1977) expanded this approach to include incorporation of ^{32}P-labeled extracellular phosphate in total microbial P as the measure of microbial growth. However, it should be pointed out that such a procedure is of limited value, as it assumes a constant value for N:P in microbial DM, as well as a constant value for net growth:total growth. Smith et al. (1978) proposed a technique based on the incorporation of ^{32}P-labeled inorganic phosphate into rumen bacterial nucleic acids.

D-alanine. Unlike DAP, D-alanine occurs in the peptydoglycan layer of most bacteria. Because it occurs in relatively constant amounts, Garrett et al. (1982) have developed an enzymatic procedure for the analysis of D-alanine. Because of limited data the use of D-alanine cannot be critically evaluated, however work is currently in progress to determine whether D-alanine is a suitable bacterial marker.

Comparative measurements between microbial markers. Comparisons in vitro for ^{15}N, ^{32}P and ^{35}S (Harmeyer et al., 1976) did not show good agreement between the various methods. Smith et al. (1978) compared nucleic acid ^{32}P:NAN ratios in related samples of rumen bacteria and duodenal contents with similar estimates using DAP and RNA as bacterial markers. Their estimates based upon ^{32}P-labeled RNA nucleotides were approximately 85% of those based upon total RNA. They attributed this difference mainly to the latter being elevated by the presence of small amounts of dietary RNA. Estimates of microbial nitrogen using the DAP method were found to be affected by rumen protozoal concentrations, because the DAP method does not account for protozoal nitrogen.

Ling and Buttery (1978) assessed the use of RNA, ^{35}S, DAP and AEP as markers of microbial nitrogen in duodenal digesta. They found AEP to be present in substantial quantities in dietary and bacterial material as well as in isolated rumen protozoa and suggested that the use of AEP as a protozoal nitrogen marker was invalid. In contrast, Dufva et al. (1982) examined bacteria and a wide variety of feedstuffs and found them to be devoid of AEP. Therefore at present the value of AEP as a protozoal marker is uncertain. Ling and Buttery (1978) also indicated that one of the major problems with DAP as an index of total microbial nitrogen was it did not account for protozoal contribution to duodenal digesta nitrogen but concluded that it would probably continue to be widely used. Where total microbial values are required, the choice of method becomes either RNA or ^{35}S. They concluded that where microbial nitrogen estimates of a more general and comparative nature are required, the use of RNA would probably be adequate, however, where more accurate estimates are required, ^{35}S would be more appropriate.

It should be emphasized that all methods, where mixed microbes are separated from rumen fluid by differential centrifugation, assume that the microbes are representative of the population entering the duodenum or abomasun. Serious doubts must be cast upon this assumption as some organisms are found free in the liquid phase of the rumen digesta, while others adhere strongly to feed particles and some are closely associated with the rumen wall. Therefore the techniques used to isolate rumen bacteria will only sample material from the liquid phase which form only a small part of the total rumen bacteria. These bacteria that are found in the liquid phase may be less metabolically active than the greater numbers of bacteria that are associated with food particles (Smith, 1975). In addition to the problems associated with microbial markers and fractionation of rumen contents into microbial fractions, Theurer (1979) determined that digesta flow markers have a more profound effect on calculation of microbial protein synthesis than microbial markers. It is concluded that for a better understanding of the principles of microbial growth and efficiency of microbial protein synthesis in the rumen, measuring techniques and analytical procedures applied in research in this field need to be improved.

FACTORS AFFECTING MICROBIAL PROTEIN SYNTHESIS

In recent years, numerous studies have been conducted to determine microbial protein synthesis in the rumen under varied conditions. As a result of these studies several factors have been found to influence microbial protein synthesis including rumen dilution rate, concentration and source of nitrogen and carbohydrate, dietary sulfur and frequency of feeding.

Dilution rate. Several in vitro studies have demonstrated a positive correlation between increased dilution rate and increased microbial growth (Hobson, 1965; Hobson and Summers, 1967; Stouthamer and Bettenhaussen, 1973; Isaacson et al., 1975). These in vitro studies have indicated that the highest yields of cells are obtained when the mean time spent in the fermentation vessel is such that the bacteria pass onwards as soon as their development is complete. Otherwise, bacteria use substrate for maintenance or for storage as carbohydrate or lipid and the efficiency of production of bacterial protein falls.

Several in vivo studies (table 2) have also noted increases in microbial protein synthesis correlated with rumen dilution rate (defined as the proportion of total rumen volume leaving the rumen per hour). Several factors such as diet, intraruminal buffer infusion, level of intake and environmental conditions

have been found to alter rumen dilution rate. Cole et al. (1976) observed increases in rumen dilution rate (.03 to .05/hr) along with increases in protein synthesis (7.5 to 11.8 g/100 g DMD) when steers were switched from an all concentrate diet to one containing only 14% roughage. Allen and Harrison (1979) revealed that feeding monensin to sheep decreased rumen dilution rate from .07 to .04/hr, with a concomitant decrease in microbial synthesis from 15.3 to 12.6 g CP/100 g OMD. Intraruminal infusion of artificial saliva in sheep increased dilution rate from .03 to .08/hr (Harrison et al., 1976). Concurrently, there was an increase of total amino acids synthesized per mole hexose fermented from 25.4 to 29.8 grams. Kennedy et al. (1976) found that sheep maintained at an environmental temperature of 18 to 21 C were less efficient in synthesizing microbial protein than were those maintained at -1 to 1 C (29.9 vs 33.7 g CP/100 g OMD, respectively). The increased efficiency of microbial production in cold-exposed sheep was positively correlated with dilution rate (.1 vs .14/hr, respectively). In a subsequent study, Kennedy and Milligan (1978) found that when sheep were maintained at 22 to 25 C and 2 to 5 C, there was an increase in dilution rate from .07 to .12/hr and a concurrent increase in microbial synthesis from 22.4 to 31.8 g CP/100 g OMD, respectively. Similar trends were observed by Hogan and Weston (1970), who showed that increased dilution rates in sheep from .06 to .1/hr were associated with microbial production rates of 19.5 and 23.1 g CP/100 g OMD, respectively.

Kennedy et al. (1976) suggested that any one, or more likely a combination, of the following factors may be involved in causing efficiency of microbial synthesis to be postively related to dilution rate: reduced autolysis of bacteria; reduced engulfment of bacteria by protozoa; changes in microbial population structure induced by a change in substrate or possibly due to washout of slow generation time organisms.

It should be noted that Crawford et al. (1980) observed in continuous culture, using a mixed microbial population with solids substrate, an increase in microbial cell yield with a decrease in solids retention time more so than with increased liquid dilution rate. This finding is important as rumen bacteria, unlike those in a chemostat with a soluble substrate, do not necessarily leave the rumen with the liquid phase and can travel with the particulate matter. Dilution rate is certainly one of the major factors affecting microbial protein synthesis and differences in dilution rate may explain the wide range of efficiencies found in the rumen (Stern and Hoover, 1979). However, as bacteria can travel with the liquid or solid phase leaving the rumen, it is important to determine which dilution rate (solids or liquid) should be used when relating microbial growth efficiency to rumen outflow.

Sulfur. Sulfur is required by rumen microorganisms for synthesis of methionine and cysteine and intake of sulfur may limit protein synthesis when large amounts of nonprotein nitrogen are used (Buttery, 1977). Studies by Hume and Bird (1970) showed that when sheep were fed a diet supplying .6 g sulfur per day (N:S = 34.3), 82 g microbial protein were produced daily in the rumen. Raising sulfur intake to 2.0 g/day (N:S = 10.9) increased protein production to 94 g/day, but there was no further increase when intake was raised to 3.4 g/day (N:S = 6.4). Based on this type of information an optimum nitrogen: sulfur ratio of 10:1 has been suggested for maximum microbial growth.

Since a large proportion of sulfur incorporated into microbial protein is probably derived from ruminal sulfide, it would seem likely that the rate of ruminal release of dietary sulfur in conjunction with nitrogen could be important (Harrison and McAllan, 1980). For example, Playne et al. (1978) found

that with alfalfa hay (N:S, 11.4:1) the rate of removal of sulfur from nylon bags suspended in the rumen was similar to that of nitrogen removal. In contrast the rate of removal of sulfur from spear grass, stylo and chloris (which had N:S ratios of 9:1, 7:1 and 3.6:1, respectively) was at least 2.5 times faster than the rate of nitrogen removal. Therefore, Harrison and McAllan (1980) noted that when formulating sulfur requirements on the basis of N:S ratios in feeds it is obvious that the total sulfur content (particularly of forages) cannot be taken as an indicator of the relative availability of nitrogen and sulfur. At the present time there are insufficient data on dietary sulfur utilization to formulate accurate sulfur requirements and more research needs to be done in this area.

Nitrogen. Rumen microbial protein synthesis requires an adequate supply of nitrogen to achieve maximum efficiency. If nitrogen level is not adequate, uncoupled fermentation may occur and this will result in fermentation without useful ATP production (McMeniman et al., 1976; Buttery, 1977). In other words, the rate of fermentation is not necessarily dependent upon the rate of microbial growth. In contrast, if the nitrogen level is excessive, energy may be the limiting factor for efficient utilization of nitrogen. Therefore, for maximal efficiency of microbial growth to occur, nitrogen and energy availability in the rumen must be balanced.

Microbial nitrogen is derived from NH_3-N and (or) performed amino acids, with the latter highly dependent on the dietary nitrogen source. The percentage of microbial nitrogen derived from ruminal NH_3-N has been reported to range from 40 to 100% under various conditions (Pilgrim et al., 1970; Mathison and Milligan, 1971; Al-Rabbat et al., 1971; Nolan et al., 1976; Al-Rabbat and Heaney, 1978). Because ammonia is an important precursor of microbial protein and is essential for the growth of certain bacteria, there has been considerable interest in the process of ammonia fixation and the concentration of ammonia required for maximal growth. Two enzymes for utilization of rumen ammonia by rumen microbes are known, glutamate dehydrogenase and glutamate synthetase. Glutamate dehydrogenase is a constitutive enzyme with a low affinity for ammonia, km = 5 mmol/l, while glutamate synthetase is induced at low ammonia concentrations and has a high affinity for ammonia, km = .2 mmol/l, (Baldwin and Koong, 1980).

Several in vitro studies have shown maximum microbial growth to occur when the NH_3-N concentration was 5 to 8 mg/100 ml (Allison, 1970; Annison, 1975; Nikolic et al., 1975; Satter and Slyter, 1974). Hume et al. (1970) observed, in vivo, that microbial growth attained a maximum level when rumen NH_3-N concentration reached approximately 9 mg/100 milliliters. In contrast, Miller (1973) found a considerably higher value of approximately 29 mg/100 milliliters. Results of a more recent in vivo study (Okorie, 1981) indicated that maximal protein synthesis was achieved when the rumen NH_3-N concentration reached 5 mg/100 ml; an observation consistent with in vitro observations of Satter and Slyter (1974). Mehrez et al. (1977) used the dacron bag technique in situ to predict that the rumen NH_3-N concentration for maximum rate of fermentation was 23.5 mg/100 milliliters. Discordant with the results of Mehrez et al. (1977), Ortega et al. (1979) found in situ that progressively increasing rumen NH_3-N from 6.3 to 27.5 mg/100 ml did not result in any significant changes in rate of fermentation. In studies examining the rumen NH_3-N concentration required for maximal rate of fermentation, it should be emphasized that this measure does not necessarily equate to maximal protein synthesis.

Although the nitrogen concentration in a diet may appear to be adequate for maximum microbial growth, resistance of the protein to ruminal degradation may result in nitrogen deficiency. McMeniman and Armstrong (1977) determined that 2.0 g of available nitrogen per 100 g OMD is the minimum amount required for efficient microbial protein production for low-roughage diets. With starchy cereal diets there are distinct possibilities of nitrogen inadequacy, particularly with corn in which the protein is highly resistant to ruminal degradation (Thomas, 1977). Depending upon various factors such as species, stage of maturity and drying, forages can also result in deficiencies of available nitrogen to the rumen microbes (Hume, 1975; Ulyatt et al., 1975; Walker et al., 1975). In addition, Armstrong (1980) indicated that microbial protein synthesis from ensiled herbages may be lower than for other forms of conservation, probably due to the fact that the carbohydrates fermented during ensiling can no longer serve as as a source of energy for the rumen microorganisms. In support of these concepts, Van Soest (1982) concluded in his review of literature that silages and high concentrate diets show lower microbial yields than do forages or mixed diets.

Microbial protein synthesis can occur in the rumen on diets in which urea is the only nitrogen source, however, efficiency of microbial growth may be limited by a deficiency of preformed amino acids. An optimum ratio of urea nitrogen (75%) to amino acid nitrogen (25%) for rumen microbial growth in vitro was reported by Maeng et al. (1976). Hume (1970) found that nitrogen provided from urea, gelatin, casein and zein resulted in microbial synthesis of 17.1, 19.8, 23.3 and 22.5 g CP/100 g OMD in the rumen of sheep, respectively. It is possible that urea resulted in the most inefficient microbial growth due to a lack of preformed amino acids. The author suggested that since degradation of both gelatin and casein in the rumen approached completion, microbial protein production on the gelatin diet may have been limited by the rate of synthesis of one or more amino acids by the rumen bacteria, since gelatin is deficient in several amino acids, including methionine. Salter et al. (1979) examined the origin of nitrogen incorporation in rumen bacteria of steers fed protein and urea-containing diets. They found that when an adequate dietary supply of preformed amino acids was available that proline, arginine, histidine, methionine and phenylalanine were derived from the medium to a greater extent than other amino acids. While synthesis of proline, arginine and histidine increased on the urea-containing diet, that of methionine and phenylalanine did not. Therefore, methionine and phenylalanine may be limiting for bacterial growth on diets low in protein and high in nonprotein nitrogen.

Carbohydrate source. Efficient utilization of degraded dietary nitrogen requires that the energy from the fermentation of dietary OM must be supplied at a rate which matches the synthetic abilities of the rumen microbes (Oldham et al., 1977). Readily available carbohydrates such as starches and sugars were found to be more effective than other carbohydrates in increasing utilization of degraded dietary nitrogen and (or) increasing microbial growth both in vitro and in vivo (Offer et al., 1978; Stern et al., 1978; Nikolic et al., 1981). When starch has been added to high cellulose diets, or replaced part of the cellulose, increased nitrogen utilization and decreased fiber digestion has been reported (Slyter et al., 1971; Offer et al., 1978; Stern et al., 1978). The efficiency of starch to promote nitrogen utilization may be related to energy yield during fermentation. Compared to other carbohydrates studied, McAllan and Smith (1976) found that fermentation of starch provided the greatest amount of energy for rumen bacteria. Although ruminal degradation of starch is generally found to be at a high level (Sutton, 1971), differences in digestion due to source of starch (such as corn vs barley) have been reported (Ørskov et al., 1971).

Stern et al. (1978) noted that dietary energy level is not the only factor limiting microbial growth. They found an increase in microbial growth in continuous cultures (15.0 to 19.5 g microbial CP/100 g DMD) in response to increased dietary nonstructural carbohydrate levels, even though diets were isocaloric and VFA production and DM digestibilities did not differ markedly among diets (table 3). It was concluded that a major factor affecting the utilization of degraded dietary nitrogen was the type and rate of availability of carbohydrates. In a subsequent study, Macgregor (1979) examined the effect of level of total nonstructural carbohydrate (TNC) on nitrogen metabolism and lactation performance in dairy cattle. He found that a high TNC ration was associated with increased total milk and decreased ruminal ammonia level (figure 3). Presumably the high TNC ration was associated with an increase in rumen microbial protein synthesis. Figure 4 shows that the TNC level of a feed is not necessarily reflected by the traditional measures of energy content such as total digestible nutrients (TDN) or metabolizable energy (ME). Stern et al. (1978) noted that in recently proposed systems of calculating protein requirements for ruminants that available energy for microbial protein synthesis has been calculated based on values for ME and TDN and suggest that TNC may offer another means of calculating energy availability for rumen microbes.

CONCLUSIONS

Because of the importance of microbial protein to protein supply in ruminants, more accurate information is needed on factors influencing its synthesis. Measuring techniques used in cannulated animals and analytical procedures using markers for microbial protein need to be improved. Otherwise the results of research in this area will yield qualitative rather than quantitative differences for the efficiency of microbial protein production. While qualitative results may provide useful information, quantitative results are required to incorporate this information into a practical feeding situation.

Table 1. Contribution of microbial protein to total protein requirement of the lactating dairy cow[a].

Microbial CP synthesis (g/100 g OM digested)[b]	Theoretical contribution of microbial protein when daily milk production (kg) equals:		
	25	35	45
	%		
15	56	47	42
20	75	62	57
25	93	78	71

[a] NRC (1978).

[b] Assumed that 50% of OM intake is apparently digested in the rumen.

Table 2. Effect of rumen dilution rate on microbial protein synthesis.

Rumen dilution rate (hr^{-1})	Microbial CP synthesis (g/100 g OM digested)	Reference
.03 - .05	7.5 - 11.8	Cole et al. (1975)
.03 - .08	25.4 - 29.8[a]	Harrison et al. (1978)
.06 - .1	19.5 - 23.1	Hogan and Weston (1970)
.1 - .14	29.9 - 33.7	Kennedy et al. (1976)
.07 - .12	22.4 - 31.8	Kennedy and Milligan (1978)
.04 - .07	12.6 - 15.3	Allen and Harrison (1979)

[a]Grams of total amino acids synthesized/mole hexose fermented.

Table 3. Effects of nonstructural carbohydrate levels on VFA production, DM digestibility, and microbial growth.

Item	Nonstructural carbohydrate (%)[a] 49.0	32.6	19.6
Total VFA			
mM	79.0	84.2	83.4
mM/g DM fed	4.3	4.6	4.5
DM digestibility, %	68.8	69.4	65.8
Ammonia, mg/100 ml	18.1[b]	22.8[c]	27.4[d]
Microbial synthesis, g CP/100 g DM digested	19.5[b]	17.5[bc]	15.0[c]
Y_{ATP}[e]	20.5[b]	17.6[c]	14.6[d]

[a]Percent of total diet DM - comprised of starches, sugars and fructosans.

[b,c,d]Values in the same row with different superscripts are significantly different ($P < .05$).

[e]Grams of microbial cells (dry wt) produced per mole ATP generated.

(Stern et al., 1978)

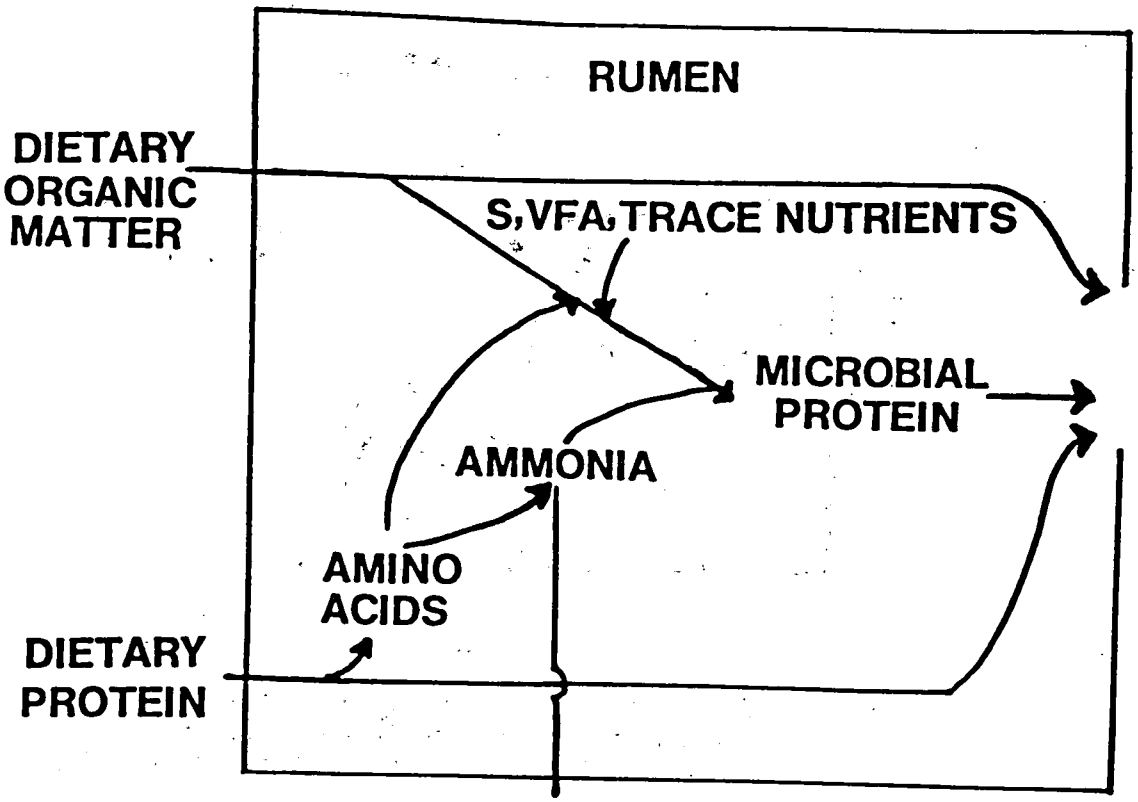

Figure 1. Microbial protein synthesis in the rumen.

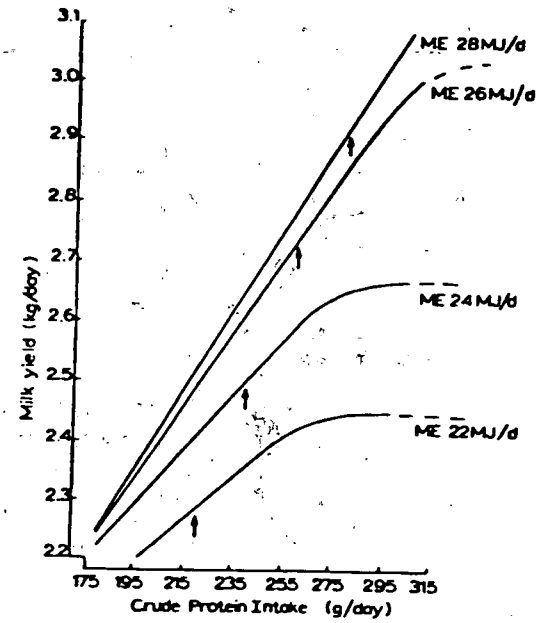

Figure 2. The effect of crude protein intake on the milk yield of ewes given different amounts of metabolizable energy (ME). The symbol ↑ indicates contribution from microbial protein, extrapolated. (Ørskov and Robinson, 1981).

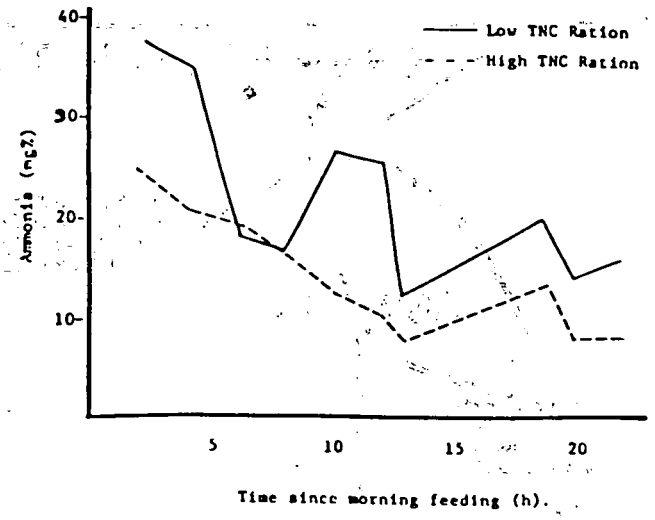

Figure 3. Rumen ammonia levels in fistulated cows fed rations differing in level of total nonstructural carbohydrate (Macgregor, 1979).

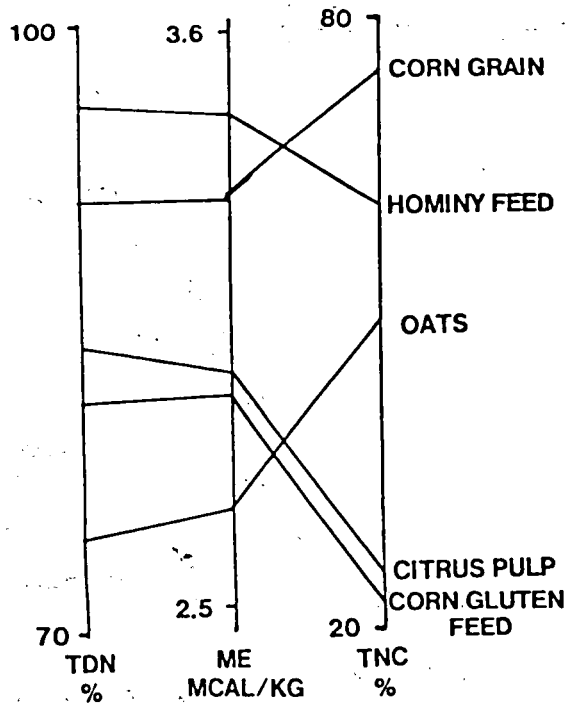

Figure 4. Comparison of TDN, ME and TNC values in five feedstuffs (Macgregor, 1979).

REFERENCES

Allen, J. D. and D. G. Harrison. 1979. The effect of the dietary addition of monensin upon digestion in the stomach of sheep. Proc. Nutr. Soc. 38: 32A. (Abstr.)

Allison, M. J. 1970. Nitrogen metabolism of ruminal micro-organisms. In A. T. Phillipson (Ed.) Physiology of Digestion and Metabolism in the Ruminant. Oriel Press Ltd., England.

Al-Rabbat, M. F., R. L. Baldwin and W. C. Weir. 1971. Microbial growth dependence on ammonia nitrogen in the bovine rumen: A quantitative study. J. Dairy Sci. 54:1162.

Al-Rabbat, M. F. and D. P. Heaney. 1978. The effects of anhydrous ammonia treatment of wheat straw and steam cooking of aspen wood on their feeding value and on ruminal microbial activity. II. Fermentable energy and microbial growth derived from ammonia nitrogen in the ovine rumen. Can. J. Anim. Sci. 58:453.

Annison, E. F. 1975. Microbial protein synthesis in relation to amino acid requirements. P. 141. Tracer Studies on Nonprotein Nitrogen for Ruminants II. FAO/IAEA.

Armstrong, D. G. 1980. In H.J. Osluge and K. Rohr (Ed.) 3rd E.A.A.P. Int. Symp. on Protein Metabolism and Nutrition, E.A.A.P., Braunschweig, W. Germany.

Baldwin, R. L. and L. J. Koong. 1980. Mathematical modelling in analyses of ruminant digestive function:philosophy, methodology and application. In Y. Ruckebush and P. Thivend (Ed.) Digestive Physiology and Metabolism in Ruminants. AVI Publishing Co. Inc., Westport, Ct.

Beever, D. E., D. G. Harrison, D. J. Thomson, S. B. Cammell and D. F. Osbourn. 1974. A method for the estimation of dietary and microbial protein in duodenal digesta of ruminants. Brit. J. Nutr. 32:99.

Brock, T. D. 1974. Biology of Microorganisms. Prentice-Hall Inc. Englewood Cliff, N.J.

Buchholtz, H. F. and W. G. Bergen. 1973. Microbial phospholipid synthesis as a marker for microbial protein synthesis in the rumen. Appl. Microbiol. 25:504.

Buttery, P. J. 1977. Aspects of the biochemistry of rumen fermentation and their implication in ruminant productivity. In W. Haresign and D. Lewis (Ed.) Recent Advances in Anim. Nutr. 1977. Butterworth Inc., Boston, MA

Buttery, P. J. and D. J. A. Cole. 1977. Chemical analysis: sources of error. Proc. Nutr. Soc. 36:211.

Cole, N. A., R. R. Johnson, F. N. Owens and J. R. Males. 1976. Influence of roughage level and corn processing method on microbial protein synthesis by beef steers. J. Anim. Sci. 43:497.

Coleman, G. S. and F. J. Hall. 1969. Electron microscopy of the rumen cilitate Entodinium caudatum, with special reference to the engulfment of bacteria and other particulate matter. Tissue and Cell 1:607.

Crawford, Jr., R. J., W. H. Hoover and L. L. Junkins, Jr. 1980. Effects of solids and liquid flows on fermentation in continuous cultures II. Nitrogen partition and efficiency of microbial synthesis. J. Anim. Sci. 51:986.

Czerkawski, J. W. 1974. Methods for determining 2-6-diaminopimelic acid and 2-aminoethylphosphonic acid in gut contents. J. Sci. Food Agr. 25:45.

Dufva, G. S., E. E. Bartley, M. J. Arambel, S. J. Galitzer and A. D. Dayton. 1982. Content of 2-aminoethylphosphonic acid in feeds, bacteria and protozoa and its role a rumen protozoal marker. J. Anim. Sci. 54:837.

Evans, R. A., R. F. E. Axford and N. W. Offer. 1975. A method for estimating the quantities of microbial and dietary protein flowing in the duodenal digesta of ruminants. Proc. Nutr. Soc. 34:65A. (Abstr.)

Forsberg, C. W. and K. Lam. 1977. Use of adenosine 5'-triphosphate as an indicator of the microbiota biomass in rumen contents. Appl. Environ. Microbiol. 33:528

Garrett, J. E., R. D. Goodrich and J. C. Meiske. 1982. Measurement of bacterial nitrogen using D-alanine. In: F. N. Owens (Ed.) Protein Requirements for Cattle: Symp. pp 23-25. Stillwater, OK.

Gawthorne, J. W. and C. J. Nader. 1976. The effect of molybdenum on the conversion of sulphate to sulphide and microbial-protein-sulphur in the rumen of sheep. Brit. J. Nutr. 35:11.

Harmeyer, H., H. Holler, H. Martens and C. von Grabe. 1976. Estimate of microbial protein synthesis in vitro by the simultaneous use of three different isotopic markers. P. 69. Tracer Studies on Nonprotein Nitrogen for Ruminants III. FAO/IAEA.

Harrison D. G., D. E. Beever, D. J. Thomson and D. F. Osbourn. 1976. Manipulation of fermentation in the rumen. J. Sci. Food Agr. 27:617.

Harrison, D. G. and A. B. McAllan. 1980. Factors affecting microbial growth yields in the reticulo-rumen. In Y. Ruckebush and P. Thivend (Ed.) Digestive Physiology and Metabolism in Ruminants. AVI Publishing Co. Inc., Westport, Ct.

Henderickx, H. 1961. The incorporation of sulfate in the ruminal proteins. Arch. Int. Physiol. Biochim. 69:449.

Hobson, P. N. 1965. Continuous culture of some anaerobic and facultatively anaerobic rumen bacteria. J. Gen. Microbiol. 38:167.

Hobson, P. N. and R. Summers. 1967. The continuous culture of anaerobic bacteria. J. Gen. Microbiol. 47:53.

Hogan, J. P. and R. H. Weston. 1970. Quantitative aspects of microbial protein synthesis in the rumen. In A. T. Phillipson (Ed.) Physiology of Digestion and Metabolism in the Ruminant. Oriel Press Ltd., England.

Hume, I. D. 1970. Synthesis of microbial protein in the rumen. III. The effect of dietary protein. Australian J. Agr. Res. 21:305.

Hume, I. D. and P. R. Bird. 1970. Synthesis of microbial protein in the rumen. IV. The influence of the level and form of dietary sulfur. Australian J. Agr. Res. 21:315.

Hume, I. D., R. J. Moir and M. Somers. 1970. Synthesis of microbial protein in the rumen. I. Influence of the level of nitrogen intake. Australian J. Agr. Res. 21:283.

Hume, I. D. 1974. The proportion of dietary protein escaping degradation in the rumen of sheep fed on various protein concentrates. Australian J. Agr. Res. 25:155.

Hume, I. D. 1975. Use of ^{35}S to estimate the proportion of dietary protein degraded in the rumen. P. 1. Tracer Studies on Nonprotein Nitrogen for Ruminants II. FAO/IAEA.

Hutton, K., F. J. Bailey and E. F. Annison. 1971. Measurement of the bacterial nitrogen entering the duodenum of the ruminant using diaminopimelic acid as a marker. Brit. J. Nutr. 25:165.

Ibrahim, E. A. and J. R. Ingalls. 1972. Microbial protein biosynthesis in the rumen. J. Dairy Sci. 55:971.

Isaacson, H. R., R. C. Hinds, M. P. Bryant and F. N. Owens. 1975. Efficiency of energy utilization by mixed rumen bacteria in continuous culture. J. Dairy Sci. 58:1645.

Kennedy, P. M., R. J. Christopherson and L. P. Milligan. 1976. The effect of cold exposure of sheep on digestion, rumen turnover and efficiency of microbial synthesis. Brit. J. Nutr. 36:231.

Kennedy, P. M. and L. P. Milligan. 1978. Effects of cold exposure on digestion, microbial synthesis and nitrogen transformations in sheep. Brit. J. Nutr. 39:105.

Kennedy, P. M., J. R. Lindsay and L. P. Milligan. 1980. In vivo measurement of rumen microbial growth using ^{35}S. J. Agr. Sci. 94:89.

Leibholz, J. 1972. Nitrogen metabolism in sheep. II. The flow of amino acids into the duodenum from dietary and microbial sources. Australian J. Agr. Res. 23:1073.

Ling, J. R. and P. J. Buttery. 1978. The simultaneous use of ribonucleic acid, ^{35}S, 2,6-diaminopimelic acid and 2-aminoethylphosphonic acid as markers of microbial nitrogen entering the duodenum of sheep. Brit. J. Nutr. 39:165.

Macgregor, Jr., C. A. 1979. Effect of level of total nonstructural carbohydrate in complete blended rations on energy and nitrogen metabolism and lactation performance in dairy cattle. Ph.D. Thesis, University of Maine, Orono.

Maeng, W. J., C. J. VanNevel and R. L. Baldwin. 1976. Rumen microbial growth rates:effect of amino acids and protein. J. Dairy Sci. 59:68.

Mathers, J. C. and E. L. Miller. 1980. A simple procedure using ^{35}S incorporation for the measurement of microbial and undegraded food protein in ruminant digesta. Brit. J. Nutr. 43:503.

Mathison, G. W. and L. P. Milligan. 1971. Nitrogen metabolism in sheep. Brit. J. Nutr. 25:351.

McAllan, A. B. and R. H. Smith. 1969. Nucleic acid metabolism in the ruminant. Determination of nucleic acid in digesta. Brit J. Nutr. 23:671.

McAllan, A. B. and R. H. Smith. 1973. Degradation of nucleic acids in the rumen. Brit. J. Nutr. 29:331.

McAllan, A. B. and R. H. Smith. 1974. Contribution of microbial nitrogen to duodenal digesta in the ruminant. Proc. Nutr. Soc. 33:41A. (Abstr.)

McAllan, A. B. and R. H. Smith. 1976. Effect of dietary nitrogen source on carbohydrate metabolism in the rumen of the young steer. Brit. J. Nutr. 36:511.

McMeniman, N. P., D. Ben-Ghedalia and D. G. Armstrong. 1976. Nitrogen-energy interactions in rumen fermentation. In D. J. A. Cole, K. N. Boorman, P. J. Buttery, D. Lewis, R. J. Neale and H. Swan. (Ed.) Protein Metabolism and Nutrition. Butterworth Inc., Boston, MA.

McMeniman, N. P. and D. G. Armstrong. 1977. Nitrogen levels in low-roughage diets for efficient rumen microbial protein production. Anim. Feed Sci. Tech. 2:225.

Mehrez, A. Z., E. R. Ørskov and I. McDonald. 1977. Rates of rumen fermentation in relation to ammonia concentration. Brit. J. Nutr. 38:447.

Merck Index. 1978. M. Windholz (Ed.), Merck and Co. Rahway, N.J.

Mercer, J. R. and E. F. Annison. 1976. Utilization of nitrogen in ruminants. In D. J. A. Cole, K. N. Boorman, P. J. Buttery, D. Lewis, R. J. Neale and H. Swan (Ed.) Protein Metabolism and Nutrition. Butterworth Inc., Boston, MA.

Miller, E. L. 1973. Evaluation of foods as sources of nitrogen and amino acids. Proc. Nutr. Soc. 32:79.

Nader, C. J. and D. J. Walker. 1970. Metabolic fate of cysteine and methionine in rumen digesta. Appl. Microbiol. 20:677.

Nikolic, J. A., M. Jovanovic and R. Filipovic. 1975. Microbial protein synthesis by bovine rumen content in relation to ammonia concentration. P. 43. Studies on Nonprotein Nitrogen for Ruminants II. FAO/IAEA.

Nikolic, J. A., M. Jovanovic and D. Djordjevic. 1981. Influence of readily digestible carbohydrates on the utilization of ammonia and sulphur for protein synthesis in rumen contents. Acta Vet. 31:289.

Nolan, J. V., B. W. Norton and R. A. Lang. 1976. Further studies of the dynamics of nitrogen metabolism in sheep. Brit. J. Nutr. 35:127.

NRC. 1978. Nutrient Requirements of Dairy Cattle. Fifth Revised Ed. National Academy of Sciences. Washington, DC.

Offer, N. W., R. F. E. Axford and R. A. Evans. 1978. The effect of dietary energy source on nitrogen metabolism in the rumen of sheep. Brit. J. Nutr. 40:34.

Okorie, A. U. 1981. Microbial protein synthesis in relation to rumen and duodenal ammonia concentrations. Nutr. Rep. Int. 24:1241.

Oldham, J. D., P. J. Buttery, H. Swan and D. Lewis. 1977. Interactions between dietary carbohydrates and nitrogen and digestion in sheep. J. Agr. Sci. Camb. 89:467.

Ørskov, E. R., R. C. Fraser and I. McDonald. 1971. Digestion of concentrates in sheep. Effects of rumen fermentation of barley and maize diets on protein digestion. Brit. J. Nutr. 26:477.

Ørskov, E. R. and J. J. Robinson. 1981. The application of modern concepts of ruminant protein nutrition to sheep production systems. Livestock Prod. Sci. 8:339.

Ortega, M. E., M. D. Stern and L. D. Satter. 1979. The effect of rumen ammonia concentration on dry matter disappearance in situ. J. Dairy Sci. Suppl 62:76. (Abstr.)

Pilgrim, A. F., F. V. Gray, R. A. Weller and C. B. Belling. 1970. Synthesis of microbial protein from ammonia in the sheep's rumen and the proportion of dietary nitrogen converted into microbial nitrogen. Brit. J. Nutr. 24:589.

Playne, M. J., Eschevarria, M. G. and R. G. Megarrity. 1978. Release of N, S, P, Ca, Mg, K and Na from four tropical hays during their digestion in nylon bags in the rumen. J. Sci. Food Agr. 29:520.

Rode, L. M. 1981. Factors affecting and measurement of microbial protein synthesis in vitro. M.S. Thesis, University of Wisconsin, Madison.

Salter, D. N., K. Daneshvar and R. H. Smith. 1979. The origin of nitrogen incorporated into compounds in the rumen bacteria of steers given protein and urea-containing diets. Brit. J. Nutr. 41:197.

Satter, L. D. and L. L. Slyter. 1974. Effect of ammonia concentration on rumen microbial protein production in vitro. Brit. J. Nutr. 32:199.

Slyter, L. L., R. R. Oltjen, E. E. Willimas, Jr. and R. L. Wilson. 1971. Influence of urea, biuret and starch on amino acid patterns in ruminal bacteria and blood plasma and on nitrogen balance of steers fed high fiber purified diets. J. Nutr. 101:839.

Smith, R. H. and A. B. McAllan. 1970. Nucleic acid metabolism in the ruminant. 2. Formation of microbial nucleic acids in the rumen in relation to the digestion of food nitrogen, and the fate of dietary nucleic acids. Brit. J. Nutr. 24:545.

Smith, R. H. and A. B. McAllan. 1974. Some factors influencing the chemical composition of mixed rumen bacteria. Brit. J. Nutr. 31:27.

Smith, R. H. 1975. Nitrogen metabolism in the rumen and the composition and nutritive value of nitrogen compounds entering the duodenum. In I. W. McDonald and A. C. I. Warner (Ed.) Digestion and Metabolism in the Ruminant. The Univ. of New England Publishing Unit, Armidale, N.S.W., Australia.

Smith, R. H., A. B. McAllan, D. Hewitt and P. E. Lewis. 1978. Estimation of amounts of microbial and dietary nitrogen compounds entering the duodenum of cattle. J. Agr. Sci. Camb. 90:557.

Stern, M. D., W. H. Hoover, R. G. Summers, Jr. and J. H. Rittenburg. 1977a. Ultrastructure of rumen entodiniomorphs by electron microscopy. J. Dairy Sci. 60:902.

Stern, M. D., W. H. Hoover and J. B. Leonard. 1977b. Ultrastucture of rumen holotrichs by electron microscopy. J. Dairy Sci. 60:911.

Stern, M. D., W. H. Hoover, C. J. Sniffen, B. A. Crooker and P. H. Knowlton. 1978. Effects of nonstructural carbohydrate, urea and soluble protein levels on microbial protein synthesis in continuous culture of rumen contents. J. Anim Sci. 47:944.

Stern, M. D. and W. H. Hoover. 1979. Methods for determining and factors affecting rumen microbial protein synthesis:A review J. Anim. Sci. 49:1590.

Storm, E. and E. R. Ørskov. 1982. Biological value and digestibility of rumen microbial protein in lamb small intestine. Proc. Nutr. Soc. 41:78A. (Abstr.)

Stouthamer, A. H. and C. Bettenhaussen. 1973. Utilization of energy for growth and maintenance in continuous and batch cultures of microorganisms. Biochim. Biophys. Acta. 301:53.

Sutton, J. D. 1971. Carbohydrate digestion and glucose supply in the gut of the ruminant. Proc. Nutr. Soc. 30:243.

Theurer, B. C. 1979. Microbial protein synthesis as influenced by diet. In W. H. Hale and P. Meinhardt (Ed.) Regulation of Acid-Base Balance. Church Dwight Co., Piscataway, N.J.

Thomas, P. C. 1977. Ruminal fermentation and flow of nitrogen compounds to the duodenum. In S. Tamminga (Ed.) Proc. of the Second Internat'l Symp. on Protein Metabolism and Nutrition. PUDOC, Wageningen, The Netherlands.

Ulyatt, M. J., J. C. MacRae, R. T. J. Clarke and P. D. Pearce. 1975. Quantitative digestion of fresh herbage by sheep. IV. Protein synthesis in the stomach. J. Agr. Sci. Camb. 84:453.

Van Nevel, C. J. and D. I. Demeyer. 1977. Determination of rumen microbial growth in vitro from ^{32}P-labelled phosphate incorporation. Brit. J. Nutr. 38:101.

Van Soest, P. J. Nutritional Ecology of the Ruminant. O&B Books, Inc. Corvallis, Oregon.

Walker, D. J. and C. J. Nader. 1968. Method for measuring microbial growth in rumen content. Appl. Microbiol. 16:1124.

Walker, D. J. and C. J. Nader. 1975. Measurement in vivo of rumen microbial protein synthesis. Australian J. Agr. Res. 26:689.

Walker, D. J., A. R. Egan, C. J. Nader, M. J. Ulyatt and G. B. Storer. 1975. Rumen microbial protein synthesis and proportions of microbial and non-microbial nitrogen flowing to the intestines of sheep. Australian J. Agr. Res. 26:699.

Wolstrup, J. and K. Jensen. 1978. Adenosine triphosphate and deoxyribonucleic acid in the alimentary tract of cattle fed different nitrogen sources. J. Appl. Bact. 45:49.

Work, E. and L. L. Dewey. 1953. The distribution of α-ξ-diaminopimelic acid among various microorganisms. J. Gen. Microbiol. 9:394.

