From Birth to Bones: Skeletal Evidence for Health, Disease, and Injury in the Gombe Chimpanzees

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Chapter 1

Introduction

The relevance of chimpanzees, skeletons, and chimpanzee skeletons to anthropological research on human evolution

What makes us human? The answers to this question are as diverse and complicated as our species; one way to approach it is by studying human origins and evolution. A primary method for interpreting human evolution is the investigation of the human fossil record, often with accompanying comparison to the skeletons of living primates. Understanding the relationship between behavior and the skeleton makes it possible to reconstruct the life histories of animals whose behavior can no longer be directly observed. Skeletons with known life histories are the context in which unknown skeletons may be interpreted, and are therefore necessary for a more complete understanding of skeletal biology.

Studying the skeletons of animals with known life histories develops our understanding of the relationship between behavior, soft tissue, and the skeleton. These relationships are often complex, reflecting their inter-connectedness. Integrating skeletal biology with behavioral biology has the potential to provide insights into individual lives that might otherwise remain invisible.

Because natural selection acts at the level of the individual, it is of particular interest to study individual life histories, especially variables that are likely to affect fitness, such as dominance rank. How these variables may be reflected by the skeleton is under-explored, primarily because suitable skeletal collections (wild animals with known

life histories) are scarce. My project, a study of wild chimpanzee skeletons with known life histories, addresses this issue.

Since 1960, Dr. Jane Goodall and colleagues have studied the chimpanzees of Gombe National Park, Tanzania. The skeletal materials obtained from chimpanzees who have died during this 50-year study represent an unparalleled opportunity to better understand the effects of a chimpanzee's life history and behavior on his or her skeleton because an extraordinary amount of detailed information is available about the lives of chimpanzees whose skeletons have been preserved (e.g. Goodall, 1986).

Only rarely are chimpanzee life history data available for the same animals for whom skeletal data are available. Because of the nature of their acquisition, museum skeletal collections do not usually include life history data (Harman, 2005) and laboratory chimpanzees, for whom behavioral data are often available, do not allow for appropriate comparisons or make good proxies for the study of primate evolution. Reasons for the decreased value of captive ape skeletons for research of this nature include the very different stresses associated with captivity compared to the wild (e.g. less nutritional stress, access to veterinary care, and often more psycho-social stress), and that captive chimpanzees achieve larger ultimate body sizes and grow up faster than their wild counterparts (Zihlman *et al.*, 2004a). Reliable sets of both skeletal and behavioral data are only obtained for wild animals from behavioral research stations such as Gombe National Park in Tanzania.

Neither modern chimpanzees nor modern humans are exactly like extinct hominoids, and neither species should be considered a direct proxy. Understanding these two closely related species, however, provides a framework for understanding the past.

Knowledge about modern adaptations, morphology, and behaviors allows for informed interpretation of the hominoid fossil record. In the case of the hominoid fossil record, we are interested in hard tissues, soft tissues, and behaviors, but only one of these variables is still observable. Establishing the relationship between these three variables helps us reconstruct what is unobservable from the data available, and also creates a more complete picture of the life histories of the organisms we study. Improving our understanding of the relationship between life history and the skeleton is an ongoing effort among physical anthropologists, and a portion of the skeletal collection from Gombe has already contributed to this understanding.

Previous studies of the Gombe skeletal collection

Previous studies of the Gombe chimpanzee skeletal material have focused primarily on chimpanzees who died between the years 1966 and 1987 (see below, and Gombe Stream Research Center (GSRC) unpublished data) and have already yielded much valuable information. Previous research covers a wide variety of topics, including degenerative joint disease (Jurmain, 1989; Jurmain, 2000), trauma (Jurmain, 1989; Jurmain, 1997), generalized skeletal pathology (Jurmain, 1989), dental pathology (Kilgore, 1989), body size (Morbeck and Zihlman, 1989), skeletal age changes (Morbeck et al., 2002; Sumner et al., 1989), the skeletal effects of poliomyelitis (Morbeck et al., 1991), sex differences in the pelvis (Morbeck et al., 1992) and the vertebrae (Galloway et al., 1996), asymmetry due to hand preference (Morbeck et al., 1994), dental maturation (Zihlman et al., 2004a; Zihlman et al., 2004b), and individual life histories as reflected in the skeleton (Zihlman et al., 1990). The sample presented here is nearly double the size