THE ASSOCIATIVE EFFECTS OF FEEDSTUFFS

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INTRODUCTION

There is a paucity of both scientific research and common understanding of the associative effects (AE) of individual feeds in ruminant diets. Because feed costs usually comprise the largest portion of total production costs, any factor that may influence the efficiency of feed utilization should not be overlooked.

An AE results from the interaction of the individual feed ingredients such that the value of the mixed ration differs from the expected value based on animal performance or nutrient utilization when the individual feed ingredients are fed alone. The AE can be either positive or negative. A positive AE results when animal performance or nutrient utilization is greater on the mixed diet compared to that predicted by performance on the single ingredient. A negative associative effect is simply a lower than expected performance on the multiple ingredient diet relative to the single ingredient diets.

The purpose of this paper is to review the research on associative effects. I will discuss the factors impinging on AE and the practical implications to ruminant nutrition and the feed industry.

EARLY STUDIES ON ASSOCIATIVE EFFECTS

The term associative effects was first presented by Ewing and Ellis (1915). Forbes published several studies that illustrated the existence of AE (Forbes et al., 1931; Forbes, et al., 1933). Considerable interest and controversy were generated by these early research reports. If AE are real and of such a magnitude that warrant alterations in feeding systems, the implication to ruminant nutrition is large. First, compensations must be made, depending on the type of AE, in terms of expected gain and/or feeding level for mixed diets. Secondly, the mass of data gathered on individual feeds would need to be adjusted when these feeds are fed in mixed diets. Indeed, several workers have proposed that individual feeds should not be evaluated, but characterizations should be made only on complete rations (Forbes, 1933; Kriss, 1943; Kromann et al., 1975).

ASSOCIATIVE EFFECTS ON DIGESTIBILITY

Perhaps the most intuitively easy to accept AE is the depression in fiber digestibility that occurs when molasses or another source of readily available carbohydrate is included in the ration (Ahmed and Kay, 1975; Chappell and Fontenot, 1968; Swift and French, 1954). Examples are available of positive AE on digestibility of fiber and other diet components. Johnson et al., (1962) determined the digestibility of soybean flakes alone or in combination with timothy hay (Table 1). An improvement (positive AE) in organic matter, cellulose and crude fiber digestibility resulted when timothy hay (40% of DM) was included in the ration. The calculated organic matter digested/100 pounds fed was 60.9,

while that actually measured was 66.6. Similar estimates for cellulose were 23.9 versus 29.2. The authors indicated that the improved digestibility of soybean flakes in the mixed diet was likely due to longer rumen retention time which would be expected when timothy hay was included in the diet.

Hintz et al. (1964) fed soybean flakes, soybean hulls or hay, and 1 to 1 combinations of hay and soybean products. The data (table 2) show significantly lower TDN and digestibility of dry matter and crude fiber for the hulls compared to the flakes. However, these large differences were eliminated when soybean products were fed with hay. The authors noted a decreased rate of passage for the hay diets.

Nelson et al. (1968) fed bermudagrass-concentrate ratios of 100:0, 75:25, 50:50, 25:75 and 0:100. These researchers noted that AE on ration digestibility increased as the proportion of concentrate increased (table 3). Energy digestibility decreased .8% below theoretical (assuming no associative effects) for each 25% increase of concentrate in the diet.

Alfalfa hay, beet pulp and a combination of the two ingredients were evaluated by Asplund and Harris (1971). Observed digestion coefficients for the individual feeds were significantly lower in many cases, compared to the values from the mixed diets that were calculated by the difference method (table 4).

The AE between corn and corn silage was investigated by Byers et al. (1976). These workers observed significant negative AE on dry matter and energy digestibility in mixed diets (1/3 or 2/3 corn) relative to those calculated from individual feed ingredient diets (table 5). Kromann et al. (1975) showed that crude protein, ether extract and nitrogen-free extract digestibility increased curvilinearly and crude fiber decreased curvilinearly as corn levels increased in alfalfa and corn diets.

Recent studies by Joanning et al. (1981) confirmed earlier observations (Byers et al., 1976) on the negative AE between corn and corn silage in mixed diets. Data in table 6 show that the digestibility depression for the mixed diets ranges from approximately 10 to 20 units below that expected based on calculations from the single ingredient diets. Starch, cell wall and protein were the major nutrients that contributed to the depression in dry matter digestibility of the mixed diets. Starch, because it was present in the greatest amount, accounted for about 67% of the depression, cell wall 28% and the value for protein was 13%.

Level of feed intake is an important consideration in feed evaluation because it is known to affect nutrient digestibility (Campling et al., 1961; Colovos et al., 1970; Moe et al., 1965; Nicholson and Sutton, 1969; Tyrrell and Moe, 1972). Data by Joanning et al., (1981) were adjusted by covariate analysis for intake effects on all diets; intake did not significantly affect dry matter digestibility (Joanning, 1979). Relationships for the individual diets are shown in figure 1. Intake had very little influence on digestibility of the corn or corn silage single ingredient diets. However, the multiple ingredient diets showed a negative regression of digestibility on intake (r^2=.7) This indicates that the digestibility depression, for the mixed diets, may involve intake effects mediated through altered rates of passage, as a component of AE. The data of Joanning et al. (1981) indicate that no depression in digestibility would occur at intakes at or below twice maintenance. Research by Raven (1972) with hay and/or barley, fed at maintenance intakes, showed no AE, thus corroborating this theory. Yet the negative AE on digestibility of corn and corn

silage diets described earlier (Byers et al., 1976) occurred at intakes just above maintenance.

ASSOCIATIVE EFFECTS ON METABOLIZABLE AND NET ENERGY

There is a developing body of evidence that indicates AE influence not only digestibility but metabolizable and net energy. Indeed, a large portion of the research conducted indicates the AE mainly affects energy availability and metabolism.

Early studies that determined the energy values of corn meal-alfalfa hay combinations indicated (table 7) that observed metabolizable energy (ME) values agreed with those calculated from data on the sole-ingredient feeds (Forbes et al., 1931). However, the calculated net energy (NE) values for corn differed from the directly determined values. The heat increment (HI) was substantially lower for the multiple ingredient diets than from either ingredient alone, indicating that AE may influence nutrient metabolism.

Forbes et al. (1933) examined the AE among forage sources and the utilization of energy in corn. Table 8 illustrates the different ME and NE values of corn when fed at 1.5 times maintenance with either oat straw, timothy hay or alfalfa hay. The ME value of corn increased as quality of the forage source decreased, the NE pattern was different and due in part to the large differences in heat increment (measured by direct calorimetry). These calorimetry studies lend support to the theory expressed by Forbes that individual feeds express their characteristic net energy values only as part of nutritively complete rations.

Kriss (1943) reviewed the theory and procedures for the evalution of feeds. This report summarized the thoughts of Morrison, Kleiber, Fraps and Carlyle. They all realized the problems associated with the determination of nutritive value of single feeds and attempted to determine the relative or replacement value of a feedstuff in a diet. In his work the net availability of ME of corn meal fed with oat straw, timothy hay or alfalfa hay was 74, 57 and 67 per cent respectively.

The NE value of blackstrap molasses showed a curvilinear decline when fed at 10, 25 or 40% of the diet (Lofgreen and Otagaki, 1960). Kromann and Ray (1967) fed 30, 60 or 90% milo with alfalfa hay. The NE of alfalfa declined from 1.12 to .84 kcal/g as milo content increased and milo NE increased from 1.52 to 1.84.

Kromann et al., (1975) failed to show any net energy AE in pelleted diets between corn grain and ground dehydrated alfalfa. Physical form of the diet may be a modifying factor since Kromann (1967) fed similar ingredients in a non-pelleted form and did observe AE on net energy.

Vance et al., (1972) fed several corn grain-corn silage combinations. Curvilinear relationships were shown between NE_{m+g} of the ration and corn silage content, but linear NE_m values were obtained. The NE_g values were nonlinear and showed significant quadratic relationships, indicating the lack of additivity of these values.

The work of Byers et al. (1976) demonstrated marked negative AE between corn grain and corn silage (table 9). The depression from predicted diet energy

values ranged from 5 to 15%. The dry matter digestibility depression averaged 5%. In this study, the NE_g value of corn silage was .49 kcal/g when added to an all corn diet, and the same silage in an all corn silage diet had an NE_g of 1.25 kcal/g. This large disparity in energy content of a given feed, which is apparently modified by the feed in the basal rations, confirmed earlier observations by Blaxter and Wainman (1964). Also, these data reiterated the concept that energy values of individual ingredients are fundamentally variable and depend to a large extent on the combination of feeds in which they are fed.

POSSIBLE MODES OF ACTION OF ASSOCIATIVE EFFECTS

The compendium of data presented thus far demonstrates the existence of AE. The farmer, researcher and feed supplier should all be aware of the potential for AE in that substantial alterations in feed efficiency and animal performance may result.

Given the present state of knowledge in animal nutrition, it is not possible to delineate the underlying mechanism of AE. Thoughful speculation may provide plausible modes of action and stimulate scientific research that will eventually ellucidate the variables involved in AE.

As mentioned earlier, both physical form and intake may be modifying factors involved in AE. Physical form connotes changes in particle size of the feed. Particle size effects are mediated through altered rate of passage of diet components. Similarly, feed intake effects are also mediated by changes in rate of passage. A faster rate of passage will allow less time for the feed to be digested at a given site in the gastrointestinal tract. In this context, as particle size decreases and/or feed intake increases, rate of passage of the feed will increase. Thus, the potential for escape of ultimately digestible feed fractions would increase and the probability of observing a digestibility depression would be higher.

It is interesting to note that multiple ingredient diets are often consumed in greater amounts than single ingredient diets, which may explain some of the observed negative associative effects. An excellent example is the aforementioned work of Joanning et al. (1981). Owens and Rust (personal communication) observed a 16% increase in feed intake relative to single component feeds for mixed corn silage, milo or corn grain diets, when AE were present. These workers contend that reduced digestibility with elevated feed intake (faster passage) could account for a sizeable portion of the negative AE (Rust and Owens, 1982). Conversely, coarse forage in the diet may slow the rate of passage of the grain portion of the diet and improve (positive associative effect) feed utilization.

Further, the combination of feed ingredients will obviously present the rumen microbiota with different nutrients compared to the single ingredient diets. Thus, alterations in microbial species distribution, metabolic propensity or rumen environment may be involved in changes in nutrient utilization and resulting animal performance.

Finally, the previously described calorimetry work on net energy values of multiple ingredient diets relative to the single feed ingredient counterparts suggests the potential for altered metabolism of derived nutrients. The complex, interacting cycle continues because present data do not allow distinction

among rate of passage, rumen environment and metabolic factors involved in associative effects.

SUMMARY

Associative effects of feeds that have been evaluated as single ingredient diets may occur in multiple ingredient diets such that the nutrient utilization differs from expected values. Associative effects influence digestibility and energy utilization in cattle finishing and dairy rations. The mode of action of associative effects is not clear. Alterations in rate of passage, rumen environment and nutrient metabolism have been suggested as factors involved in associative effects. Scientific effort in this area will lead to improved understanding of nutrient utilization and conversion of feed to saleable products which will result in a more efficient ruminant agriculture.

LITERATURE CITED

Ahmed, F. A. and M. Kay. 1975. A note on the value of molasses and tapioca as energy supplements to forage for growing steers. Anim. Prod. 21:199.

Asplund, J. M. and L. E. Harris. 1971. Associative effects on the digestibility of energy and the utilization of nitrogen in sheep fed simplified rations. J. Anim. Sci. 32:152.

Blaxter, K. L. and F. W. Wainman. 1964. The utilization of the energy of different rations by sheep and cattle for maintenance and for fattening. J. Agric. Sci. 63:113.

Byers, F. M., D. E. Johnson and J. K. Matsushima. 1976. Associative effects between corn and corn silage on energy partitioning by steers In: Energy metabolism of farm animals. M. Vermoral (ed.) E.A.A.P. publication No. 19. pg. 253.

Campling, R. C., M. Frier and C. C. Balch. 1961. Factors affecting the voluntary intake of food by cows. 2. The relationship between the voluntary intake of roughages, the amount of digesta in the reticulorumen and the rate of passage disappearance of digesta from the alimentary tract. Brit. J. Nutr. 15:531.

Chappell, G. L. M. and J. P. Fontenot. 1968. Effect of level of readily-available carbohydrates in purified sheep rations on cellulose digestibility and nitrogen utilization. J. Anim. Sci. 27:1709.

Colvos, N. F., J. B. Holter, R. M. Does, W. E. Urban, Jr. and H. A. Davis. 1970. Digestibility, nutritive value and intake of ensiled corn plant (Zea Mays) in cattle and sheep. J. Anim. Sci. 37:994.

Ewing, P. V. and C. A. Ellis. 1915. The associative digestibility of corn silage, cottonseed meal and starch in steer rations. Georgia Ag. Exp. Sta. Bull. 115:271.

Forbes, E. B. 1933. The law of maximum normal nutritive value. Science 77:306.

Forbes, E. B., W. W. Braman, M. Kriss, R. W. Swift, A. Black, D. E. Frear, O. J. Kohlenberg, F. J. McClure and L. Voris. 1933. The associative effects of feeds in relation to the utilization of feed energy. J. Ag. Res. 46:753.

Forbes, E. B., W. W. Braman, M. Kriss, R. W. Swift, R. C. Miller, R. B. French, T. V. Letonoff and G. R. Sharpless. 1931. The metabolizable energy and net energy values of corn meal when fed exclusively and in combination with alfalfa hay. J. Ag. Res. 43:1015.

Hintz, H. F., M. M. Mathias, H. F. Ley, Jr. and J. K. Loosli. 1964. Effects of processing and of feeding hay on the digestibility of soybean hulls. J. Anim. Sci. 23:43.

Joanning, S.W. 1979. Digestibility of corn silage, corn grain and mixtures by steers. Ph.D. Thesis. Colorado State Univ., Fort Collins.

Joanning, S. W., D. E. Johnson and B. P. Barry. 1981. Nutrient digestibility depressions in corn silage-corn grain mixtures fed to steers. J. Anim. Sci. 53:1095.

Johnson, R. R., E. W. Klosterman and H. W. Scott. 1962. Studies on the feeding value of soybean flakes for ruminants. J. Anim. Sci. 21:406.

Kriss, M. 1943. Evaluation of feeds on the basis of net available nutrients. J. Anim. Sci. 2:63.

Kromann, R. P. 1967. A mathematical determination of energy values of ration ingredients. J. Anim. Sci. 26:1131.

Kromann, R. P. and E. E. Ray. 1967. Energy metabolism in sheep as influenced by interactions among nutritional and genetic factors. J. Anim. Sci. 26:1379.

Kromann, R. P., E. T. Clemens and E. E. Ray. 1975. Digestible, metabolizable and net energy values of corn grain and dehydrated alfalfa in sheep. J. Anim. Sci. 41:1752.

Lofgreen, G. P. and K. K. Otagaki. 1960. The net energy of blackstrap molasses for fattening steers as determined by a comparative slaughter technique. J. Anim. Sci. 19:392.

Moe, P. W., J T. Reid and H. F. Tyrrell. 1965. Effect of level of intake on digestibility of dietary energy by high-producing cows. J. Dairy Sci. 48:1053.

Nelson, B. D., H. D. Ellzey, E. B. Morgan, and M. Allen. 1968. Effects of feeding lactating dairy cows varying forage-to-concentrate ratios. J. Dairy Sci. 51:1796.

Nicholson, J. W. G. and J. D. Sutton. 1969. The effect of diet composition and level of feeding on digestion in the stomach and intestines of sheep. Brit. J. Nutr. 28:585.

Raven, A. M. 1972. The effect of different proportions of hay and fortified rolled barley in the diet on digestible-energy value and urinary-energy by cattle. J. Ag. Sci., Camb. 79:99.

Rust, S. R. and F. N. Owens. 1982. Effect of intake and roughage level on digestion. Animal Science Research Report, MP-112, Oklahoma Ag. Exp. Sta.

Swift, R. W. and C. E. French. 1954. Energy Metabolism and Nutrition. Scarecrow Press. New Brunswick, N. J.

Tyrrell, H. F. and P. W. Moe. 1972. Net energy value for lactation of a high and low concentrate ration containing corn silage. J. Dairy Sci. 55:1106.

Vance, R. D., R. L. Preston, V. R. Cahill and E. W. Klosterman. 1972. Net energy evaluation of cattle-finishing rations containing varying proportions of corn grain and corn silage. J. Anim. Sci. 34:851.

Table 1. The digestibility of organic matter, cellulose and crude fiber in soybean flakes and timothy when fed separately or in combination.[1]

	Diet		
Digestibility %	Soybean flakes	Soybean flakes:timothy[2]	Timothy
Organic matter	70.3	70.8	55.9
Cellulose	74.1	77.9	53.1
Crude fiber	72.1	74.5	50.7

[1]From Johnson et al. (1962).

[2]60:40 combination.

Table 2. The TDN and digestibility of dry matter and crude fiber in soybean flakes, soybean hulls and hay when fed separately or in equal combinations[1].

	Diets				
Item	Soybean flakes	Soybean hulls	Soybean flakes & hay	Soybean hulls and hay	Hay
TDN	66.7[b]	60.7[c]	70.8[a]	69.2[ab]	48.8
DM digestibility (%)	75.8[a]	69.3[c]	80.0[a]	78.5[ab]	56.3
Crude fiber digestibility (%)	75.5[a]	66.3[b]	80.7[a]	80.5[a]	49.8

[1]From Hintz et al. (1964).

[a,b,c]Means in the same row with uncommon superscripts differ ($P < .05$).

Table 3. Influence of forage to concentrate ratio on actual and calculated digestibilities of energy and crude protein.[1]

	Forage: concentrate					
	75:25		50:50		25:75	
Digestibility %	actual	calculated	actual	calculated	actual	calculated
Energy	48.96	49.76	57.66	59.14	66.14[a]	68.36[b]
Crude protein	62.2[a]	56.2[b]	72.2[a]	65.0[b]	76.3[a]	73.3[b]

[1]From Nelson et al. (1968).

[a,b]Means in the same row, for a given forage: concentrate ratio, with uncommon superscripts differ ($P < .05$.)

Table 4. Alfalfa hay and beet pulp digestion coefficients determined directly versus those calculated by difference.[1]

Item	Alfalfa		Beet pulp	
	Observed	Calculated	Observed	Calculated
Dry matter	59[a]	61[b]	86[a]	88[b]
Energy	58[a]	60[b]	85	87
Ether extract	28	15	-57	-96
Crude protein	69[a]	71[b]	63	66
Crude fiber	52[a]	54[b]	84	87
NFE	66[a]	71[b]	94[a]	96[b]

[1]From Asplund and Harris (1971).

[a,b]Means, for a given feed, in the same row with uncommon superscripts differ ($P < .05$).

Table 5. Associative effects between corn and corn silage on dry matter and energy digestibility.[1]

Item	Corn silage	1/3 corn	2/3 corn	corn
DM digestibility, measured	67.65	68.34	70.78	79.19
Predicted	-	71.64	75.39	-
% decrease	-	4.83	6.24	-
Digestible energy, measured	2.96	2.98	3.04	3.44
Predicted	-	3.13	3.28	-
% decrease	-	4.79	7.40	-

[1]From Byers et al. (1976).

Table 6. Expected and observed digestibilities of mixed corn silage corn grain diets.[1]

Item	Expected	Observed	Depression
Dry matter	78.6	69.8	11.2
Energy	78.2	68.6	12.2
Starch	97.0	87.4	10.0
Crude protein	69.7	61.4	11.9
Cell wall	55.8	45.0	19.9

[1]From Joanning et al. (1981).

Table 7. Methane production, heat increment and energy values of single ingredient or mixed diets.[1]

Diet	Methane (g/kg)	Metabolizable energy (kcal/kg)	Heat increment (kcal/kg)	Net energy (kcal/kg)
Alfalfa	23.6	1.94	.67	1.27
Alfalfa-corn	30.4	2.57	.37	2.21
Corn	23.0	3.34 (3.26)[2]	.53 (.14)[2]	2.80 (3.13)[2]

[1]From Forbes et al. (1931)
[2]Values for corn in parentheses were calculated from the mixed diet.

Table 8. Influence of forage source on utilization of feed energy.[1]

Forage	DM intake (kg) Total	DM intake (kg) Corn	Metabolizable energy (kcal/kg) Total	Metabolizable energy (kcal/kg) Corn	Heat increment (kcal/kg corn)	Net energy (kcal/kg corn added
Oat straw	4.9	1.1	11.90	3.36	.88	2.49
Timothy hay	4.9	1.1	11.77	3.06	1.30	1.75
Alfalfa hay	5.7	1.2	13.70	2.99	1.01	2.02

[1]From Forbes et al. (1933).

Table 9. Energy values of corn and corn silage diets.[1]

Item	Diet: Corn Silage	1/3 corn	2/3 corn	Corn
ME, Kcal/g				
measured	2.53	2.55(7.4)[2]	2.61 (12.1)[2]	3.18
predicted	-	2.75	2.79	-
NE_m, Kcal/g				
measured	1.69	1.84 (4.7)[2]	1.84 (4.8)[2]	2.39
predicted	--	1.93	2.16	-
NE_g, Kcal/g				
measured	1.24	1.38 (10.4)[2]	1.60 (12.3)[2]	2.11
predicted	--	1.54	1.82	--

[1]From Byers et al. (1976).
[2]Indicates the % depression from the predicted value

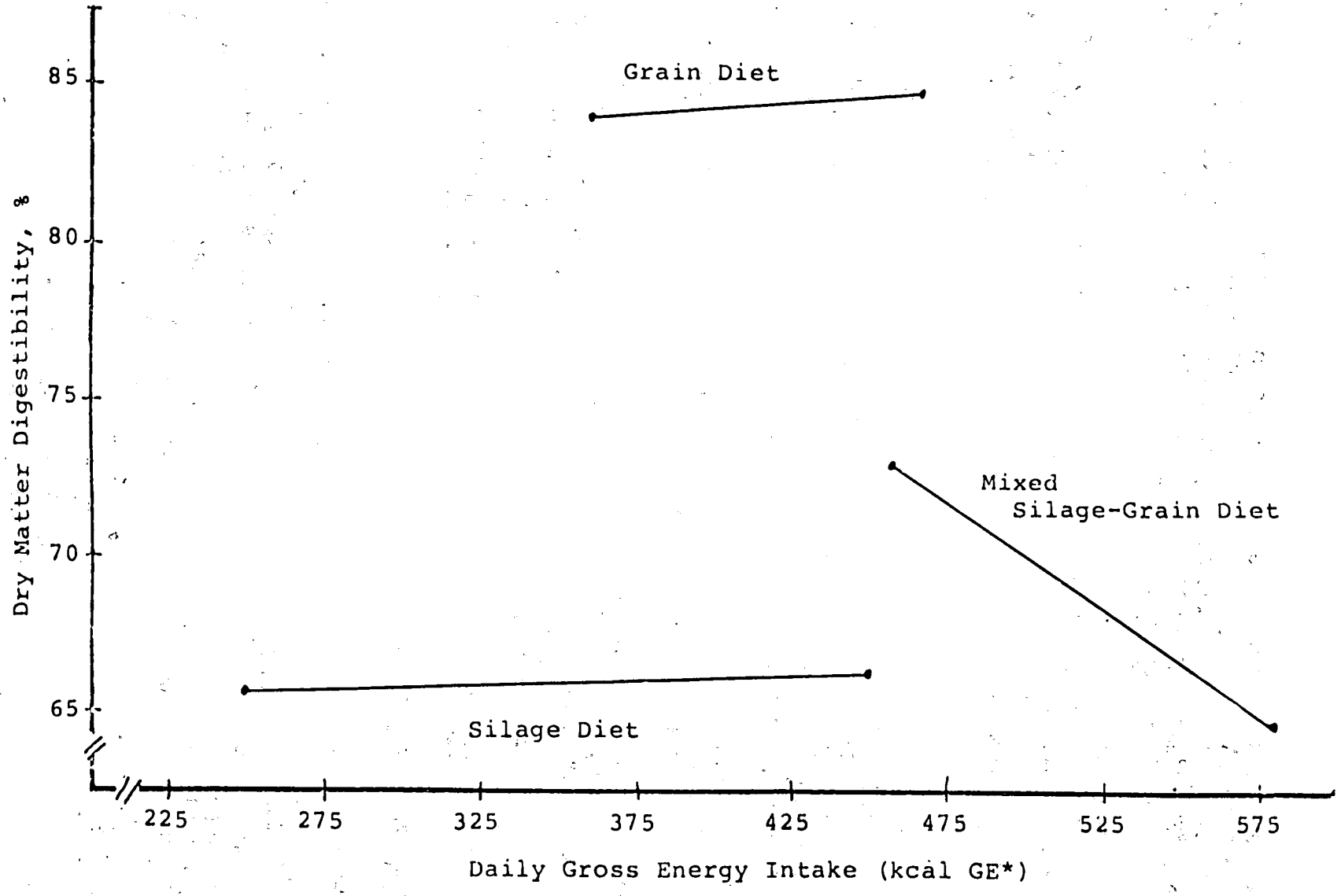

FIGURE 1. LEVEL OF INTAKE EFFECT ON DRY MATTER DIGESTIBILITY.

METHIONINE HYDROXY ANALOG FOR LACTATING DAIRY CATTLE

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INTRODUCTION

During the past 15 years, several experiments have been conducted on supplementation of dairy cattle diets with methionine hydroxy analog (M-analog). Although milk yield has been influenced in some trials, the most consistent response to M-analog has been an increase in milk fat percent and/or yield. However, some experiments have not shown any response to M-analog and for this reason, nutritionists have been hesitant to recommend the use of M-analog. The intent of this paper is to summarize work with M-analog including recent work at Minnesota and present practical feeding recommendations for its inclusion in dairy cattle diets.

SUMMARY OF LACTATION TRIALS

A summary of lactation trials conducted with M-analog is in Table 1. It is evident from these data that positive responses have not always occurred. With a few exceptions (13,18,32,33) increases in milk fat and/or fat-corrected milk have been observed in continuous lactation experiments where M-analog was fed during early lactation (2,3,4,8,15,16,20,21,25,29,34). This is the period when mobilization of body fat plays an important role in meeting nutritional demands for milk production. Most experiments conducted during mid-lactation or switchback and Latin square design experiments have not shown positive results for milk production or composition, indicating that the period of lactation when M-analog is fed is a factor in its efficacy. Carry over effects of M-analog between experimental periods may be a problem in switchback and Latin square design experiments.

Optimum amounts of M-analog appear to be 25 to 35 g/cow/day or approximately .25 to .3% of the concentrate mix (air dry basis). Bhargava et al. (2) included M-analog at either .1, .2, or .3% of the concentrate fed and observed the greatest increase in milk fat at .3%, or about 26 g/day. Intakes in excess of 45 g/day of M-analog have been reported to decrease total feed intakes (25). This is probably because the objectionable odor of M-analog may reduce palatability.

A 2 to 3 week prepartum adjustment period to M-analog has been employed in most continuous lactation experiments, although the data do not indicate whether or not this is necessary. This practice may not be practical in on-farm usage. Unpublished data from Minnesota indicated no differences in milk fat response when M-analog was included in diets beginning either 2 weeks prepartum or 2 weeks postpartum.

Cows fed high energy (high concentrate) diets seem to respond most to M-analog (2,12,16,20,21,25). High concentrate diets may tend to depress milk fat test. Rowe et al. (28) observed increases in milk fat percent when cows were slightly fat depressed by low (10% ADF) fiber diets, but M-analog had no effect when milk fat tests were depressed greater than 20% below normal.

Chandler et al. (8) reported a greater response in milk fat production when M-analog was fed in low protein (12.5% of DM) contrasted to higher protein (15.5%) diets. A protein sparing effect could be expected if M-analog were simply supplying a limiting amino acid for tissue protein synthesis. However, a lack of response to dietary protein in this study (8) together with the fact that milk fat was the only production parameter which increased indicated that other mechanisms were involved. Jenny et al. (20) reported significant increases in milk, fat-corrected milk, and milk fat production with inclusion of M-analog in both 13.5 and 15.5% crude protein diets.

Methionine

Relatively few experiments have been conducted with the amino acid methionine in lactating cow diets. Remond et al. (26) fed 50 g of DL-methionine beginning 2 weeks prepartum and continuing through 5 weeks postpartum and observed a significant increase in milk fat production. This supports the contention that M-analog elicits its effect via methionine. No differences in production were observed by Williams et al. (36) when 12 g/day of an encapsulated methionine which by-passed the rumen was fed, suggesting the response to methionine may be ruminal. However, milk fat percent did increase approximately 10% ($P < .05$) with intravenous injections of 8 g/day of L-methionine (7). Milk protein increased significantly in an experiment conducted by Fisher (11) when 11.2 and 24.8 g DL-methionine were administered intravenously. In an experiment conducted at Minnesota, M-analog or DL-methionine was fed at .1, .2, and .3% of the concentrate to cows in early lactation. While no differences in production were observed between M-analog and D,L methionine supplemented cows, both groups had higher fat tests than controls at each level of supplementation. Highest fat percentages were obtained with .3% M-analog (3.73) and with .2% DL-methionine (3.72) compared to 3.34% fat for unsupplemented cows.

MECHANISMS OF ACTION

As evidenced from the previous discussion, the mode of action of M-analog is not understood. Belasco (1) has reported greater resistance to microbial degradation in the rumen for M-analog than for methionine and biotransformation of the analog to methionine in ruminant tissues. Emery (10) administered M-analog and DL-methionine through a rumen fistula and reported similar disappearance rates between 2 and 4 hours postadministration. Patton et al. (24) reported increases in protozoal numbers in the rumen of cows fed M-analog. The high lipid content of protozoa (17) may be related to increases in milk fat production. Changes in rumen volatile fatty acids have been observed with M-analog. Rosser et al. (27) reported increased ruminal acetate and butyrate and decreased amounts of propionate with M-analog supplementation. Rowe et al. (28) observed an increase in the ruminal acetate:propionate ratio from 2.53 to 2.86 ($P < .05$) when M-analog was fed in low fiber diets.

Altered lipid metabolism via changes in serum lipoprotein fractions has been observed with M-analog supplementation (22,25). Methionine is involved in the assembly of lipid and protein moieties in lipoprotein synthesis (31). The question of whether the M-analog effect is simply as a source of dietary sulfur

was addressed by Gil and Shirley (14) who found that M-analog and sulfur containing amino acids stimulated growth of rumen bacteria while inorganic sulfur additions did not. Increases in milk fat production have been observed with M-analog supplementation when all diets were either isosulfur or were at or above NRC sulfur requirements (2,5,8,16,34).

MINNESOTA RESEARCH

Work at Minnesota has concentrated on defining practical conditions under which M-analog feeding would be most beneficial. Bhargava et al. (2) showed that .2 to .3% M-analog in the concentrate elicited optimal response from cows fed diets of 60% concentrate, but moderate in protein content. Subsequently, three experiments were designed and conducted in consecutive years to study the effects of dietary energy and protein on production responses to M-analog.

Trial 1 was designed to determine the effects of M-analog supplementation at two dietary protein percentages. Forty-five lactating Holsteins were fed either 13.2 or 15.8% crude protein diets (DM basis). The dietary dry matter consisted of approximately 40% concentrate and 60% forage (corn silage, alfalfa hay). M-analog was fed at either 0 or .25% of the concentrate (air dry basis) beginning 2 weeks prepartum and continuing through 16 weeks postpartum. All diets met NRC requirements for sulfur. Milk production and composition were not significantly influenced by M-analog at either dietary protein percentage. However, milk fat percentage was slightly higher overall for cows fed M-analog compared to controls (3.65 vs 3.44%).

The objective of Trial 2 was to study the interaction of concentrate level on M-analog response in low and high protein diets. Concentrate was maintained at either 40 or 60% of ration dry matter and dietary protein was either 13.5 or 17.3%. M-analog supplementation, concentrate composition and forages fed were similar to those in Trial 1.

M-analog had no effect on milk production or composition when fed with the 40% concentrate diets (Table 2). However, when the 60% concentrate diets were supplemented with M-analog, milk fat percent increased significantly when compared to the unsupplemented diet (3.42 vs 3.66%). Milk fat yield and fat corrected milk increased by .07 and 1.1 kg/day, respectively for cows fed M-analog in the 60% concentrate diets compared to controls. No significant M-analog by protein interaction was found, although the increase in fat test was higher for cows fed low protein. Milk fat percentages of cows on low protein, high concentrate were 3.46 for controls versus 3.88 with M-analog, whereas fat tests of cows fed high protein, high concentrate diets were 3.39 and 3.45 for control and M-analog treatments, respectively. Analysis of rumen fluid revealed that on the high concentrate diets, cows fed M-analog had significantly higher molar percentages of acetate and lower propionate than controls, which may be related to the increase in milk fat test. Blood β-hydroxybutyrate levels were essentially the same for all cows at 2 weeks prepartum, however at 2 weeks postpartum this blood constituent was significantly higher for cows fed M-analog and remained slightly higher at 4 and 6 weeks postpartum. Since ruminal butyrate was not affected by M-analog, the increase in plasma β-hydroxybutyrate may have resulted from increased mobilization of body fat and its subsequent conversion to ketones.

Because optimum response to M-analog was achieved with the high concentrate diet in Trial 2, concentrate was maintained at 60% of ration dry matter in Trial 3 to further investigate the response to M-analog at varying dietary protein levels (13.0, 15.0, and 17.5% of ration DM). Composition of the concentrate and forage and M-analog supplementation were similar to the first two experiments.

Milk fat increased ($P < .05$) an average of .26 percentage units with M-analog supplementation (Table 3). This is similar to the response observed on the 60% concentrate diet in Trial 2. The higher fat test was consistent at each dietary protein percentage. Milk fat percentages for cows fed control and M-analog diets are shown in Figure 1 through 28 weeks postpartum. M-analog was fed through 16 weeks only, however fat percentages of these cows remained higher after supplementation was discontinued. Since milk fat was similar at the start of the experiment for control and M-analog cows, a carry over effect of M-analog may be involved. Lowest fat tests during the treatment period occurred during week 12. Cows fed control diets tested 3.16% during this period, while those fed M-analog maintained a fat test of 3.39. Milk fat and fat-corrected milk yields averaged .07 and 1.1 kg/day higher for cows fed M-analog. No differences in dry matter intake, milk yield, SNF, or milk protein were observed.

Combined Lactation Study

The results from Trials 1, 2, and 3 indicate that a response to M-analog supplementation can be expected from cows fed high concentrate diets, regardless of dietary protein level. Because cow numbers per treatment in the individual experiments were small, data from 179 lactations over a period of 6 years were combined and analyzed for effects of M-analog on milk production and composition. These data were from 2 published trials by Bhargava et al. (2), Trials 2 and 3 reported in this paper, and 2 unpublished experiments conducted at Minnesota. All animals used were fed similar diets of approximately 60% concentrate, 40% forage in which M-analog was the dietary variable, fed at either 0 or .25 to .3% of the concentrate from 2 weeks prepartum through 16 weeks postpartum. Dietary protein varied from 13 to 17.5% of dry matter among the 6 trials, but amount of protein fed was balanced for cows fed M-analog and control diets within trials.

Results are presented in Table 4. Approximately one-third of the animals represented were in their first lactation. No M-analog by age interactions occurred for any of the parameters measured, so results from 2-year olds and older cows are combined in the table. Milk yields were similar for cows fed M-analog and control diets. Milk fat, however, averaged .35 percentage units higher ($P = .0001$) when M-analog was included in the diets. Daily milk fat production was .09 kg/day higher ($P = .0005$) and 4% fat-corrected milk averaged 1.1 kg/day higher ($P = .04$) for cows fed M-analog compared to controls. Solids-not-fat percentage was higher ($P = .04$) but yields of SNF were similar on M-analog and control diets. Milk protein was not influenced by M-analog.

SUMMARY AND RECOMMENDATIONS

If M-analog is to be recommended for use in a commercial dairy operation, supplementation must be profitable. Utilizing the results obtained from the

combined lactation study and a milk price of $13.00/cwt with a .17 butterfat differential, milk from cows fed M-analog was worth 60¢/cwt more than that of the control cows. Supplementation with 30 g/cow/day of M-analog costs about 10¢/cow/day at current prices. Cows fed M-analog in this study showed a net profit of about 19¢/day or approximately $21.25/cow more than controls during the first 16 weeks of lactation. It must be emphasized that this is under conditions in which the maximum milk fat response to M-analog occurred.

A milk fat response to M-analog supplementation cannot always be expected as evidenced by the results of the experiments summarized in Table 1. Care must be taken that M-analog is not used as a "cure-all" for fat test problems. However, M-analog appears to be beneficial for improving milk fat production of lactating dairy cattle fed high grain diets (50 to 60% of DM) during early lactation. Efficacy does not appear to be limited by ranges in dietary protein normally fed to dairy cattle. M-analog can be included at .25 to .3% of the concentrate or about 25 to 35 g/cow/day. It should be well mixed with other ration components to obtain a maximum response without reducing ration palatability. Future research should be concerned with establishing the mode of action of the M-analog response. A better understanding of the biochemical mechanisms involved would further aid in recommending situations for M-analog use.

Table 1. Effects of methionine hydroxy analog on milk and milk fat production.

Reference	No. of cows	Amount of M-analog	Actual milk	4% FCM	Milk fat	Milk fat	Experimental design	Type of diet
			(kg/day)	(kg/day)	(kg/day)	(%)		
Griel, 1968 (15)	42	0		27.6[a]			Continuous lactation, -21 to 56 days postpartum	Haylage, corn silage, grass hay
		40 g/cow/day		29.6[b]				
		80 g/cow/day		28.1[a]				
Polan, 1970 (25)	24	0		31.1		3.76[a]	Continuous lactation, -14 to 90 days postpartum	50:50 concentrate: corn silage
		.2% of grain		34.7		4.16[b]		
		.4% " "		34.1		4.30[c]		
		.8% " "		29.9		4.61[d]		
Burgos, 1970 (6)	16	0	15.7	14.7	.55	3.72	Switchback, 4-wk periods	Corn silage, hay
		40 g/cow/day	15.4	14.2	.57	3.76		
Hutjens, 1971 (19)	12	0	18.3		.50	2.50	3x3 Latin square, 5-wk periods	3:1 concentrate: hay
		.22% of grain	17.9		.40	2.40		
	12	0	18.4		.70	3.60	3x3 Latin square, 5-wk periods	2:1 concentrate: hay
		.22% of grain	18.4		.70	3.60		
Rosser, 1971 (27)	16	0	18.3	15.8			4-wk period during midlactation	85% concentrate, 15% hay
		40 g/cow/day	19.3	19.4				
	6	0	27.6[b]	25.0	.81	3.00	Latin square	85% concentrate, 15% hay
		40 g/cow/day	25.1[a]	22.8	.80	3.00		
Bishop, 1971 (3)	185	0					Continuous lactation, 0 to 90 or 120 days postpartum	
		30-40g/cow/day	+374*	+463*	+21*			

[a,b,c,d]Means within experiments differ ($P < .05$).

*Difference in year to year deviation of treated and control ME production ($P < .05$).

Table 1. Effects of methionine hydroxy analog on milk and milk fat production.

Reference	No. of cows	Amount of M-analog	Actual milk (kg/day)	4% FCM (kg/day)	Milk fat (kg/day)	Milk fat (%)	Experimental design	Type of diet
Holter, 1972 (16)	14	0	27.0	23.6[a]	.85[a]	3.10[a]	Continuous lactation, -14 to 168 days postpartum	55 to 65% concentrate, alfalfa hay, corn silage
		.36% of grain	26.4	24.4[b]	.92[b]	3.50[b]		
Whiting, 1972 (35)	12	0	25.2			2.88	4-wk periods, 1-wk adjustments, mid-lactation	45% concentrate, 55% cubed alfalfa
		40 g/cow/day	26.1			2.78		
		80 g/cow/day	25.2			3.00		
Bishop, 1972 (4)	85	0					Continuous lactation 305 days	Milo, barley, alfalfa hay
		.22% of grain	+554*	+554*	+22*			
Fosgate, 1973 (12)	10	0	20.1			[a]	12-wk double reversal	65% corn, 35% cottonseed hulls and alfalfa pellets
		25 g/cow/day	21.2			+.26 units[b]		
Bouchard, 1973 (5)	4	0	22.1			3.40	Split plot 4x4 Latin square	Corn, corn silage
		25 g/cow/day	22.4			3.70		
Olson, 1974 (23)	36	0	17.4	15.8	.59	3.42	Switch-back, 5-wk periods, mid-lactation	Corn, corn silage, hay
		16.6g/cow/day	17.6	16.0	.59	3.43		
		23.2g/cow/day	17.8	16.2	.60	3.42		
		27.9g/cow/day	17.7	16.1	.60	3.44		
Fuquay, 1974 (13)	55	0	22.4			4.78	Continuous lactation, 0 to 90 days	Pasture, hay
		25 g/cow/day	23.6			4.63		

[a,b]Means within experiments differ ($P < .05$).
*Difference in year to year deviation of treated and control ME production ($P < .05$).

Table 1. Effects of methionine hydroxy analog on milk and milk fat production.

Reference	No. of cows	Amount of M-analog	Actual milk (kg/day)	4% FCM (kg/day)	Milk fat (kg/day)	Milk fat (%)	Experimental design	Type of diet
Van Horn, 1975 (32)	36	0	18.0			3.92	Continuous lactation, 0 to 84 days	Corn, sugarcane bagasse, citrus pulp
		25 g/cow/day	18.1			3.84		
Wallenius, 1975 (34)	24	0	32.5			3.20	Continuous lactation, 13 to 139 days	48% concentrate, 42% alfalfa hay, 10% alfalfa-grass silage
		25 g/cow/day	30.4			3.30		
Hutjens, 1975 (18)	42	0	18.3		.86		Continuous lactation -14 to 305 days	
		25 g/cow/day	17.1		.83			
	87	0	23.0		.91		Continuous lactation -14 to 305 days	
		25 g/cow/day	22.9		.87			
Chandler, 1976 (8)	144	0	23.5		.84	3.61^{a}	Continuous complete lactation	Varied for location 12.5 or 15.5% CP
		.125% of DM	23.1		.86	3.73^{b}		
Stanley, 1977 (29)	503	0	25.5^{a}	21.4^{a}	$.74^{a}$	3.00^{a}	Continuous lactation, 0 to 110 days	
		30 g/cow/day	26.8^{b}	23.0^{b}	$.82^{b}$	3.11^{b}		
Bhargava, 1977 (2)	47	0	26.3	23.4	$.86^{a}$	3.30^{a}	Continuous lactation -14 to 116 days	60% concentrate, 40% corn silage + alfalfa hay
		.1% of grain	24.7	23.2	$.89^{ab}$	3.57^{ab}		
		.2% of grain	25.8	24.1	$.92^{ab}$	3.56^{ab}		
		.3% of grain	26.8	26.2	1.03^{b}	3.87^{b}		
		0	26.2	23.4^{a}	$.87^{ab}$	3.29^{a}	Continuous lactation -14 to 116 days	
		.1% of grain	28.2	25.8^{ab}	$.97^{ab}$	3.45^{ab}		
		.2% of grain	27.3	25.8^{ab}	$.99^{ab}$	3.62^{ab}		
		.3% of grain	26.8	26.3^{b}	1.04^{b}	3.83^{b}		

^{a,b}Means within experiments with different superscripts differ ($P < .05$).

Table 1. Effects of methionine hydroxy analog on milk and milk fat production.