of the earlier sample, and therefore allows for statistical analyses not previously possible. The sample also includes chimpanzees of a wider variety of ages, which allows for a closer look at behavior's relationship to the skeleton at multiple stages of life history. I examine the relationship between life history variables available from the behavioral research data and skeletal markers of health and stress. These skeletal markers of stress include trauma, pathology, and enamel hypoplasia. The life history variables include age, sex, and dominance rank.

Dominance rank

Dominance rank plays an important role in the social lives of chimpanzees. It affects access to resources (Murray *et al.*, 2006; Pusey *et al.*, 2005), inter-individual relationships (Foster *et al.*, 2009; Goodall, 1986), and reproductive success (Constable *et al.*, 2001; Pusey *et al.*, 1997; Wroblewski *et al.*, 2009), among other variables. It seems obvious to assume that higher ranking chimpanzees are "healthier" in some way than their lower ranking counterparts, but long-term observations and analysis of chimpanzee behavior reveal that the relationship between rank and various markers of health is complex.

For example, body mass in living chimpanzees at Gombe is affected by food availability. Chimpanzees commonly experience a seasonal fluctuation in body mass that is correlated with the availability of ripe fruit. Higher ranking female chimpanzees tend to have larger body masses than lower ranking females. This is not true for male chimpanzees, however, for whom dominance rank and body mass are not correlated (Pusey *et al.*, 2005).

Hormone analysis, particularly of cortisol, is a long-standing technique for evaluating stress levels in wild primates as well as how stress levels are affected by an animal's own social status and the statuses of conspecifics (e.g. Sapolsky, 2005). If higher ranking primates are healthier, we might expect that they would experience lower levels of stress. Conversely, if it is stressful to be high ranking, because of increased agonistic encounters, for example, we might expect that lower ranking chimpanzees would have lower cortisol levels, reflecting lower levels of stress.

The interaction between cortisol levels and social status or dominance rank is complex; it is affected by a large number of variables, including the social structure of a particular species (Sapolsky, 2005). In other words, it is not safe to assume that all species will demonstrate the same relationship between rank and cortisol levels. This relationship may also change over time, even within the same group of animals because of factors such as divergent dominance "styles" of different animals (Foster *et al.*, 2009; Ray and Sapolsky, 1992) or instability in the dominance hierarchy (Bergman *et al.*, 2005; Sapolsky, 1983). Dominance rank often changes over the course of a chimpanzee's life, particularly for males (Kawanaka, 1990; Takahata, 1990), meaning that stress levels will not be consistent throughout life history. In addition, dominance rank is not the only variable that influences cortisol levels (Lane, 2006), so the effects of diet, time of day, estrus cycle (*Ibid.*), energetic costs (Muller and Wrangham, 2004), and other behaviors (e.g. rate of consortships and number of aggressive interactions) (Muller and Wrangham, 2004; Schino *et al.*, 2007; Virgin and Sapolsky, 1997), and other behavioral styles or personality traits (Anestis, 2005; Anestis *et al.*, 2006) should be considered in addition to the effects of the social hierarchy.

In chimpanzees in particular, high male dominance rank has been found to positively correlate with urinary cortisol levels, indicating that maintaining high rank is costly in some way. Rates of male aggression as well as lower abundance of fruit were both correlated with higher cortisol levels in male chimpanzees (Muller and Wrangham, 2004), suggesting that both social and energetic factors influence stress. In addition, lower-ranking male chimpanzees have higher urinary C-peptide concentrations, indicating a better energetic condition than higher ranking males. The benefits of access to prime feeding sites that high rank confers therefore seem to be more than offset by the energetic costs of the stress of the aggressive interactions needed to achieve and maintain high rank (Thompson *et al.*, 2009). In other words, dominant male chimpanzees may spend more time maintaining their rank and less time feeding (Muller and Wrangham, 2004; Thompson *et al.*, 2009), and the benefits to reproductive success resulting from this strategy are presumed to outweigh the energetic costs (Muller and Wrangham, 2004). High ranking female chimpanzees, on the other hand, do not have elevated urinary cortisol levels compared to lower ranking females. In particular, immigrant female chimpanzees, who are low ranking during the period immediately following immigration, experience high levels of intra-sexual aggression. This is correlated with high stress levels as measured by urinary cortisol (Kahlenberg *et al.*, 2008).

If we now have improved data on how hormone levels are affected by specific stressors as well as how these variables correlate to dominance rank, this still leaves many questions about longer term trends in health status as well as the direction of causality. Hormone levels of any kind are, by nature, more ephemeral and time specific than skeletal markers of health and stress. Skeletal trauma and pathology incidents

accumulate over the life time of an organism; even old injuries may still leave a sign on the skeleton. Examining skeletal markers of health and stress therefore has great potential for studying the cumulative effects of stress over the course of a chimpanzee's entire life, and how these effects may be related to dominance rank.

A correlation between something like cortisol levels and dominance rank does not contain information on causality. Does having a higher rank result in higher cortisol levels for male chimpanzees? Alternately, could elevated cortisol levels (and perhaps a greater tolerance for stress (Chichinadze and Chichinadze, 2008)) make a chimpanzee more likely to achieve high rank? If skeletal markers of stress, which can accumulate during early development, correlate well with dominance rank achieved late in life, it may be possible to determine direction of causality. Are chimpanzees with higher levels of developmental stress more or less likely to achieve high rank? I examine this question in relationship to enamel hypoplasia as well as potential stress events accumulated over a longer period of time (skeletal trauma and pathology).

The skeletal markers of stress or developmental instability examined here include: incidences of trauma and pathology, severity of enamel hypoplasia on the canine, and degree of fluctuating asymmetry in the posterior dentition. The relationship of trauma, pathologies, and enamel hypoplasia with dominance rank is explored through regression analysis. From previous research on cortisol levels as a measure of stress and its relationship to dominance rank, I expect that:

1. Higher ranking males will have more skeletal signs of stress than their low ranking counterparts.

2. Lower ranking females will have more skeletal signs of stress than their high ranking counterparts.

Skeletal markers of stress

Pathology and trauma, enamel hypoplasia, and fluctuating asymmetry are skeletal markers of stress I studied in the Gombe skeletal collection. Trauma and pathology reflect sources of morbidity such as falls, inter-individual violence, and joint disease (Carter *et al.*, 2008; Jurmain, 1989; Jurmain, 1997; Kilgore, 1989; Lovell, 1990; Lovell, 1991), and also help to paint the story of an chimpanzee's life history (Zihlman *et al.*, 1990). Studying these phenomena at the level of the individual is particularly useful because natural selection acts at this level. Understanding the relationship between fitness and instances of trauma and pathology is therefore of great interest. Chapter 2 addresses this topic in detail. I expect, because of the high costs of agonistic encounters associated with high rank in male chimpanzees, that high ranking males (or males who once achieved high rank) will have the highest levels of skeletal trauma of any demographic group. Lower ranking females should be the next most affected group. Chapter 3 offers a case study in trauma: the skeletal damage observable on the skeletons of chimpanzee infanticide victims. This is a demographic that has the potential to skew perceptions of trauma and pathology frequencies relative to other demographic groups and how they may be related to dominance rank.