Reference	No. of cows	Amount of M-analog	Actual milk (kg/day)	4% FCM (kg/day)	Milk fat (kg/day)	Milk fat (%)	Experimental design	Type of diet
Clay, 1979 (9)	104	0 (10.5% CP)	28.3			3.41	Continuous lactation 15 to 84 days postpartum	50% grain, 35% corn silage, 15% hay
		.22% of grain	28.8			3.60		
		0 (12.5% CP)	32.4			3.42		
		.22% of grain	31.3			3.42		
		0 (14.5% CP)	32.7			3.40		
		.22% of grain	34.3			3.48		
		0 (16.5% CP)	34.8			3.31		
		.22% of grain	33.7			3.55		
Vandersall, 1980 (33)			Yr 1 Yr 2		Yr 1 Yr 2			
	28	0	28.8 26.4		1.07 1.02		Continuous lactation, -14 to 112 days	2:1 concentrate: corn silage + 10% alfalfa hay
		.1% of grain	29.5 29.5		1.10 1.16			
		.2% of grain	26.2 30.9		.95 1.12			
		.3% of grain	29.2 30.5		1.08 1.14			
Jenny, 1980 (20)	24	0 (13.5% CP)	27.5^{a}	24.3^{a}	$.90^{a}$		Continuous lactation, -14 to 112 days	60% concentrate, 40% forage
		.25% of grain	29.8^{b}	28.7^{b}	1.12^{b}			
		0 (15.5% CP)	24.4^{a}	22.5^{a}	$.85^{a}$			
		.25% of grain	28.3^{b}	26.3^{b}	1.00^{b}			
Stokes, 1981 (30)	24	0 (low protein)	28.1	24.7	.89	3.29	Incomplete block switchback, mean 40 days postpartum	Corn, corn silage, hay
		25 g/cow/day (low protein)	28.3	25.3	.93	3.42		
		0 (high protein)	30.1	26.7	.98	3.31		
		25 g/cow/day (high protein)	29.6	26.4	.97	3.27		

^{a,b}Means within experiments with different superscripts differ ($P < .05$).

Table 1. Effects of methionine hydroxy analog on milk and milk fat production.

Reference	No. of cows	Amount of M-analog	Actual milk	4% FCM	Milk fat	Milk fat	Experimental design	Type of diet
			(kg/day)	(kg/day)	(kg/day)	(%)		
Jenny, 1982 (21)	24	0	25.1	21.4	.75		Continuous lactation, -14 to 112 days	60% concentrate, 40% forage
		.25% of grain (solid)	27.1	24.2	.89			
		.25% of grain (liquid)	28.3	24.8	.90			
Rowe, 1982 (28)	28	0				2.96[a]	Double reversal	Low fiber (10% ADF)
		34 g/cow/day				3.14[b]		

[a,b] Means within experiments differ ($P < .05$).

Table 2. Effects of M-analog on intake, milk production, and milk composition (Trial 2).

Item	Concentrate, %	40		60	
	M-analog, %	0	.25	0	.25
Dry matter intake (kg/day)		16.9	17.3	18.1	17.8
Milk (kg/day)		24.7[a]	24.0[a]	27.9[b]	28.0[b]
Milk fat (%)		3.76[b]	3.68[b]	3.42[a]	3.66[b]
Milk fat (kg/day)		.92[ab]	.88[a]	.95[ab]	1.02[b]
4% FCM (kg/day)		23.7[a]	22.8[a]	25.4[ab]	26.5[b]
SNF (%)		8.48	8.32	8.40	8.51
SNF (kg/day)		2.09[a]	2.00[a]	2.34[b]	2.37[b]
Milk protein (%)		3.28	3.23	3.25	3.32
Milk protein (kg/day)		.81[a]	.77[a]	.90[b]	.92[b]

[a,b]Means within rows with different superscripts are different ($P < .05$).

Table 3. Effect of M-analog on milk fat percent at three dietary crude protein percentages (Trial 3)

Item	M-analog (%)	Dietary protein (%) 13	15	17.5	Mean
Milk fat (%)	.00	3.42	3.33	3.53	3.43[a]
	.25	3.71	3.59	3.75	3.69[b]

[a,b]M-analog means differ ($P < .05$).

Table 4. Influence of M-analog on milk production and milk composition (Combined data from six lactation studies)

Item	M-analog (%) 0	.25-.30	SE	P
Milk (kg/day)	26.5(92)[a]	26.3(87)	.47	.70
Milk fat (%)	3.36(92)	3.71(87)	.05	.0001
Milk fat (kg/day)	.88(92)	.97(87)	.02	.0005
4% FCM (kg/day)	23.9 (92)	25.0 (87)	.43	.04
SNF (%)	8.65(92)	8.74(87)	.03	.04
SNF (kg/day)	2.29(92)	2.29(87)	.04	.92
Milk protein (%)	3.22(68)	3.25(64)	.03	.40
Milk protein (kg/day)	.87(68)	.87(64)	.02	.83

[a]Number of animals.

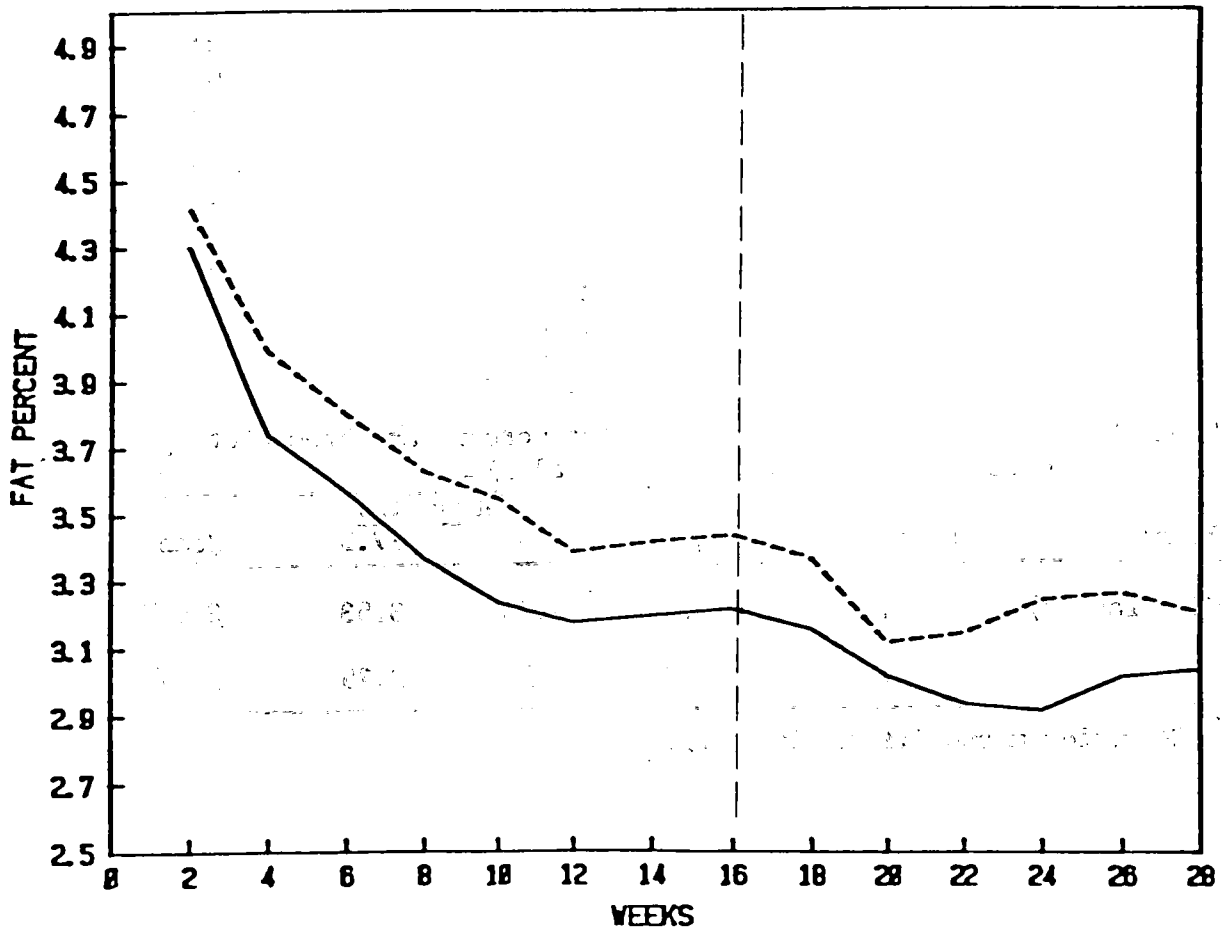

Figure 1. Influence of M-analog on milk fat percent, Trial 3. 0% M-analog———; .25% M-analog ---.

LITERATURE CITED

1. Belasco, I. J. 1980. Fate of carbon-14 labeled methionine hydroxy analog and methionine in the lactating dairy cow. J. Dairy Sci. 63:775.

2. Bhargava, P. K., D. E. Otterby, J. M. Murphy, and J. D. Donker. 1977. Methionine hydroxy analog in diets for lactating cows. J. Dairy Sci. 60:1594.

3. Bishop, R. B. 1971. Effect of methionine hydroxy analog on complete lactation of dairy cows. J. Dairy Sci. 54:1240. (Abstr.)

4. Bishop, R. B., and W. D. Murphy, Jr. 1972. Effect of continuous methionine hydroxy analog supplementation on complete rations. J. Dairy Sci. 55:711 (Abstr.)

5. Bouchard, R., and H. R. Conrad. 1973. Supplementary value of hydroxy analog of methionine and sulfates in diets of lactating cows. J. Dairy Sci. 56:665. (Abstr.)

6. Burgos, A., and H. H. Olson. 1970. Effects of 40 g of methionine hydroxy analog on yield and composition of milk. J. Dairy Sci. 53:647 (Abstr.)

7. Chamberlain, D. G., and P. C. Thomas. 1982. Effect of intravenous supplements of L-methionine on milk yield and composition in cows given silage-cereal diets. J. Dairy Res. 49:25.

8. Chandler, P. T., C. A. Brown, R. P. Johnston, Jr., G. K. Macleod, R. D. McCarthy, B. R. Moss, A. H. Rakes, and L. D. Satter. 1976. Protein and methionine hydroxy analog for lactating cows. J. Dairy Sci. 59:1897.

9. Clay, A. B., and L. D. Satter. 1979. Milk production response to dietary protein and methionine hydroxy analog. J. Dairy Sci. 62:75 Suppl. 1 (Abstr.)

10. Emery, R. S. 1971. Disappearance of methionine from the rumen. J. Dairy Sci. 54:1090.

11. Fisher, L. J. 1972. Response of lactating cows to the intravenous infusion of amino acids. Can. J. Anim. Sci. 52:377.

12. Fosgate, O. T., C. M. Clifton, J. Z. Aframe, and P. R. Fowler. 1973. Addition of methionine hydroxy analog to high urea all-in-one rations. J. Dairy Sci. 56:307. (Abstr.)

13. Fuquay, J. W., W. H. McGee, and E. W. Caster. 1974. Methionine hydroxy analog during early lactation in a commercial Guernsey herd making maximum use of annual grazing crops. J. Dairy Sci. 57:132. (Abstr.)

14. Gil, A., and R. L. Shirley. Effect of methionine hydroxy analogue-Ca (MHA) on rumen bacteria, in vitro. J. Anim. Sci. 34:359.

15. Griel, L. C., Jr., R. A. Patton, R. D. McCarthy, and P. T. Chandler. 1968. Milk production response to feeding methionine hydroxy analog to lactating dairy cows. J. Dairy Sci. 51:1866.

16. Holter, J. B., C. W. Kim, and N. F. Colovos. 1972. Methionine hydroxy analog for lactating dairy cows. J. Dairy Sci. 55:460.

17. Hungate, R. E. 1966. The rumen and its microbes. p. 146-390. Academic Press, NY.

18. Hutjens, M. F., and K. M. Nold. 1975. Two-year field study of methionine analog supplementation. J. Dairy Sci. 58:779. (Abstr.)

19. Hutjens, M. F., and L. H. Schultz. 1971. Addition of soybeans or methionine analog to high-concentrate rations for dairy cows. J. Dairy Sci. 54:1637.

20. Jenny, B. F., H. J. van Dijk, L. W. Grimes, and G. D. O'Dell. 1980. Effect of methionine hydroxy analog and two levels of protein in complete feeds fed to cows in early lactation. J. Dairy Sci. 63:182. Suppl. 1 (Abstr.)

21. Jenny, B. F., H. J. van Dijk, L. W. Grimes, and G. D. O'Dell. 1982. Effect of methionine as the hydroxy analog or hydroxy acid in complete feeds fed to cows in early lactation. J. Dairy Sci. 65:217. Suppl. 1 (Abstr.)

22. McCarthy, R. D., G. A. Porter, and L. C. Griel, Jr. 1968. Bovine ketosis and depressed fat test in milk: A problem of methionine metabolism and serum lipoprotein aberration. J. Dairy Sci. 51:459.

23. Olson, H. H., and W. R. Grunbaugh. 1974. Effect of methionine hydroxy analog feeding on yield and composition of bovine milk. J. Dairy Sci. 57:695.

24. Patton, R. A., R. D. McCarthy, L. G. Keske, L. C. Griel, Jr., and B. R. Baumgardt. 1970. Effect of feeding methionine hydroxy analog on the concentration of protozoa in the rumen of sheep. J. Dairy Sci. 53:933.

25. Polan, C. E., P. T. Chandler, and C. N. Miller. 1970. Methionine hydroxy analog: Varying levels for lactating cows. J. Dairy Sci. 53:607.

26. Remond, B., C. Champredon, C. Decaen, R. Pion, and M. Journet. 1971. Effect of a supplement of DL-methionine for cows at the start of lactation on production of milk and composition of blood. Ann. Biol. Anim. Biochim. Biophys. 11:455.

27. Rosser, R. A., C. E. Polan, P. T. Chandler, and T. L. Bibb. 1971. Effects of whey components and methionine analog on bovine milk fat production. J. Dairy Sci. 54:1807.

28. Rowe, S. A., A. H. Rakes, W. J. Croom, Jr., A. C. Linnerud, and J. H. Britt. 1982. Effects of methionine hydroxy analog on milk production and ruminal and blood parameters in dairy cows fed low-fiber diets. J. Dairy Sci. 65:118. Suppl. 1 (Abstr.)

29. Stanley, R. W., and W. Toma. 1977. Methionine supplementation for lactating dairy cattle. J. Dairy Sci. 60:70. Suppl. 1 (Abstr.)

30. Stokes, M. R., J. H. Clark, and L. M. Steinmetz. 1981. Performance of lactating dairy cows fed methionine or methionine analog at two concentrations of dietary crude protein. J. Dairy Sci. 64:1686.

31. Trams, E. G., E. A. Brown, and C. J. Lauter. 1966. Lipoprotein synthesis. I. Rat plasma lipoprotein composition and synthesis from radioactive precursors. Lipids. 1:309.

32. Van Horn, H. H., S. P. Marshall, C. J. Wilcox, P. F. Randel, and J. M. Wing. 1975. Complete rations for dairy cattle. III. Evaluation of protein percent and quality, and citrus pulp-corn substitutions. J. Dairy Sci. 58:1101.

33. Vandersall, J. H., E. Russek, and L. W. Douglass. 1980. The effect of graded levels of methionine hdyroxy analog on milk production, milk composition and feed intake of high producing dairy cows. J. Dairy Sci. 63:140. Suppl. 1 (Abstr.)

34. Wallenius, R. W., and R. E. Whitchurch. 1975. Methionine hydroxy analog or sulfate supplementation for high producing dairy cows. J. Dairy Sci. 58:1314.

35. Whiting, F. M., J. W. Stull, W. H. Brown, and B. L. Reid. 1972. Free amino acid ratios in rumen fluid, blood plasma, milk, and feces during methionine and methionine hydroxy analog supplementary feeding. J. Dairy Sci. 55:983.

36. Williams, L. R., F. A. Martz, and E. S. Hilderbrand. 1970. Feeding encapsulated methionine supplement to lactating cows. J. Dairy Sci. 53:1709.

NITROGEN REQUIREMENT AND METABOLISM IN FISH: A COMPARATIVE PERSPECTIVE

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Gross protein requirements have been determined for a number of coldwater and warmwater fish, generally in the freshwater environment (NRC, 1981; 1977). Results from studies with first feeding fry, fingerling, and yearling fish show that gross protein requirements are highest in fry and decrease as fish size increases. The minimum requirement for protein of the young growing and adult salmonid is reported to be 49 and 37 percent of the metabolizable energy (ME), respectively. This is considerably higher than the requirements of other animals as shown in Table 1. Beef cattle, dog and the rat need

Table 1. Protein requirement of different young and adult animals

Animal	Protein energy as a percent of dietary metabolizable energy (ME)	
	Young	Adult
Cat[1]	28	19
Mink[2]	38	27
Dog[3]	12	5
Rat[4]	13	5
Chicken[5]	25	17
Turkey[5]	40	17
Pheasant[5]	43	24
Pig[6]	30	16
Beef cattle[7]	12	6
Trout & salmon[8]	*49*	*37*

[1]NRC, 1978; [2]NRC, 1982; [3]NRC, 1974; [4]NRC, 1978; [5]NRC, 1977; [6]NRC, 1979; [7]NRC, 1976; [8]NRC, 1981.

considerably less protein than do the trout, mink, and cat. The growing trout needs about four times more protein than the growing rat while the sexually mature trout needs about eight times the protein of the adult rat. The requirement of the mink and cat is more in line with that of the trout. Examination of the data leads one to question whether the efficiency of utilization of nitrogen is lower

in trout than other animals -- especially the more mature or adult fish. This seems intuitively true since the near mature or adult animal is near nitrogen balance (neither gaining or losing nitrogen) and we know the fish requires more protein than other animals. However, fish readily use protein for energy as well as for tissue deposition since they are very efficient in eliminating nitrogenous wastes in the form of soluble ammonia compounds through the gill tissue directly into the water environment (Cowey and Sargent, 1972; Smith *et al.*, 1978). It has been shown that readily-digested high-protein materials have higher ME values for fish than is reported for other monogastric animals. Table 2 compares experimentally determined ME values for trout to published ME values for swine and chicken (Smith and Rumsey, 1976). Comparatively speaking, with the exception

Table 2. Metabolizable energy of some high protein feedstuffs for trout, chickens and swine.

	Metabolizable energy (kcal/kg)		
Feedstuff	Trout[a]	Chicken[b]	Swine[c]
Fish meal			
Herring	3900	3190	3090
Anchovy	3808	2640	2780
Dried fish solubles	3083	2970	2824
Soybean meal	3038	2530	3249
Casein	4500	4120	2984

[a](Smith and Rumsey, 1976).

[b](NRC, 1977).

[c](NRC, 1979).

of soybean meal in the case of swine, the ME values for fish are approximately 20-30% higher and protein appears to have more energy for fish than for mammals or birds. Work at our laboratory, employing direct calorimetry, show that less than 5% of the ME is lost as heat increment (HE) in fish (Table 3) (Smith *et al.*, 1978). This is not surprising when the differences in nitrogen waste excretion are considered. The HE of protein can be derived from three sources: 1) the cost of metabolizing the non-nitrogen part of the amino acids; the carbon and hydrogen atoms enter many of the same metabolic pathways as the atoms from carbohydrate and fat. The energy cost of this function could be expected to be similar to that for carbohydrate and fat metabolism. 2) The cost of synthesis of the excreted waste. 3) The energy cost of concentrating and excreting the waste products by the kidneys. Since the latter two functions either do not occur or require little energy in teleost (bony) fish, the HE associated

Table 3. Energy distribution of protein by ammonotelic, ureotelic, and uricotelic animals.*

Fraction	Excreted product (kcal/g)		
	Ammonia	Urea	Uric acid
Gross energy (IE)	5.7	5.7	5.7
Digestion loss (8%)	0.46	0.46	0.46
Digestible energy (DE)	5.24	5.24	5.24
Metabolic loss (UE and ZE)	0.72	0.86	1.31
Metabolizable energy (ME)	4.52	4.38	3.93
Heat increment (HE)			
Waste product synthesis	0.0	0.51	0.44
Waste product concentration and excretion	0.0	0.22	0.28
Metabolism of non-nitrogen	0.28	0.28	0.28
Total (HE)	0.28	1.01	1.01
Net energy (NE_g)	4.24	3.37	2.92

*Smith *et al.*, 1978.

with protein consumption can be expected to be similar to that of carbohydrate consumption. For comparative purposes in considering Table 3, digestion loss was assumed to be equal at 8% for the three types of animals. The metabolic loss reflects the differences in the heats of combustion of the excreted ammonia, urea, and uric acid. The theoretical cost of urea synthesis from 1 g of protein is 0.505 kcal (1 g protein x 0.16 g N/g protein x 1 mole N/14 g N x 88.4 kcal/mole urea x 1 mole urea/2 mole N = 0.505 kcal). Using similar calculations, the cost of uric acid synthesis is 0.44 kcal/g of protein. If the energy cost of metabolizing the nonprotein portion of the protein is considered equal in the three types of animals, the differences between the measured HE associated with dietary protein are due to the heat of synthesis of urea or uric acid plus the energy cost of concentration and excretion of these compounds in the homeotherms.

These observations make another interesting comparison possible when the energy/protein ratios are calculated from the various NRC publications (Table 4). Keeping in mind that ratios vary with age, size, and level of production, it can be shown that fish require a lower energy/protein ratio in the diet than other farm animals. Smith (1981) has shown that fish require less energy to metabolize protein than do other animals. He postulated that (sic) "what appears to be a high protein requirement is really a low requirement for energy" which is another way of expressing the earlier data on the high protein needs of fishes. Comparatively-speaking, it has been

Table 4. Energy/protein ratios (kcal/g protein) of different young and adult animals.

Animal	Ratio
Cattle[1]	23-40
Swine[2]	15-23
Chicken[3]	13-17
Trout and catfish[4]	8-11

[1]NRC, 1976; [2]NRC, 1979; [3]NRC, 1977; [4]NRC, 1977, 1981.

shown that fish are among the most efficient users of dietary energy of all animals (Smith *et al.*, 1978; Reid *et al.*, 1980). This superior efficiency is attributed to, in addition to their unique nitrogen metabolism, the low energy requirements for body temperature control, locomotion, and reproduction. Irrespective, it is worthwhile to examine the high protein requirement of fish and the possible reasons for same.

A summary of the essentiality of amino acids for fish is shown in Table 5. Cystine and taurine are listed as conditionally essential

Table 5. Essentiality of amino acids in salmonids.[1]

No synthesis (essential)	Conditional synthesis (sparing action)	Adequate synthesis (dispensable)
Arginine	Cystine	Alanine
Lysine	Taurine	Glutamine
Histidine	Tyrosine	Proline
Isoleucine		Serine/glycine
Methionine		Aspartic acid
Leucine		Glutamic acid
Valine		
Phenylalanine		
Threonine		
Tryptophan		

[1]Adapted from Ketola (1980).

even though in most studies, methionine has been able to satisfy the requirement for both. However, when methionine is deficient or marginally present, cystine appears to become limiting (Ogino, 1980; Rumsey, 1981) -- indicating a conditional need of methionine for the synthesis of cystine. While the function of cystine could include synthesis of taurine, more work is needed to determine if a specific

requirement for cystine (or taurine) exists. A summary of the amino requirements of salmonids and other animals is shown in Table 6. The values are expressed as a percent of the protein in order to compare species having different total protein needs.

Table 6. Comparative amino acid requirements, as percent of dietary protein.

				Salmonid		
Amino acid	Chicken[1]	Pig[2]	Rat[3]	Mertz[4]	Arai[5]	Ogino[6]
Arginine	6.1	1.5	1.0	6.0	5.1	3.5
Histidine	1.7	1.5	2.1	1.8	2.7	1.6
Isoleucine	2.2	4.4	4.6	2.2	3.4	2.4
Leucine	3.9	6.7	4.6	3.9	6.4	4.4
Lysine	5.0	6.1	4.7	5.0	7.4	5.3
Methionine	4.0	4.4	3.0	4.0	2.9	1.8
Phenylalanine	5.1	7.2	3.6	5.1	3.8	3.1
Threonine	2.2	3.3	3.0	2.2	4.4	3.4
Tryptophan	0.5	1.1	0.8	0.5	1.2	0.5
Valine	3.2	4.4	3.1	3.2	4.0	3.1

[1]NRC, 1977; [2]NRC, 1979; [3]NRC, 1978; [4]Mertz, 1972; [5]Arai, 1981; [6]Ogino, 1980.

The values of Mertz for salmonids are almost universally used, even though Ogino (1980) and Arai (1981) have reported more recent data. Although Arai's amino acid data are based on the carcass amino acid profile of coho salmon, it is worth noting that he reported excellent growth on a diet with 33% protein and 10% fat which was equal to or better than 40 and 47% protein diets. Ogino's work (1980) is noteworthy in that some of the essential amino acid requirements reported are much lower than previously reported (Mertz, 1972). The sum of the percentages of essential amino acids in the Ogino dietary recommendations would correspond to a highly digestible diet with 29% protein. These data are important, since they illustrate the importance of amino acid balance and how less protein appears to be necessary when the diet is well balanced.

All these animals require similar levels of amino acids (expressed as percent of protein), except for arginine. Fishes and chickens both require higher levels of arginine since they lack the "metabolic machinery" that produces arginine; however, the requirement for protein is low in the chicken but high in fish. It does not follow that an unusually high requirement for one or more amino acids could explain the high protein requirement of fish.

The other possible reason could be that the fish requires high dietary protein because of an unusually high requirement for nitrogen. Most animals can adapt to various levels of protein intake (Harper, 1965; Kaplan and Pitot, 1970). When an animal is fed a lower than normal level of protein, many tissue amino acid catabolic enzymes decrease, so that a lower proportion of the amino acids from the diet are lost. This is a protective-survival mechanism that allows the animal to conserve essential amino acids, and ultimately survive in spite of lower than normal protein intake. The opposite is true when an animal is fed a high protein diet -- i.e., the amino acid degrading enzymes increase. These enzyme adaptations facilitate disposal of excess nitrogen. Concurrent with these adaptations of amino acid catabolic enzymes, there are, in a number of animals, increases in gluconeogenic enzymes and decreases in glycolytic enzymes as dietary protein increases and dietary carbohydrate decreases. Some of these changes occur within hours whereas others take several days (Szepesi and Freedland, 1967; Das and Waterlow, 1974).

Until recently, few data have been reported on variation in diet and the effects on tissue enzyme activities in trout. However, recent work at the Tunison Laboratory and published information (Cowey *et al.*, 1981) allow some interesting insight. It is generally held that trout and salmon are "strict carnivores." It is also known that the trout has a protein requirement for both growth and maintenance that is higher than that of any other animal. It could be postulated that the fish's high protein requirement might be partly explained by the nature and extent to which its tissue enzymes adapt to gross dietary changes.

Indeed, there appears to be little difference in activities of enzymes involved in amino acid degradation of rainbow trout fed diets low or high in protein (Table 7). However, activity of the glucose degrading enzyme (pyruvate kinase) and glucose forming enzyme (fructose diphosphatase) did change significantly. Summarily, increased dietary protein had no effect on amino acid degrading enzymes, whereas the activity of enzymes that store glucose increased and that of enzymes that degrade glucose decreased (Cowey, 1981).

It is unusual to find that the enzymes involved in amino acid degradation do not change in fish that are shifted to high protein diets (Table 8). In contrast, enzyme activity in the rat, as in other omnivorous animals, increases consistently as the level of dietary protein is increased above normal, and decreases as the level of dietary protein is decreased below normal. Amino-acid-degrading enzyme activity in the rat increased from 175 to 1200% after a change from a low to a high protein diet, whereas the largest increase for the trout was only 25%. The cat is shown for comparison (Table 8) because it represents a "strict carnivore" in the mammalian world and, remarkably like the fish, cannot appreciably adjust its amino acid degradative enzyme activity. The ability of trout to exert metabolic control over glucose storage and degradation contrasts with their inability to control amino acid degradation. In this sense, the trout's regulatory mechanism is similar to that of omnivorous animals

Table 7. Effect of dietary protein on activities[1] of some select salmonid enzymes involved in amino acid and carbohydrate metabolism.

Enzyme	Dietary protein[2] Low	High
Glutamic dehydrogenase[3]	49	59
Arginase[3]	33.3	3.5
Branched chain aminotransferase[3]	0.84	0.75
Histidine deaminase[4]	3.9	4.9
Urocanase[4]	4.4	4.5
Pyruvate kinase[4] (glycolytic)	404	265
Fructose diphosphatase[4] (gluconeogenic)	18	77

[1]Activities are expressed as µmol/min/g protein at 8° in Tunison data and µmol/hr/g tissue at 15° in Cowey *et al.* (1981) study.

[2]Low and high protein levels were 20 and 60%, respectively, in Tunison Lab data and 22 and 54% in Cowey *et al.* (1981) study.

[3]Tunison Lab (Rumsey, 1981).

[4]Cowey *et al.* (1981).

Table 8. Comparison of enzyme activities[1] in fish, cat, and rat fed *high protein diets.*

	Rat[2] (omnivore)	Fish[2] (carnivore)	Cat[2] (carnivore)
Ala aminotransferase	1,200	125	80
Glu dehydrogenase	175	120	100
Arginase	360	106	109
Pyruvate kinase	26	66	92
Fructose diphosphatase	143	428	94

[1]Activity expressed as a percentage of activity on low protein diets.

[2]Rat and cat activity data from Rogers *et al.* (1977) and Das and Waterlow (1974). Fish enzyme activity from Cowey *et al.* (1981) and Tunison Laboratory (Rumsey, 1981).

(e.g., the rat) in that glucose storage enzymes increase and glucose degrading enzymes decrease in response to a high protein intake. Most animals (herbivores and omnivores) have the mechanisms for control of blood glucose concentration in their livers (i.e., the liver plays a homeostatic role -- taking up glucose by virtue of glucose phosphorylating enzymes [hexokinase] or, vice-versa, releasing glucose by hydrolyzing glucose phosphate [glucose-6-phosphatase] -- as the situation dictates). Cowey (1977) reported that there was little or no glucokinase (glucose phosphorylating) activity in trout liver -- providing an explanation of the inability of trout to metabolize large amounts of dietary carbohydrate. Since release of glucose from the trout liver is very slow, Cowey (1977) postulated that regulatory control over glucose storage is particularly important in trout because it could be operating to meet glucose demand.

There are other convincing illustrations of a lack of fine control of amino acid metabolism in fish (Table 9) (Cowey and Sargent, 1980).

Table 9. Effect of dietary protein level on amino acid oxidation in the fish[1] and rat[2].

Amino acid[3] and protein level		Oxidation[4] (percent AA oxidized) Fish	Rat
Leucine	Low	24	10
	High	29	50
Phenylalanine	Low	24	0
	High	20	10
Glutamic acid	Low	56	35
	High	59	40
Alanine	Low	57	88
	High	49	78

[1]Cowey and Sargent, 1980.

[2]McFarlane and Holt, 1969.

[3]Leucine and phenylalanine are essential; glutamic acid and alanine are non-essential amino acids.

[4]Production of $^{14}CO_2$ in expired air after feeding ^{14}C-labeled amino acids.

Appreciable quantities of the essential amino acids leucine and phenylalanine were oxidized irrespective of dietary protein level. This oxidation contrasts with the reduction in pheynylalanine and leucine oxidation in the rat when dietary protein intake was reduced. These same investigators (data not shown) found no change in rate of incorporation of amino acids into fish tissue proteins in response to dietary protein levels.

Even though the studies to date are not exhaustive in scope, and comparison of enzyme maximum velocities (Vmax's) at different temperatures, pH's, and between species is somewhat risky, the salmonid does appear to have highly active amino acid degrading enzymes. These high activities of amino acid catabolic enzymes indicate that the fish is "geared" to handle a substantial quantity of dietary protein and does not change its existing levels of enzyme activity to meet changing dietary demands. These results also suggest that the fish does not have the capability to conserve amino acids or nitrogen.

Summarily, it seems that the reason the fish has an unusually high protein requirement is that the amino acid or nitrogen catabolic enzymes are permanently set to handle a high protein diet. The fish does not have significant ability to adjust these nitrogen catabolic enzyme activities -- resulting in a high obligatory nitrogen loss when fish are fed a low protein diet. The only real option is to eat a high protein diet. The data also show that the "functional fate" of the obligatory degraded amino acids is toward glycogen synthesis which makes sense when one considers that the diet of the carnivore contains little carbohydrate, and glucose would have to be produced by glycogen synthesis to meet the requirement of the animal.

LITERATURE CITED

Arai, S. 1981. A purified test diet for coho salmon, Oncorhynchus kisutch fry. Bull. Jpn. Soc. Sci. Fish. 47(4):547-550.

Cowey, C. B. and J. R. Sargent. 1972. Fish nutrition. Adv. Mar. Biol. 10:383-492.

Cowey, C. B. and J. R. Sargent. 1980. Bioenergetics and growth in Vol. 8, Fish Physiology (Hoar, Randall and Brett, eds.). Academic Press, NY.

Cowey, C. B., M. Higuera and J. W. Adron. 1977. The effect of dietary composition and of insulin on gluconeogenesis in rainbow trout. Br. J. Nutr. 38:285-395.

Cowey, C. B., D. Knox, M. J. Walton and J. W. Adron. 1977. The regulation of gluconeogenesis by diet and insulin in rainbow trout. Br. J. Nutr. 38:463-470.

Cowey, C. B., D. J. Cooke, A. J. Matty and J. W. Adron. 1981. Effects of quantity and quality of dietary protein on certain enzyme activities in rainbow trout. J. Nutr. 111:336-345.

Das, T. K. and J. C. Waterlow. 1974. The rate of adaptation of urea cycle enzymes, aminotransferases and glutamic dehydrogenase to change in dietary protein intake. Br. J. Nutr. 32:353-373.

Harper, A. E. 1965. Effect of variations in protein intake on enzymes of amino acid metabolism. Can. J. Biochem. 43:1589-1603.

Kaplan, J. H. and H. C. Pitot. 1970. The regulation of intermediary amino acid metabolism in animal tissues. In Munro (ed.) Mammalian Protein Metabolism, IV, pp. 388-443. Academic Press, NY.

McFarlane, L. G. and C. von Holt. 1969. Metabolism of amino acids in protein-calorie-deficient rats. Biochem. J. 111:557-563.

Mertz, E. T. 1972. Protein and amino acid requirements. Chap. 3 in Fish Nutrition (J. E. Halver, ed.). Academic Press, NY.

National Research Council. 1974. Nutrient requirements of the dog. No. 8. National Academy of Sciences, Washington, D. C.

National Research Council. 1976. Nutrient requirements of beef cattle. No. 4. National Academy of Sciences, Washington, D. C.

National Research Council. 1977. Nutrient requirements of poultry. No. 1. National Academy of Sciences, Washington, D. C.

National Research Council. 1977. Nutrient requirements of warmwater fishes. National Academy of Sciences, Washington, D. C.

National Research Council. 1978. Nutrient requirements of laboratory animals. No. 10. National Academy of Sciences, Washington, D. C.

National Research Council. 1978. Nutrient requirements of cats. No. 13. National Academy of Sciences, Washington, D. C.

National Research Council. 1979. Nutrient requirements of swine. No. 2. National Academy of Sciences, Washington, D. C.

National Research Council. 1981. Nutrient requirements of coldwater fishes. National Academy of Sciences, Washington, D. C.

National Research Council. 1982. Nutrient requirements of furbearing animals. National Academy of Sciences, Washington, D. C.

Ogino, C. 1980. Requirements of carp and rainbow trout for essential amino acids. Bull. Jpn. Soc. Sci. Fish. 46(2):171-174.

Reid, J. T., O. D. White, R. Anrique and A. Fortin. 1980. Nutritional energetics of livestock: Some present boundaries of knowledge and future research needs. J. Anim. Science 51(6):1393-1415.

Rogers, Q. R., J. G. Morris and R. A. Freedland. 1977. Lack of hepatic enzymatic adaptation to low and high levels of dietary protein in the adult cat. Enzyme 22:348-356.

Rumsey, G. L. 1981. Significance of nitrogen metabolism: Why does the salmonid require a high protein diet? Salmonid 5(4):20-24.

Smith, R. R. 1981. Energy metabolism in fishes. Symposia from the XII International Congress of Nutrition, pp. 945-953. A. R. Liss Inc., New York, NY 10011.

Smith, R. R. and G. L. Rumsey. 1976. Nutrient utilization by fish. Proceedings, First International Symposium on feed composition, animal nutrient requirements, and computerization of diets. Utah State Univ., Logan. pp. 320-326.

Szepesi, B. and R. A. Freedland. 1967. Alterations in the activities of several rat liver enzymes at various times after initiation of a high protein regimen. J. Nutr. 93:301-306.

VITAMIN A AND CAROTENE FOR RUMINANTS

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Since the 1930's and 1940's when the dietary requirement of vitamin A for cattle was first established, many changes have occurred in ruminant feeding practices. Some of these changes have affected vitamin A utilization and may also affect the requirement for health, growth, production and reproduction. Cattle do not synthesize vitamin A in the body and, thus require supplementation of this vitamin.

Many factors, however, influence vitamin A nutrition in cattle under practical field conditions. Rumen functions, including rumen synthesis of various B-vitamins, depend on the ratio consumed. Additional factors that influence vitamin nutrition in cattle include:

- Stress, disease, parasites and other conditions that lower feed intake and reduce intestinal absorption of vitamins.
- Diseases and other conditions that impair rumen function.
- Bioavailability and stability of vitamins.
- Least-cost feed formulations may result in reduction or exclusion of various vitamin-rich feedstuffs.
- Quality of forage and grain.

REQUIREMENTS

In the early 1940's, the vitamin A requirement of cattle was determined based on vitamin A activity from beta-carotene.[1] Test results have shown that carotene is not efficiently converted to vitamin A activity in cattle.[2] An optimum vitamin A use rate of 2,000 I.U. per 100 pounds of body weight was suggested for beef cattle. The vitamin A requirement of breeding, growing, and finishing beef cattle can be met by (1) provitamin A (carotene) in feedstuffs, or (2) by supplementary vitamin A either by intramuscular injection or orally, or (3) by combinations of (1) and (2).

The minimum vitamin A requirement for normal growth may be lower than those required for higher rates of gain, resistance to various diseases, and normal bone development and nervous system function in cattle.[3] It was suggested that: (a) calves born with low vitamin A liver stores should receive a minimum of 7,500 I.U. of vitamin A per 100 lbs. of body weight and (b) 3 to 5 times this level--22,500 to 37,500 I.U. per 100 lbs. of body weight--may be necessary for adequate vitamin A liver stores in calves during the critical first few months of life.

Test results showed that the vitamin A requirement of calves was increased by as much as sevenfold, depending on the criteria used to determine it.[4] The vitamin A requirement values (I.U. per 100 lbs. of body weight) were 1,200 for adequate weight gains, 2,400 for increased weight gains and 8,000 for optimum weight gains and vitamin A liver stores.

In a 168-day experiment, moderate to extremely high vitamin A levels were fed to fattening cattle; 40,000 I.U. per head daily was needed to maintain initial vitamin A liver stores.[5] Cattle fed 2.5 million I.U. of vitamin A per head daily, which totalled 420 million I.U. per head for 168 days, showed no evidence of vitamin A toxicity.

CAROTENES

Before commercially-synthesized, stabilized vitamin A was available for animal feed use, cattle were dependent on various carotenes in feedstuffs as sources of vitamin A activity.

Carotenes occur naturally in green and dry roughages, silages, and, to a lesser extent, in some grains including corn. Various carotenes are precursors of vitamin A, i.e., they have provitamin A activity. They are converted to vitamin A activity primarily in the intestinal wall of animals. All-trans beta-carotene is the most biologically active form.[6] (Table 1).

Beta-carotene is converted to vitamin A activity in rats at the rate of 1,667 I.U. of vitamin A activity per mg of beta-carotene (the reference standard). In cattle, however, the conversion rate is 400 I.U. of vitamin A activity per mg of beta-carotene when fed at low to moderate levels. As the beta-carotene level fed increases, conversion to vitamin A activity decreases.

Studies have shown that carotene levels from feedstuffs in the diet often did not meet the vitamin A requirements of cattle.[8-12]

Carotene levels vary widely within a given class of feedstuff, depending upon stage of growth at harvest, curing, preservation and storage conditions, and length of storage time.[13,14] (Table 2).

In a study, the average carotene level in samples of light-weight corn, as determined by assay, was 26.3% lower than that in sound corn[15] (Table 3). Moldy corn samples were also assayed and found to average 98.7% less carotene than normal corn.

Several factors have been identified as affecting vitamin A nutrition of the ruminant.

PROTEIN

Test results suggest that dietary protein levels may affect the vitamin A needs of ruminants. Vitamin A and protein plasma levels in sheep fed inadequate protein were lower than in sheep receiving adequate (10.4%) protein.[16]

NITRATE OR NITRITE

Moderately high nitrate or nitrite intake can adversely affect conversion of carotene to vitamin A activity in ruminants, and very high intake can also produce nitrite toxicity in these animals. Cattle can consume nitrate or nitrite from various forages and grains and water.

A review of the effect of nitrates on animal metabolism reported that conversion of carotene to vitamin A activity in ruminants can be influenced by the level of nitrate or nitrite intake, pH of the rumen, ratio of nitrate or nitrite level to beta-carotene or vitamin A level and possibly the energy level of the ration.[17]

In a study, cattle receiving high-nitrate corn silage containing 5 mg of carotene per lb. and providing about 100 mg of carotene per head per daily developed vitamin A deficiency.[18]

PHOSPHORUS

Evidence suggests that phosphorus deficiency can reduce conversion of carotene to vitamin A activity in cattle.

In a study with steers and sheep fed low or high phosphorus rations supplemented with carotene, plasma carotene levels were higher in phosphorus-deficient steers than in the controls. Plasma and liver vitamin A levels were lower in phosphorus-deficient steers than in the controls. In sheep, the opposite effect occurred. Plasma and liver vitamin A levels were higher in the phosphorus-deficient lambs than in the controls. Plasma and liver carotene levels were negligible in the lambs. This suggests a difference between cattle and sheep in response to vitamin A during phosphorus deficiency.[19]

In another study, sheep and rats fed carotene or vitamin A had greater liver vitamin A stores when fed low phosphorus rations than when fed high phosphorus rations.[20]

HIGH ENERGY RATIONS

Feeding either high energy or high roughage rations with low carotene content did not affect weight gain or cattle in a 180-day experiment. However, at the end of the experiment, vitamin A plasma levels of cattle fed the high energy ration were lower than those receiving the high roughage rations.[21]

Increasing the dietary energy or protein content increased rate of depletion of vitamin A from the liver.

HEAT STRESS

Test results showed that cattle exposed to environmental temperatures of 110°F lost three times as much vitamin A from the liver as those at 75°F to 89°F.[22] In addition, cattle without shade lost 2½ times as much of their vitamin A liver stores as those with shade at the same temperature.

INFECTIOUS DISEASE AND INTERNAL PARASITES

Some evidence indicates that inadequate vitamin A intake may lead to increased incidence of various infectious diseases as well as certain internal parasitic infections in ruminants.

In a study, high calf mortality due mostly to scours and pneumonia was attributed to inadequate vitamin A intake.[23] Other test results showed that supplemental vitamin A reduced the incidence and severity of lungworm infection in sheep.[24]

REPRODUCTION

Adequate vitamin A is essential for reproduction in cattle. Early signs of vitamin A deficiency in pregnant cows include shortened gestation periods, high incidence of retained placentas, and birth of dead, incoordinated or blind calves.[7]

Adequate vitamin A is necessary for fertility in cows[25,26,27] and bulls.[28] Increased semen volume and sperm count and reduced incidence of abnormal sperm occurred in breeding bulls receiving supplemental vitamin A added to rations containing normal amounts of carotene.[28] (Tables 4 and 5). Recent studies suggest that beta-carotene influences reproductive performance in cows independent of the vitamin A activity.[29] (Table 6). The possible role of beta-carotene in improving fertility in dairy cows is reviewed in a recent report on this subject.[30]

VITAMIN A ALLOWANCES

The intake of vitamin A will vary depending on stage of growth, production and reproduction. The quality of feedstuffs often will determine the need for supplementation under various management conditions. Guidelines for vitamin A in cattle is presented in Table 7.