Enamel hypoplasia is a period of decreased ameleoblast activity, and is one skeletal proxy for physiological stress such as nutritional deficit or severe illness (Berbesque and Doran, 2008a; Corruccini *et al.*, 2005; Goodman and Rose, 1990;

Hannibal and Guatelli-Steinberg, 2005; Lukacs, 2001). An enamel hypoplasia manifests on the tooth as a region of the enamel that is thinner compared to the enamel on the unaffected part of the tooth. Because hypoplasias accumulate during the period of tooth formation, they are a reflection of specific stress events during early life history. Examining the relationship between dominance rank and severity of enamel hypoplasia therefore has the potential to inform about the causality of stress versus rank. Whether chimpanzees with greater severity of enamel hypoplasia more or less likely to achieve high rank is addressed in Chapter 4.

Fluctuating asymmetry is an indicator of an organism's ability to buffer against developmental instability. It is a non-directional deviation in symmetry between bilateral structures (e.g. Wells et al. 2006). Increased magnitude of fluctuating asymmetry has been linked to several factors, including poor maternal health (Kohn and Bennet, 1986; Singh and Rosen, 2001), higher levels of reactive aggression (Benderlioglu et al., 2004), and shorter stature (and therefore poorer nutrition) (Perzigian, 1977). Other lines of evidence that point to symmetry as being a useful skeletal marker of relative fitness are that human females tend to prefer symmetrical male faces (Gangestad and Thornhill, 2003; Thornhill and Gangestad, 2006) and that symmetrical females tend to have higher potential fertility (Jasienska et al., 2006). All of this suggests that fluctuating asymmetry is likely to be a valuable skeletal proxy for fitness. Dental metrics are especially useful in assessing fluctuating asymmetry because these will not reflect locomotor habits (as directional asymmetry in the limb bones may (e.g. Churchill and Formicola, 1997; Sarringhaus *et al.*, 2005; Sladek *et al.*, 2007). Unlike trauma/pathology and enamel hypoplasia, fluctuating asymmetry measures the ability to buffer against stress rather than

recording specific stress events. That fluctuating asymmetry measures a different phenomenon is likely to be part of the reason that fluctuating asymmetry sometimes correlates with other markers of physiological insult (such as enamel hypoplasia) (DeLeon, 2007; Palubeckaite and Jankauskas, 2001), but does not always demonstrate this pattern (Hoover et al., 2005; Smith et al., 1982). Whether levels of stress, as reflected by fluctuating asymmetry, change over the course of a chimpanzee's life is addressed in Chapter 5.

Materials and Methods

In this section, I discuss the entire Gombe chimpanzee skeletal collection, skeletonization procedure, and the assessment of dominance rank from behavioral data. Methodologies for recording and analyzing trauma and pathology, enamel hypoplasia, and fluctuating asymmetry are provided in the appropriate chapters. Each chapter only assesses a portion of the entire skeletal collection; the specific chimpanzees analyzed in each case are presented therein.

Forty-eight chimpanzees are represented in the Gombe skeletal collection; of these, at least 28 are complete or nearly complete skeletons. Most of the chimpanzees have known life histories, and many of those who were born after 1960 include complete life histories from birth until death. Protocols for behavioral observation have been outlined elsewhere (e.g. Goodall, 1986). Part of the skeletal collection, chimpanzees who died prior to 1987, was being curated and analyzed by Dr. Adrienne Zihlman and colleagues at the University of California Santa Cruz. This portion of the collection is currently housed at the University of Minnesota, in the Evolutionary Anthropology

Laboratory. The other part of the sample is curated in Gombe National Park, Tanzania, which is located on the shore of Lake Tanganyika in western Tanzania.

Table 1.1 is a demographic summary of the skeletal collection. Age categories in Table 1.1 are after Goodall (1986). Table 1.2 is a summary of the chimpanzees presented here, including completeness of skeleton, birth and death dates (GSRC), and location of curation.