Table 1. Relative Provitamin A activity of Various Carotenes

Carotene	Provitamin A Activity (%)
All-trans beta-carotene	100
Neo beta-carotene B	53
Neo beta-carotene	38
All-trans alpha carotene	53
Neo alpha carotene B	16
All-trans gamma carotene	28-45
Neo gamma carotene P	19
All-trans cryptoxanthin	57
Neo-cryptoxanthin	42

Table 2. Carotene Content of Feedstuffs

Feedstuffs	Carotene (mg/lb.) Average	Range
A. Grains:		
Corn	1.5	0.1-6.0
Corn Dent #2	0.9	*
Corn Gluten Meal	15.0	*
Sorghum (Milo)	0.2	*
B. Dry Roughages:		
Alfalfa Hay	27.7	0-321.2
Alfalfa Hay, Suncured (S.C.)	17.2	1.1- 65.0
Alfalfa Dehy. 17%	70.0	0-288.4
Brome Hay	16.7	1.0- 84.0
Clover Hay	30.7	0.5-149.1
Alfalfa-Timothy Hay	6.0	1.5- 16.7
Alfalfa-Brome Hay	8.9	4.8- 16.0
Grass-Legume	12.5	0.5-127.3
C. Green Roughages:		
Alfalfa	90.2	33.1-177.5
Blue Stem	53.7	0.3-173.7
Brome	141.0	30 -264.4
Clover	128.6	34.5-253.1
Fescue	153.1	36.3-285.8
Timothy	101.6	34.0-215.0
Wheat	184.7	81.5-433.2
D. Silages:		
Alfalfa	40.7	0.2-133.3
Brome	32.3	12.2- 47.6
Clover	49.8	14.5-213.2
Corn	7.1	3.6- 10.6
Corn Dent	23.6	2.1- 25.6
Sorghum	15.3	0.3- 65.1
Alfalfa-Brome	6.2	5.1- 7.3

* Range not published.

Table 3. Carotene Deficits in Moldy and Light-Weight Corn Compared to Sound Corn

Corn Samples	Average Carotene Content mg/lb.	% Below Sound Corn
Sound	1.9	-
Moldy	0.025	98.7
Light-Weight*	1.4	26.3

* Below U. S. No. 2 standard for test weight.

Table 4. Influence of Vitamin A Supplementation on Semen Production

Semen Characteristics	Control	45,000 I.U. Vitamin A/ Head/Day
Volume (ml)	5.4	5.9
Sperm Concentration (10^6)	963	1010
Methylene Blue Reduction Time, Minute	4.8	4.5
Abnormal Sperm, %	11.8	9.7

Table 5. Influence of Vitamin A on Conception Rate

Age Group	Control Group No. Animals	Control Group % Pregnant	Vitamin A* Treated Group No. Animals	Vitamin A* Treated Group % Pregnant
Mature Cows	582	70.1	1,097	84.5
First Calf Heifer	129	74.9	241	83.0
Replacement Heifers	107	64.5	261	79.3

* 2 M.U., IM Injection

Table 6. Effect of Supplemental Beta-Carotene on Conception Rate and Services per Conception in Dairy Cows Fed Adequate Vitamin A

Per Cow	Vitamin A 220 IU/kg B.W.	Beta-Carotene plus Vitamin A 0.3 mg plus 100 I.U./kg B.W.	Improvement from Beta-Carotene Plus Vitamin A vs. Vitamin A Alone
Average Conception Rate from:			
First service	40%	68.4%	28.4% diff.
Second service	15%	21.1%	6.1% diff.
Average No. of Services/ conception	2.0± 0.91	1.42 ± 0.69	29% reduction

Table 7. Guidelines for Vitamin A Allowance for Beef and Dairy Cattle (IU/Head/Day)

Dairy Cattle	NRC 1978	Field Survey Levels (Total)	Roche (Supplemental)
Calf Starter	4,400[1]	3,200-20,800	20,000
Lactating Cows and Breeding Bulls	60,320[2]	30,000-100,000	80,000-100,000
Dry Cows	48,000[3]	30,000- 80,000	60,000- 80,000
Growing Heifer and Bulls	20,650[4]	5,000- 60,000	25,000- 35,000
Beef Cattle	**NRC 1976**	**Field Survey Levels (Total)**	**Roche (Supplemental)**
Growing and Finishing Steers and Heifers	17,962[5]	24,000-107,000	40,000-60,000
Dry Cows and Preg. Heifers	25,400[6]	30,000-75,000	40,000-60,000
Lactating Cows and Breeding Bulls	46,000[7]	38,000-250,000	50,000-70,000

1 Based on calf weighing 165 lbs. consuming 4.4 lbs. of concentrate daily.

2 Based on 1430 lb. cow producing 55 lbs. 4% fat corrected milk daily with dry matter intake of 2.9% of B.W.

3 Based on maintenance and last 2 months of gestation of 1400 lb. cow.

4 Based on 880 lb. heifer gaining 1.5 lb of body weight daily.

5 Based on 700 lbs. B.W. and daily feed consumption of 18 lbs.

6 Based on 1100 lbs. B.W. and daily feed consumption of 20 lbs.

7 Based on bulls weighing 1400 lbs and lactating cow weighing 1100 lbs. each consuming 26 lbs. of feed daily.

LITERATURE CITED

1. Moore, L.A., 1941, Some Ocular Changes and Deficiency Manifestations in Mature Cows Fed a Ration Deficient in Vitamin A, J.Dairy Sci., 24:893.

2. Guilbert, H.R., C.E. Howell and G.H. Hart, 1940,Vitamin A and Carotene Requirements of Mammalian Species, J.Nutr., 19:91.

3. Weichenthal, B.A., R.J. Emerick, F.W. Whetzal and L.B. Embry, 1963, Influence of Sodium Nitrate Vitamin A and Protein Level on Feedlot Performance and Vitamin Status of Fattening Cattle, J. Anim. Sci. 22: 979.

4. Lewis, J.M., and L.T. Wilson, 1945, J. Nutr., 30:467.

5. Hale, W.H., F. Hubbert, Jr., R.E. Taylor, T.A. Anderson and B.J. Taylor, 1961, The Effect of Feeding High Levels of Vitamin A to Beef Cattle Upon Performance and Tissue Vitamin A Levels, J. Anim. Sci., 20: 668.

6. Tiews, J., 1963, Wiss. Verottentl. Dtsch. Ges. Ernahr, 9:235.

7. Nutrient Requirements of Dairy Cattle No. 3, 5th rev. ed., National Research Council/National Academy of Sciences, 1978.

8. Mitchell, G.E., Jr., A.L. Neumann, R.R. Garrigus, W.M.Duidle and K.A. Kendall, 1960, Another Look at Vitamin A in Cattle Feeding, Univ. of Illinois, Agric. Expt. Sta. Cattle Feeders Day Report.

9. Perry T.W., W.M. Beeson and R. C. Holz, 1960, Levels of Supplemental Vitamin A for Fattening Beef Steers, J. Anim. Sci, 19:1283.

10. Smith, G.S., A.L. Neumann, W.G. Huber,M.A. Jordan and O.B. Ross, 1961, Avitaminosis A in Cattle Fed Silage Rations Supplemented with Vitamin A, J. Anim. Sci., 20:952.

11. Jordan, H.A., G.S. Smith, A.L. Neumann, J.E. Zimmerman and G.W. Breneman, 1963, Vitamin A Nutrition of Beef Cattle Fed Corn Silage, J. Anim. Sci., 22: 738.

12. Perry, T.W., W.M. Beeson, W.H. Smith and M.T. Mohler,1967, Injectable vs. Oral Vitamin A for Fattening Steer Calves, J. Anim. Sci., 26:115.

13. Composition of Cereal Grains and Forages, 1958, National Research Council, National Academy of Sciences, Pub. 585.

14. Atlas of Nutritional Data on U. S. And Canadian Feeds, 1971, National Research Council, National Academy of Sciences.

15. Adams, C. R., H.J.Eoff and C.R. Zimmerman, 1975, Protecting Feeds from Vitamin E and A Deficits in Light-weight, Moldy and Blighted Corn, Feedstuffs, 47 (36):24.

LITERATURE CITED (continued)

16. Anderson, T.A., F. Hubbert, Jr., C.B. Roubicik and R.E. Taylor, 1962. Influence of Protein Depletion on Vitamin A and Carotene Utilization by Vitamin A Deficient Sheep, J. Nutr., 78:341.

17. Beeson, W.M., Effect of Nitrates on Animal Metabolism, 1964, Purdue Cattle Feeders Day Report.

18. Klosterman, E.W., D.W. Eolin, M. Light and W.R. Dinusson, 1952, Relationship Between Inorganic Phosphorus and Vitamin A in the Rat and Sheep, Science, 116:665.

19. Gallup, W.D., O.O. Thomas, O.B. Ross and C.K. Whitehair, 1953, Carotene and Vitamin A Metabolism in Cattle and Sheep on Phosphorus-Deficient Rations, J. Anim. Sci., 12:716.

20. Klosterman, E.W., D.W. Eolin, M. Light and W.R. Dinusson, 1952, Relationship Between Inorganic Phosphorus and Vitamin A in the Rat and Sheep, Science, 116:665.

21. Anthony, W.R., R.R. Harris and J.S. Starling, 1961, J. Anim. Sci. 20:924.

22. Page, H.M., E.S. Erwin and G.E. Nelms, 1959, Effect of Heat and Solar Radiation on Vitamin A Utilization by the Bovine Animal, Amer. J. Physiol., 196:917.

23. Jones, T.J., R.M. Hough and W.T. Gerard, 1962, Vitamin A Studies in the Dairy Calf, Can. Vet. J., 3:8.

24. Akopjan, W.P., 1956, Veterinarija (Moscow) 1:129.

25. Byers, J.H., I.R. Jones and J.F. Bones, 1956, Carotene in the Ration of Dairy Cattle, J.Dairy Sci., 39: 1556.

26. Moore, L.A., 1939, Carotene Intake, Level of Blood Plasma Carotene and the Development of Papillary edema and Nyctalopia in Calves, J. Dairy Sci., 22:803.

27. Ronning, M.E., R. Berousek, A.H. Kuhlman and W.D. Gallup, 1953, The Carotene Requirements for Reproduction in Guernsey Cattle, J. Dairy Sci., 36:52.

28. Roussel, J. D., T.E. Patrick, H.C. Kellgren, D.F. Randel and L. L. Rusoff, 1963, Influence of High Level of Vitamin A Supplementation on Semen Characteristics and Blood Composition of Breeding Bulls, J. Dairy Sci., 46:583.

29. Lotthammer, K.H., L. Ahlswede and H. Meyer, Dtsch. Tierarztl. Wochenschr., 83:353, 1976.

30. Hemken, R.W. and D.H. Bremel, 1982, Possible Role of Beta-Carotene in Improving Fertility in Dairy Cattle. J.Dairy Sci. 65:1069.

B VITAMINS FOR RUMINANTS

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INTRODUCTION

In 1913 only a fat soluble and a water soluble vitamin had been discovered. E.V. McCollum who was born on a Kansas farm and received his early education at the University of Kansas proposed the name fat soluble A and water soluble B for these two vitamins. We now know that there are several B vitamins. These are almost collectively concerned with the transfer of energy in the body. Niacin is a component of coenzymes (NAD, NADP) which are involved in energy transfer. Thus, niacin aids in energy release from carbohydrates, fats and proteins. Riboflavin is also essential to the metabolism of carbohydrates, fats and proteins and is a part of two flavoprotein coenzymes (FAD, FMN). Thiamin is a component of two enzymes (TPP, ThPP) and is involved in cellular decarboxylations. Pantothenic acid is a part of coenzyme A and is concerned with the metabolism of acetate. Pyridoxine functions as a coenzyme for many enzyme systems involved in amino acid metabolism. Folic acid and vitamin B_{12} function in the metabolism of single carbon units and aid in the synthesis of RNA and DNA. Biotin functions as a coenzyme for carbon dioxide, and plays a key role in gluconeogenesis, in the oxidation of fatty acids, and is required for the synthesis of dicarboxylic acid.

B VITAMIN REQUIREMENTS OF RUMINANTS

It is well known that the microorganisms in the rumen synthesize B vitamins (Hungate, 1966). Virtanen (1963) and Oltjen (1969) successfully raised ruminants on purified diets free of B vitamins. For years we have been instructing our students that rumen microorganisms sythensize B vitamins in quantities adequate to meet the animal's requirements and that B vitamin deficiencies do not occur in ruminants like they do in nonruminants. However, recent research with some B vitamins suggests that we reassess the idea that the rumen microorganisms can synthesize enough B vitamins to meet the host animal's requirements. Presently ruminant nutrition research is focusing on niacin and thiamin. This paper will examine the recent research dealing with these two vitamins. There is not enough research to suggest that adult ruminants require dietary supplements of the other members of the vitamin B-complex.

NIACIN

It is well known that rumen microorganisms can synthesize niacin (Hungate, 1966). Niacin synthesis by rumen microbes has been considered to be adequate for optimum animal performance (National Research Council, 1978). However, recent evidence suggests that this may not be true for

animals stressed for high production.

NIACIN FOR LAMBS. Shields and Perry (1981) added 100 ppm niacin to the diet of lambs receiving either soybean meal (SBM) or urea as nitrogen supplements. During the growing phase niacin improved rate of gain, feed efficiency and nitrogen retention with both SBM and urea diets. During the finishing phase niacin increased rate of gain only with the SBM diet. However, niacin increased feed efficiency and nitrogen retention with both the SBM and urea diets.

In a second study with ram lambs, Shields, Perry and Schaefer (1981) observed that the addition of 100 ppm niacin appeared to be beneficial in adapting the lambs to a urea diet. Mizwicki et al. (1975) observed that 500 ppm of supplemental niacin improved feed efficiency of lambs fed a high-concentrate diet containing urea.

NIACIN FOR BEEF CATTLE. Byers (1980) recently summarized the niacin research with beef cattle. A summary of 14 studies indicated that niacin supplementation of beef cattle was beneficial during the period of acclimation to feedlot diets (first 21 to 38 days). Growth rate and feed efficiency were improved in most trials with response to gain and feed efficiency to 50-250 ppm niacin averaging 9.7 and 10.9%, respectively. Supplementing 500 ppm niacin was too high a dose and produced negative effects on gain and feed efficiency. In two studies where plant protein or urea were compared, gains and efficiency were better with plant protein plus niacin than with urea plus niacin. However, in several other studies where urea was the only source of supplemental nitrogen responses to niacin were good.

Byers (1980) summarized the overall response to niacin in feedlot cattle. In 22 studies, primarily with yearling cattle, and with feeding periods of 73 to 176 days in both university research facilities and commercial feedlots, the 50 ppm level of niacin was ineffective and the 500 ppm level appeared excessive. Overall, the 100 ppm was the most effective level and enhanced daily gain and feed efficiency by 3.6 and 3.7%, respectively.

NIACIN FOR DAIRY CATTLE. Piva et al. (1976) observed an increase in milk production when cows were fed urea-containing diets supplemented with 265 ppm niacin. Kung et al. (1980) used lactating Holsteins in early or midlactation. The effect of niacin was tested in these cows fed either SBM or nonprotein nitrogen from ammonia treated corn silage. Niacin response was greater in the early-lactation cows fed rations containing all natural protein.

In one of two lactation studies (Riddell et al., 1981) with cows in midlactation fed urea-containing diets, we observed a slight increase in milk production attributable to niacin (6 g per cow per day). In the other study a slight improvement in milk protein production was attributable to niacin. In a third study, but with fresh cows, milk production increased significantly (2.9 kg milk per cow per day) in cows receiving niacin (ca.

200 ppm of diet or 5 g niacin per cow per day) and SBM, but not in those receiving niacin and urea (table 1). In a fourth study with fresh cows fed SBM, cows receiving niacin (5 g per cow per day) gave slightly more milk than those receiving none.

It is clear that the response to niacin is greater in fresh cows than in those in midlactation and is greater in cows fed natural protein than in those fed urea.

NIACIN AND KETOSIS. It is possible that the response to niacin is greater with fresh cows than with those in midlactation because niacin may play a role in ketosis. Fronk and Shultz (1979), who gave 12 g niacin to cows with subclinical or clinical ketosis, observed significant increases in blood glucose, and milk production and significant decreases in betahydroxybutyric acid and free fatty acids (table 2). While Fronk and Shultz (1979) attempted to treat ketosis with niacin we (G. S. Dufva and E. E. Bartley, unpublished) attempted to prevent ketosis. To induce ketosis, energy intake of 20 cows that had just calved was reduced below requirement. Ten cows were given 12 g niacin per cow per day and 10 served as controls. The cows receiving niacin had slightly higher blood glucose concentrations, higher milk production, lower betahydroxybutyric acid concentrations, and lower concentrations of nonesterfied fatty acids (table 3). There were two cases of clinical ketosis in the controls and none in those receiving niacin. Additional studies are in progress to determine if the increase in milk production due to niacin supplementation of fresh cows is due to an effect on ketosis.

EFFECT OF NIACIN ON THE RUMEN FERMENTATION. Recent studies have demonstrated that niacin supplementation of cattle diets affects the rumen fermentation. Mizwicki (1976), Bartley et al.,(1979) and Riddell et al., (1980, 1981) showed increased microbial protein synthesis in vitro from the addition of niacin to the substrates.

We (Riddell et al., 1980 and Arambel et al. 1982), observed that the feeding of niacin to rumen fistulated cattle increased bacterial protein production in the rumen and increased the percentage of rumen propionate.

In a later study, we (Riddell et al., 1981) observed that microbial protein synthesis in vitro was greater with niacin and SBM as a substrate than with niacin and urea plus ground corn as a substrate (table 4). Because niacin is involved intimately in energy metabolism, it was expected that the response to niacin would be greater with urea than with SBM. Niacin can be synthesized from tryptophan by bacteria (Giesecke and Hendrickx, 1973). Because rumen ciliate protozoa differ from bacteria in that they cannot synthesize niacin (Hungate, 1966; Jones, 1974; Kidder, 1967) and, therefore, must obtain their niacin from bacteria or feed, we conjectured that heat treating SBM as occurs in commercial processing might reduce the availability of either niacin or tryptophan for bacteria, thus reducing the supply of niacin to protozoa. To test that, we compared the effect of heated (conventional process) or unheated SBM, with or without niacin, on protozoal numbers (Dennis et al., 1982). Rumen protozoal numbers

increased when niacin was added to diets containing heated SBM but not when they contained unheated SBM (table 5). In a companion study, Arambel et al. (1982) observed that rumen bacterial nitrogen synthesis was higher in cattle fed unheated SBM (738 mg bacterial-N g total N) compared with cattle fed heated SBM (554 mg/g total N). Niacin supplementation of cattle fed heated SBM increased bacterial N synthesis 10.9%. Microbial efficiency of duodenal samples was improved in cattle fed heated SBM and niacin (22.2 versus 16.6 g bacterial-N per kg dry matter). Thus, niacin appears to be a limiting nutrient for rumen microorganisms when cattle are fed diets containing heated (conventional processed) SBM.

SYNTHESIS AND DEGRADATION OF NIACIN IN THE RUMEN. While it is well known that rumen bacteria can synthesize niacin (Hungate, 1966), there is a paucity of information pertaining to what extent supplementary dietary niacin interferes with ruminal synthesis of niacin and to what extent dietary niacin is degraded in the rumen. We (Riddell et al., 1982) studied these effects in vitro. Niacin synthesis was greatest with no niacin supplementation. Small quantities of supplemental niacin (.5 ppm) decreased niacin synthesis, and large quantities (2-8 ppm) of supplemental niacin were degraded during fermentation. Byers (1981) postulated that there is an optimum concentration of niacin in the rumen for bacteria below which synthesis will occur and above which no net synthesis occurs. Excess niacin may be degraded for other purposes. Our results support the hypothesis of Byers.

Supplementing diets fed to duodenal cannualted cattle with 2g niacin per feeding (30 ppm) resulted in higher niacin concentrations in both ruminal and duodenal digesta. The flow of niacin to the small intestine and absorption of niacin from the small intestine increased with niacin supplementation. Niacin supplementation resulted in a slight increase in niacin excreted in the feces.

Thus, while supplemental niacin may affect ruminal synthesis of niacin and some may be degraded in the rumen, there is a considerable quantity of supplementary niacin that reaches the duodenum and is absorbed.

Levitas et al. (1947) indicated that niacin in blood is found almost entirely in erythrocytes and exists in these cells as the nicotinamide part of the pyridine nucleotides. We (Riddell et al., 1982) found that the niacin concentration of erythrocytes in cows not supplemented with niacin dropped after calving (table 6). However, this drop did not occur after calving in cows supplemented with niacin (12 g per cow per day).

DISCUSSION. Supplementing ruminant diets with niacin appears to have a beneficial effect on production. The beneficial effect of niacin may be ruminal, systemic or a combination of both. It is evident that microbial protein synthesis is enhanced in the rumen by the addition of niacin to the diet.

Niacin supplementation appears to be especially beneficial to stressed animals - beef cattle being adapted to high grain diets and lactating cows

who have just calved. It is well known that high producing cows if not manifesting clinical ketosis are often on the border of being ketotic. Supplementing fresh cows with niacin (12 g per cow per day) reduces the incidence of ketosis. Kronfeld and Raggi (1964) reported a reduction in pyridine nucleotides in mammary tissue of ketotic cows. In our studies (Riddell et al., 1982) supplementary niacin was absorbed and prevented the drop in niacin concentration of erythrocytes in fresh cows. Handler and Kohn (1943) reported a rapid absorption of naicin from the small intestine of humans and an increased pyridine nucleotide concentration of blood after continuous dosing with niacin. Also, adding niacin to the diet of rats increased blood pyridine nucleotide concentration (Morrison et al., 1963). The increased milk production in early lactation observed with niacin supplementation could be due to niacin providing an adequate supply of pyridine nucleotides for tissue metabolism.

Recommended levels of supplementary niacin for ruminant diets are 100 ppm for feedlot cattle and 200 ppm for fresh dairy cows. Approximately 400 ppm of supplementary niacin in the diet may be required to prevent ketosis in high producing cows.

THIAMIN

The interest in thiamin has centered around the central nervous system condition, polioencephalomalacia (PEM)(Cerebrocortical necrosis, or CCN in Great Britain). PEM is often characterized by circling, head pressing, opisthotonus (in which the head is drawn back over the neck), blindness, convulsions, and death if the animal is not treated rapidly. Early workers found that there was a dramatic improvement in the condition if animals were given a mixture of B-complex vitamins. Later, the condition was found to respond to thiamin specifically. But if the feed contained thiamin, and if the rumen synthesized even more, how could the victims possibly be thiamin deficient? The apparent answer was arrived at by Edwin et al. (1968) who proposed the involvement of a thiaminase enzyme. Two types of thiaminase are described. Thiaminase II simply cleaves the vitamin at the methylene bridge between the thiazole and the pyrimidine rings, yielding free thiazole, free pyrimidine, and of course, less thiamin. Thiaminase I substitutes a new base for the thiazole ring. This, leads to less thiamin, but it also gives rise to a thiamin analog composed of the pyrimidine ring of the original thiamin, and another ring from the "cosubstrate". That thiamin analog could then be absorbed, and quite possibly inhibit thiamin-requiring reactions.

The working hypothesis for (PEM) is: Thiaminase I, in the presence of a cosubstrate, produces a thiamin analog, which in turn, blocks thiamin reactions. Because the brain depends on glucose utilization via glycolysis, and because thiamin pyrophosphate is a cofactor in decarboxylations, it seems reasonable that the central nervous system should be first to show symptoms. Several years ago, Lusby (1971), in our laboratory, was able to induce PEM in 3 to 4 days, using a continuously infused, liquid, high-cabohydrate diet. His work supported the thiaminase I hypothesis, because it is very difficult to envision animals developing primary thiamin

deficiency in that short a time. So the hypothesis has been in use for several years and although it generally explains how (PEM) occurs, recent literature has shown some exceptions. The hypothesis is not as "clean" as we once thought.

Bacterial thiaminase is often described as an exoenzyme. That is, it is bound to the surface of the cell. Sapienza (1981), in our laboratory, found that if rumen fluid is taken from normal animals, adjusted to pH 6.8, and assayed for thiaminase, little, if any activity is found. In fact, more thiamin may be recovered than is added due to thiamin synthesis. However, if samples were "acid shocked" to about pH 4.5, then adjusted back to pH 6.8 before assay, thiaminase was present. Apparently, the thiaminase had been separated from the bacterial cell. In rumen fluid from PEM cases, the enzyme is found in the supernatant, and no more enzyme is released by acid shocking. However, in our PEM model, rumen acidosis has already accomplished the acid-shock procedure.

Where does thiaminase I come from if it is the culprit? Clostridium sporogenes and bacillus thiaminolyticus have both been isolated from the rumen of PEM cases. Both organisms produce thiaminase I. However, that does not mean that these organisms are the source of thiaminase I, especially since no one has been able to establish populations of those organisms in the rumens of cattle. Also, several workers have reported that the characteristics of thiaminase from those organisms are not like that found in spontaneous PEM. Spontaneously occurring thiaminase has two pH optima, and that from C. sporogenes and B. thiaminolyticus has only one. Their electrophoretic mobilities also differ, as do their estimated molecular weights.

Thiaminase I can also come from a number of plant species. This has been a special problem in Australia, where PEM occurs under pasture conditions, apparently being derived from some of the fern species. In the U.S., PEM generally occurs in feedlot cattle, and frequently about 3 wk after a ration change. If PEM is seen in pasture cattle, thiaminase I from a plant source should receive serious consideration.

The sulfite ion will also cleave thiamin at the methylene bridge, and analytically, will mimic thiaminase. Several cases of PEM have occurred when gypsum has been used as a feed intake limiter. It would appear that the sulfate ion of gypsum, during its conversion to sulfide, must pass through sulfite, which may destroy thiamin. However, we have been unable to create PEM by giving large doses of sulfate ion.

There may be a whole series of specific thiaminase I enzymes. That becomes especially likely when we look at cosubstrate specificity. The preferred cosubstrate for thiaminase I from spontaneous cases of PEM is analine. Niacin holds special interest, since supplemental niacin is being added to rations to improve animal performance. Can the extra niacin acting as a cosubstrate precipitate a case of PEM if the other conditions are already met?

Our group has postulated that rumen acidosis may set the stage for PEM. Histamine is generally synthesized in the rumen whenever acidosis is present. Can histamine serve as the cofactor for thiaminase I?

If we assume that a thiamin analog is the agent that blocks thiamin reactions, we need to look at the thiamin analogs that might be involved. Amprolium is an excellent coccidiostat, apparently because it inhibits thiamin phosphorylation. Lowe and Dunlap (1972) found that high levels of amprolium (considerably above the levels needed to prevent coccidiosis) could produce the physical symptoms and the histological lesions of PEM. It is doubtful that thiaminase I produces amprolium, but amprolium's ability to induce PEM adds credence to the idea that a thiamin antagonist is involved in the disease. Since the amprolium molecule is similar to thiamin, and can apparently inhibit some thiamin-requiring reactions, is it possible that lower doses of amprolium can inhibit thiaminase I?

Research on PEM is a frustrating experience. Although Lusby, in our laboratory, was able to produce PEM in 4 days, and never had a failure, recently, we have been unable to produce the condition, using the same system. We may speculate that in Lusby's case a few bacteria capable of producing PEM were present in the rumen and proliferated when a liquid diet was infused. Perhaps those bacteria are no longer present. Several workers have been unable to successfully introduce C. sporogenes or B. thiaminolyticus into the rumen although they can be isolated from other rumens. We have also shared the frustration of failing to establish those known thiaminase producers.

In a review (Brent, 1976), it was postulated that lactic acidosis was probably a precursor to PEM. If that is true, our (Nagagaraja et al, 1981) recent finding that the polyether antibiotics lasalocid and monensin effectively prevent lactic acidosis should mean that monensin should effectively prevent PEM. Though it has been claimed that there are fewer cases of PEM in feedlots since the introduction of monensin, a farmer just west of Manhattan recently lost six head of feeder cattle to PEM, and they had received monensin since they arrived in his yard.

We have assembled a few pieces of the PEM puzzle, but the remaining pieces are going to be rather difficult to assemble. For example, the presence of thiaminase in the rumen is not specifically diagnostic for PEM! Probably the easiest place to check for thiaminase is in the feces. Apparently if there is thiaminase in the feces, there is also thiaminase in the rumen. However, there is considerable thiaminase found in the feces of perfectly healthy animals. Why don't they have PEM? If a thiamin analog, exists whose target is the central nervous system, then there must be some sort of mechanism protecting animals against the analog. Several authors have postulated that there is enough thiaminase in the rumen to rapidly hydrolyze all the thiamin present. Perhaps thiaminase is routinely present, but the cosubstrate limits the reaction rate, or perhaps there is some sort of protective mechanism that prevents the analog from acting on the target organ. Or perhaps, the absence of thiaminase is an artifact.

After examining some of the new data on these two vitamins, it is easy to make a case for new research projects that will carefully examine the assumption of adequacy of B-vitamins in the rumen, and the ruminant.

LITERATURE CITED

Arambel, M. J., E. E. Bartley, S. M. Dennis, D. O. Riddell, J. L. Camac, J. F. Higginbotham, G. G. Simons, and A. D. Dayton. Effect of heated soybean meal with or without niacin on rumen fermentation, passage rate of duodenal digesta, and digestibility of nutrients. J. Anim. Sci. submitted.

Bartley, E. E., E. L. Herod, R. M. Bechtle, D. A. Sapienza, and B. E. Brent. 1979. Effect of monensin or lasalocid, with and without niacin or amicloral, on rumen fermentation and feed efficiency. J. Anim. Sci. 49:1066.

Brent, B. E. 1976. Relationship of acidosis to other feedlot ailments. J. Anim. Sci. 43:930.

Byers, F. M. 1980. Niacin for ruminants. Feed Management. July.

Byers, F. M. 1981. Another look at niacin. Anim. Nutr. Health. 36(6):36.

Dennis, S. M., M. J. Arambel, E. E. Bartley, D. O. Riddell, and A. D. Dayton. 1982. Effect of heated or unheated soybean meal with or without niacin on rumen protozoa. J. Dairy Sci. (In Press).

Edwin, E. E., G. Lewis, and R. Allcroft. 1968. Cerebrocortical necrosis: A hypothesis for the possible role of thiaminase in its pathogenesis. Vet. Rec. 83:176.

Fronk, T. J., and L. H. Schultz. 1979. Oral nicotinic acid as a treatment for ketosis. J. Dairy Sci. 62:1804.

Giesecke, D., Hendrickx, H. K. Biologie und Biochemie der mikrobielle Verdauung BLV Verlugsgesellschaft (1973) 240.

Handler, P., and H. I. Kohn. 1943. The mechanism of sozymase synthesis in the human erythrocyte: a comparison of the roles of nicotinic acid and nicotinamide. J. Biol. Chem. 151:447.

Hungate, R. E. 1966. The rumen and its microbes. Academic Press Inc., New York, NY.

Jones, A. R. 1974. The Ciliates. Hutchinson & Co. Ltd. London.

Kidder, G. W. 1967. Chemical Zoology I. Protozoa. Academic Press Inc., New York.

Kronfeld, D. S., and F. Raggi. 1964. Nicotinamide coenzyme concentration in mammary biopsy samples from ketotic cows. Biochem. J. 90:219.

Kung, L., K. Gubert, and J. T. Huber. 1980. Supplemental niacin for lactating cows fed diets of natural protein or nonprotein nitrogen. J. Dairy Sci. 63:2020.

Levitas, N., J. Robinson, F. Rosen, J. W. Huff, and W. A. Perlzweig. 1947. The fluorescent condensation production of N-methylnicotinamide and acetone. III. A rapid fluorometric method for the determination of the total pyridine nucleotides in the red blood cells. J. Biol. Chem. 167:169.

Loew, F. M., and R. H. Dunlop. 1972. Induction of thiamin inadequacy and polioencephalomalacia in adult sheep with Amprolium. Amer. J. Vet. Res. 33:2195.

Lusby, Keith S. 1971. Hyperalimentation in lambs: A model for the study of polioencephalomalacia. M.S. Thesis. Kansas State University, Manhattan.

Mizwicki, K. L. 1976. Niacin and nitrogen metabolism in sheep. M.S. Thesis. University of Illinois, Urbana.

Mizwicki, K. L., F. N. Owens, H. R. Isaacson, and B. Shockey. 1975. Supplemental dietary niacin for lambs. J. Anim. Sci. 41:411 (Abstr.).

Morrison, M. A., M. S. Reynolds, and A. E. Harper. 1963. Effect of increments of tryptophan and niacin on growth and on the concentration of blood and liver pyridine nucleotides. J. Nutr. 63:441.

Nagaraja, T. G., T. B. Avery, E. E. Bartley, S. J. Galitzer, and A. D. Dayton. 1981. Prevention of lactic acidosis in cattle by lasalocid or monensin. J. Anim. Sci. 53:206.

National Research Council. 1978. Nutrient requirements of domestic animals. No. 3. Nutrient requirements of dairy cattle. 5th edition. National Acad. Science 43:329.

Oltjen, R. R. 1969. Effects of feeding ruminants non-protein nitrogen as the only nitrogen source. J. Animal Sci. 28:673.

Piva, G., M. Amerio, R. Fabbri, E. Bergonzini, and F. Martillotti. 1976. Su taluni effecti della nicotinamide nella produzine del latte. Scienza E. Tecnica Lattiero-Casearia 27:287.

Riddell, D. O., E. E. Bartley, and A. D. Dayton. 1980. Effect of nicotinic acid on rumen fermentation in vitro and in vivo. J. Dairy Sci. 63:1429.

Riddell, D. O., E. E. Bartley, and A. D. Dayton. 1981. Effect of nicotinic acid on microbial protein synthesis in vitro and on dairy cattle growth and milk production. J. Dairy Sci. 64:782.

Riddell, D. O., M. J. Arambel, G. S. Dufva, E. E. Bartley, A. D. Dayton, T. G. Nagaraja, and G. W. Miller. J. Dairy Sci. submitted.

Sapienza, D. A. 1981. A hypothesis for the etiology of polioencephalomalacia. Ph.D. Dissertation. Kansas State University, Manhattan 66506.

Shields, D. R., T. W. Perry, and D. M. Schaefer. 1981. Niacin supplementation in lamb diets during adaptation to urea. Purdue Agr. Exp. Sta. Sheep Day Report, April 11, pages 7-10.

Shields, D. R. and T. W. Perry. 1981. Effect of supplementation niacin on protein digestion in growing and finishing lambs. Purdue Agr. Exp. Sta. Sheep Day Report, April 11, pages 3-5.

Virtanen, A. I. 1963. Produktion der Kuhmilch ohne Protein mit Harnstoff und Ammoniumslazen als Stickstoffquelle und gereingten Kohlenhydraten als Energiequelle. Biochem. Z. 338:443.

TABLE 1. Effect of niacin on average daily feed intake and milk production in fresh cows.

DIETARY PROTEIN	PROTEIN SUPPLEMENT	NIACIN	INTAKE		MILK[a]
			GRAIN	HAY	
%		(g)	(kg)		
15.9	SBM	5	16.5	7.5	31.6[b]
15.8	SBM	0	15.7	7.3	28.7[c]
16.6	UREA	5	14.2	7.2	26.6[d]
16.5	UREA	0	15.1	7.1	28.2[c]
8.8	NONE	0	14.0	7.5	25.5[d]

[a]AVERAGE DAILY MILK PRODUCTION (8 COWS/GROUP) FOR WEEKS 1-7 AFTER CALVING.

[b]MEANS WITHIN COLUMNS SHOWING DIFFERENT SUPERSCRIPTS DIFFER ($P<.05$).

TABLE 2. Effect of nicotinic acid treatment on ketosis (mean of eight cows). (Fronk and Schultz, 1979)

MEASUREMENT	DAY 0		DAY 7	
	$\bar{X}$	SE	$\bar{X}$	SE
MILK (KG/DAY)	30.1	1.47	34.1	3.64**
GLUCOSE (MG/100 ML)	43.1	8.1	50.6	7.69*
β-HYDROXYBUTYRATE (MG/100 ML)	10.3	2.96	6.7	3.09**
FREE FATTY ACIDS (MG/100 ML)	21.1	10.5	11.3	5.27**

[a]12 g NICONTINIC ACID ADMINISTERED DAILY DIVIDED INTO TWO EQUAL AM AND PM DOSES, STARTING ON DAY 0.

*P<.10.

**P<.05.

TABLE 3. Effect of niacin on milk production, and blood glucose, betahydroxy butyric acid and nonesterified fatty acids in fresh cows.

MEASUREMENT	TREATMENT	WEEKS AFTER CALVING 1	2	3	4
MILK (kg)	CONTROL[a]	21.4	26.8	28.2	29.0
	NIACIN[b]	19.5	28.6	30.9	32.3
BLOOD GLUCOSE	CONTROL	57	59	60	62
(mg/100 ml)	NIACIN	62	61	63	64
BETA-HYDROXY-	CONTROL	8.5	10.0	7.0	5.7
BUTYRATE (mg/100 ml)	NIACIN	6.0	5.3	4.5	4.3
NEFA (mEq/l)	CONTROL	.59	.38	.37	.27
	NIACIN	.45	.38	.25	.24

[a] 10 COWS PER GROUP

[b] 10 COWS PER GROUP - 12g NIACIN/COW/DAY

TABLE 4. Effect of high concentrate or high roughage substrates containing soybean meal or urea, with or without niacin, on microbial protein synthesis in vitro.

NUTRIENT CONTENT OF SUBSTRATE			NIACIN CONCENTRATION	MICROBIAL PROTEIN SYNTHESIS DIFFERENCE[b]	
ROUGHAGE	GROUND CORN	NITROGEN SOURCE			
(%)			PPM	(mg/g)	
				±	SE
20	57	SBM 23	0		
			100	2.89	.68
20	77	UREA 3	0		
			100	-1.10	.61
80	00	SBM 20	0		
			100	2.26	.54
80	17	UREA 3	0		
			100	- .72	.78

[a]Mean of 9 in vitro runs (3 animals each used 3 times) in duplicate.
[b]Difference in synthesis (100 ppm - 0 ppm niacin).

TABLE 5. Effect of heated soybean meal, with or without niacin, on rumen protozoal counts obtained between 0 and 6 h after feeding.

DIET	HOLOTRICHS	ENTODINIOMORPHS	TOTAL
	($X10^3$/ml)		
UNHEATED SBM WITHOUT NIACIN	28.9^{ab}	568^{c}	597^{c}
UNHEATED SBM WITH NIACIN	29.0^{ab}	605^{c}	634^{c}
HEATED SBM WITHOUT NIACIN	21.9^{a}	437^{d}	459^{d}
HEATED SBM WITH NIACIN	32.6^{b}	695^{e}	728^{e}

^{a,b}MEANS IN SAME COLUMN WITH DIFFERENT SUPERSCRIPTS DIFFER ($P<.05$)

^{c,d,e}MEANS IN SAME COLUMN WITH DIFFERENT SUPERSCRIPTS DIFFER ($P<.01$)

TABLE 6. Effect of niacin on niacin concentration of red blood cells and daily milk production.

	NIACIN IN RED BLOOD CELLS		MILK PRODUCTION	
	CONTROL	TREATMENT	CONTROL	TREATMENT
	(g/ml)		(g/ml)	
PREPARTUM	15.8	15.5		
WEEKS POSTPARTUM				
1	13.2[b]	15.4	21.8	23.2
2	14.3[c]	15.3	27.1	30.0
3	14.4[c]	15.3	27.1	32.7
4			29.2[d]	33.7[e]

[a]Eight animals per group.

[b]Different from prepartum ($P<.01$).

[c]Different from prepartum ($P<.05$).

[d,e]Paired means within rows showing unlike superscripts differ ($P<.08$).

Chronic Vitamin D_3 Toxicity in Dairy Cattle

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Vitamin D supplementation is required for all animals housed in absence of sunlight. Exposure to sunshine 1-2 hours per day will prevent rackets. The requirements for dairy cattle range from 600 IU per day of Vitamin D in calves to 6000 in lactating dairy cattle. Its primary functions are to regulate calcium absorption from the gut and resorption and mineralization of bone. Its hormonal metabolites function in concert with parathyroid hormone and calcitonin to maintain serum calcium between 8 and 10 mg/dl. Common recommendations are for 2 to 4 times the NRC requirements, 12-25,000 IU of Vitamin D per day. There is no data available to show that feeding over 12,000 IU per cow per day will improve calcium availability or improve milk production.

The rationale for supplementing higher or "mega" levels come from at least 5 different reasons.

1. **Milk fever prevention**

   Oral injections of 20 to 30 million IU of Vitamin D_2 (Hibbs and Conrad) or 10 million IU of D_3 administered in a single intramuscular injection will reduce the incidence of milk fever (Manston and Payne). Soft tissue calcification became a serious side effect unless oral dosage was discontinued after 7 days or a balance in calcium phosphorus and magnesium intake was controlled. Hibbs and Conrad reported that feeding 35,000 IU of Vitamin D_2 per lb. of concentrate year around reduced the incidence of milk fever in problem herds, however cows with no previous history of milk fever had an increased incidence of milk fever.

2. **Overage in supplements to insure meeting label claims**

   Of unknown importance is the excess Vitamin D concentration formulated into various feeds in order to meet label claims. This was a possible source because of studies by Waibel and others that some sources of Vitamin D were of lower biological activity than the chemical assay indicated. Current practices are unknown, however we do not know of any instances where this is a serious problem.

3. **Several supplemental sources or accidental toxicity**

   On a few occasions in field problem investigations, several sources of supplemental vitamins were present, resulting in Vitamin D levels over 300,000 IU per day based on label claims.

4. **To increase milk production**

   In the arena of nutritional consultants and ration balancing

there is the "claim" by a few nutritionists and veterinarians that when high amounts of Vitamin D are supplemented (200,000 IU per day or more) there is an increase in blood levels of calcium and that this in turn will increase milk production. The data to substantiate this claim are not known.

5. **"If a little is good, a lot is better"**

   Shotgun and excessive dosages of vitamins and drugs are rarely critically evaluated other than clinical impressions and often are accompanied by other changes which makes scientific evaluation impossible.

Diagnosis of Vitamin D toxicity is difficult and requires a thorough investigation of the feeding program and laboratory confirmation. Classically, soft tissue mineralization or calcification must be demonstrated. In experimental studies a variety of symptoms or problems may be encountered which mimic many other diseases (Capen, Conrad, Hibbs, Payne, Manston, Littledyke & Horst). The signs include anorexia, depression, polydypsia, poyuria, firm dry feces, weight loss, shedding of winter hair coat, abnormal cardiac function, decreased milk production and apparent muscle or joint stiffness manifested by cows getting up slowly and moving with a stiff or slow gait. Experimentally, sudden deaths have been observed with no signs of Vitamin D toxicity prior to death (Littledyke and Horst). The etiology of these signs are not well understood, but may be due to cellular toxic effects of Vitamin D or its metabolites or to persistant hypercalcemia. Vitamin D_2 toxicity has been produced experimentally by feeding 20-30 million IU of D_3 per day for more than 7 days. Signs and lesions of D toxicity were observed in 3-4 weeks (Hibbs and Conrad). Littledyke and Horst produced Vitamin D_3 toxicity by administering 2 injections of 15 million IU of Vitamin D_3, 7 days apart. The mortality rate was 58% (Table 5).

Chronic toxicity was not thought to be a problem as evidenced by the work on metabolized milk where up to 700,000 IU of Vitamin D_2 were fed daily year around to achieve over 400 IU of Vitamin D activity per quart of milk. Hibbs and Conrad fed 162,000 IU of D_2 per day year around and observed no problems. The first suggestion that Vitamin D_3 may cause a chronic toxicity syndrome was in 1974 when it was suggested as the cause of a herd problem when 50-90,000 IU of D_3 was being fed plus injections of D_3 were being administered at drying off and just before parturition. At that time the metabolic profile test revealed hypercalcemia and hyperphosphatemia. A histopathologic diagnosis of hypervitaminasis D was made in a bone specimen from a 16 month old bull from that farm. This diagnosis of chronic Vitamin D_3 toxicity was not accepted by the farmer or the scientific community.

Over the past 8 years, poor production, sudden deaths, emaciation and possible increased susceptibility to common diseases were observed in several herds on an unusual feeding program (Table 1). The feeding program provided a balancer to be fed with corn silage to make a complete ration. Assessment of its nutritional quality is provided in Tables 2 and 3. It is evident that the ration is deficient in energy, protein and phosphorus for 70 lbs. of milk production. In addition Vitamin D_3 and Vitamin A were fed at 200 and at 50 times respectively of the NRC recommended allowances. All of these herds were assessed by metabolic profile testing (Stevens et al). Every herd was anemic. Statisically significant hypercalcemia and hyperphosphatemia was present in individual herds. All calcium, phosphorus, magnesium, and hematocrit values

from 5 herds were combined according to milk production and analyzed (Table 4). The increased calcium and phosphorus concentrations were highly significant. Magnesium was slightly lower and enemia was highly significant in cows over 40 lbs. of milk production.

Plasma Vitamin D_3 and metabolite concentrations were highly elevated in these herds receiving over 1×10^6 IU of Vitamin D_3 per cow per day (Table 5). It is apparent that chronic feeding of excess levels of Vitamin D_3 for 1 year or more results in elevated plasma levels of all of the metabolites except for $1,25(OH)_2$ D_3, when compared to control and acute toxicity studies by Littledyke and Horst. The consistancy of the data between herds and comparing the composite data to the acute toxicity is evidence for chronic toxicity. The lack of soft tissue calcification may be explained by the presence of 2.4×10^6 IU of Vitamin A and the deficient supplementation of minerals and protein. The severe anemia may be due to either Vitamin D or Vitamin A toxicity or protein and/or phosphorus deficiency. Iron deficiency was determined not to be a problem because of adequate iron content in bone marrow specimens.

Causes of high incidences of sudden death are unknown in these herds. Anemia and hypercalcemia may be related to the observed cardiac abnormalities however there is speculation that the presence of 25 OH, 23-26 lactone may be involved in the sudden death syndrome here. The lactone ring (formed from the 23 and 26 carbons on the 25,OH cholecalciferol side chain, Horst) is identical to the lactone ring in digitalis. The importance of this observation is unknown.

The immediate and obvious question because of wide spread Vitamin D_3 supplementation is "how much is too much". Continuing studies in field problem herds have revealed that when 80,000 IU of Vitamin D_3 are fed daily and injections of Vitamin D_3 are administered, variable hypercalcemia, hyperphosphatemia and anemia have been observed along with feet and leg problems and above average mortality rates. The problems have as a clinical observation been relieved when vitamin supplementation was reduced to recommended levels.

In Table 6 are the Vitamin D_3 and metabolite concentrations in herds receiving 1×10^5 to over 2.5×10^5 IU of Vitamin D_3 per cow per day. Hypercalcemia and hyperphosphatemia was present in varying significance in all of these herds.

Certainly, feeding 80,000 IU of Vitamin D_3 per cow per day can be associated with higher blood concentrations of the vitamin and its metabolites, and serum calcium concentrations. The NRC recommended level is 6000 IU per day. Our best guess is that whenever supplementation exceeds 100,000 IU per cow per day there is a potential for problems and this potential may increase with each incremental increase in supplementation and/or if parenteral injections are administered.

The claim for any milk production benefit has not been substantiated by scientific studies. On the contrary studies by Conrad have shown no greater calcium digestability when Vitamin D supplementation was at 40,000 IU per day versus 12,000 IU per day.

Thus the most commonly recommended range of 10-30,000 IU per cow per day seems to be realistic and allow for a safety factor in a wide range of feeding programs.

Literature Cited

Capen, C.C., C.R. Cole, and J.W. Hibbs (1968). Influence of Vitamin D on calcium metabolism and the parathyroid gland, Fed. Proc., 27:143.

Conrad, H.R., Personal communication.

Hibbs, J.W. and H.R. Conrad (1960). Studies on milk fever in dairy cows. III. Effect of three prepartal dosage levels of Vitamin D on milk fever incidence. J. Dairy Sci., 43:1124.

Horst, R.L. (1979). 25 OH D_3 26-23 Lactone: A metabolite of Vitamin D_3 that is 5 times more potent than 25 OH D_3 in the rat plasma competitive binding assay. Biochem. Biophys, Res. Comm. 89:286.

Littledyke, T. and R.L. Horst (1982). Vitamin D_3 toxicity in dairy cattle. J. Dairy Sci. 65:715.

NAS/NRC Nutrient Requirements of Dairy Cattle, 1977.

Payne, J.M. and R. Manston (1967). The safety of massive doses of Vitamin D_3 in the prevention of milk fever. Vet. Rec. 79:215.

Stevens, J.B., J.F. Anderson, W.G. Olson, et al (1976). Metabolic profile test design. Technical International Congress.

Waibel, P.E., H.S. Yang and J. Brenes (1971). Biopotency of Vitamin D_3 supplements. Proceedings. Minn. Nutr. Conf.

TABLE 1. HERDS INVESTIGATED

HERD	DATE	BREED	RHA	COMPLAINTS	NO. OF DEATHS	NO. OF NECROPSIES	YEARS ON PROGRAM
A	1974	G	?	THIN POSTPARTUM COWS, POOR PRODUCTION	?	?	?
B	1979	G	7,000	HIGH DEATH LOSSES, REPRODUCTION PROBLEMS, LOW PRODUCTION	7*	4	2
C	1980	H	11,000	UNEXPLAINED DEATH LOSSES, LAMENESS OR FOOT PROBLEMS	20	2	3
D	1980	G	?	HIGH DEATH LOSS DUE TO PNEUMONIA, DEPRESSED IMMUNE SYSTEM	(15)**	4	22
E	1981	H	12,000	HIGH DEATH LOSS, MASTITIS SUDDEN DEATHS UNEXPLAINED	15†	5	3
F	1981	H	11,800	HIGH DEATH LOSS, SUDDEN DEATHS UNEXPLAINED	23‡	4	5

*AS OF MAY, 1979.

**1979 TO OCTOBER, 1980.

†IN PREVIOUS 18 MONTHS.

‡15 IN 1980 AND 1981.

TABLE 2. NUTRITIONAL PROGRAM FOR 1300-LB COW PRODUCING 70 LB OF FCM

Ingredient	As Fed (lb)	DM Basis				
		Pro (%)	C. Pro (lb)	Net Energy (M cal)	Ca (gm)	P (gm)
Corn silage	33	8	2.64	24	51	30
Balancer	6	34	2.04	5	56	28
TOTAL	39		4.68	29	107	58

Vitamin D_3, 1.2×10^6 IU/day

Vitamin A, $> 2.4 \times 10^6$ IU/day

TABLE 3. PROGRAM PROVIDES PERCENT OF (%) OR TIMES (X) THE NRC REQUIREMENTS (1300-LB COW PRODUCING 70 LB FCM)

	Net Energy (M cal)	C. Pro (lb)	Ca (gm)	P (gm)	Vit. D. (IU)	Vit. A (IU)
NRC requirement	34	7.15	104	86	6,000	50,000
Program provides	29	4.68	107	58	1,200,000	2,400,000
% or X	84%	65%	102%	65%	200X	48X

Table 4

Comparison of Metabolic Profile Test Parameters in All Cows Sampled from 5 Herds Receiving > 1 x 10^6 IU Vit D_3 Per Day Compared to Base Population of 1502 Cows

Parameter	Production Level: Over 41 lbs FCM		Production Level: 20-40 lbs FCM		Dry Cows	
	Base	Field Case 5 herds	Base	Field Case 5 herds	Base	Field Case 5 herds
Ca	9.0	10.0	9.1	9.9	9.06	10.5
	± .38	± .35	± .39	± .49	± .38	± .85
	2.47 SDS	= .99	2.10 DSs	= .96	3.82 SDs	= .99
P.i	5.7	7.2	5.8	7.0	5.7	7.4
	± .82	± .07	± .72	± .60	± .73	±1.06
	1.8 SD	= .93	1.63 SD	= .90	2.33 SD	= .98
Mg	2.2	2.0	2.1	2.0	2.1	1.9
	± .20	± .29	± .20	± .31	± .16	± .14
	0.74 SD	= .54	0.54 SD	= .41	1.21	= .77
PCV	31.8	26.5	32.8	26.8	34.4	31.0
	± 1.64	± .71	± 2.03	1.79	± 2.03	
	3.22 SDs	= .99	2.97 SDs	= .99	1.65 SD	= .90

Table 5
Calcium Phosphorus and Vitamin D_3 and Metabolites in Blood of Cows Receiving Normal and Toxic Amounts of Vitamin D_3

	Normal[a]	Acute Toxicity NADC Study	Chronic Toxicity[b]
Death Rate	----	58%	0 - 25%
Calcium mg/100 ml	9.0 ± .38[c]	11.5 (10.5-13.0)	10.0 ± .35
Phosphorus mg/100 ml	5.8 ± .8[c]	7.5 (6.0-9.0)	7.2 ± .7
Vitamin D_3 ng/ml	1.7 ± .67	27 ± 6	95 (8-262)
25 OH D_3 ng/ml	37 ± 7.1	205 ± 18	298 (218-424)
1,25 $(OH)_2$ D_3 pg/ml	38 ± 10	187 ± 43	44 (24-74)
24,25 $(OH)_2$ D_3 ng/ml	2.6 ± 1.3	40 ± 8	41 (29-101)
25, OH-26-23 Lactone ng/ml	0	13 ± 2.6	12 (0.5-34)

a 5 adult cows receiving diet containing NRC requirements for calcium, phosphorus, and Vitamin D_3.

b .8 to 1.4 x 10^6 IU of Vitamin D_3 per cow per day for 1 year or more.

c Normal values for 1502.

TABLE 6

Observed Range of Vitamin D_3 and Metabolite Levels in Plasma from Cows Receiving 100,000 to 250,000 IU of Vitamin D_3 Per Cow Per Day

Herd	IU/day	N	D_3	25-OHD_3	1,25$(OH)_2D_3$	Lactone
			ng/ml			
CA	10^5	7	3.0-6.6	31-76	.073-.133	.04-.13
RR	1.5×10^5	4	10.0-24.5	40-83	.058-.077	.00-.12
GC	10^5	7	9.4-31.3	35-69	.046-.103	.00-.06
MM	2.5×10^5	7	37-48	56-175	.021-.070	0

VITAMIN E NUTRITION OF RUMINANTS

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Vitamin E is recognized as an essential nutrient for all species of animals. However, differences of opinion exist among research workers as to the conditions under which vitamin E supplementation is required and at what levels it should be fed.

Vitamin E for many years after its discovery was believed to be the fertility vitamin. Subsequent investigations over the past 50 years have disclosed that vitamin E is essential for a variety of metabolic functions and is required for the prevention of a number of deficiency conditions in different species of animals (Table 1).

A discussion of some of the chemical and biological properties of vitamin E may give us a better idea of how to evaluate vitamin E nutrition of ruminants.

The chemical name for vitamin E is tocopherol and it is found in nature as a high molecular weight alcohol. We generally think of vitamin E as a single entity, but there are several known forms occurring in nature. The most important and one of greatest practical significance is alpha-tocopherol which should be considered true vitamin E because it has the greatest biological activity of all known forms. The other forms vary in biological potency from one-third to one-hundredth that of alpha-tocopherol (Table 2).[1]

Unless the alpha-tocopherol content of a feedstuff is known, the biologically available vitamin E present in a ration can be overestimated. There are few feedstuffs available which contain high levels of alpha-tocopherol. Alfalfa meal is an exception.[2] See Table 3 for the biologically active tocopherol content of several common feedstuffs used in cattle feeds.

Several factors are interrelated with the function of vitamin E. Some are classified as anti-metabolites or antagonists, while others are recognized as co-factors or sparing agents.

In consideration of the antagonists of vitamin E, the discovery that the addition of readily oxidized and/or unsaturated fat to an otherwise vitamin E adequate diet would cause encephalomalacia in chicks. This condition could be prevented by the addition of vitamin E to the diet, indicating that fats would either destroy the vitamin E present or interfere with its proper utilization.[3]

Later it was found that the polyunsaturated fatty acids increased the physiological requirement for vitamin E.

It is now well established that the unsaturated fat, linoleic acid, is primarily responsible. Injections of linoleic acid into the body will increase the vitamin E requirement, indicating that at least part of the antagonistic action occurs within the animal body and is not related to the effect of fat on the stability of vitamin E in the ration.[4]

Another vitamin E antagonist of unknown identity has been extracted from alfalfa. It is an alcohol-soluble, heat resistant compound which reduces by about two-thirds the availability of naturally occurring vitamin E for the chick. A similar factor has been found in red kidney beans which reduces vitamin E utilization in chicks and sheep. Evidence indicates that these factors may exist in certain other forages as well.

Of the several compounds identified as vitamin E co-factors or sparing agents, selenium is the most important. Perhaps it is best to describe vitamin E and selenium as co-factors which are interdependent, although they may have different metabolic roles. Research data demonstrating this interrelationship has shown that selenium alone is not completely effective in preventing nutritional muscular dystrophy in chicks, rabbits, and lambs. In some trials, selenium treatment has initially improved white muscle disease in lambs, but failed to maintain improvement unless vitamin E was given also.[5]

Earlier research reported that muscular dystrophy in lambs and calves, when induced by adding unsaturated fat to the ration or with spontaneous outbreaks in the field, could be corrected by treatment with vitamin E. In other occurrences, particularly as experienced in the Northwest, the best response to nutritional muscular dystrophy in lambs is obtained with selenium treatment.[6] It would appear that nutritional muscular dystrophy and other disorders can be caused by either a deficiency of selenium, vitamin E, or both (Table 4).[7]

REQUIREMENTS

The 1978 NRC vitamin E requirement of dairy calves is 300 I.U./kg (136.4 I.U./lb) of ration. Vitamin E requirements for the other classes of dairy cattle are not included in the 1978 NRC publication.[5]

The 1976 NRC vitamin E requirements of beef cattle range from 15 I.U./kg to 60 I.U./kg (6.8 I.U./lb to 27.3 I.U./lb) of ration.[8]

PERFORMANCE

The performance of growing beef cattle supplemented with vitamin E has varied depending on feeding program. Several earlier reports have indicated improved rate of growth and in most cases improved feed conversion when cattle received supplemental vitamin E. More recent observations have shown no improvement in growth when finishing rations were supplemented with vitamin E at 100 I.U./head/day especially when total vitamin E intake from feedstuffs was increased.[9-16]

In a 110 day growing trial to determine the effects of supplementing vitamin E at 100 I.U./head/day, steers were fed processed feedstuffs (steamed-flaked grain sorghum, pelleted supplements, ammoniated residue and corn silage). Control steers gained less ($p < .05$) and used feed less efficiently during the first 66 day of the trial when they consumed 0.47 I.U. of E per kg of body weight than did the supplemented animal receiving 0.74 I.U. of vitamin E per kg of B.W. During the final 44 days of the trial ammoniated corn stover were substituted for ammoniated wheat straw. During this period, control steers consumed more vitamin E (0.86 I.U./kg B.W./day than they did during the first 66 days. At the end of the trial control steers and supplemented steers had similar gains and feed efficiency. The total vitamin E intake was 148 and 295 I.U./head/day for the control steers during the first period and second period, respectively. Total intake was 233 and 357 I.U./head/day for supplemented steers for the same periods.[27]

The best criteria for evaluating the vitamin E status of cattle has not been determined. Unlike calves, older cattle do not usually show gross symptoms of vitamin E deficiency, such as nutritional muscular dystrophy, thus a more accurate diagnostic method for assessing the vitamin E status of older cattle is needed.

Several investigators have reported plasma tocopherol values for normal animals and those suspected of being deficient. Cows producing calves with muscular dystrophy had plasma tocopherol levels of approximately 0.24 mg/100 ml.[17,18] Cattle responding to vitamin E supplementation in one field trial had plasma levels of less than 0.3 mg alpha-tocopherol per loo ml plasma or less.[19] Cattle on pasture appear to consume adequate vitamin E. Plasma values for pastured cattle are usually above 0.5 mg/100 ml of plasma.[20]

A field survey conducted with dry lot beef cattle indicated that plasma alpha-tocopherol levels ranged from 0.01 to 2.2 mg/100 ml. Sixty percent (60%) of the total 286 individual samples assayed below 0.3 mg alpha-tocopherol per 100 mg and nearly 40% of the samples assayed below 0.2 mg/100 ml. In one case, cattle fed high roughage rations had the lowest plasma alpha-tocopherol level ranging between 0.06 to 0.13 mg/100 ml for 9 animals sampled. The vitamin E intake calculated from alpha-tocopherol assay of the total diet was approximately 61 I.U./head/day.

In a separate location cattle fed a low roughage ration consumed approximately 190 I.U. vitamin E/head/day and their plasma alpha-tocopherol ranged between 0.17 to 0.28 mg/100 ml with an average value of 0.22 mg/100 ml for seven animals sampled.[17]

Feeding supplemental vitamin E is reported to be helpful in preventing oxidized flavor in milk.[21-24] In one study, 30 mcg of tocopherols/g of milk fat was necessary to protect against oxidized flavor.[25]

The vitamin E status of dairy cows was monitored over a four year period to determine if feeding dairy cows only stored feeds could cause a deficiency of vitamin E. Low E cows were fed stored feeds all year; whereas, the pastured cows grazed during summer. There were no signs of vitamin E deficiency although plasma tocopherol of low E cows was lower than that of cows pastured. Total tocopherol in milk from low E cows remained below 25 mcg/g fat except during the fall when crop hay and silage contained more vitamin E than feeds stored longer. Total-tocopherols in milk from pastured cows increased to about 50 mcg/g fat but declined to less than 30 mcg/g fat by winter. There were no differences between low E and pastured cows in incidences of retained placentas or reproductive efficiency.[26]

Table 1. Conditions Resulting from a Vitamin E Deficiency

Testicular degeneration
Embryo degeneration
Exudative diathesis
Anemia
Red Blood cell fragility
Encephalomalacia
Muscular degeneration
Necrotic liver degeneration
Kidney tubule degeneration
Stomach ulcers
Steatitis (yellow fat disease)
Oxidative rancidity-meat & milk
Bone deformity
Depigmentation of teeth
Nerve degeneration
Impaired utilization of vitamin A and carotene
Impaired utilization of protein

Table 2. Relative Biological Activity of the Tocopherols (Rats)

Form Tocopherol	Relative Biopotency	Primary Source
Alpha	100	wheat germ, alfalfa
Beta	35	wheat germ
Gamma	1	corn
Delta	1	soybean
Epsilon	5	bran
Zeta	30-100	rice & barley
Eta	1	rice

Table 3. Alpha-Tocopherol Content of Several Feedstuffs

Feed	No. Samples	High Value	Low Value	Avg. All Samples	I.U. lb.
Alfalfa Hay	2	27.7	23.8	23.9	35.6
Barley	6	19.4	9.85	16.5	24.6
Corn	17	15.87	5.17	9.03	13.5
Cottonseed Meal	6	7.3	1.14	4.18	6.2
Milo	12	7.2	4.63	6.76	10.1
Molasses	3	3.9	1.54	2.45	3.6
SBM-44%	8	2.22	0.68	1.36	2.0
Wheat	4	6.62	1.45	5.04	7.5
Wheat Bran	2	8.63	6.9	7.76	11.6

Table 4. Vitamin E and Selenium Deficiency Disease of Ruminants

Disease	Animal	Tissue Affected	Prevented by Dietary Vit. E	Se
Reproductive Failure				
Fetal death, resorption	Cow, Ewe	Embryonic Vascular system	X	X
Nutritional Myopathies				
Stiff lamb disease	Newborn lamb	Striated muscle		X
NMD	Sheep,goat, calf	Striated muscle	X	X
Systemic disorders				
Accumulation of ceroid	Calf,lamb	Adipose	X	not confirmed

Table 5. Guidelines for Vitamin E Allowance for Dairy and Beef Cattle I.U./head/Day

Dairy Cattle	NRC 1978	Field Survey Levels (Total)	Roche (Supplemental)
Calf starter feed	-	0-19.5	20.0
Lactating cows and Breeding bulls	-	0-100	80-100
Dry Cows	-	0-100	60-80
Growing Heifers and Bulls	-	0-10	25-35
Beef Cattle	**NRC 1976**	**Field Survey Levels (Total)**	**Roche (Supplemental)**
Growing and Finishing Steers and Heifers	122-490	190-750	20-50
Pregnant Cows and Heifers	136-544	6-584	20-100
Breeding Bulls and Lactating Cows	177-708	80-1146	20-100

LITERATURE CITED

1. Draper, H. H. Fat Soluble Vitamins, International Encyclopedia of Food and Nutrition, Vol. 9. Edited by R. A. Morton. Elmsford, N.Y. Pergamon. 1970.

2. Bunnell, R.H., J. P. Keating and A. J. Quaresimo, 1968. Alpha-Tocopherol Content of Feedstuffs. J. Agr. Food Chem. 16: 659.

3. Marusich, W. 1967. Supplemental Vitamin E Reduced Incidence of Encephalomalacia in Broilers. J. Poultry Sci. 46 (3) 541.

4. Dam, H., E. Sondergaard, G. Holmer, and E. Leerbrock, 1966. Production of Encephalomalacia in Chicks with Diets Containing Vegetable Oils. Sonderdruck Aus, Zeitschrift für Ernahrung-swissenschaft 7:50.

5. Nutrient Requirement of Dairy Cattle No. 3. 5th Rev. Ed., National Research Council/National Academy of Science, 1978.

6. Muth, O. H., J. E. Oldfield, L. F. Remmert, and J. P. Schubert 1958. Effect of Selenium and Vitamin E on White Muscle Disease. Science 128:1090.

7. Scott, M. L. 1979. Advances in our Understanding of Vitamin E. Federation Proceedings. 39 (10) 2736.

8. Nutrient Requirement of Beef Cattle, No. 4. 5th Rev. Ed., National Research Council, National Academy of Science. 1976.

9. Perry, T.W., W.M. Beeson, M.T. Mohler and B. R. Tonroy, 1972, Ann. Indiana Cattle Feeders Day Report, Purdue University, p.25.

10. Perry,T.W., W.M. Beeson, M.T. Mohler and B.R. Tonroy, 1973, Ann. Indiana Cattle Feeders Day Report, Purdue University, p. 33.

11. Stob, M., W.M. Beeson, T.W. Perry and M.T. Mohler, 1974, Ann. Indiana Cattle Feeders Day Report, p. 33.

12. Chapman, H.L., Jr., R.L. Shirley, A.Z. Palmer, and J.W. Carpenter, 1971, Bulletin 748, Fla. Agri. Exp. Sta., Univ. of Florida.

13. Beeson, W.M., T.W. Perry, W.H. Smith and M.T. Mohler, 1962, J. Anim. Sci., 21: 988.

14. Beardsley, D.W., 1968, Proc. Univ. of Fla. Beef Cattle Short Course.

15. Iowa State University, 1964, Proc. Cattle Feeder Day.

LITERATURE CITED

16. Cattle Feeder's Day 1982. Garden City Branch Agricultural Experiment Station. Kansas State Univ.

17. Adams, C.R., 1982. Feedlot Cattle Need Supplemental Vitamin E. Feedstuffs 54 (18) 24-25.

18. Safford, J.W. and K.F. Swingle, 1955. Amer. J. Vet. Res. 16:64.

19. Sheldon, J.J. 1980. Proc. Roche Seminar, Vitamin Nutrition for Cattle, AFMA Liquid Feed Symposium, Dallas, Texas.

20. Technical Bulletin # 139, Virginia Agr. Expt. Station, 1954.

21. Krukovsky, V.N. and J.K. Loosli, 1952, Further Studies on the Influence of Tocopherol and Supplementation on the Vitamin Content of Milk Fat, Stability of Milk, and Milk and Milk Fat Production, J.Dairy Sci. 35:834.

22. Neilsen, J., A.N. Fisher and A.H. Pedersen, 1953, the Influence of Feeding Tocopherol to Dairy Cows on the Yield of Milk and Milk Fat and on the Tocopherol Content and Keeping Quality of Butter, J. Dairy Res. 20:333.

23. Krukovsky, V.N., F. Whiting and J.K. Lossli, 1950, Tocopherol, Carotenoid, and Vitamin E Content of Milk Fat and the Resistance of Milk to the Development of Oxidized Flavors as Influenced by Breed and Season, J. Dairy Sci. 33: 791.

24. Dunkley, W.L., M. Ronning, A.A. Franke and J. Robb, 1967, Supplementing Rations with Tocopherol and Ethoxyquin to Increase Oxidative Stability of Milk, J. Dairy Sci., 50:492.

25. Kurkovsky, V.N., J.K. Loosli and F. Whiting, 1949, The Influence of Tocopherols and Cod Liver Oil on the Stability of Milk, J. Dairy Sci. 32:196.

26. Schingoethe, D.J., J. G. Parsons, F.C. Ludens, W.L. Tucker and H.J. Shave. 1978. Vitamin E Status of Dairy Cows Fed Stored Feed Continuously or Pastured During Summer. J. Dairy Sci. 61:1582.

27. Davis, G.V. 1982. Vitamin E Supplementation of Growing Rations Containing Ammoniated Residues, Cattle Feeder's Day Report of Progress 416. Kansas State University, p. 24.

EGG SHELL QUALITY

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How much money is lost each year due to poor egg shell quality? It is impossible to give an exact answer but it is estimated that between 361 and 550 million dollars are lost annually. These estimates are based on reports of 11 researchers (Table 1). The percentage of eggs lost range from a low of 4% to a high of 12.15%. Based on a loss value of 40 cents per dozen, 290 million hens and 20 dozen eggs per hen per year, the low estimate would be 93 million dollars and the high estimate would be 282 million dollars with an average loss of 148 million dollars. It is believed that these estimates include only eggs which were broken between collection and consumer and did not include shell-less eggs or eggs that fall through the cage into the manure. In 1977, studies by Roland indicated that for every 100 eggs collected, 7.77 eggs are not collected. Because these eggs have no salvage value, they were valued at 60 cents per dozen instead of 40 cents. Thus, we have another 268 million dollar loss, which gives estimates ranging from 361 million to 550 million dollars with an average of 416 million dollars per year.

Realizing that this value is only an estimate influenced by several variables and can vary between producers, it still gives a definite indication of the severity of the shell quality problem.

It is impossible with current knowledge to correct all shell quality problems, but it is possible to make significant reductions in the number of eggs lost due to poor shell quality. The purpose of this presentation is to give an idea of how much of the shell quality problem (cracked eggs due to thin shells or excess pressure and body-checks) can possibly be eliminated and to describe a five step program as to how this can be achieved.

STEP 1: ELIMINATE THE 20 MOST COMMON MISMANAGEMENT PRACTICES ADVERSELY AFFECTING SHELL QUALITY

There are two major causes of broken and body-checked eggs. The first and perhaps major cause is by man through mismanagement, and the second is the natural decline in shell quality caused by aging. In many instances the shell quality problem caused by man's mismanagement of pullets and hens is overlooked when attempting to solve a shell problem. The reason is that some producers want to believe that the cause is due to some unknown, complicated problem. Although this is true in some instances, I believe that at least 50% of all cracked and body-checked eggs are due to common mismanagement practices and could be eliminated by proper management using a good total shell quality program. This is not a simple program. It involves more than waiting until you have a shell quality problem and then trying to solve it. It will take considerable effort from everyone in a company.

The mismanagement practices that can affect shell quality can be further divided into two categories. First are the mismanagement practices that affect the health of the bird causing a pre-mature decline in shell thickness. Second are the mismanagement practices that cause eggs to break due to excess force.

Obviously, all mismanagement practices and other factors affecting shell quality cannot be listed; however, 20 of the most common mismanagement practices are listed in Table 2 and examples of each will be discussed during the presentation.

I believe that if these 20 common mismanagement practices are eliminated much of the shell breakage could be corrected.

STEP 2: DO NOT OVER FEED HENS SULFUR AMINO ACIDS WHICH CAN CAUSE GREATER THAN NEEDED EGG SIZE RESULTING IN A PREMATURE REDUCTION IN SHELL THICKNESS

Although many factors are known to cause a premature decline in shell quality, it was not until recently that the major reason for the decline in shell quality due to aging was determined. In 1977, it was discovered that a disproportionate increase in egg size and shell deposition was responsible for the decline in shell quality.

When shell weight was determined on hens 3, 6, 9, and 12 months in lay, it was found that shell weight at 3 months in lay (5.23 g) did not decrease but remained constant or even slightly increased to 5.38 g at 12 months in lay (Table 3). However, egg weight increased from 55.9 g to 64 g. Because the hens had to spread this fairly constant amount of shell around 8 more grams of egg, the specific gravity decreased. This increases egg breakage for two reasons. First egg shell thickness declines and second, a larger egg is more susceptible and more easily broken than a smaller egg.

Since many studies that tried to increase shell deposition above normal have failed, it was believed that it would be easier to improve shell quality by reducing egg size and maintain shell deposition. In other words, can we reduce egg size from an extra large or jumbo egg to a large egg, 57 g, the weight at 6 months in lay, and maintain it at this size without reducing the amount of shell deposited or egg production? This should return or improve egg shell quality to that at the same stage.

In one experiment, dietary protein levels of 20, 16, 13.5 and 11.5% with 3% calcium and 2.84 ME cal/g were used. Decreasing the dietary protein from 16 to 11.5% decreased egg weight 3.3 g within 8 weeks (Table 4). However, shell weight decreased as the protein content of the diet decreased which resulted in no improvement in egg specific gravity.

So in another experiment, the same rations were fed except that the calcium level was increased from 3 to 4.25%, hoping that this would prevent any reduction in shell weight as egg size decreased, thereby improving shell quality. Similar effects were also observed. Egg weight was significantly reduced in hens fed 11.5% protein. Shell weight also declined as egg weight decreased which resulted in no improvement in specific gravity. Eight other dietary treatments were also used in other experiments in which we were able to reduce egg weight; however, shell weight went down in the same proportion as egg weight, resulting in no change in shell quality.

The next question was, since it appears difficult to reduce egg weight and increase shell quality, could one decrease the normal rate of increase in egg weight in young birds and prevent the decline in shell quality? Yes, when the increase in egg size was reduced by dietary manipulation no decrease in shell quality was observed as the hen aged; however, we were

unable to accomplish this without adversely affecting production.

Recently, C. F. Peterson and co-workers at Idaho University conducted experiments similar to these; however, they waited until egg size reached a large egg, then fed low levels of methionine and were successful in improving shell quality. However, they did not reduce egg size. They simply reduced the normal increase in egg size without reducing shell deposition. They did this in two separate experiments without adversely affecting production. Peterson and co-workers obtained the greatest improvement in shell quality with an average intake of 255 mg methionine per hen per day. This level of methionine should only be taken as a guide because many factors can influence the requirement (previous levels fed, egg size desired, condition, and age of hen, etc.).

It was concluded from those studies that egg breakage can be reduced by controlling the increase in egg size. It was also suggested that reduced levels of protein and sulfur amino acids can result in a substantial savings in feed cost.

STEP 3: ENSURE THAT HENS CONSUME THEIR CALCIUM REQUIREMENT

Of all the nutrients, none will have a greater or faster adverse affect on shell quality than a deficiency of calcium. For example, a hen cannot go a single day or even a few hours (depending upon time of calcium deficiency) without dietary calcium and still maintain shell quality. Although calcium has been associated with shell quality for over 75 years, it is still the most common culprit either directly or indirectly involved in almost all thin shell problems. There are two reasons for this. First is the difficulty in establishing the calcium requirement, and second are the numerous mismanagement practices or factors that can prevent the hen from consuming the required amount of calcium even though the diet may be formulated correctly. The most common mismanagement practices associated with getting the correct amount of calcium into the hen are presented in Table 2. The most probable reasons for the difficulty in determining the hen's calcium requirement are listed below.

Changing genetics of the hen and the continued improvement in production capability;

Differences in calcium requirement among hens within strains and among strains;

Interrelationship of calcium with other nutrients, especially with phosphorus and vitamin D_3;

Interrelationship of particle size of calcium carbonate to calcium availability and palatability of feed;

Ability of the hen to partially adjust feed intake to meet her calcium requirement;

Calcium requirement has been reported by many researchers in terms of percentage in the diet without taking into account the variation in feed intake as influenced by energy level, environmental temperature, strains or age of bird, and a fear of the adverse effects of excess or inadequate consumption of calcium.

Because of the above factors, establishing the hens calcium requirement has been one of the most difficult to determine of the major nutrients. With this in mind, the calcium requirement recommendations in the following sections are based on the authors interpretation of published data and research conducted at Auburn during the past few years.

To ensure maximum shell quality, it is recommended that hens consume a minimum of 3.75 g of calcium per day. For older hens or hens with shell quality problems, the calcium intake should be increased by as much as 1 g depending on age and the severity and type of shell quality problem. The following guide is a general recommendation.

Starting at 1 week prior to the first egg until peak production, the birds should be fed a diet containing 3.75% calcium. This would normally be from approximately 19 to 28 weeks of age. From peak production until 36 weeks of age, feed 3.75 g Ca/hen/day. From 36 to 52 weeks of age the hens should be fed 4.0 g Ca/hen/day. From 53 weeks until the end of the production cycle the hens should be fed 4.25 g Ca/hen/day. Any time after peak production that shell thickness becomes abnormally thin, it is suggested that the calcium intake be increased to a maximum of 4.75 g /hen/day and/or part of the calcium supplied in the form of pullet or hen sized $CaCO_3$. An explanation for this particular program will be discussed in the presentation.

STEP 4: DETERMINE AND FOLLOW EGG SPECIFIC GRAVITY VALUES AT REGULAR INTERVALS JUST AS YOU DO EGG PRODUCTION AND FEED CONSUMPTION

When it is determined that an abnormal undergrade problem is due to thin shells we know that mismanagement practices associated with nutrition or health have adversely affected calcium metabolism. This can be either a direct effect caused by the hen consuming inadequate calcium or an indirect effect which alters calcium absorption and deposition. It is usually much easier to determine the cause of the direct effects and correct these type of problems than the indirect ones. For example, there may be many factors that may have adversely affected the bird before she reaches maturity (disease, management, nutrition) and these effects may not be noticeable or cause a severe problem until the hen has been in lay for several months. Because of this delayed effect it has been very difficult to pinpoint which mismangement practices prior to maturity are the major causes of subsequent shell quality problems. For this reason, it is very important to determine peak shell quality and follow shell quality at regular intervals just as you do production and feed consumption. By doing this it will help one to determine whether the problem occurred to the hens prior to maturity or after maturity. Over a period of time involving different flocks one can associate the cause of shell quality problems with specific mismanagement practices occurring to the birds prior to maturity.

STEP 5: ELIMINATE ENVIRONMENTAL FACTORS THAT CAN INCREASE THE INCIDENCE OF BODY-CHECKS

Body-checked eggs (BCE) are one of the major types of shell quality problems confronting the egg industry. They are of inferior quality because it takes 20% less pressure to break a BCE than a non-BCE. Until recently, little was known concerning the cause or methods of correcting BCE. In 1977, research conducted at Auburn indicated that the incidence of BCE was directly related to cage density and hen age with most BCE being laid between 6 am and 8 am.

Based on these results, it was hypothesized that most BCE were laid during the early morning because of a relationship between photoperiod, hen activity, and the critical period of shell calcification (CPSC). The CPSC is defined as the first few hours of shell calcification when the shell is ultra-thin and most susceptible to breakage. Results of additional experiments at Auburn indicated that this hypothesis was correct because by increasing or decreasing the photoperiod, the incidence of BCE could be increased or decreased. Also, the time of lay of BCE could be completely reversed by reversing the photoperiod.

Although reducing the length of the photoperiod in older hens for short periods significantly reduced the incidence of body-checkes, reducing the photoperiod was believed to be risky as far as egg production was concerned and could only be recommended under extreme conditions. Also, since light can't be reduced during the afternoon in an open-type house in Alabama in the summer, these data suggested that current lighting programs should be re-examined. Can hens obtain maximum production when supplied 14 or 15 hours light instead of 17-18 hours typically supplied and recommended by most management guides? Also, would a shorter photoperiod throughout lay continue to reduce the incidence of BCE?

To answer these questions, two experiments were conducted. A stimulight program starting with 15 hours total light and increasing 15 minutes at regular intervals until a maximum of 18 hours was compared to a constant 15-hour photoperiod. Fifteen hours was chosen because that is the shortest light day that one can have in an open-type house in Alabama without having increasing and decreasing light.

There were no significant differences in production for 12 months associated with photoperiod (Table 5). However, the hens supplied the shorter photoperiod in both cage densities laid approximately half as many BCE (Table 6). For example, at the end of 8 months, the hens housed three per cage and exposed to 15 hours of light laid only 4.4% BCE versus 9.7% for hens lighted 18 hours. The reason for this was that the hens exposed to 18 hours of light laid earlier in the day. For example, 34% of their eggs were laid before 7 am versus only 18.8% for the hens that received 15 hours of light. Laying later in the day decreased the number of hens with ultra-thin shells during the late afternoon, thus decreasing the possibility of breakage.

In a summary, to reduce a body-check problem; 1) Do not overcrowd hens. 2) Eliminate activity or noise in the hen house after approximately 4 pm to keep hens as still as possible. 3) Use a shorter photoperiod.

In conclusion, the five point program for maintaining maximum shell quality is as follows:

STEP 1: Eliminate the 20 most common mismanagement practices adversely affecting shell quality.

STEP 2: Do not over feed sulfur amono acids which can cause greater than needed egg size which will cause a premature reduction in shell thickness.

STEP 3: Ensure that hens consume their calcium requirement.

STEP 4: Determine and follow egg specific gravity values at regular intervals just as you do egg production and feed consumption.

STEP 5: Eliminate environmental factors that can increase the incidence of body-checked eggs.

Table 1. Estimates of eggs lost due to poor egg shell quality

Estimator	Year	% eggs lost[1]
Petersen et al.[2]	1960	5.00
Wallace	1965	7.00
Sanford	1966	5.00
Forsythe	1966	4.00
Elson	1968	5.80
Sunde	1971	6.50
Harwood and Morris	1972	5.21
Sullivan	1972	6.50
Bezpa	1972	12.15
Swanson and Johnson	1973	6.50
	Average =	6.37

[1] It is believed that these estimates included only body-checked eggs, pimpled eggs, and eggs which were broken between collection and consumer and do not include shell-less eggs or eggs which fall through cage into manure. In 1977, Roland found (Poultry Sci. 56:1517-1527) that for every 100 eggs collected, 7.77 eggs are not collected. This suggest that the shell quality problem is more than twice as severe as previously determined.

[2] Complete references are given in Poultry Science 56:1517.

Table 2. Most common mismanagement practices associated with poor shell quality

I. Mismanagement Practices Affecting Bird Health

1. Feed misformulated (Example: calcium, phosphorus, vitamin D)
   a. Requirement not known or misinterpreted
   b. Math error
   c. Inaccurate nutrient composition of feed ingredients
   d. Nutrient formulation not adjusted to change in feed intake
2. Milling errors
   a. Scale inaccuracy
   b. Error in addition of supplement or ingredient
   c. Nutrient in wrong bin
3. Wrong feed to birds
4. Feeders or waters out of adjustment
5. Interruption of feed or water supply
6. Feed contamination; Example: mycotoxins, insecticides
7. Ingredient separation
8. Wrong particle size of calcium carbonate
9. Disease and/or possible adverse vaccine reactions
   a. Newcastle disease
   b. Infectious bronchitis
   c. Adeno-virus
10. Pullet mismanagement
   a. Failure to have a consistent shell quality evaluation program

II. Mismanagement Practices Causing Breakage By Excess Force to Egg Shells

1. Excess cage density
2. Cage design or condition of cages
   a. Too much slope or not enough slope
3. Eggs stacked too high and egg carton condition
4. Frequency of egg collection inadequate
5. Clumsy egg collector
6. Automatic egg collection equipment out of adjustment
7. Poor road conditions or worn shocks on egg truck
8. Egg processing equipment out of adjustment
9. Not separating jumbo eggs
10. Excess Light

Table 3. Influence of bird age on shell quality

Bird age (months)	Egg weight (g)	Shell weight (g)	Specific gravity	Shell thickness (g)
8 (3)[1]	55.91[d][2]	5.23[b]	1.089[a]	.367[a]
11 (6)	57.20[c]	5.27[ab]	1.084[b]	.366[a]
14 (9)	59.13[b]	5.34[ab]	1.082[c]	.356[b]
17 (12)	64.03[a]	5.38[a]	1.077[d]	.353[b]

[1]Values in parenthesis represent months in lay.

[2]Means followed by different letters in the same column are significantly different ($P \leq 0.05$).

Table 4. Egg weight, shell weight, and specific gravity as influenced by dietary protein

Protein	Egg weight (g)	Shell weight (g)	Specific gravity
20.0	65.0[a][1]	5.38[a]	1.078[a]
16.0	62.9[ab]	5.34[a]	1.079[a]
13.5	63.4[ab]	5.28[ab]	1.078[a]
11.5	59.6[c]	4.92[b]	1.077[a]

[1]Means followed by different letters in the same column are significantly different ($P \leq 0.05$).

Table 5. Influence of photoperiod and cage density on production

Experiment	Photoperiod	Cage density	Production[2] % hen day
1	15	Single	81[ab][1]
	18	Single	83[a]
2	15	Triple	82[ab]
	18	Triple	80[b]

[1]Values followed by different letters are significantly different ($P \leq 0.05$).

[2]Average production for 12 months.

Table 6. Influence of photoperiod and cage density on body-checks

Experiment	Photoperiod	Cage density	Body-checks (%) months in lay 5th	8th	11th
1	15	Single	.6[c][1]	2.2[b]	1.6[c]
	18	Single	2.7[b]	4.4[b]	4.0[b]
2	15	Triple	2.3[b]	4.4[b]	4.9[b]
	18	Triple	5.5[a]	9.7[a]	8.8[a]

[1]Values followed by different letters in the same column are significantly different ($P \leq 0.05$).

Feeding and Managing Capons for Successful Production

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Introduction:

A capon is an unsexed male chicken. By surgical removal of the testes (caponizing) the meat becomes the finest and most delicious of all poultry. In this country only about one of 1000 people have ever tasted capon. Sometimes a roaster, however delicious, is mistaken for capon in the market place. The capon is larger, ideally weighing about 7½ lbs. dressed. This relates to a liveweight of 9¼ to 9½ lbs. in approximately 18 weeks. During this time it consumes 35-40 lbs. of feed, even though for 8 weeks the capon is on a restricted diet program.

The capon is a specialty item in the food market and restaurant trade. There are only approximately 1 million capons raised annually in the U.S. Grade is very important! There are 3 grades; "A", "B" and "C" along with "parts missing". Payment is made on grade and this will be explained in detail later in this paper. The capon population is fairly well-controlled with most of the capons of the past season sold before the new season begins.

Contrary to other types of meat-type poultry producing programs, capons should never be full-fed with high energy broiler chicken-type formulas. This is most important because if they are full-fed they will become obese and spend most of their time sitting between meals. This will result in a breast blister and downgrading. You must build skeletal growth on a lean frame from 8 through 14 weeks in order to increase the chance of Grade A carcasses.

Getting Prepared for Baby Chicks:

This is the same procedure as for replacement pullets, broilers, turkeys or for any other type of poultry. The house, feeders and waterers should be thoroughly cleaned and disinfected at least a week before chicks are due to arrive. Spread dry, soft litter at least 4 inches deep several days before receiving chicks. Wood shavings are excellent litter. Start heating units at least 48 hours before arrival so they can be regulated and litter will be warm. Have feeders and waterers filled and ready. Be sure that water is at room temperature.

When the Chicks Arrive:

The chicks should arrive early in the morning so that they have the entire day to learn to eat and drink. If they do arrive late in the day be sure that continuous light is used for the first 48 hours.

Brooders: Start 500 chicks under a 6 ft. hover, 750 under an 8 ft. hover, or 750 under a 40,000 BTU heater.

Feeders: Allow capons 2 inches of trough space through 6 weeks and 3 inches until 9 weeks.

Waterers: Provide two chick founts per 100 chicks at the start of the brooding period. Later, each capon should have 1 inch of waterer space when troughs are used. With pans supply about 1/3 less bird space.

Lights: Use continuous light the first 48 hours, then provide about 0.5 foot candle of illumination at bird level to supplement natural daylight or about 1 foot candle in environmentally controlled houses.

Care During First and Second Days:

Keep the house warm.

Be sure all the chicks are eating and drinking.

Do not let chicks crowd against brooder guards.

Check birds often the first two days.

Keep water founts free from debris.

Watch for starve-outs. Find the trouble and remedy it.

Check brooder temperatures at night.

Be sure there is a coccidiosis control program.

Debeaking:

Debeaking is recommended when chicks are raised in confinement. Most birds are being debeaked day-old at the hatchery. For small flocks, it should be done at 5 or 6 days with an electric debeaker. It is a good idea to add soluble vitamin K to the drinking water the 3rd, 4th and 5th days in preparation for debeaking.

Vaccinations:

Extreme caution must be used if vaccinating chicks for Newcastle/ Bronchitis before caponizing. If birds are vaccinated before caponizing it should be done at day-old by using B_1-Connecticut vaccine via the eye-drop method. Do not attempt to vaccinate chicks through the drinking water before caponizing as usually the birds will be going through a stress period at caponizing time and severe losses will result. Chicks can be vaccinated through the drinking water after the stress of caponizing.

Third and Fourth Days:

Discontinue bright all-night lights after 48 hours.

Check brooder temperature at night.

Expand brooder guards each day.

Clean waterers daily; disinfect them once a week.

Start using larger feeders on the fourth day, but continue the feeder lids.

Fifth Through Seventh Day:

Keep brooder warm and start to lower house temperature.

Supply large waterers.

Start to remove first waterers and feeders.

Remove brooder guards.

Second Week:

Start to raise feeders and waterers.

Watch for feed wastage.

Check feed intake daily.

Increase ventilation rate.

Caponizing Time - 2½ to 3½ Weeks of Age:

Three or four days before caponizing start adding a soluble antibiotic (1 tablespoon) and 2 teaspoons of soluble vitamin K (Clotin) per 5 gallons of drinking water. Continue with this up to caponizing time. The day before caponizing take all feed from birds for entire day, but continue with medicated water until evening, then also eliminate all water until after birds are caponized. Check all birds closely for signs of sickness and colds as sick birds cannot be caponized.

Day of Caponizing:

No feed or water until after birds have been caponized. Make sure fresh water is readily available and the inclusion of a soluble vitamin mix in the water the remainder of the day is recommended. Take dry feed away from the birds. Make a wet mash from the Nutrena Complete Chick Starter and do not overfeed the chicks. Scatter small amounts of wet mash frequently to keep birds moving around. Keeping birds active will help prevent lameness and windpuffs and chicks will recover quicker from the shock and stress of caponizing. This is a very critical period.

The grower or his key help should be present to assist with penning and catching to speed up the process. The caponized birds need to be watched carefully and stirred at least every half hour to get them eating and drinking. Keeping birds active will help prevent lameness and windpuffs and chicks will recover quicker from the shock and stress of caponizing. Keeping light on during the night of caponizing plus 1 or 2 extra nights should also help to keep them active instead of sitting in the litter all night. Be sure to keep the birds warm during and after caponizing.

Windpuffs:

The developing of windpuffs in capons is not uncommon. Should they occur, don't bother the birds for about 10 days, then puncture only the large puffs with a sharp instrument and let the air out. If a bird seems in obvious pain from windpuffs after caponizing then use surgery immediately.

Days Following Caponizing:

Put chicks back on regular feed. Continue with vitamins in the water for next 3 or 4 days. Your birds are now full-fledged capons and are ready for a capon growing program.

Fifth and Sixth Weeks:

Feed a chick starter-type diet (20% protein) for the first 5 weeks, then start on your feed restriction program. During the 6th week change to Developer I and feed 20 lbs./100 birds. Make sure there is plenty of feeder space (about 50% more than when on full feed).

Seventh Week:

Worm capons with piperazine water wormer. Limit the feeding to 22 lbs./100 birds/day. You can control feed in many ways.

1. Hand weighing and feeding.
2. Use of an automatic timer on the feeder.
3. Lowering and raising hanging feeders.
4. Running in feed in the dark in chain-type feeders.
5. By use of sophisticated new feeding equipment that pre-weighs feed then drops it into feeder pan. This is best system for large operations.

Eighth Week:

Continue feeding Developer I but increase the amount of feed to 25 lbs./100/day.

Ninth and Tenth Week:

Change to Developer II but still feed 25 lbs./100/day.

Eleventh, Twelfth and Thirteenth Weeks:

Continue feeding Developer II but change feeding rate to 28 lbs./100/day.

Worm again during the 12th week.

Fourteenth Week:

Continue feeding Developer II and change rate to 30 lbs./100/day.

Grit Feeding:

We recommend the feeding of grit to capons. It can be put into hanging feeder or put in the feed about once per week.

Fifteenth Week to Marketing:

Change to Capon Finisher ration. Gradually increase amount of feed given birds daily so that at the beginning of the 16th week they will be on full-feed. Continue on full feed until marketing, usually at 18 weeks.

Grade Pricing:

The going market price is used to establish the premium incentive program. At 50% Grade A the current market price is used (say, for example, the market price is 45¢). For every 1% above 50% Grade A the price to the producer increases by 0.1 of a cent. If a flock owner comes in with 52% Grade A he will receive 45.2¢/lb. liveweight for his capons. If they are at 60% Grade A, they will receive 46¢/lb. at 70%; 47¢/lb. and at 80%; 48¢ or 3¢/lb. more than the standard.

Grade A capons must be well-finished with good body conformation and no breast blisters or other blemishes.

Grade B capons are well-finished birds with small blisters that can be sewn.

Grade C capons usually have large blisters (non-infected) that have to be cut away, taking most of the skin from the breast.

Parts Missing - same as for broilers and turkeys.

NDM:DE

TABLE 1

CAPON FEEDING PROGRAM

Weeks to Feed	Type of Feed
0-5	20% Complete Chick Starter. Should contain a coccidiostat.
6-8	Complete feeding: Capon Developer No. 1 - high in fiber - low in protein - should contain coccidiostat.
	Concentrate feeding:
	41% Capon Concentrate 20%
	Ground corn 50%
	Whole oats 30%
9-14	Complete feeding: Capon Developer No. 2 - high in fiber - low in protein - should contain coccidiostat.
	Concentrate feeding:
	41% Capon Concentrate 15%
	Ground corn 42.5%
	Whole oats 41.5%
	Dicalcium phosphate 1.0%
15 to Market	Complete feeding: Capon Finisher - low in protein - high in energy - should contain coccidiostat.
	Concentrate feeding:
	41% Capon Concentrate 20%
	Ground corn 77%
	Dicalcium phosphate 1%
	Fat 2%

TABLE 2

CAPON DATA

Taken From Actual Capon Experiments

	Expected Body Weight Gain (In Pounds)				
Period: (weeks)	0-5	6-8	9-14	15-18	0-18
	2.17	1.83	3.94	1.96	9.89

	Feed Per Capon				
Period: (weeks)	0-5	6-8	9-14	15-18	0-18
	3.99	5.45	15.36	10.71	35.51

	Feed Required/Pound Gain				
Period: (weeks)	0-5	6-8	9-14	15-18	0-18
	1.84	3.01	3.90	5.45	3.87

TABLE 3

STARTER AND DEVELOPER I REQUIREMENTS

Nutrients:	Starter	Devel. I
Protein, %	20	16
CME/kg.	2850	2719
Calcium, %	1.00	1.10
Avail. Phos., %	0.50	0.50
Lysine, %	1.05	0.75
TSAA, %	0.75	0.56
Sodium, %	0.17	0.15
Weeks Fed	0-5	6-8

TABLE 4

DEVELOPER II AND FINISHER REQUIREMENTS

Nutrients:	Devel. II	Finisher
Protein, %	16	15.5
CME/kg.	2509	3200
Calcium, %	0.83	0.94
Avail. Phos., %	0.55	0.50
Lysine, %	0.72	0.69
TSAA, %	0.52	0.50
Sodium, %	0.15	0.17
Weeks Fed	9-14	14-Mkt.

TABLE 5

HOW PAYMENT IS MADE FOR GRADES

1. 50% Grade A Market Price

   For example: liveweight price is 45¢/lb.

2. Each 0.1% above 50% increases price by 0.1 cent.

3. Each 0.1% below 50% decreases price by 0.1 cent.

4. For example:

   55% Grade A, price @ 45.5¢/lb.

   45% Grade A, price @ 44.5¢/lb.

   40% Grade A, price @ 44.0¢/lb.

   70% Grade A, price @ 47.0¢/lb.

TABLE 6

Group 1 = Fed like broilers

No Restriction

Group 2 = Fed like broilers

Restriction of Grower

Group 3 = Regular capon method of

feeding w/restriction

TABLE 7

RESULTS OF CAPON STUDY

(Day-Old - 18 Weeks)

	Group 1	Group 2	Group 3
Type	Br. FF	BR. RF	Ca. RF
Body Wt., kg.	5.00	4.63	4.14
Feed/Capon, kg.	16.82	15.43	13.15
Feed/Gain, kg.	3.43	3.41	3.25
Weak legs, %	22.8	10.2	5.3
Grade A's, %	56.7	71.2	80.6

TABLE 8

FIELD REPORT

Purchased . . . . . 6,000 head

9.5% mortality

Production cost, total $16,111.28

or 31.42¢/lb.

Average body weight . . . . . 9.4 lbs.

Feed cost, total $9721.39

18.96 feed cost/lb. meat

Grade A . . . 74.8%, Grade B . . . 22.5%

Thrift . . . . . 2.7% = 100%

Return . . . . . Capons $20,642.35

Pullets 141.96

$20,784.31

Costs 16,111.28

$4,673.03

Profit/Bird Packed 5327 Head = 87.72¢/Capon

TABLE 9

EXPERIMENTS - ITEMS TESTED

1. Mold control
2. Appetite depressants
3. Vitamin levels (3)
4. Energy levels (2)
5. Different fiber sources
6. Calcium:Phos. levels
7. Lactobacillus cultures
8. Antibiotics
9. Trace mineral levels
10. High levels vitamin D_3
11. Grains, sunflower and wheat
12. Feeding programs

INFLUENCE OF ENVIRONMENTAL TEMPERATURE ON THE LYSINE REQUIREMENT OF GROWING TURKEYS

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INTRODUCTION

Environmental and Temperature and Protein-Amino Acid Requirements

As environmental temperature exerts an effect on growth and feed consumption of poultry (de Albuquerque et al., 1978; Deaton et al., 1978; Milligan and Winn, 1964; Prince et al., 1961), it has been suggested that a nutritional adjustment should be made in the diet due to altered energy requirements (Combs, 1970; Waibel, 1976; Wilgus, 1973). The adjustment usually made is in dietary protein or amino acid concentration in relation to dietary available energy to compensate for the change in feed intake. At cool temperatures, then, a lower protein diet should be adequate, while at warm environmental temperatures, higher protein diets will be needed to maximize growth.

Review of the literature concerning nutrient requirements as related to environmental temperature has not shown a consistent relationship between requirements and temperature. Lower protein requirements have been demonstrated with Large White male turkeys reared at cooler environmental temperatures. Waibel et al. (1975) reported that turkeys 4-20 weeks of age and reared at 51°F needed lower dietary protein compared to the turkeys kept at 72°F. In another experiment lower protein requirements were seen for turkeys (6-20 weeks of age) reared at 49 or 59°F (Waibel et al., 1976); turkeys reared at warmer temperatures (69° or 81°F) grew better when dietary protein was higher, but growth was still depressed compared to turkeys in the cooler environments. Data of March and Biely (1972) indicated a greater concentration of lysine was needed to maximize gains of broilers from 2-4 weeks of age at 31.1°C compared to chicks kept at 20°C. Hurwitz et al. (1980) also indicated changing amino acid requirements with temperature. Calculated daily amino acid requirements (mg/cal) of 6-week old broilers were shown to increase as temperature increased from 12 to 27°C. From 28 to 34°C there was a decline in the requirement. The lower requirement was reasoned to be due to a slower growth rate and increased caloric need at 34°C to remove body heat.

Other studies have shown no difference in requirement with ambient temperature. Adams and co-workers (1962a, 1962b, and 1968) found similar protein and sulfur amino acid requirements for broilers (4-8 weeks of age) held at ambient temperatures of 21 or 29°C. Murillo and Jensen (1976) saw no difference in methionine requirements for turkeys at low to moderate temperatures. Recent work by Charles et al. (1981) with finishing broilers held at temperatures varying from 18 to 27°C indicated no interaction of protein level with ambient temperature.

Lysine Requirements of Market Turkeys

In turkey diets, methionine and lysine are used to determine the protein level in diet formulation due to their limiting nature. In young poults, methionine and lysine are first and second limiting amino acids, respectively, in corn-soybean meal type diets (Fitzsimmons and Waibel, 1962; Waibel, 1959). Corn-soy diets for turkeys, then, can be supplemented with methionine and lysine and have lower protein contents (Carter et al., 1962; Balloun, 1962; Balloun and Phillips, 1957). Since supplemental methionine is usually economically feasible for meeting the methionine requirement, it becomes critical to establish lysine requirements for use in diet formulation to set the protein level.

For older turkeys, reports are few concerning the lysine requirement. For the 8-12 week old male turkey the NRC (1977) states a requirement of 1.35% (.45%/Mcal) in a diet containing 22% protein. Potter et al. (1981) found the same requirement level experimentally (1.34% or .45%/Mcal). Kratzer et al. (1956) reported a very low lysine requirement of .85% in a 20% protein diet in an early study.

For the 16-20 week old turkey a lysine level of .80% (.25%/Mcal) in a 16.5% protein diet is recommended by the NRC (1977). Kratzer et al. (1956) reported a lower requirement of .56% lysine using a diet containing 17.8% protein. Summers et al. (1966) found turkeys fed a 16.6% protein diet with .68% lysine grew as well as those fed the same diet containing supplemental lysine or a diet with additional protein. Jensen et al. (1976) reported two rather different lysine requirements depending on basal diet type used in the experiment (.825 or .730%). The lower requirement was thought to be due to a higher available lysine content in the basal diet used in that particular trial.

A few reports have indicated the lysine requirement to be much higher for the 16-20 week old turkey. Warnick (1979) estimated the requirement to be .90% in an 18% protein diet. Potter et al. (1981) reported a requirement of .97% lysine for the 16-20 week male turkey.

The objectives of the two experiments presented in this paper were to determine the effect of environmental temperature on the lysine requirement of growing turkeys, ages 8-12 and 16-20 weeks. These ages were selected as available information on the lysine requirements is quite variable.

EXPERIMENTAL METHODS

The two experiments were completed at the Turkey Research Facility at the Rosemount Agricultural Experiment Station. Both experiments utilized male Large White turkeys of the Nicholas strain in floor pens. The birds were reared under an intermittent lighting program. The experimental periods were from 8-12 and 16-20 weeks of age. When the turkeys were not on experiment they were fed diets typical for their age composed mainly of corn, soybean meal and fat with vitamin and mineral supplementation. During the week prior to the start of each experimental period, the birds were weighed and sorted into the pens such that the average body weight of the pens was similar. Poults from poor treatments in the previous experimental period were discarded.

In each experiment, a lysine deficient diet containing sesame meal and corn as the only protein sources was used to assay the requirement. Diets for Experiment 1 contained 54.6 and 23.6% sesame meal for the 8-12 and 16-20 week experimental periods. In experiment 2 the amounts were 46.4 and 23.6% respectively for the two age periods. The remainder of the diet was composed of corn, and supplements of vitamins and minerals. The protein content of the basal diets was slightly high to assure adequate levels of the other amino acids. Varying amounts of feed grade L-lysine·HCl (98%) were added to the basal diet to form seven levels of supplementation. As an additional control, an eighth treatment group in each experiment was fed a corn-soy diet.

In experiment 1, two environmental temperatures (45° and 75°F) were utilized in each period. In experiment 2, three temperatures were used. In the 8-12 week period the temperatures were 45, 70 and 80°F. In the 16-20 week experimental period the temperatures were 45, 60 and 75°F. Room temperatures were recorded during the experimental periods with hygrothermographs.

Individual bird weights were taken at the beginning and end of each period. Feed consumption and mortality records were kept. Body weight, average daily gain and feed efficiency were the main criteria by which the effects of the environmental temperature and lysine supplementation were measured. Only data for average daily gain will be presented herein. The experimental design was factorial with ambient temperature and level of lysine supplementation as factors. The lysine requirement within each environment and age period was determined using the broken-line least squares method (Robbins et al, 1979).

RESULTS AND DISCUSSION

Experiment 1.

The effect of lysine supplementation and environmental temperature on body weight gain is seen in Figures 1 and 2 for the 8-12 and 16-20 week experimental periods, respectively. Ambient temperature and dietary treatment significantly affected gain and feed intake. Turkeys at 75° grew slower and consumed less feed than those at 45°F. The severity of growth and feed intake depression was much worse for the turkeys at 75°F in the 16-20 week experimental period. Average daily gain and feed intake for the turkeys fed high levels of lysine at 75°F were depressed to 81% and 79% of that seen for turkeys kept at 45°F, respectively. Turkeys fed the sesame meal basal diet with adequate lysine grew as well as the corn-soy control birds.

To determine the lysine requirement turkeys were fed the sesame meal basal diet supplemented with different levels of lysine and the growth response measured. To estimate the requirement with the broken-line method the response curve needs to exhibit certain characteristics. The initial part of the growth curve should show a linear response in growth for each incremental addition of nutrient. The growth curve should plateau and no additional response be seen in growth after the requirement is met. The broken line method determines the requirement point (break-point) as the intersection of the sloping line with the plateau of the growth curve.

The data for the 8-12 and 16-20 week experimental periods appear to fit this general form with some imperfections. At 75° (both ages) the growth response

to additional lysine fits the broken line model better than the curve representing the growth response at 45°F. The growth response at 45°F tends to show some curvature near the break-point where growth rate is slowing.

The calculated lysine requirements for maximum gain for each age period are given in Table 1 and indicate requirement differences between environments. For the 8-12 week old turkey the lysine requirements were 1.26 and 1.14%, respectively, at 75 and 45°F. The estimated requirements are lower than the current NRC (1977) recommendation of 1.35% (.45% Mcal). The requirements are much lower when related to energy content of diet, being .35 and .32%/Mcal, respectively.

For the 16-20 week old turkey, the total lysine requirement for maximum gain was .80 and .70% for the 75 and 45°F environments. The estimate of .80% (.25%/Mcal) is the same as reported by the NRC (1977) although as related to dietary energy content, requirements for gain were .23 and .20%/Mcal.

The lower requirement in relation to energy at 8-12 weeks could be due to overestimation of the metabolizable energy content of the sesame meal. The metabolizable energy (ME) content of the sesame meal (3990 kcal/kg) was calculated according to the method presented by Carpenter and Clegg (1956) based on chemical analyses. Calculated energy content of a similiar sample of sesame meal yielded larger value compared to the conventional ME value (Devegowda, 1981).

Experiment 2.

The effect of lysine supplementation and environmental temperature on body weight gain of turkeys is seen in Figures 3 and 4 for the 8-12 and 16-20 week experimental periods, respectively. Ambient temperature and dietary treatment significantly affected gains and feed intake. For the 8-12 week period gains at 70 and 80°F were depressed to 91 and 84% of those seen at 45°F while feed intake was depressed to 88 and 82% respectively. For the 16-20 week period gains at 60 and 75° were depressed to 94 and 82% of those seen at 45°F while feed intake was depressed to 92 and 84%.

Growth responses (Figure 3) to lysine addition at 8-12 weeks for all three environments show some curvature in the break-point area where growth rate is slowing. The estimated lysine requirement (Table 1) for the 8-12 week old turkey at 45, 70 and 80°F was 1.11, 1.10, and 1.24% respectively. A higher requirement was seen at 80°F with similiar requirements at 45 and 70°F.

For the 16-20 week old turkey the growth responses to lysine at the three temperatures are more variable especially for the turkeys held at 45°F as seen in Figure 4. Turkeys at 45 and 60°F gained similiarly at suboptimum levels of lysine; however, with adequate lysine the turkeys at 45°F outperformed those at 60°F. The similarity in response for the turkeys at 45 or 60°F led to approximately equal requirements of .73 and .71% (Table 1). At 75° the lysine requirement was .78%.

The requirements determined in Experiments 1 and 2 agree well with each other and tend to show that environmental temperature did affect the requirement. The similiarity of requirement for Experiment 2 at 45 and 70°F for the 8-12 week period and at 45 and 60°F for the 16-20 week period may reflect the variation

about the requirement estimate. Lysine requirements in both experiments for the 8-12 week period appear to be lower than the NRC recommended level. The 16-20 week estimate also appears to be slightly low in contrast to the NRC value and much lower than values presented by Potter et al. (1981) and Warnick (1979).

The low requirement estimates could be due to the method used of calculating the requirement. The broken-line method has been suggested by Morris (1981) to underestimate the requirement. In a sulfur amino acid requirement study with laying hens, regression analysis indicated an increase in production to additional amounts of SAA after the determined break-point. In the above experiments statistically significant slopes in the plateau region were seen twice, in the 8-12 week period for the 45° environment (Experiment 1) and for the 70° environment (Experiment 2). The degree to which the broken line method may or may not underestimate the requirement is unknown as few comparative reports are available for study.

Another comparison may be made to demonstrate environmental effects on nutrient requirements by determining approximate levels of lysine needed to produce a certain amount of gain in each environment. For example, using the 8-12 week data in Experiment 2 (Figure 3), equal gains of approximately 102 gms can be seen for the three temperatures. Interpretation from the gain to the dietary lysine level would yield dietary lysine requirement levels of 1.02, 1.18 and 1.38% at 45, 70 and 80°F, respectively. In practice the gains might not be the same; if so, this comparison may not be valid. The comparison does point to large differences in the need for dietary lysine where gain is not variable.

In summary, lysine requirements of Large White male turkeys aged 8-12 and 16-20 weeks were determined using a corn-sesame meal diet in two experiments. Environmental temperatures were found to influence the lysine requirement as determined by the broken-line method. At 45°F the requirement (% of diet) was approximately 10% lower than that determined at 75 or 80°F for both age groups. At 8-12 weeks the estimated requirement for lysine was lower compared to the reported value in the NRC (1977), while the requirement at 16-20 weeks, with warm ambient temperatures, was similiar to the NRC value.

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LITERATURE CITED

Adams, R.L., F.N. Andrews, J. C. Rogler and C.W. Carrick, 1962a. The protein requirement of 4-week old chicks as affected by temperature. J. Nutr. 77: 121-126.

Adams, R.L., F.N. Andrews, J.C. Rogler and C.W. Carrick, 1962b. The sulfur amino acid requirement of the chick from 4 to 8 weeks of age as affected by temperature. Poultry Sci. 41:1801-1806.

Adams, R.L. and J.C. Rogler, 1968. The effects of environmental temperature on the protein requirements and response to energy in slow and fast growing chicks. Poultry Sci. 47:579-586.

de Albuquerque, K., A.T. Leighton, Jr., J.P. Mason, Jr. and L.M. Potter, 1978. The effects of environmental temperature, sex and dietary energy levels on growth performance of Large White Turkeys. Poultry Sci. 57:353-362.

Balloun, S.L., 1962. Lysine, arginine, and methionine balance of diets for turkeys to 24 weeks of age. Poultry Sci. 41:417-424.

Balloun, S.L. and R.E. Phillips, 1957. Lysine and protein requirements of bronze turkeys. Poultry Sci. 36:884-891.

Carpenter, K.J. and K.M. Clegg, 1956. The metabolizable energy of poultry feeding stuffs in relation to their chemical composition. J. Sci. Fd. Agric. 1:45-51.

Carter, R.D., E.C. Naber, S.P. Touchburn, J.W. Wyne, V.D. Chamberlin and M.G. McCartney, 1962. Amino acid supplementation of low protein turkey growing rations. Poultry Sci. 41:305-311.

Charles, D.R, C.M. Groom and T.S. Bray, 1981. The effects of temperature on broilers: interactions between temperature and feeding regime. Brit. Poultry Sci. 22:475-481.

Combs, G.F., 1970. Feed ingredient composition and amino acid standards for broilers. Proc. Maryland Nutrition Conf., p. 81-89.

Deaton, J.W., F.N. Reece and J.L. McNaughton, 1978. The effect of temperature during the growing period on broiler performance. Poultry Sci. 57:1070-1074.

Devegowda, G., 1981. Evaluation of feedstuffs for true metabolizable energy and bioavailable amino acids in turkeys. Ph.D. Thesis, University of Minnesota.

Fitzsimmons, R.C. and P.E. Waibel, 1962. Determination of the limiting amino acids in corn-soybean oil meal diets for young turkeys. Poultry Sci. 41: 260-268.

Hurwitz, S., M. Weiselberg, U. Eisner, I. Bartov, G. Riesenfeld, M. Sharvit A. Niv and S. Bornstein, 1980. The energy requirements and performance of growing chickens and turkeys as affected by environmental temperature. Poultry Sci. 59:2290-2299.

Jensen, S.L., B. Manning, L. Falen and J. McGinnis, 1976. Lysine needs of rapidly growing turkeys from 12-22 weeks of age. Poultry Sci. 55:1394-1400.

Kratzer, F.H., P.N. Davis and B.J. Marshall, 1956. The protein and lysine requirements of turkeys at various ages. Poultry Sci. 35:197-202.

March, B.E. and J. Biely, 1972. The effect of energy supplied from the diet and from environment heat on the response of chicks to different levels of dietary lysine. Poultry Sci. 51:665-668.

Milligan, J.L. and P.N. Winn, 1964. The influence of temperature and humidity on broiler performance in environmental chambers. Poultry Sci. 43:817-824.

Morris, T.R., 1981. Response curves for estimating the optimum amino acid intake for laying pullets. Proc. Maryland Nutr. Conference, pp. 26-35.

Murillo, G.M. and L.S. Jensen, 1976. Methionine requirement of developing turkeys from 8-12 weeks of age. Poultry Sci. 55:1414-1418.

National Research Council, 1977. Nutrient requirements of poultry. No. 1. In "Nutrient requirements of domestic animals." 7th ed. Nat. Acad. Sci. Washington, D.C.

Potter, L.M., J.R. Shelton and J.P. McCarthy, 1981. Lysine and protein requirements of growing turkeys. Poultry Sci. 60:2678-2686.

Prince, R.P., L.M. Potter and W.W. Irish, 1961. Response of chickens to temperature and ventilation environments. Poultry Sci. 40:102-108.

Robbins, K.R., H.W. Norton and D.H. Baker, 1979. Estimation of nutrient requirements from growth data. J. Nutr. 109:1710-1714.

Summers, J.D., W.F. Pepper and S.J. Slinger, 1966. The protein and lysine requirements of large white turkeys during the growing and finishing period. Can. J. An. Sci. 46:51-57.

Waibel, P.E., 1959. Methionine and lysine in rations for turkey poults under various dietary concentrations. Poultry Sci. 38:712-720.

Waibel, P.E., 1976. Turkey nutrition and flexible feed formulation. Feedstuffs 48(2):33-35.

Waibel, P.E., M.E. El Halawani, and B.R. Behrends, 1975. Ambient temperature and protein requirements of turkeys. Univ. of Minn. Agr. Exper. Sta. Misc. Report 134:77-84.

Waibel, P.E., M.E. El Halawani and B.R. Behrends, 1976. Growth and efficiency of large white turkeys in relation to dietary protein and environmental temperatures. Proc. 37th Minn. Nutr. Conf. p.119-127.

Warnick, R.E., 1979. Lysine levels in turkey grower diets. Poultry Sci. 58:1120.

Wilgus, H.S., 1973. Temperature-programmed feeding schedules and other means of conserving protein in market turkey production. Feedstuffs 45(27): 27-31.

Table 1. Dietary lysine requirement for maximum gain of Large White male turkeys as affected by environmental temperature

Experiment Number	Temperature (°F)	Age Period (weeks) 8-12	16-20
Experiment 1			
	45	1.14 (.32)[1]	0.70 (.20)
	75	1.26 (.35)	0.80 (.23)
Experiment 2			
	45	1.11 (.32)	0.73 (.21)
	60	-	0.71 (.21)
	70	1.10 (.31)	-
	75	-	0.78 (.23)
	80	1.24 (.35)	-

[1]Requirements expressed as % of diet and, in parenthesis, as %/Mcal metabolizable energy.

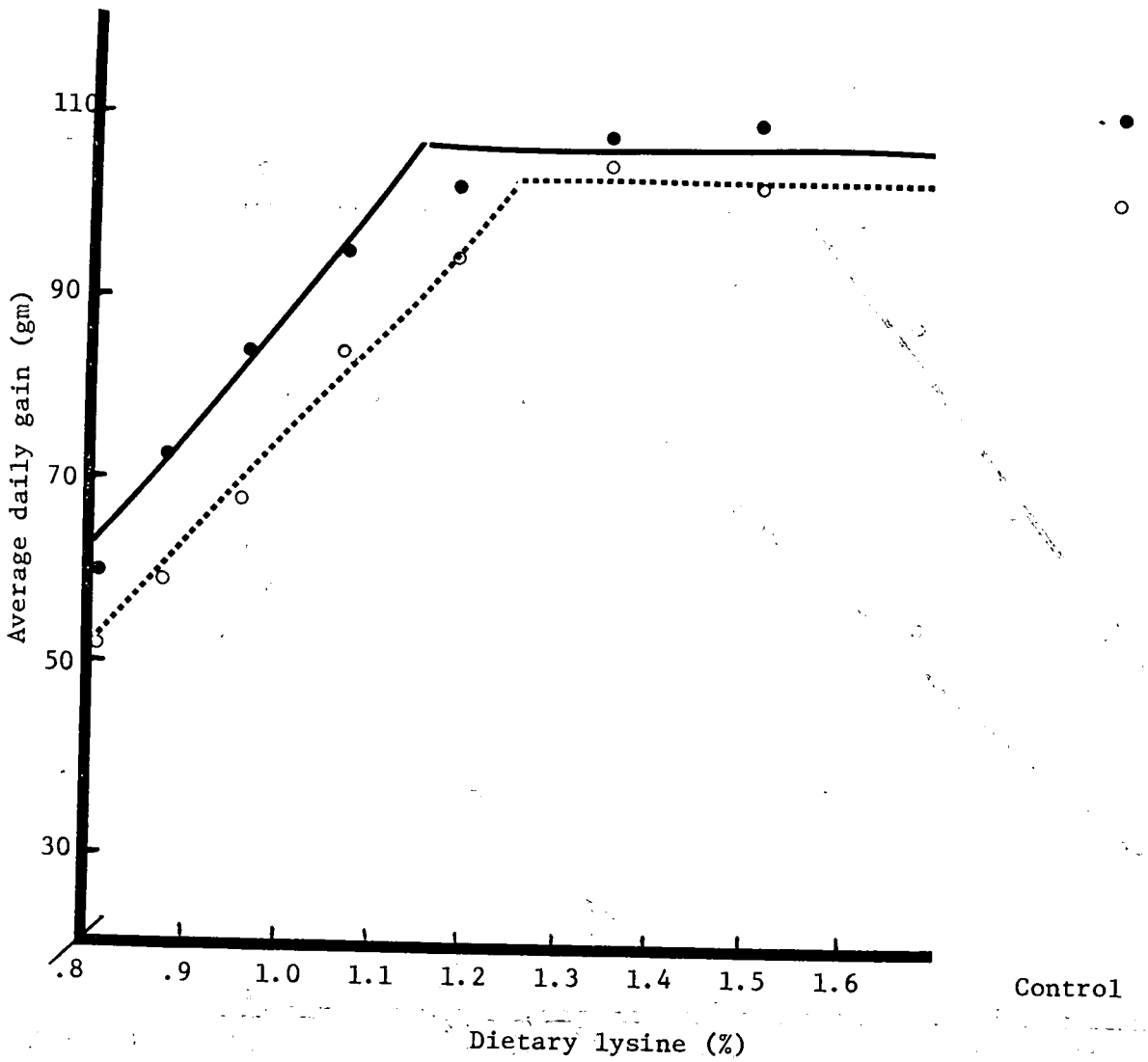

Figure 1. Effect of dietary lysine on the growth of male turkeys (8-12 weeks of age) at temperatures of 45°F (●) and 75°F (○). Lines represent the fitted broken-line used to estimate the requirement at 45°F (——) and 75°F (••••).

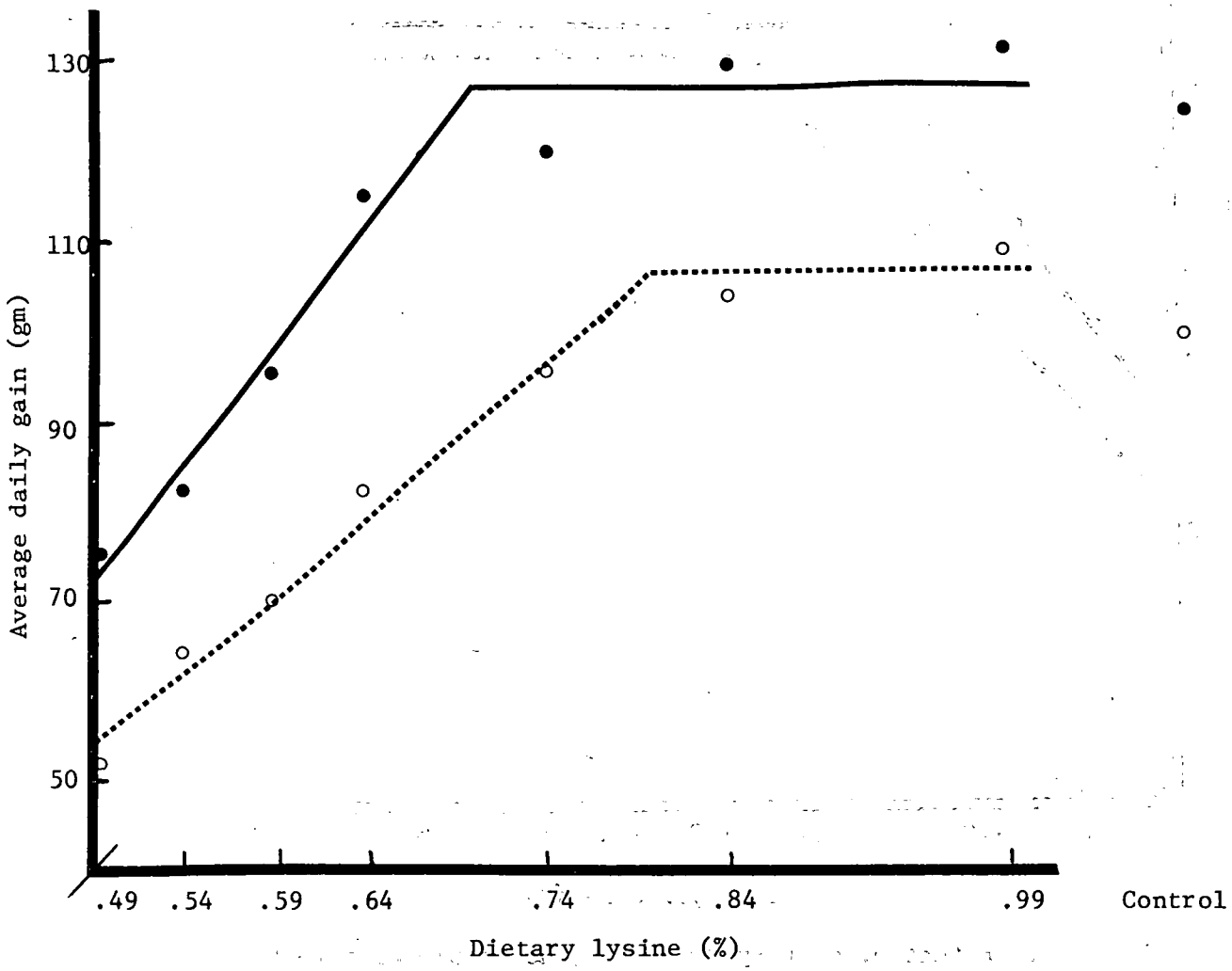

Figure 2. Effect of dietary lysine on the growth of male turkeys (16-20 weeks of age) at temperatures of 45 F°(●) and 75°F (○). Lines represent the fitted broken-line used in determining the requirement at 45°F (——) and 75°F (••••••).

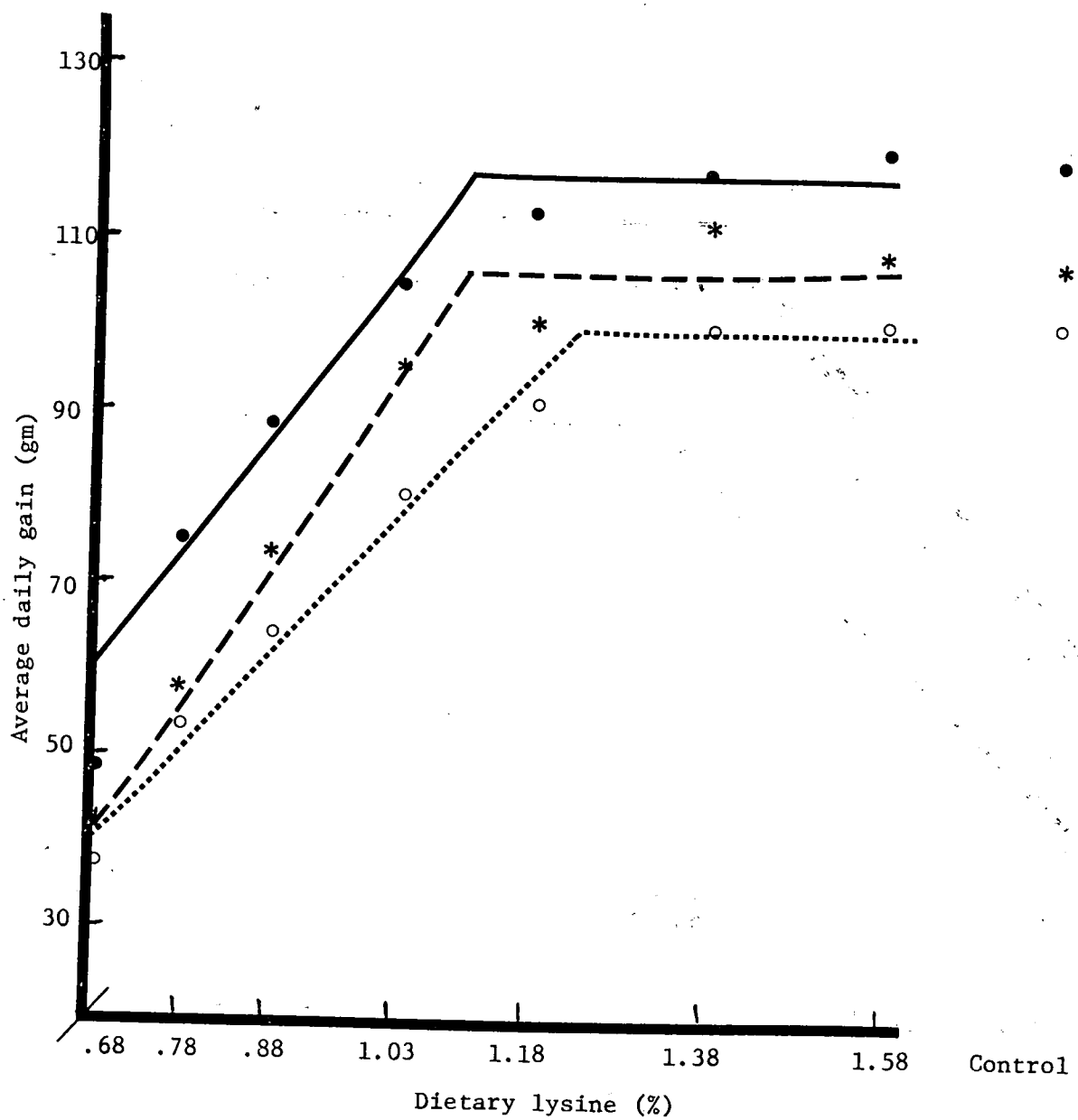

Figure 3. Effect of dietary lysine on the growth of male turkeys (8-12 weeks of age) at temperatures of 45° (●), 70° (✱) and 80°F (o). Lines represent the fitted broken-line used in determining the requirement at 45° (——), 70° (– – –) and 80°F (·····).

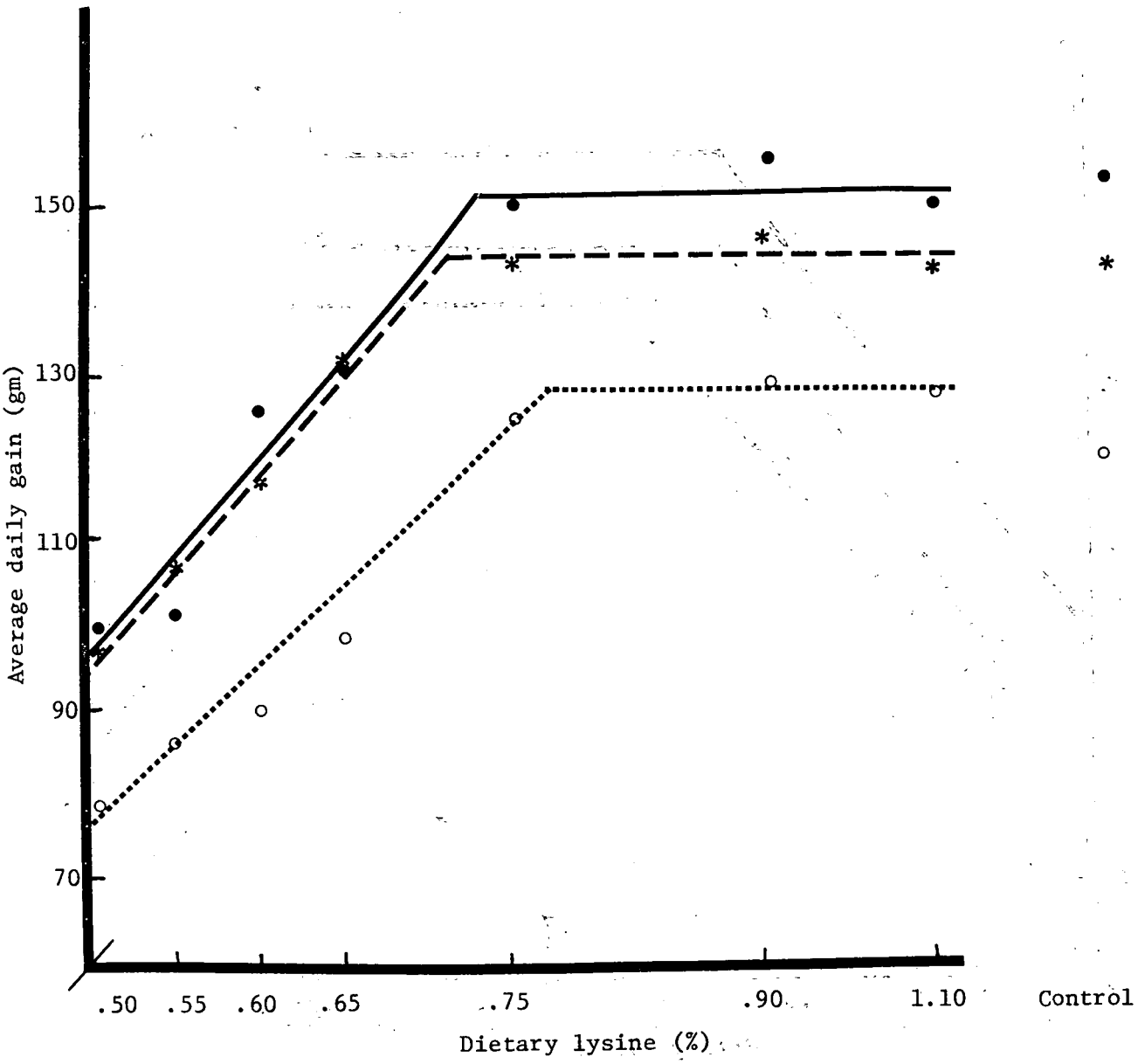

Figure 4. Effect of dietary lysine on the growth of male turkeys (16-20 weeks of age) at temperatures of 45°F (●), 60°F (*), and 75°F (o). Lines represent the fitted broken-line used in determining the requirement at 45° (——), 60° (— —) and 75°F (·····).

SOYBEAN MEAL USE IN ANIMAL FEEDS

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Soybean meal is the most widely used source of supplemental protein for animal and poultry feeds of any feed ingredient in the world. Soybean meal got its start as a protein for human food in the Far East centuries ago. The earliest recordings of soybean meal usage go back to about 3,000 years before Christ. However, its use as an important protein source for poultry and livestock feeds in this country goes back only about 40 years. Soybeans were first imported into this country in the early 1800's and were grown as a novelty crop for almost a hundred years. After the turn of the century soybeans were used just prior to World War II as a fill-in crop for the declining cottonseed oil production in Southeastern United States. Some were also imported from China to be crushed in a plant on the West Coast. After World War I the crop was raised primarily as a hay crop.

During the 30's, soybeans began to be used as an oilseed crop. After World War II the value of the crop became apparent and soybeans were raised for the oil and meal content. Their use as a hay crop was phased out. The acreage expanded by leaps and bounds and it has now evolved into the largest single cash export crop in the United States. The standard pattern around the world is for new importing countries to start with soybean meal and import their needs. As their volume increases to the point they can utilize large quantities of both soybean meal and soybean oil, they will build a crushing plant. They then become soybean customers. From 1965 to 1981 soybean exports increased almost four-fold. Soybean meal exports expanded to a somewhat lesser extent and increased at a rate of just over three times. Soybean oil exports increased approximately 50 percent. At the present time, our number one problem is utilizing all the soybean oil. There are many other oilseed crops around the world that compete directly for the edible oil market and as a result soy oil is the primary product to promote and export. These figures are found in Table 1 (18).

At the present time, over 50 percent of the crop is exported each year. This will vary from year to year depending on the size of the soybean and other oilseed crops and monetary conditions in the importing countries of the world. In addition, many countries of the world have oilseed crops of their own and soybeans are used to fill in the extra protein tonnage needed to balance out their requirements.

Table 1. U.S. Soybean/Meal/Oil Exports Calendar Year

	Soybeans (Mil. bu.)	Soybean Meal (000 St)	Soybean Oil (Mil Lb.)
1965	227.7	2,170	1,219
1970	435.0	4,035	1,501
1975	435.2	4,170	786
1980	800.1	7,726	2,416
1981	802.0	6,692	1,803
Av. Annual Growth	15.8	13.0	2.9

Source: Oil World 4/30/82

If we look at the tons of oilseeds produced throughout the world we find that soybeans supply over 50 percent of the total. Many of these oilseeds contain large quantities of oil so the amount of oilseed meals is reduced to substantially less tons than indicated by the raw seed figures in Table 2 (18).

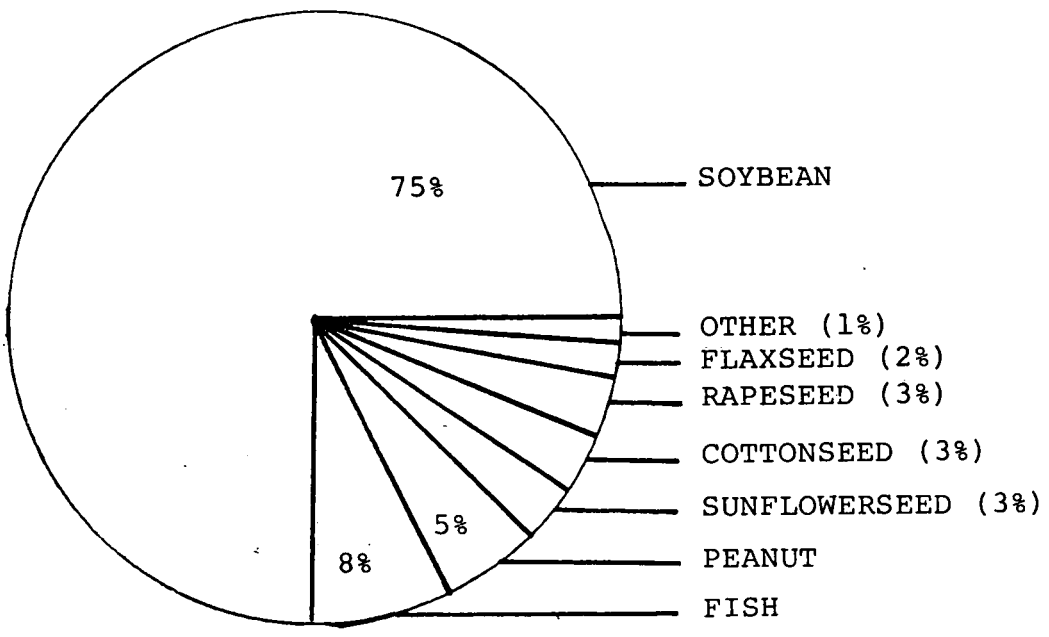
Table 2. World Oilseed Production 1981/82

	1000 MT	%
Soybeans	87,170	52.4
Cottonseed	27,590	16.6
Peanuts	12,480	7.5
Sunflowerseed	14,228	8.6
Rapeseed	12,775	7.7
Sesameseed	1,985	1.2
Copra	5,135	3.1
Palmkernels	1,590	1.0
Linseed	2,373	1.4
Castorseed	895	.5
	166,221	100.0

Source: Oil World 4/30/82 No. 17 XXV:119

Those that find their way into world trade are illustrated in Figure 1 (9).

FIGURE 1 WORLD TRADE



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Of the countries of the world that grow soybeans, the United States produces almost two-thirds of the total. Brazil is the second largest producer with approximately one-fourth the tonnage of the United States. Brazil's figure below is probably a little high for the current year; we now think Brazil produced just over 12 million metric tons. China is the third largest producer of soybeans in the world and practically all of it goes directly to human use; however, they are exporting some to other countries in Southeast Asia to earn hard currency. Argentina and Paraguay are well down the line. All of the rest of the countries of the world contribute less than 4 percent of the total tons. These figures are found in Table 3 (18).

Table 3. World Soybean Production 1981/82

	000 MT	%
USA	54,800	62.9
Brazil	13,000	14.9
China	9,245	10.6
Argentina	4,300	4.9
Paraguay	700	.8
Indonesia	690	.8
Canada	631	.7
USSR	500	.6
All others	3,304	3.8
	87,170	100.0

Source: Oil World 4/30/82 No. 17XXV:A49

Swine consume about a third of the soybean meal crush in the United States. (Table 4) (2). Poultry of all kinds consume almost half of it. Ruminants are just below 20 percent and this is declining because byproduct ingredients, low quality protein meals and other materials including non-protein nitrogen also fill the protein needs for cattle and sheep. This is not necessarily true for hogs and poultry. All the other types of animals which include fish, pet foods, fur-bearing animals and so forth, consume about 4 percent.

Table 4. Soybean Meal Usage in US 1981/82

	%	
Swine		32.6
Layers	12.6	
Pullets	4.7	
Broilers	21.1	
Turkeys	7.9	46.3
Dairy	8.4	
Beef	9.0	
Sheep	.1	17.5
Other		3.6
		100.0

One of the earliest methods of processing soybean meal was to boil the beans and run them through a hand hydraulic press. This process is still being used in China. The oil is squeezed out, the result is a cake about three inches thick and 2 feet in diameter. It contains from 7-9 percent fat. The oil obtained is normally consumed directly into human food in its unrefined state.

The hydraulic press was used in the United States in early days; however, it is not being used at this time. After the hydraulic press in this country came the expeller system. At the present time this system of production is almost discontinued in soybean processing; however, it is used in prepressing part of the oil from sunflowerseed and rapeseeds. In this system the soybeans are ground, tempered with steam, and forced through an expeller that squeezes the oil out of the product. The meal comes out in large flat flakes as big as pie plates or larger, and about a quarter of an inch thick. Steam and friction heat become the toasting/cooking process. These flakes are then ground and moisture is added back to the original content. It is ground and marketed as expeller soybean meal. This product still contains about 3 to 3½ percent fat and normally is 41 percent protein.

Modern soybean processing uses the solvent extraction process in which the soybeans are cleaned, dried to less than 12 percent moisture and stored until they are ready to use. When needed, they are taken out of storage, run over a scalper and each bean is cracked into several particles. (The ideal is 6-8 particles per bean.) Prior to going through the roller mills, the hulls are taken off. There are two methods of production. One leaves the hulls in with the bean particles and all go through processing, the other removes the hulls and by-passes them around the extraction process.

The bean particles are then warmed up to about 160°F at which point they become soft and rubbery. They then run through smooth rollers which flatten the particles to about 10/1,000 of an inch thick. This breaks down the cell wall structure and allows the solvent to penetrate the flattened flakes.

The full-fat flakes are then ready to be washed with a solvent. In the United States, hexane has been the most popular solvent. The solvent dissolves the oil out of the flake. The dissolved oil is removed. Normally the amount of residual oil left in the flakes is reduced to about one-half percent or less. The oil portion is subjected to heat and vacuum, which removes the solvent. The solvent is recycled and reused. The raw oil is diverted to storage and further processing. The meal portion contains about 30 to 40 percent solvent. It goes through a unit called a de-solventizer-toaster. This unit adds steam heat to the meal and as the temperature rises the solvent evaporates and is removed by vacuum and recycled. The heat continues to cook the meal portion. The temperature gets up to about 220°F over a period of time. As the meal comes out of the de-solventizer-toaster it is then ready to be cooled, dried and ground.

Tests normally used to determine the effectiveness of the processing include one for moisture, primarily because this determines whether or not the product will keep. The moisture content should be 12 percent or less. Ideally processors do not like to sell soybean meal much below the 12 percent level. A flash point test is usually run to determine whether or not all of the solvent has been removed. Fat content is often determined to ascertain whether or not the fat was removed from the meal and to measure the efficiency of operation. Ideally it is not desirable to leave high priced oil in the finished product. Protein is determined to measure the marketability. A minimum of protein has to be guaranteed. If the protein level is too high additional hulls can be added to standardize the product. Residual urease activity is quite often determined to measure the apparent adequacy of heat treatment. Ideally we would like to have a urease index between .02 and 0.02.

There has been some interest in the last few months in certain parts of the world in full-fat soybean meal, particularly in areas where there is a shortage of protein and high energy feed grains. One area is Western Europe where there is such a high duty on feed grains, importation is almost eliminated. They do permit the importation of grain by-products which have a relatively low energy level. The use of the full-fat soybeans compliments this situation quite nicely.

Tropical areas of the world suffer from both protein and energy problems as these are not good grain growing areas. The energy materials they have to work with are comparatively low calorie compared to corn or grain sorghum.

Dry extruders have been used to process soybeans in these countries. These got their start about 20 years ago as a method of preparing soybean meal on the farm using the farmer's own beans. One of the units, called the Brady Crop Cooker, could be pulled by a power takeoff on the tractor. Another unit is powered by an electric motor. This unit has less mobility but is probably somewhat more efficient. Neither of these two units requires outside steam or moisture to process soybeans. The heat treatment is accomplished by the friction of the meal going through the extruder.

With steam extrusion, the product is ground and steam is added to it in a conditioning chamber prior to going to the extruder. These units are an integral part of a large mill requiring steam, conditioning chambers, the extruder, coolers, driers and bagging equipment. These are used primarily for human food, snack foods, petfoods, fish foods and in some cases for starter feeds for calves and pigs. At the present time these units are made by several suppliers.

Another type of heat treatment is dry roasting. The first unit of this type on the market was called the Roast-A-Tron. It employed a heating chamber where a stream of soybeans was passed through hot air and in the process was roasted to the right internal temperature.

With the Roast-A-Tron there was quite a lot of variation between the heat treatment of the various kernels depending on how close they got to the heat source. In some cases they were burned or charred to the point their feed value was destroyed. This unit is no longer in production.

This was followed by a large unit called a Jet-Sploder which employs a jet of hot air flowing through a stream of soybeans. It takes about two minutes for the beans to pass through the unit. At the end of the process, the beans are run through a roller mill that flattens them out into nice flat flakes. Three things happen in this process. First, the beans are heated; second, the rolling process breaks the cell walls of the beans so the digestive juices can better penetrate; third, it reduces product density. This method is more efficient as it can produce several tons of product per hour.

The same type of production is available from a unit made at Winona, Minnesota. Again, a stream of hot air is passed through a layer of beans and cooking takes place in the process. These are primarily designed for dairy cattle or at a lower temperature for hogs and poultry. It has been well established that the optimum heat requirement for ruminants is higher than it is for poultry or swine.

Another type of dry heat treatment got its start at Colorado State University by Dr. Harper and his group (19). There they used a bed of salt heated to approximately 521°F (272°C). Soybeans were blended into this super-heated salt and left in there for approximately 20 seconds. This allowed heat to penetrate through the kernel and cook the product. The soybeans were then held at this temperature for another 120 seconds. The degree of heat treatment can be regulated by the temperature of the salt, the length of time the soybeans are allowed to stay in the salt, and also the amount of holding time prior to cooling. The heat treatment of the end product can be well controlled. A prototype of this process is being produced experimentally in the Minneapolis area.

As we look ahead we see nothing on the horizon that will replace soybean meal; however, additional research will have to be conducted to seek the proper heat treatment for maximum utilization. Dr. McNaughton and his group at Mississippi State University (13) did some very interesting time and temperature research on soybean meal for broilers. They found that commercial soybean meal had to be heat treated another 10 minutes for optimum growth in broilers. Their results indicated the optimum urease index should be more than .02 but less than 0.10, and the trypsin inhibitor level should be less than 7 micrograms per milligram of protein. They also found samples of soybeans ground to pass through a 20 mesh screen but held on a 40 mesh screen should show a +a color value of approximately +3.20. If the soybean meal in the Southeastern part of the United States is under-cooked for poultry, it probably is in the rest of the country also. At this time we do not have a good estimate of the status of heat treated soybean meal in all parts of the country.

Some research was conducted at Texas A&M with cannulated pigs. Two different heat treatments were used. One was normally heated and the other overheated. Both heat treatments produced the same weight gains and amino acid utilization in hogs. This research is being repeated and further evaluated.

We have known for several years there is an allergenic factor in soybean meal that sensitizes baby calves (3)(4)(10)(21)(22). This detrimental effect dissipates after the calves are several weeks old but is a real problem in the beginning. Individual calves can be sensitized to this allergenic factor. Some research first conducted in Europe (21)(22) indicated hot ethanol extraction prevented this allergenic effect. Treating soybean meal with acid or alkali or additional heat (6, 7, 8, 11, 12, 14, 15 17) improved growth rate of young calves when these products were used in a milk replacer. Soybean concentrate (65-70% protein) has been an ingredient widely used in calf milk replacers (1, 6, 16, 20). The soy concentrate contains much lower levels of the antinutritional and allergenic factors than soy flour or soybean meal.

We have known for some time through research at Kentucky (11, 12) and Iowa State (23, 24) and other universities that soybean meal was readily digested or broken down in the rumen. This ease of breakdown prevented large quantities from bypassing the rumen to be further digested from the abomosum on down. It was also found that higher heat treatment of soybean meal reduced the solubility of the protein (11, 12 17) and allowed more of it to bypass the rumen. It was also found that treatment with tannic (17) acid or formaldehyde (17, 23, 24, 25) would allow the soybean protein to bypass the rumen. Formaldehyde has never been approved by the Food and Drug Administration and the use of tannic acid is quite unpredictable. This highlights another need for additional research to be done on soybean meal.

In closing, soybean meal requirements are correlated to meat consumption. See Table 5 (5). In the last five years beef consumption has decreased from approximately 125 pounds to 104. This is carcass weight. Veal consumption has also decreased. Lamb consumption has pretty well leveled off. Pork has been going up; however, it has not gone up as much as beef consumption has come down. Nevertheless, we would expect pork production to pick up some in the next couple of years because it is at a low point in the cycle at the present time.

Table 5. Per Capita Consumption:
Livestock, Poultry, Dairy Products and Eggs, United States [1]
1977-1981

Item	1977	1978	1979	1980	1981
			Pounds per person		
Red meat					
Beef	125.9	120.1	107.6	103.3	104.2
Veal	3.9	3.0	2.0	1.8	1.9
Lamb & mutton	1.7	1.6	1.6	1.6	1.6
Pork (exclud. lard)	61.5	61.4	70.2	73.5	70.0
TOTAL	193.0	186.1	181.4	180.2	177.7
Poultry *[2]					
Broilers & other chickens	44.8	47.5	51.5	51.1	51.9
Turkeys	9.3	9.3	10.1	10.4	10.7
Dairy Products					
Butter	4.3	4.5	4.6	4.4	4.3
All cheese	16.2	17.2	17.6	17.6	18.1
Cottage cheese	4.8	4.8	4.6	4.5	4.3
Ice cream	17.8	17.7	17.5	17.3	17.2
Ice milk	7.9	7.9	7.5	7.1	7.0
Non-fat dry milk	3.4	3.2	3.4	3.2	3.3
Canned milk	4.5	4.2	4.3	4.0	4.1
Fluid milk:					
Whole	170.0	161.0	156.0	144.0	138.0
Lowfat and skim	88.4	91.5	94.6	97.9	99.9
Cream and mixtures	5.6	5.6	5.6	5.7	5.8
Milkfat	20.1	20.4	20.4	19.9	19.9
Milk solids/non-fat	38.3	38.0	38.4	36.6	36.4
Eggs(# per person)	272	278	284	272	264

*1: Civilian population excludes military
*2: Ready to cook basis

Broiler and chicken consumption has increased to over 50 pounds per person per year. Turkey consumption has also gone up substantially. These animals are large consumers of soybean meal. Nothing indicates these consumption levels will go down and at the present time we look for an expanding market for soybean meal for poultry, turkeys and swine.

Fluid milk consumption has been decreased for several years and low-fat or skim milk consumption has been increasing, but not at a rapid enough rate to compensate for the decline in whole milk consumption. Butter has pretty well leveled off. One factor involved in soybean meal usage in dairy cattle has been the three or four months of production a dairy cow cannot consume enough feed of any kind to maintain its body weight and adjust to a high rate of production. The diet should contain a minimum of non-protein nitrogen and the protein should be supplied from natural sources, thus maintaining a market for soybean meal or other natural proteins.

The world-wide use of soybean meal has expanded at a rate of about 17 percent a year over the last 12 years (9). We would expect this to continue but at a lower rate as there are quite a few countries in the world that are just becoming soybean customers. These countries are evolving from back-yard production into commercial type poultry or swine production. The people who have their money invested in large operations will not tolerate poor performance. As a result, good rations have to be fed and this will require soybean meal.

Regardless in which country of the world we are located, the name of the game is the same -- economical food -- and production of this food will require the most judicious use of scarce resources.

REFERENCES

1. Akinyele, I.O., R. B. Rindsig, J. G. Velu and K. E. Harshberger. Soy Protein Concentrate in Milk Replacers for the Calf. 1975 Proceedings of Dairy Science Annual Meeting 58:741.

2. Allen, G. 1982 Personal Communication. Economic and Statistical Service, USDA.

3. Barratt, M. E., P. J. Stachan and P. Porter. Antibody Mechanism Implicated in Digestive Disturbances Following Ingestion of Soya Protein in Calves and Piglets. 1978 Clinical Experimental Immunology 31: 305-312.

4. Barratt, M. E., P. J. Strachan and P. Porter. Immunologically Medicated Nutritional Disturbances Associated with Soya Protein Antigens, 1979 Proceedings of Nutritional Society 38:143-150.

5. Brown, R., D. Murfield, W. L. Pratt, M. D. Humphrey, Jr., R. T. Bass 1981 Texas Livestock, Dairy and Poultry Statistics:57 Texas Department of Agriculture.

6. Coblentz, E, J. L. Morrill, D. B. Parrish and A. D. Dayton, Nutritive Value of Thermalkali-Processed Soy Materials for Young Calves and Rats. 1976 Journal of Dairy Science 59:481-490.

7. Colvin, B. M. and H. A. Ramsey. Growth of Young Calves and Rats Fed Soy Flour Treated with Acid or Alkali. Journal of Dairy Science 52:270-273.

8. Daniels, L. B., J. L. Davis, O. T. Stallcup, J. M. Rakes and M. M. Campbell. Nutritive Value of Fll-Fat Soybeans Fed to Dairy Calves as Influenced by Length of Cooking Time. 1981 American Society of Animal Science Abstracts: 125.

9. Davenport, G. and E. Fryer. World Production and Trade of the Major High Protein Meals 1981 Fats and Oil Situation And Outlook FOS 304: 12-23.

10. Gardner, R. W., D. L. Martin and D. J. Weber, Allergenicity of Soybean with Milk Replacers to Calves, 1982 Proceedings of Dairy Science Annual Meeting (Abstract) 65:122.

11. Glimp, H. A., M. R. Karr, C. O. Little, P. G. Woolfolk, G. E. Mitchell, Jr., and L. W. Hudson. Effect of Reducing Soybean Protein Solubility by Dry Heat on the Protein Utilization of Young Lambs, 1967 Journal of Animal Science 26:858:861.

12. Hudson, L. W., H. A. Glimp, C. O. Little, and P. G. Woolfolk. Effect of Level and Solubility of Soybean Protein on its Utilization by Young Lambs. 1969 Journal of Animal Science. 28:279-282.

13. McNaughton, J. L. and F. N. Reece. Effect of Moisture Content and Cooking Time on Soybean Meal Urease Index, Trypsin Inhibitor Content, and Broiler Growth. 1982 Poultry Science 59:2300-2306.

14. Mielke, C. D. and D. J. Shingoethe. Heat Treated Soybeans for Lactating Cows. 1980 American Dairy Science Annual Meeting (Abstracts) 63: 138-139.

15. Morrill, J. L., K. C. Behnke and A. D. Dayton. Processed Soybeans for Young Calves. 1981 Journal of Dairy Science (Abstracts) 64:125-126.

16. Morrill, J. L., S. L. Melton, E. J. Guy and M. J. Pallansch. Performance of Calves Fed Milk Replacers Containing a Soybean Protein Concentrate and High Levels of Whey. 1969 Journal of Dairy Science 52:932.

17. Nishimuta, J. F., D. G. Ely and J. A. Boling. Ruminal Bypass of Dietary Soybean Protein Treated with Heat, Formalin and Tannic Acid. 1974 Journal of Animal Science 39:952-957.

18. Oil World - 4/30/82 No. 17 XXV:119 and A 49.

19. Raghavan, G. S. V., J. M. Harper and E. W. Kienholz. Nutritive Value of Salt-Bed Roasted Soybeans for Broiler Chicks, 1974 Poultry Science 53:547-553.

20. Schmutz, W. G., W. W. Cravens, W. L. Soldner and D. L. Hughes. Evolution of a Soybean Protein Concentrate in Calf Milk Replacers. 1967 Journal of Dairy Science 50:993.

21. Sissons, J. W. and R. H. Smith. The Effect of Different Diets Including Those Containing Soya-Bean Products, on Digesta Movement and Water and Nitrogen Absorption in the Small Intestine of the Pre-Ruminant Calf. 1976 British Journal of Nutrition. 36:421-438.

22. Smith, R. H. and J. W. Sissons. The Effect of Different Feeds, Including Those Containing Soya-Bean Products, on the Passage of Digesta from the Abomasum of the Preruminant Calf. British Journal of Nutrition. 33:329-349.

23. Thomas, E., A. Trenkle and W. Burroughs. Evaluation of Protective Agents Applied to Soybean Meal and Fed to Cattle I. Laboratory and Measurements, 1979 Journal of Animal Science 49:1337-1345.

24. Thomas, E., A. Trenkle and W. Burroughs. Evaluation of Protective Agents Applied To Soybean Meal and Fed to Cattle II. Feedlot Trials, 1979 Journal of Animal Science 49:1346-1356.

25. Yu, Y. Effect of Treating Full-Fat Whole Soybeans with Dry Heat or Formaldehyde on Digestibilities of Nitrogen and Polyenoic Acids. 1978 Journal of Dairy Science 61:128-131.

SULFUR AMINO ACID NUTRITION OF POULTRY: AN UPDATE

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The limiting nutrogenous factor in practical-type, grain-soybean meal diets for poultry is sulfur amino acids (SAA); ie., methionine plus cystine. Thus, DL-methionine or the hydroxy analogue of CL-methionine is generally fed at supplemental levels to both chickens and turkeys. Numerous reviews have been written that deal with sulfur nutrition of poultry (cf. Baker, 1977). It is my intention in this review to update recent relevant research with SAA that is of practical significance to poultry nutrition.

NEW SOURCES OF METHIONINE ACTIVITY

Methionine hydroxy analogue (Ca) is not new. It has existed as a marketable form of methionine activity for some time. It contains about 83% total SAA activity on a methionine activity basis, but somewhat less than this when assessed on a biological availability basis. With purified diets, DL-OH-met (Ca) contains about 60 to 70% bioactivity (weight basis) relative to DL-met (Katz and Baker, 1975; Featherston and Horn, 1974; van Weerden et al., 1982). With practical diets, the bioactivity of DL-OH-met (Ca) appears to be higher than this. The liquid form of LH-met (Monsanto) exists as the free acid and contains about 88% methionine activity, but less than this on a bioavailability basis (Boebel and Baker, 1982; van Weerden et al., 1982), although Romoser et al. (1976) and Waldroup et al. (1981) observed similar molar efficacies of DL-OH-met (Ca) and DL-OH-met.

The work from our laboratory was designed to delineate possible reasons for the lesser bioefficacy of the DL-OH-met products than of DL-methionine itself. While the D-isomer of methionine is slightly less efficacious than the L-isomer, the reverse is true with OH-methionine. Hence, L-OH-met (Ca) is less active biologically than D-OH-met (Ca), cf. Baker and Boebel (1980). The newer liquid form of DL-OH-met contains several OH-met polymers which exist in both linear and cyclic forms, and are in equilibrium with each other. Boebel and Baker (1982) studied a freeze-dried preparation of OH-met polymers consisting of 21% OH-met monomer, 35% dimer, 30% timer, 12% higher-order oligomers and 2% H_2O. This product was decidedly inferior as a source of methionine bioactivity as compared with liquid OH-met itself (which also contained polymers, but not in such a concentrated form). Hence, this suggests that some of the polymerized OH-met present in commercial OH-met may not be efficiently utilized by the chick. Data from our laboratory (Boebel and Baker, 1982) comparing several forms of methionine are presented in tables 1 and 2.

Because broiler feed mills have adapted to the technology of adding methionine to poultry diets in a liquid rather than a dry form, a new liquid form of DL-methionine existing as the sodium salt was recently introduced by Degussa Corporation. The product is 45.9% methionine (Na). On a sulfur basis, DL-met (Na) is equal in bioefficacy to DL-met itself (van Weerden et al., 1982; Anderson and Dobson, 1982). Thus, in using this product, a methionine activity value of 40% should be assumed.

Table 1. Efficacy of methionine and methionine hydroxy analog for chicks fed a chemically defined diet[1]

SAA source and level[2]	Gain(g)[4]	Gain/feed	% Efficacy (molar basis)
.20% DL-met	12.5^{a}	.22^{a}	
.30% DL-met	33.9^{c}	.33^{b}	
.40% DL-met	65.1^{d}	.50^{e}	100.0^{a}
.48% DL-OH-met (Ca)[3]	39.9^{c}	.41^{cd}	87.4^{b} ±4.49
.46% DL-OH-met (free acid)[3]	33.6^{c}	.36^{bc}	78.0^{bc} ±4.72
.38% DL-OH-met (free acid polymer)[3]	23.3^{b}	.30^{b}	69.4^{c} ±5.97
Pooled SEM	2.44	.022	

^{a,b,c,d,e}Means within a column not having a common superscript differ significantly ($P<.05$).

[1]Data are means of triplicate groups of five male chicks during the period 8 to 17 days posthatching; average initial weight was 53 g.

[2]The basal crystalline amino acid diet (Baker et al., 1979) was devoid of SAA.

[3]Isosulfurous to .40% DL-met.

[4]Multiple regression of chick gain (g) on supplemental met, OH-met, OH-met free acid, and OH-met free acid polymer intake was $Y = -3.4 + 19.1\ X_1 + 16.7\ X_2 + 14.9\ X_3 + 13.3\ X_4$ ($r = .99$), where Y represents chick gain and X_1, X_2, X_3, and X_4 represent DL-met, OH-met, OH-met free acid, and OH-met free acid polymer intake, respectively.

Table 2. Efficacy of methionine and methionine hydroxy analog for chicks fed a semipurified feather meal diet[1]

Treatment	Gain(g)[4]	Gain/feed	% Efficacy (molar basis)
Basal (B)[2]	5.7^{a}	.12^{a}	
B + .10% DL-met	32.9^{b}	.38^{b}	
B + .20% DL-met	89.2^{d}	.61^{d}	100.0^{a}
B + .24% DL-OH-met (Ca)[3]	62.5^{c}	.52^{c}	84.0^{b} ±2.31
B + .23% DL-OH-met (free acid)[3]	57.1^{c}	.49^{c}	76.5^{c} ±2.30
Pooled SEM	2.43	.010	

^{a,b,c,d}Means within a column not having a common superscript differ significantly ($P<.05$).

[1]Data are means of sextuplicate groups of five male chicks during the period 8 to 16 days posthatching; average initial weight was 84 g.

[2]The basal semipurified feather meal diet contained .11% methionine and 1.12% cystine from cystine and lanthionine.

[3]Isosulfurous to .20% DL-met. (Footnote 4 on next page).

[4]Multiple regression of chick gain (g) on supplemental met, OH-met, OH-met free acid, and OH-met free acid polymer intake (mmoles) was $Y = 6.4 + 42.4\ X_1 + 35.6\ X_2 + 32.4\ X_3$ ($r = .99$), where Y represents chick gain and X_1, X_2, and X_3 represent DL-met, OH-met, and OH-met free acid intake, respectively.

FACTORS ALLEGED TO SPARE OR ANTAGONIZE METHIONINE

Cystine. There is little question that dietary cystine can spare methionine. The literature is rather clear that cystine can provide up to 50% (by weight) of the broiler chick's dietary SAA requirement (Sasse and Baker, 1974). With diets first limiting in cystine (rather than methionine) and containing less than 200 mg/kg inorganic sulfate (from feed and water supplies), supplemental inorganic sulfate (eg., Na_2SO_4 or K_2SO_4) may elicit a small gain and efficiency response. Generally, however, the combination of feed ingredients (eg., fish products; dicalcium phosphate) plus water provide in excess of 200 mg/kg SO_4 such that a response in practice will not likely occur (Baker, 1977).

Poultry by-product meal (if it contains the feather meal) and feather meal are products rich in cystine. They also contain another sulfur amino acid - lanthionine - which has some bioavailable cystine activity (Baker et al., 1981). Thus, with ingredients such as these it is extremely important to give cystine its full replacement-value activity when computer formulating on a least-cost basis.

Choline. Recent work by Pesti et al. (1979, 1980) has been interpreted to suggest that excess dietary choline can spare the dietary need for supplemental methionine in poultry diets. Were this true, it would make sense to increase the level of supplemental choline chloride and decrease the level of supplemental DL-methionine used in poultry diets, since choline is less expensive at present than methionine. Teleologically, because both choline (in oxidized form, as betaine) and methionine (in adenylated form, as S-adenosyl-methionine) are involved metabolically in transmethylation reactions, it would seem reasonable that choline might exert a sparing effect on the dietary methionine requirement, and vice versa. In practice, however, recent evidence from Australia (Derilo and Balnave, 1980), Utah (Anderson and Dobson, 1982), and Illinois (Baker et al., 1982) has indicated that excess choline does not, in fact, spare methionine in this manner.

The Illinois work involved 1,230 battery-reared crossbred chicks. Experiment 1 employed a crystalline amino acid diet in an effort to establish the SAA and choline need for maximal feed efficiency of chicks during the period 8 to 18 days posthatching. Six graded levels of SAA (50:50 weight ratio of methionine and cystine) and five of choline (0 to 1216 mg/kg) were employed in a 6 x 5 factorial treatment arrangement. Gain/feed ratio was maximized at 608 mg/kg choline at all levels of SAA and at between .50 and .55% SAA at all levels of choline.

Table 3. Performance of chicks fed a crystalline amino acid diet containing graded levels of choline and methionine (Experiment 1)

Dietary	Dietary choline (mg/kg)[2]									
	0		304		608		912		1216	
SAA (%)	G	G/F[4]	G	G/F[4]	G	G/F[4]	G	G/F[4]	G	G/F[4]
.40[3]	35.6	415	48.3	430	63	488	69	511	62	489
.45	51.8	504	76.6	566	99.9	595	102	592	102	583
.50	68.2	558	83.1	606	110	659	120	688	112	654
.55	70.1	594	101	643	107	668	124	721	121	665
.60	61.0	565	110	679	103	660	127	703	113	635
.65	69.7	619	107	667	120	698	117	682	115	702

[1] Data represent means of triplicate groups of seven male chicks during the period 8 to 18 days posthatching; chicks weighed 61 g at day 8. All chicks had been pretested from days 5 to 8 on a choline-free purified diet (table 1) containing .35% DL-methionine and .20% L-cystine. Tabular data, ie., gain (G) and gain/feed (G/F) are expressed in g and g/kg feed, respectively. Pooled SEM for G and G/F were 4.7 g and 23.5 g/kg, respectively.

[2] Supplied by crystalline choline chloride.

[3] Contained .20% DL-met and .20% L-cys; additions were made with DL-met.

[4] Broken line SAA requirement estimates based upon gain/feed ratio were: .52% (r^2 = .94); .52% (r^2 = .95); .50% (r^2 = .96); .50% (r^2 = .97) and .50% (r^2 = .92) for chicks fed choline levels of 0, 304, 608, 912 and 1216 mg/kg, respectively.

Another experiment with the purified diet involved three levels of dietary DL-methionine: 0, .10 and .27% (in the presence of .27% L-cystine; .54% SAA representing the minimal SAA requirement for birds fed this diet), together with three levels of dietary choline: 0, 868 and 4340 mg/kg. Neither gain nor feed efficiency responded to choline supplementation at the two deficient levels of methionine, but a dramatic response to choline occurred at .27% dietary methionine.

Table 4. Effect of choline supplementation on performance of chicks fed two deficient and an adequate level of methionine[1]

Dietary	Dietary choline (mg/kg)[2]					
	0		868		4340	
DL-methionine (%)[3]	G	G/F	G	G/F	G	G/F
0	-5.3	-162	-3.9	-119	-4.7	-135
.10	8.7	155	9.9	181	8.6	159
.27	59.7	492	119.7	640	103.4	571

(Footnotes on next page).

[1]Data represent means of triplicate groups of seven male chicks during the period 8 to 18 days posthatching; average initial weight was 61 g at day 8. Gain (G) and gain/feed (G/F) data are expressed as g and g/kg feed, respectively.

[2]Supplied by crystalline choline chloride. The choline response was significant (P<.01) at the adequate level of methionine (.27%), but not at the two deficient levels.

[3]The basal diet was choline-free and contained .27% L-cystine. The methionine response was significant (P<.01) at all levels of choline.

Two trials with a practical-type corn-soybean meal diet (8 to 25 days posthatching) employed five graded levels of supplemental methionine (0 to .20%) and three of supplemental choline (0, 217 and 434 mg/kg). Optimal performance occurred at .10% supplemental methionine regardless of choline level, and at 217 mg/kg choline at supplemental methionine levels of 0 and .05%. At the .10, .15 and .20% supplemental methionine levels, no gain or gain/feed response to supplemental choline was evident. It was thus apparent that with practical diets, methionine could spare choline but that choline could not likewise spare the dietary need for methionine.

Table 5. Performance of chicks fed a corn-soybean meal diet with graded increments of supplemental choline and methionine (Experiments 2 and 3)[1]

Supplemental methionine (%)[3]	Supplemental choline (mg/kg)[2]					
	0		217		434	
	G	G/F[4]	G	G/F[4]	G	G/F[4]
0	276	581	273	591	274	590
.05	279	599	298	611	293	619
.10	292	622	299	628	298	624
.15	293	629	300	635	294	628
.20	302	644	298	640	294	625

[1]Data represent means of eight groups of five chicks (four male groups in Exp. 2 and four female groups in Exp. 3) during the period 8 to 25 days posthatching; male chicks averaged 62 g and females 74 g at day 8. Tabular data, ie., gain (G) and gain/feed (G/F) are expressed in g and g/kg feed, respectively. Pooled SEM for G and G/F were 3.8 and 4.5, respectively. Gain/feed responded (P<.01) to both methionine and choline addition.

[2]Supplied by a 60% choline chloride premix.

[3]The basal unsupplemented diet contained, by calculation, .38% methionine and .39% cystine (.77% SAA); and 980 mg/kg choline (using values of 1820 and 530 mg/kg choline for dehulled SBM and corn, respectively).

[4]The following interaction between methionine and choline for G/F was significant (P<.01): 0 and .05% vs. .10, .15 and .20% methionine X 0 vs. 217 and 434 mg/kg choline.

Lasalocid. The ionophorous anticoccidial drug, lasalocid, has been shown to exert a sparing effect on SAA (Patel et al., 1979; Marusich and DeYoung, 1979). Work from our laboratory (Willis and Baker, 1980) has confirmed that lasalocid can indeed spare SAA, but only when the diet is severely deficient in SAA; ie., just above maintenance levels. At moderate deficiencies of SAA, or of methionine, a SAA-sparing effect of lasalocid was not observed when either a crystalline amino acid diet or a corn-soybean meal diet was fed. Interestingly, the SAA level x lasalocid interaction could not be duplicated using monensin, a similar ionophorous anticoccidial drug. In any event, the interaction is fascinating from an academic standpoint, but appears to have no relevance to practical nutrition of broiler chickens.

Coccidiosis. Confusion exists with regard to the effect of coccidiosis on SAA requirements (Ruff, 1974; Ruff et al., 1976; Murrillo et al., 1976; Harms et al., 1967). A recent study involving duodenal coccidiosis caused by Eimeria acervulina infection indicated rather clearly that neither severe nor moderate infection with E. acervulina either increased or decreased the chick's requirement for SAA (Willis and Baker, 1981a). At most levels of dietary SAA, E. acervulina oocyst inoculation every three days brought about markedly depressed performance. But similar to the situation with 125 mg/kg lasalocid, oocyst inoculation of birds fed diets severely deficient in SAA yielded a marked gain and gain/feed response (Willis and Baker, 1981b).

Copper. Copper as $CuSO_4 \cdot 5H_2O$ is fed at 100 to 250 mg/kg to both swine (for growth promotion) and poultry (as an antifungal to help control crop mycosis). Because in vivo, copper can complex with the free sulfhydral group of cysteine or glutathione, excess copper may exacerbate SAA deficiency of chickens when marginal SAA levels are present in the diet. Robbins and Baker (1981a, b) have demonstrated that the SAA requirement of chicks is increased when 250 mg/kg copper, and especially when 500 mg/kg copper, is fed. Duodenal coccidiosis caused by E. acervulina increases copper absorption and deposition in tissues (Southern and Baker, 1982). Thus, it is possible that the SAA-antagonizing effect of copper may be even more severe in the presence of duodenal coccidiosis.

LITERATURE CITED

Anderson, J. O. and D. C. Dobson, 1982. Comparison of DL-methionine and its sodium salt in water solution in broiler starter diets with two choline levels. Poultry Sci. (In press).

Baker, D. H., 1977. Sulfur in Nonruminant Nutrition. NFIA Publ. Co., Des Moines, Iowa, 123 p.

Baker, D. H. and K. P. Boebel, 1980. Utilization of the D- and L-isomers of methionine and methionine hydroxy analogue as determined by chick bioassay. J. Nutr. 110:959-964.

Baker, D. H., R. C. Blitenthal, K. P. Boebel, G. L. Czarnecki, L. L. Southern and G. M. Willis, 1981. Protein-amino acid evaluation of steam-processed feather meal. Poultry Sci. 60:1865-1872.

Baker, D. H., K. M. Halpin, G. L. Czarnecki and C. M. Parsons. The choline-methionine interrelationship for growth of the chick. Poultry Sci. (In press).

Baker, D. H., K. R. Robbins and J. S. Buck, 1979. Modification of the level of histidine and sodium bicarbonate in the Illinois crystalline amino acid diet. Poultry Sci. 58:749-750.

Boebel, K. P. and D. H. Baker, 1982. Efficacy of the calcium salt and free acid forms of methionine hydroxy analogue for chicks. Poultry Sci. 61: 1167-1175.

Derilo, Y. L. and D. Balnave, 1980. The choline and sulphur amino acid requirements of broiler chickens fed on semi-purified diets. Brit. Poultry Sci. 21:479-487.

Featherston, W. R. and G. W. Horn, 1974. Studies on the utilization of the α-hydroxy acid of methionine by chicks fed crystalline amino acid diets. Poultry Sci. 53:680-686.

Harms, R. H., B. L. Damron and R. E. Bradley, 1967. Influence of artificially induced coccidiosis on the methionine requirement of laying hens. Avian Dis. 11:556-558.

Katz, R. S. and D. H. Baker, 1975. Factors associated with utilization of the calcium salt of methionine hydroxy analogue by the young chick. Poultry Sci. 54:584-591.

Marusich, W. L. and W. DeYoung, 1979. The effect of graded methionine levels fed with lasalocid or monensin on broiler performance, feathering, pigmentation and processing parameters. Poultry Sci. 58:1082 (Abstr.).

Murrillo, M. G., L. S. Jensen, M. D. Ruff and A. P. Rahn, 1976. Effect of dietary methionine status on response of chicks to coccidial infection. Poultry Sci. 55:642-649.

Patel, M. B., K. O. Bishawi and J. McGinnis, 1979. Effect of drug additives and type of diet on methionine requirement of chicks for growth and feed efficiency. Poultry Sci. 58:549-554.

Pesti, G. M., A. E. Harper and M. L. Sunde, 1979. Sulfur amino acid and methyl donor status of corn-soy diets fed to starting broiler chicks and turkey poults. Poultry Sci. 58:1541-1547.

Pesti, G. M., A. E. Harper and M. L. Sunde, 1980. Choline/methionine nutrition of starter broiler chicks. Three models for estimating the choline requirement with economic considerations. Poultry Sci. 59:1073-1081.

Robbins, K. R. and D. H. Baker, 1980a. Effect of high-level copper feeding on the sulfur amino acid need of chicks fed corn-soybean meal and purified crystalline amino acid diets. Poultry Sci. 59:1099-1108.

Robbins, K. R. and D. H. Baker, 1980b. Effect of sulfur amino acid level and source on the performance of chicks fed high levels of copper. Poultry Sci. 59:1246-1253.

Romoser, G. L., P. L. Wright and R. B. Grainger, 1976. An evaluation of the L-methionine activity of the hydroxy analogue of methionine. Poultry Sci. 55:1099-1103.

Ruff, M. D., 1974. Reduced transport of methionine in intestines of chickens infected with Eimeria necatrix. J. Parasitol. 60:838-843.

Ruff, M. D., D. R. Whitlock and R. R. Smith, 1976. Eimeria acervulina and E. tenella: effect on methionine absorption by the avian intestine. Exp. Parasitol. 39:244-251.

Sasse, C. E. and D. H. Baker, 1974. Sulfur utilization by the chick with emphasis on the effect of inorganic sulfate on the cystine-methionine interrelationship. J. Nutr. 104:244-251.

Southern, L. L. and D. H. Baker, 1982. Eimeria acervulina infection in chicks fed excess copper in the presence or absence of excess dietary methionine. J. Anim. Sci. 54:989-997.

van Weerden, E. J., H. L. Bertram and J. B. Schutte, 1982. Comparison of DL-methionine, DL-methionine-Na, DL-methionine hydroxy analogue-Ca and DL-methionine hydroxy analogue-free acid in broilers by using a crystalline amino acid diet. Poultry Sci. 61:1125-1130.

Waldroup, P. W., C. J. Mabray, J. R. Blackman, P. J. Slagter, R. J. Short and Z. B. Johnson, 1981. Effectiveness of the free acid of methionine hydroxy analogue as a methionine supplement in broiler diets. Poultry Sci. 60:438-443.

Willis, G. M. and D. H. Baker, 1980. Lasalocid-sulfur amino acid interrelationship in the chick. Poultry Sci. 59:2538-2543.

Willis, G. M. and D. H. Baker, 1981a. Eimeria acervulina infection in the chicken: sulfur amino acid requirement of the chick during acute coccidiosis. Poultry Sci. 60:1892-1897.

Willis, G. M. and D. H. Baker, 1981b. Interaction between dietary protein/amino acid level and parasitic infection: morbidity in amino acid deficient or adequate chicks inoculated with Eimeria acervulina. J. Nutr. 111:1157-1163.

AMINO ACID BALANCE AND IMBALANCE: IMPORTANCE IN SWINE RATIONS

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Much has been written in the experimental nutrition literature about amino acid balance and imbalance. In general, the term "amino acid imbalance" means a pattern of dietary amino acids that is out of balance. This could take the form of a single amino acid deficiency (eg., lysine in sesame meal) or of multiple amino acid deficiency (eg., gelatin), but it could just as easily occur as a result of severe amino acid excesses over and above certain marginal amino acid levels (eg., excess lysine in relation to arginine and excess leucine in relation to isoleucine in blood meal). In fact, no intact protein even comes close to having a perfect amino acid balance.

Amino acid antagonism is a kind of amino acid imbalance in which, in addition to a depression in the voluntary intake of feed, a specific amino acid is antagonized or rendered less useful. Several examples of antagonism exist in the chick and rat literature. Thus, in both chicks and rats, excess lysine antagonizes arginine, excess dietary leucine antagonizes isoleucine and valine and excess methionine antagonizes threonine and glycine. In the chick, it is known that excess lysine (1) induces kidney arginase activity, (2) inhibits liver transamidinase activity and (3) competes with arginine for reabsorption from the kidney tubule. Also, it has been shown in both chicks and rats that excess dietary methionine induces liver threonine dehydratase activity and increases the rate of threonine catabolism. With excess leucine, no clear-cut explanation has been advanced to explain its interference with isoleucine and valine utilization.

The practical relevance of amino acid balance and imbalance has been dealt with before. Nonetheless, since several feed companies are currently advertising swine feeds on the basis of minimization of excess amino acids, it would be instructive to deal specifically with the potential ramifications of amino acid imbalance for swine.

AMINO ACID IMBALANCE IN SWINE DIETS

Is amino acid imbalance a practical problem in swine feed formulation? Obviously, extreme examples can be cited where excess amino acids or imbalanced amino acid patterns have caused reductions in rate and efficiency of swine weight gain. But what about excess amino acids in the (already well balanced) corn-soybean meal diet?

Among the indispensable amino acids found in excess when corn-soybean meal diets are fed at protein levels designed to meet the need for the limiting amino acid (lysine), leucine, arginine and phenylalanine-tyrosine are found in greatest excess. Do they cause problems at these excess levels? Will performance be improved by minimizing the levels of these excess amino acids? The answer is: we simply don't know! Thus, despite the suggestion by some that diets with "minimized excess amino acids" will perform better in the feedlot than conventional corn-soybean meal diets, no convincing and unequivocal evidence has been published to support this contention. The primary reason for our not having the hard evidence pro or con is that it is extremely difficult (and very costly) to

obtain solid evidence in this area of research, and feed companies are generally not inclined to put hard dollars into university research that will answer these kinds of questions. This is understandable given the competitive nature of their business. It is also, however, unfortunate.

LEUCINE IMBALANCE

Leucine costs about $36.00 per kilogram. To feed excess levels of leucine similar to those found in corn-soybean meal diets (and higher levels would be desirable, also) in experiments with meaningful numbers of pigs would be exceedingly costly. Even then, one could question the extrapolative relevance since the completeness and time of absorption from gut to bloodstream would be different for crystalline leucine than for the leucine bound in peptide linkage in intact protein. Leucine, isoleucine and valine concentrations in some common swine feedstuffs are shown in Table 1.

TABLE 1. LEUCINE, ISOLEUCINE AND VALINE CONTENT OF INGREDIENTS COMMONLY USED IN SWINE DIETS

		% of ingredient			Ratio	
	CP	Leucine(L)	Isoleucine(I)	Valine(V)	L/I	L/V
Protein supplements						
Blood meal	80	10.3	0.9	5.2	11.4	1.98
Corn gluten meal	60	9.4	2.3	2.7	4.09	3.48
Corn gluten feed	21	1.9	0.6	1.0	3.17	1.90
Feather meal	85	7.8	2.7	4.6	2.89	1.70
DDGS	27	2.7	1.0	1.3	2.70	2.08
Peanut meal	45	3.6	1.8	2.6	2.00	1.38
Poultry by-pdt meal	58	4.4	2.3	2.7	1.91	1.64
Meat and bone meal	50	3.2	1.7	2.3	1.88	1.39
Cottonseed meal	41	2.4	1.3	1.8	1.85	1.33
Meat meal	55	3.5	1.9	2.6	1.84	1.35
Alfalfa meal	17	1.1	0.6	0.6	1.83	1.83
Distillers solubles	27	2.1	1.2	1.2	1.75	1.75
Soybean meal	44	3.4	2.5	2.4	1.36	1.42
Fish meal	62	3.4	2.8	3.4	1.21	1.00
Grains						
Corn	8.5	1.00	0.37	0.42	2.70	2.38
Sorghum	10.0	1.40	0.60	0.53	2.33	2.64
Barley	11.5	0.80	0.42	0.62	1.90	1.29
Oats	11.0	0.90	0.53	0.62	1.70	1.45
Wheat	11.0	0.80	0.50	0.50	1.60	1.60

There is ample evidence with laboratory species fed purified diets that excess levels of dietary leucine can impair the body's capacity to utilize isoleucine and valine. The limited work done with pigs, however, has been less than conclusive. Thus, Minnesota workers found that while excess leucine reduced plasma levels of isoleucine and valine, very little reduction in rate and efficiency of gain occurred (Oestemer et al., 1973). French workers (Henry et al., 1976) observed the same results in plasma, but concluded that a doubling of the ratio of leucine to

isoleucine in pig diets had no deleterious effect on performance. Neither of these studies, however, involved a great excess of leucine, 1.15% total dietary leucine being the highest level fed. Thus, though it would be costly, pig studies with higher levels of leucine need to be carried out before definitive conclusions can be drawn concerning leucine imbalance.

ARGININE IMBALANCE

L-arginine (free base) costs about $28.00 per kilogram. Hence, it, too, is very expensive when used for swine studies designed to assess the effects of excess arginine on pig performance. Data on arginine and lysine concentrations and their ratio in some common swine feedstuffs are shown in Table 2. It is obvious that arginine is found in excess in most feed ingredients, particularly in relation to the pig's estimated arginine requirement of about 0.24% of the diet (recent evidence from our laboratory, however, suggests the requirement may be almost double this level, cf. Southern and Baker, 1982). Some feed companies have suggested that excess arginine in swine rations antagonizes lysine and that this manifests as reduced rate and efficiency of weight gain. If this were true, it would seem wise to incorporate either blood meal or fish meal into the diet as a replacement for some of the soybean meal. Whether this would, in fact, improve pig performance is questionable. In fact, careful study of Tables 1 and 2 reveals that use of blood meal as a replacement for some of the soybean meal would lower the dietary arginine:lysine ratio, but it would also elevate the leucine:isoleucine ratio. Hence, to minimize both arginine:lysine and leucine:isoleucine ratios, fish meal would seem a better choice than blood meal, although it should be noted that soybean meal, itself, has a minimum of excess leucine in relation to either isoleucine or valine. Also worthy of note, blood meal is extremely rich in phenylalanine + tyrosine, the other pair of amino acids which are in great excess already in corn-soybean meal swine diets. Hence, because of the above factors, because solid data has not been presented, and because blood meal is known to be variable in quality and generally rather high in price, caution should be exercised in the use of blood meal for swine diets.

Certainly, with chicks and rats, it is excess lysine antagonizing arginine rather than arginine antagonizing lysine that represents the greater problem (Allen and Baker, 1972; Austic and Scott, 1975). In a recent University of Illinois experiment (Hevea et al., 1980) 5% excess arginine did not depress growth of rats fed a 15% CP methionine-fortified casein-sucrose diet! On the other hand, lysine at 5% was growth depressing, and the depression could be alleviated by 1% additional arginine. Thus, excess dietary arginine seems to be rather innocuous when administered to rats.

What about the situation in pigs? Little published data exist, but some work has been done at the University of Illinois (Southern and Baker, 1981), Texas A&M University (Tanksley, 1981), Michigan State University (Miller et al., 1981) and South Dakota State University (Hagemeier et al., 1982). The results from these stations, taken as a whole, suggest that the arginine level found in common swine weaner diets (ie., 1.3%) is not antagonistic to lysine (a common lysine level in swine weaner diets is 1.0 to 1.1%). In fact, crystalline arginine additions to bring total dietary arginine to a level of 1.6 to 1.8% (arginine-lysine ratio of 1.6 to 1.8) has had no deleterious effects on swine performance. Levels of arginine above 1.8% begin to depress feed intake and thereby also depress weight gain. But the response pattern indicates simple amino acid imbalance rather than amino acid antagonism.

TABLE 2. LYSINE AND ARGININE CONTENT OF INGREDIENTS COMMONLY USED IN SWINE DIETS

Ingredient	% of ingredient			Ratio
	CP	Arginine(A)	Lysine(L)	A/L
Protein supplements				
Feather meal	85	3.9	1.0	3.90
Peanut meal	45	4.7	1.6	2.94
Cottonseed meal	41	4.7	1.7	2.76
Corn gluten meal	60	1.9	1.0	1.90
Corn gluten feed	21	1.0	0.6	1.67
DDGS	27	1.0	0.6	1.67
Poultry by-pdt meal	58	3.8	2.6	1.46
Meat + bone meal	50	3.4	2.6	1.31
Meat meal	55	3.7	3.0	1.23
Soybean meal	44	3.4	2.9	1.17
Distillers solubles	27	1.0	0.9	1.11
Alfalfa meal	17	0.6	0.6	1.00
Fish meal	62	3.2	4.7	0.68
Blood meal	80	2.4	5.3	0.45
Grains				
Corn	8.5	0.55	0.24	2.29
Oats	11.0	0.80	0.40	2.00
Sorghum	10.0	0.40	0.25	1.60
Wheat	11.0	0.40	0.30	1.33
Barley	11.5	0.50	0.50	1.00

A recent study (Table 3) from the Illinois Station (Southern and Baker, 1981) took a different approach in that a basal semipurified diet containing 1.3% arginine and 1.0% lysine (Diet 2) was modified to contain only .80% arginine together with the 1.0% lysine (Diet 1). This lowering of the arginine level had no effect on swine performance. In fact, addition of 1.0% L-arginine to give a total dietary arginine concentration of 1.8% (Diet 3) likewise had no deleterious effects on either growth rate or feed efficiency. Only at total dietary arginine levels of 2.3% and 2.8% did rate of gain decrease due entirely to a decrease in voluntary feed intake, since feed efficiency was unaltered by these high (and totally unphysiologic) levels of arginine. The performance data of this experiment are strongly supportive of amino acid imbalance rather than amino acid antagonism being responsible for the decreased performance in pigs fed the two highest levels of arginine.

Plasma arginine and ornithine were increased linearly as dietary arginine level increased; plasma lysine, on the other hard, decreased as arginine in the diet increased. Several other amino acids in plasma decreased linearly as well (aspartate, glutamate, glutamine, proline, glycine, asparagine, tyrosine, alanine, methionine and leucine) such that it cannot be concluded that plasma lysine was affected, specifically, by excess dietary arginine.

Urinary excretion of the basic amino acids was increased markedly by the addition of 2.0% arginine. Twenty-four-hour output of cystine and histidine was likewise increased by the highest level of arginine addition.

TABLE 3. PERFORMANCE, PLASMA AND URINARY AMINO ACID CONCENTRATIONS OF PIGS GRADED LEVELS OF EXCESS ARGININE[1]

Dietary Addition	Daily gain(g)[2]	G/F[3]	Plasma AA (nmole/ml) Arg[4]	Lys[4]	Orn[4]	Urinary AA (μmole/24 hr.) Arg[5]	Lys[5]	Orn[5]	Cit[5]
1. Basal (B)[6]	407	.579	108	158	125	21	94	29	3
2. B + .5% L-Arg	407	.575	178	123	180	177	480	209	29
3. B + 1.0% L-Arg	397	.595	230	104	237	167	381	244	34
4. B + 1.5% L-Arg	347	.585	265	82	268	220	144	135	11
5. B + 2.0% L-Arg.	342	.555	367	83	319	2145	1638	1757	120
Pooled SEM	11	.014	31	14		108	113	220	24

[1]Data are means of four replicates of 4 (performance), 3 (plasma) or 2 (urine) pigs each; average initial weight was 6.9 kg; 26-day trial.
[2]Diets 1 to 3 not significantly different; diets 3 to 5 - linear depression (P<.01).
[3]No significant differences among means.
[4]Arginine linear effect significant (P<.01) using diets 1 to 5.
[5]The mean of diet 5 was greater (P<.03) than the pooled means of diets 1 to 4.
[6]Contained 14.7% CP, 1.0% lysine and .80% arginine.

POTASSIUM

It has been known for some time that under certain dietary conditions, supplemental potassium can alleviate lysine imbalances brought about by adding (excess) L-lysine·HCl to diets for laboratory animals. Recent work from Cornell had suggested that (excess) potassium might exert a sparing effect on the dietary requirement for lysine. With practical diets, however, no benefits were noted in Iowa and Michigan work from adding K_2CO_3 to low lysine diets for weanling pigs (cf. Zimmerman, 1982; Froseth et al., 1982).

It seems logical that minimization of excess amino acids in swine rations might prove beneficial to swine performance. However, to accomplish this in practice without materially increasing the cost of the ration is difficult. Moreover, the array of ingredients that might be used to accomplish a diet with minimum excesses could introduce other variables and thus other problems (eg., palatability, bioavailability of amino acids and other nutrients, excesses of minerals, processing variability, inhibitor factors). Hence, the simple fortified corn-soybean meal diet should still be considered the standard for comparison. Certainly, the complementary effects of combining soy protein with corn protein bring about an excellent pattern of amino acids for meeting swine amino acid requirements, resulting in only modest amino acid excesses. It would therefore seem prudent to formulate to given levels of lysine, using corn and soybean meal, keeping a watchful eye on dietary crude protein level and total dietary energy level. The available evidence suggests it would be premature at this time to formulate swine feeds on the basis of minimization of either leucine or arginine.

LITERATURE CITED

Allen, N. K., D. H. Baker, H. M. Scott and H. W. Norton. 1972. Quantitative effect of excess lysine on the ability of arginine to promote chick weight gain. J. Nutr. 102:171-180.

Austic, R. E. and R. L. Scott. 1975. Involvement of food intake in the lysine-arginine antagonism in chicks. J. Nutr. 105:1122-1131.

Froseth, J. A., P. K. Ku and E. R. Miller. 1982. Addition of potassium carbonate to swine diets containing varying levels of lysine. Proc. Midwest ASAS, p. 97.

Hagemeier, D. L., G. W. Libal and R. C. Wahlstrom. 1982. Effect of excess arginine on lysine utilization in weaned pigs. Proc. Midwest ASAS, p. 96.

Hevia, P., E. A. Ulman, F. W. Kari and W. J. Visek. 1980. Serum lipids of rats fed excess L-lysine and different carbohydrates. J. Nutr. 110:1231-1239.

Miller, E. R., J. Skomial and P. K. Ku. 1981. Nitrogen and energy balance of pigs fed corn-soy or balanced amino acid diets. J. Anim. Sci. 53 (Suppl 1):255.

Southern, L. L. and D. H. Baker. 1981. Performance and plasma amino acids of young pigs fed excess lysine and/or arginine. J. Anim. Sci. 53 (Suppl 1):264.

Southern, L. L. and D. H. Baker. 1982. Arginine requirement of young pigs. J. Anim. Sci. (Suppl 1) - In press.

Tanksley, T. D. 1981. Protein and amino acid nutrition of young pigs. Feed Management 32(2):16-17.

Zimmerman, D. R. 1982. Lysine and potassium levels in pig starter diets. Proc. Midwest ASAS, p. 97.

ANTIBACTERIALS AND SWINE MANAGEMENT

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For three decades, antibacterial feed additives have played a major role in the growth and development of the swine industry. Antibacterials are used extensively as growth promotors in swine diets. Improved gains and feed efficiency are observed. These improvements are generally considered economically beneficial to swine production systems. Several antibacterials are available, each having been tested in numerous experiments. Research to date includes: level of antibacterial; comparisons among antibacterials and response of pigs during various stages of production. Modes of action are still under question. One theory put forward deals with the ability of antibacterials to suppress or control subclinical diseases. As production stresses increase, the pig is predisposed to greater incidence of disease. Antibacterials function to inhibit the growth of harmful microorganisms keeping diseases in check.

Several management factors related to disease control will effect the degree to which pigs respond to antibacterials. Claims and disclaimers have been made without considering some of these variables of production. These factors should be determined through research efforts. The results could then be considered when recommendations are made to the pork producers that involve the use of antibacterials to control subclinical disease.

STAGE OF GROWTH

The stress on the postweaning pig increases dramatically as weaning age decreases. This has become especially evident as producers move toward more intensive production systems that require weaning at an early age. The pig must adapt to new facilities and pen mates, a change in diet and a greater pathogen level. The combination of these stresses results in a growth depression (postweaning slump) and a greater incidence of disease. It is during this phase, that pigs respond best to antibacterials used as growth promotants in the diet. The percent response decreases dramatically as the pig becomes older. Hays (1979) summarized several trials involving the use of antibacterials at various stages of growth. The percent response in pig gains was much greater during starter phase (16%) as compared to the combined starter and grower phases (10%) and the combined starter, grower and finisher phases (4%). Supplementing the diet with high levels of copper results in a similar decrease in percent response with increasing age of the pig. Wallace (1967) reported that starter, grower, and finisher pigs fed diets supplemented with copper gained 22.1, 6.5 and 3.6 percent faster than pigs fed control diets.

DISEASE LEVEL - ENVIRONMENT

The environment, especially disease level, surrounding the pig must be considered when evaluating the pig's response to an antibacterial. The response of the antibacterial is inversely proportional to the disease level of the pig and environment. Thus, a high disease level would afford a greater response. Hays and Speer (1960) investigated the effect of two management systems and the additon of an antibacterial to the diet for starter pigs (5.4 kg) on commercial

swine farms. One management system involved emptying and cleaning the house, after which all pigs were moved in and started on trial at the same time. In the other management system, individual pens were cleaned as pens of pigs were allotted to the test at different times. The house was not empty of other pigs prior to or during the trial. The addition of an antibacterial (spiramycin, 50 gm/ton) to the diet improved pig gains in both management systems. However, the percent response (as compared to control) was significantly greater for pigs in the suspected poor environment (75%) than for pigs in the good environment (33%). Wachholz and Heidenriech (1970) reported a greater response to tylosin in starter, grower and finisher pigs raised on dirt lots that had been used previously for pig production as compared to a new environmentally controlled confinement facility (Table 1).

Table 1. Antibiotic x Environment in Growing Pigs[a]

	% Response in Gain		
	Starter	Grower	Finisher
New confinement facility	13	1	4
Old dirt lots	21	17	10

[a]Adapted from Wachholz and Heidenriech, 1970.

The response of antibacterials as affected by environment has raised discussions of various intensity about the "true" response in growing pigs. Research conducted at universities have consistently showed lower responses to antibacterials than research conducted by pharmaceutical companies on commercial farms in the field. The latter group contends that the lower response observed by the universities is due to a generally better management. The response to tylosin is dependent upon type of facility as reported by Natz (1975) from a review of tylosin finisher efficacy data conducted by Elanco. On station, university and field trials reported a percent improvement of 2.0, 3.4 and 6.0% in gain to market weight and 0.7, 2.9 and 5.0% in feed efficiency, respectively.

A trial was conducted at the University of Minnesota Sourthern Experiment Station at Waseca to investigate the effects of the addition of antibacterials to the diet of starter pigs subjected to a less than ideal environment. After weaning at approximately 32 days of age, starter pigs were allotted to partially slatted concrete pens with open partitions in a facility designed for grow-finishing pigs. The feeders were too large, but the pigs were able to eat with some difficulty. No pigs died during the 28-day trial but gains were low (.49 pounds/day). Pigs fed antibacterials gained 33.3% faster than pigs fed control diets. This percent response is higher than would be expected for starter pigs fed antibacterials in more ideal conditions.

Improvements in performance can be much greater in the field under practical conditions than in a controlled research facility, the latter having more hours of labor per pig, more all in all out systems, longer clean up time, fewer pigs per pen, and only healthy pigs on trials. Sainsbury (1975) has reported that percent improvement in gain is higher for unhealthy pigs (30%) than healthy pigs (10%).

The relative improvement in growth rate resulting from the addition of antibacterials to the diet is inversely proportional to the growth rate of the control pigs

when the improvements are expressed as a proportion of the controls (Braude et al., 1953 and Melliere et al., 1973). In contrast, Jones and Tarrant (1982) have reported evidence that the improvements in gain from the addition of tylosin to the diet is constant over a normal range of growth rates of control pigs if the improvements are expressed in absolute terms (gm/day) and not as a proportion of the controls. These results suggest that the efficacy of tylosin is not dependant on the performance of the controls. Nevertheless, producers can expect a decrease in response to antibacterials as improvements are made in the genetics, nutrition, herd health and facilities of their production systems. In fact, a well managed system may produce an economical benefit for the reduction of antibacterial usage, especially during the stages of growth where the response is already low (e.g. finishing).

SPACE ALLOWANCES

Optimum floor space allowances have been established for various weights of pigs housed in confinement. Pig buildings are designed with a predetermined number and size of pens to allow the appropriate space per pig and to accommodate the flow of pigs through the building. However, even in the normal management of a swine operation, crowded conditions can occur and may be due to: 1) the inflexibility of a permanent pen arrangement, 2) the need to keep the buildings in constant use which results in periodic overproduction of pigs, or 3) certain groups of pigs may experience an extended number of days to market weight.

Depending on the severity, a crowded condition will decrease feed intake, gain and feed efficiency of growing pigs. Therefore, the establishment of the optimum space requirement for pigs is essential to maintain maximum performance. Accurate assessment of space allowance may be dependant on a number of factors such as: group size, season/environmental temperature, floor type, air movement, feed and water availability and social structure of a pen of pigs. Because of the greater stress, pigs experiencing crowded conditions may have a greater response to the addition of an antibacterial to the diet than pigs allowed optimum floor space. This station has conducted several trials with starter and grow-finishing pigs to determine the interacting effects of antibacterials and space allowance.

Three trials (15 replications) were conducted at two stations using starter pigs in raised nursery decks (Moser et al., 1982b). Pigs were allowed either 1.5 or 2.5 sq. ft. per pig and fed either a corn-soybean meal control diet or control plus ASP-250 providing 100 g chlortetracyclin, 100 g sulfamethazine and 50 g penicillin per ton during a 28-day feeding period. Pigs fed ASP-250 had greater gains, feed intakes, and feed efficiency than pigs fed the control diet. Space allowance had no effect on the performance of pigs during the first week. For the total test period, pigs allowed 1.5 sq. ft. per pig had lower feed intakes and gains than pigs allowed 2.5 sq. ft. per pig. The response of pigs to antibacterials was similar for both space allowances, 11.9% improvement in gain for pigs allowed 1.5 sq. ft. per pig and 17.8% for pigs allowed 2.5 sq. ft. per pig (Table 2). Even though percent response was not greatly affected, the performance of crowded pigs fed the antibacterial was similar to that of pigs allowed optimal floor space and fed the control diet. These data suggest that the addition of antibacterials to the diet would help to overcome the poor performance resulting from the crowding of pigs; however, even greater improvement in performance can be achieved if pigs are fed antibacterials and allowed optimal floor space.

Table 2. Space Allowance and Antibacterials for Starter Pigs[a]

Performance	1.5 sq. ft./pig		2.5 sq. ft./pig	
	Control	C + ASP-250	Control	C + ASP-250
Week 1				
Avg. daily gain (lbs)[b]	.17	.21	.17	.30
Avg. daily feed (lbs)	.59	.60	.61	.68
Gain/feed	.29	.35	.28	.44
Total 28-day period				
Avg. daily gain (lbs)[bc]	.67	.75	.73	.86
Avg. daily feed (lbs)[bc]	1.23	1.34	1.34	1.45
Gain/feed	.55	.56	.55	.59

[a]Summary of three trials; 15 replications.
[b]Significant effect of antibacterial (P .05).
[c]Significant effect of space allowance (P .05).

Grow-finishing pigs experiencing a crowded condition show no advantage in improved performance when an antibacterial is fed. Three trials (7 replications) were conducted at two stations (Moser et al., 1982a). Pigs were allowed 4, 3.5 or 3 sq. ft. per pig during the growing period and 8, 7 or 6 sq. ft. per pig during the finishing period and fed either a control diet (C) or control plus virginiamycin (V) at 10 grams/ton. Space allowances were acheived by varying pen size. Number of pigs (9) and feeder space were constant for all pens. Pig gains and feed intakes decreased linearly as space allowance decreased (Table 3). The addition of virginiamycin to the diet had no effect on pig performance. Pigs fed a diet with or without antibacterials responded similarly to varied space allowance. These data suggest that the addition of virginiamycin to the diet was ineffective in overcoming the decreases in performance in grow-finishing pigs experiencing crowded conditions. It might be postulated that a response to antibacterials would be observed if the number of pigs per pen were increased to more closely mimic practical conditions. A study is in progress to investigate the response of pigs fed tylosin and experiencing crowded conditions with 19 pigs per pen.

Table 3. Space Allowance and Antibacterials for Grow-finisher Pigs[a]

Performance	Space allowed (grower-finisher) per pig (sq.ft.)					
	4 - 8		3.5 - 7		6 - 3	
	C	V	C	V	C	V
Avg. daily gain (lbs)[b]	1.59	1.52	1.45	1.48	1.48	1.45
Avg. daily feed (lbs)[b]	4.81	5.20	4.76	4.63	4.69	4.61
Gain/feed	.32	.30	.31	.30	.31	.32

[a]Summary of three trials; 7 replications.
[b]Significant effect of space allowance.

COPPER AND ANTIBACTERIALS

High levels of copper in the diet for growing pigs have been used for several years to improve growth rate and feed efficiency for growing swine. The most widely accepted theory for its mode of action is its bacteriostatic effect on the intestinal microflora of the pig. Recently, this station has conducted several trials to compare the response of copper and an antibacterial and to determine if an additive response can be achieved by feeding copper and an antibacterial in combination (Hagen, 1982).

Table 4 shows the results of five trials conducted at four locations with starter pigs fed various levels and types of antibacterials. Pigs responded similarly to copper and the antibacterial with the exception of trial 2. In four of five trials even greater improvements in performance were shown when the combination was fed. The copper - carbadox combination showed no added response. The greatest response to the combination was in pigs experiencing the poorest environment for starter pigs (trial 5). It can be concluded that starter pigs fed high levels of copper showed improved performance similar to pigs fed antibacterials, and that the combination of copper and certain antibacterials promote an even greater response than either fed alone.

Table 4. High Level Copper and Antibacterials for Starter Pigs

	% Response in Gain		
Trial	Antibacterials	Copper	Combination
1 (St. Paul)	11	13	21
2 (St. Paul)	0	30	37
3 (Rosemount)	13	18	35
4 (Rosemount)	31	30	31
5 (Waseca)	23	26	51

Trial 1 - copper, 125 ppm; chloretetrocycline, 20 gm/ton.
Trial 2 - copper, 250 ppm; chloretetracycline, 50 gm/ton.
Trials 3 & 5 - copper, 250 ppm; chloretetracycline, 100 gm/ton; sufamethizine, 100 gm/ton; penicillin, 50 gm/ton.
Trial 4 - copper, 250 ppm; carbadox, 50 gm/ton.

CONCLUSION

Even though the efficacy of antibacterials is widely accepted, the degree to which pigs respond will vary and is dependant on several environment and management-related factors. Pigs respond better to antibacterials during the starter phase, with the response decreasing with increasing age. The response is greater if the pigs are unhealthy and/or subjected to an environment that is less than ideal. In these situations, the subclinical diseases are controlled and improvements in performance are observed. Antibacterials benefit crowded pigs during the starter phase but seem to have no benefit for grow-finishing pigs experiencing crowded conditions. Copper and antibacterials fed in combination result in an additive response in pig performance as compared to the response when either is fed alone.

REFERENCES

Braude, R., D.H. Wallace and T.J. Cunha. 1953. The value of antibiotics in the nutrition of swine, a review. Antibiotics and Chemotherapy 3:271.

Hagen, C.D. 1982. The effect of copper sulfate and intibiotics on the performance of weaned pigs. M.S. Thesis. University of Minnesota.

Hays, V.W. 1979. Effectiveness of feed additive usage of antibacterial agents in swine and poultry production. Prepared for the Office of Technology Assessment, United States Congress.

Hays, V.W. and V.C. Speer. 1960. Effect of spiramycin on growth and feed utilization of young pigs. J. Anim. Sci. 19:938.

Jones, P.W. and M.E. Tarrant. 1982. The effect of various factors on the efficacy of tylosin as a growth promotor in clinically healthy pigs. Anim. Prod. 34:115.

Mellier, A.L., H. Brown and R.P. Rathmacher. 1973. Finishing swine performance and responses to tylosin, J. Anim. Sci. 37:286.

Moser, R.L., S.G. Cornelius, J.E. Pettigrew, H.E. Hanke and C.D. Hagen. 1982a. Response of grow-finishing pigs to varying space allowance and the addition of antibiotics to the diet. Abstract submitted to Annual Meeting of American Society of Animal Science, University of Guelph, Ontario, Canada.

Moser, R.L., S.G. Cornelius, H.E. Hanke and C.D. Hagen. 1982b. Response of starter pigs to varying space allowance and the addition of antibiotics to the diet. Minnesota Swine Report, p. 18.

Natz, D. 1973. Report measures economic gains from feed antibiotics. Feedstuffs 45:2:16.

Sainsbury, D. 1975. Making better use of antibiotics as growth promotors. Pig Farming 23:4:27.

Wachholz, D.E. and C.J. Heidenriech. 1970. Effect of tylosin on swine growth in two environments. J. Anim. Sci. 31:1014.

Wallace, H.D. 1967. High level copper in swine feeding. International Copper Research Assoc., Inc. New York.

Modeling Swine Growth

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Prefactory Remarks

"Order and simplification are the first steps toward mastery of a subject. The actual enemy is the unknown."

Thomas Mann

The concept of simulation is both simple and appealing. The operator of a simulation can experiment with systems (both real and imaginary) controlling or altering processes in a manner otherwise impossible or impractical. As a result of these experiments, the operator becomes more familiar with the behavior and operation of the system if it exists, or of the faults of the imaginary system without requiring its physical existence. Recall the near flawless first flight of the space shuttle, a network of complex systems for the most part designed and tested through the application of simulation techniques. Simulation thus becomes one of the most powerful analysis tools available for use by those responsible for the design and operation of complex processes. Are the biological processes of the pig any less complex than physical processes of the space shuttle?

Simulation can serve to enlighten or mislead. Like any tool with some dependance on art for its proper application, simulation is capable of producing either very good or very bad results depending on how it is used. It is of paramount importance that the operator (and to a lesser extent the consumer of the results) of a simulation be painfully aware of the implications of the various assumptions which must be made, the strengths and weaknesses of the approach and the sundry pitfalls, in addition to the more apparent benefits.

Scientists tend to have simple goals and are in fact forced to that end by a major precept of the scientific method which requires that the objective of an experiment be resolved to the simplest possible level. Producers have complex goals. The attainment of these goals requires synthesis of information obtained from intermediate objectives. To succeed, the producers must somehow consider all factors in the correct relationship and simultaneously.

Presenting advice on the relatively simple intermediate goals such as growth rate or feed conversion ratio is not adequate from the producer's viewpoint. In addition it is an unsatisfactory method of transmitting and interpreting the amount of scientific information we currently have available.

The Model

The mathematical formulation of the model currently being studied at the University of Minnesota is a composite of a swine growth model developed in Scotland (Whittemore and Fawcett, 1974; Wittemore, 1976) and a model of pig heat loss in relation to environmental factors (Bruce and Clark, 1980). Extension and enhancement of the model has proceeded primarily in the direction of increasing

the live weight range over which the model is useful. Our particular concern is with the weanling pig in the 5 to 20 kg weight range and with the response of the pig to cold environments.

The various parameters and inputs available for alteration by the user are shown in table 1 and the predicted outputs are listed in table 2. The association of inputs with outputs is not done through a collection of empirical relationships but rather are derived from an analysis of the causal forces indicated by the body of knowledge regarding the biology of the pig.

Nutritional information is provided to the model in terms of protein and energy densities of the diet. Although this may seem too simplistic. It appears quite satisfactory within the narrow range of ingredients commonly used. Protein quality considerations are made through the use of chemical score and digestibility adjustments to dietary protein thus determining the amount of protein available for synthetic processes.

Metabolizable energy available for use is estimated from the digestible energy content of the diet after adjustment for the energy contained in protein and that gained from deamination of unused protein.

With respect to the environmental considerations, three avenues of heat loss from the pig are considered: radiation, convection and conduction. Heat loss via each of these avenues is estimated from the physical parameters of the environment which impinge on that mode of heat loss (e.g., air velocity influences convection but not conduction).

The driving variable in the model is food intake. Once the quantity of food consumed is known, the available energy is partitioned towards maintenance, protein synthesis and lipid synthesis. The metabolic inefficiencies of these processes give rise to heat which when summed estimate the heat production of the animal. Heat loss is predicted from the physical component of the model. The influence of thermal environment on pig growth is mediated through the pig's requirement for homeothermy. Since the animal must maintain homeothermy, heat loss must equal heat production below the lower critical temperature. Thus, at low environmental temperatures, this requirement may force decreased rate of gain and increased use of energy for heat production (cold thermogenesis). Once thermal balance is achieved, the predicted lipid and protein gains are empirically converted to lean and fat estimates; live weight is appropriately adjusted and a new day dawns. These iterations are performed until the live weight of the modeled animal reaches that requested by the user.

The Reason for Being

The model described in this paper was designed to mimic the responses of the growing pig with regard to changes in nutrient supply and thermal environment.

As described, it is useful at the scientific level to point out the shortcomings of our current knowledge and aid in experimental design, extension and interpretation. At the practical level, a similar model could aid in determining management targets and allow optimization of feeding strategies. It has been developed as a research tool and thus many of the parameters associated with the biology of the pig are not generally thought of as characteristics of growing

swine. In addition, no variables associated with the cost of the various inputs or the value of the product are included. Extension of the model to include these various factors is however possible. The Edinburgh Model Pig (Whittmore, 1980) is an excellent example of this ability to easily relate "apples and oranges" (e.g. soybean meal prices and lean tissue growth capacity).

The enhancement and extension of the model is proceeding as a joint effort of the Departments of Animal Science and Agricultural Engineering with assistance from a USDA grant.[1] The short term goal is to provide a mechanism for evaluating various building designs and temperature-ventilation schemes which minimize energy usage.

What Would Happen If...?

The output from the model described above does not present an optimum solution. It only attempts to simulate the operation of the system (pig) and thus inform the operator of the results of the alteration of parameters. To determine optimum strategy a number of runs are required and individual responses mapped. Although this seems laborious, it may be very beneficial as solutions with similar responses and less convenience may be removed from the decision set. For example, one could easily couple the output of this model with for example a least cost formulation scheme to evaluate diets on the basis of maximum profit rather than unit cost.

The examples presented here are of more simplistic nature. Figure 1 includes several curves which demonstrate the general validity of the model with regard to prediction of growth as influenced by dietary level of energy and protein. The predicted and actual results agree well over a wide range of energy and protein concentrations.

Figure 2 shows the response of pigs of two different lean growth potentials fed diets of varying energy:protein ratios. As expected, response to greater energy: protein ratios is greater in pigs with a higher growth potential than in the poorer performing group. Pigs with poorer growth potential gain at rates similar to those with a higher growth potential if the "better" pigs are fed diets of increased protein density without concomittant increases in energy density (points A and B, figure 2).

Closing Comments

Although only one or two factors have been altered in the above examples, it should be remembered that impact of all factors included in table 1 were evaluated in the response prediction.

It is impractical in a brief overview to relate the myriad of possibilities wherein a mathematical model of the system under investigation may be useful. A carefully applied combination of simulation and real world experimentation can frequently lead to a more complete understanding of the behavior of a system. The impact of alternate strategies may be easily evaluated. Transmission of knowledge is easily accomplished. However, the most important outcome of such an effort may be the disclosure of gaps and incongruencies in our current knowledge of the system.

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[1]USDA project number 59-2271-0-2-039-0, Swine production using no fossil fuels, K. A. Jordan and S. G. Cornelius.

Literature Cited

Bruce, J. M. and J. S. Clark. 1980. Models of heat production and critical temperature for growing pigs. Anim. Prod. 28:353.

Whittemore, C. T. 1980. The Edinburgh Computer Model Pig. Pig News and Information. 1:343.

Whittemore, C. T. 1976. A study of growth responses to nutrient inputs by modeling. Proc. Nutr. Soc. 35:383.

Whittemore, C. T. and R. Fawcett. 1976. Theoretical aspects of a flexible model to simulate protein and lipid growth in pigs. Anim. Prod. 22:87.

Table 1. Inputs to the swine growth model.

Biological:

- Initial live weight (kg)
- Mature protein mass (kg)
- Protein accretion limit (grams)
- Initial body protein (kg)
- Initial body fat (kg)
- Initial body ash (kg)
- Obligatory fat:protein ratio
- Final weight desired

Nutritional:

- Digestible energy in feed (MJ/kg)
- Crude protein in feed (%)
- Crude protein digestibility (%)
- Chemical score of feed protein

Environmental:

- Environmental temperature (^{o}C)
- Air velocity (m/sec)
- Floor insulation (watts/m^2)
- Floor contact fraction
- Number of animals per pen

Table 2. Outputs predicted by the swine growth model

Description
Live weight
Empty body weight
Live gain
Empty body gain
Lean gain
Fat gain
Ash gain
Gain/Feed
Gain/Energy
Gain/Protein
Lean gain/Feed
DE retention
Critical temperature
Heat production
Cold thermogenesis
Feed intake
Protein intake
Energy intake
Metabolizable energy
Available ME for gain
Urine energy
Deaminated protein
Protein turnover
Protein lost due to turnover
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Fat:protein ratio
Protein accretion
Lipid accretion
Latent heat production
Tissue resistance
Pig surface area
Energy used for protein synthesis
Final carcass composition

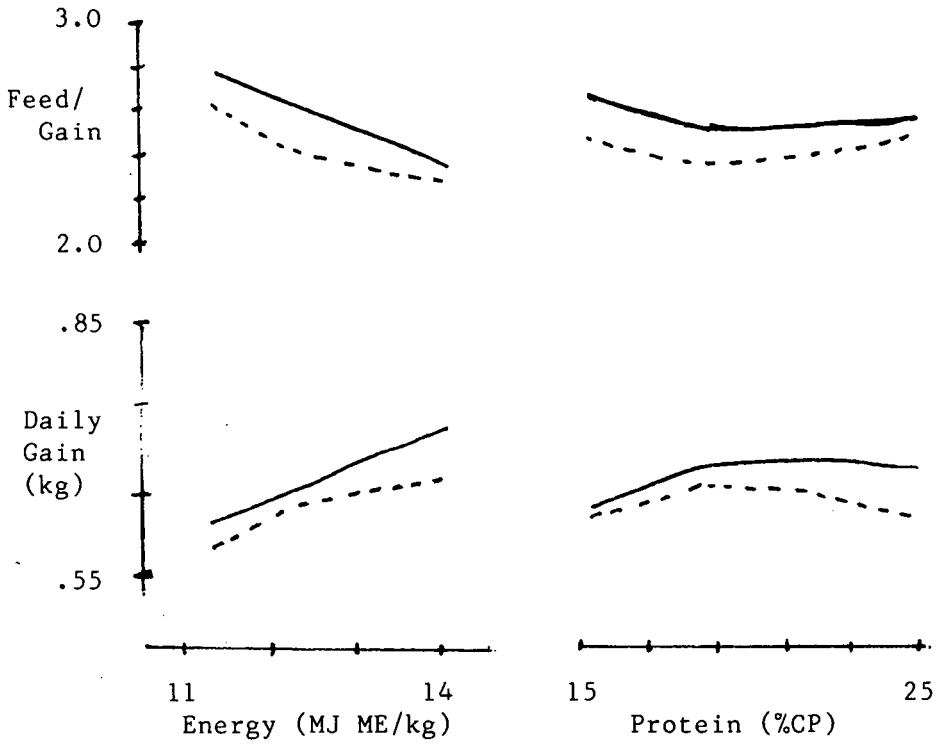

Figure 1. Performance of model pig (——) vs. actual (---)

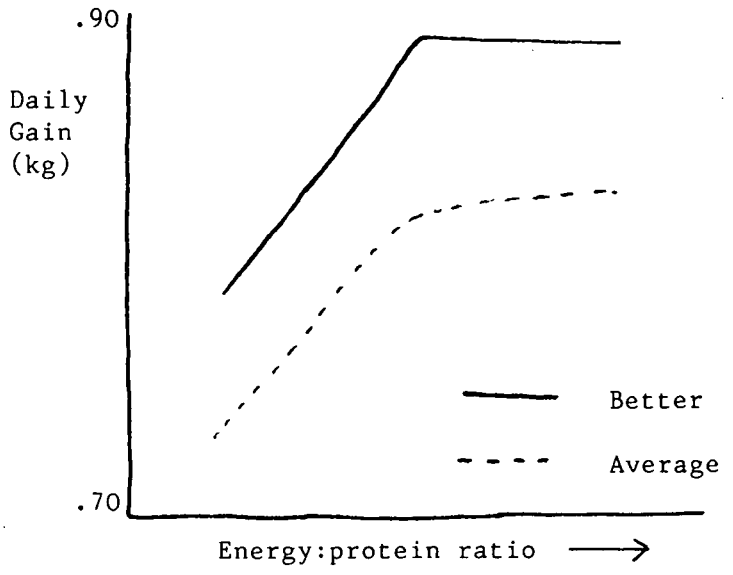

Figure 2. Influence of energy:protein ratio in pigs of two different lean growth potentials