Table 1.1: Demographic summary of the Gombe chimpanzee skeletal sample

Age category	**Females**	**Males**	**Unknown sex**	**Total**
Infant (0-4 years)	2	7	3	12
Juvenile (5-7 years)	0	0	0	0
Adolescent (females: 8-14 or 15, males: 8-15)	1	7	0	8
Adult (females: 14 or 15-33, males: 16-33)	10	9	0	19
Old adult (34+)	4	5	0	9
Total	17	28	3	**48**

Table 1.2: The Gombe chimpanzee skeletal sample

Chimpanzee (? Tentative ID)	Sex	Community	Birth Date (*estimate)	Death Date	Age at death (years) (*estimate)	Completeness of remains	Location
Melissa's baby	m	Kasekela	1/6/1976	1/8/1976	0	In transit	MN
Gaia's infant	m	Kasekela	7/16/2008	7/23/2008	0.01	complete	Gombe
Patti's baby	?	Kasekela	4/15/1978	4/21/1978	0.01	In transit	MN
Rejea	f	Mitumba	12/22/1992	3/30/1993	0.27	nearly complete (perimortem trauma)	Gombe
November infanticide	f	Kahama or Kalande		Nov-75	0.67*	complete	Dar es Salaam
October infanticide	m	Kahama or Kalande		Oct-1975	0.75*	nearly complete (perimortem trauma)	Dar es Salaam
Andromeda	f	Mitumba	11/18/2004	8/13/2005	0.73	nearly complete (perimortem trauma)	Gombe
Gyre	m	Kasekela	10/21/1977	8/13/1978	0.81	In transit	MN
Fred	m	Kasekela	9/5/1996	7/29/1997	0.9	complete	Gombe
Groucho	m	Kasekela	7/1/1985	10/12/1986	1.28	In transit	MN
Plato	m	Kasekela	9/7/1970	4/18/1973	2.61	In transit	MN
Getty	m	Kasekela	5/21/1982	4/12/1986	3.89	In transit	MN
Ebony	m	Mitumba	11/9/1996	1/17/2005	8.19	complete	Gombe
Flint	m	Kasekela	3/1/1964	9/17/1972	8.55	In transit	MN
Jackson	m	Kasekela	9/16/1989	2/14/2000	10.41	complete	Gombe
Mel	m	Kasekela	1/24/1984	10/11/1994	10.71	complete	Gombe
Sugar	f	Kasekela	7/2/1976	5/31/1987	10.91	complete	Gombe

Chimpanzee (? Tentative ID)	Sex	Community	Birth Date (*estimate)	Death Date	Age at death (years) (*estimate)	Completeness of remains	Location
Galahad	m	Kasekela	4/5/1988	2/7/2000	11.84	complete	Gombe
Michaelmas	m	Kasekela	10/7/1973	10/3/1986	12.99	In transit	MN
MacDee	m	Kasekela	7/2/1953*	12/15/1966	13.45*	In transit	MN
Sherehe	f	Kasekela	1/25/1991	11/5/2006	15.78	complete	Gombe
Gilka	f	Kasekela	7/2/1960	5/16/1979	18.87	complete	MN
Echo	f	Kalande	7/2/1972*	2006	34*	complete	Gombe
Yolanda	f	Kasekela	1983*	7/11/2007	24*	complete	Gombe
Kidevu?	f	Kasekela	6/1/1966	1/4/1992	25.59	complete	Gombe
Charlie	m	Kahama	7/2/1951*	5/15/1977	25.87*	complete	MN
Pallas	f	Kasekela	7/2/1955*	9/11/1982	27.2*	complete	MN
Madam Bee	f	Kahama	7/2/1947*	9/19/1975	28.22*	complete	MN
Vincent	m	Mitumba	7/2/1976	12/22/2004	28.47	complete	Gombe
Winkle?	f	Kasekela	7/2/1959*	5/13/1988	28.87*	cranium only	Gombe
Satan	m	Kasekela	7/2/1957*	5/3/1987	29.83*	incomplete	MN
Tumaini	m	Kalande	1972*	8/13/2002	30*	nearly complete (perimortem trauma)	Gombe
Passion	f	Kasekela	7/2/1951*	2/10/1982	30.61*	complete	MN
Jomeo	m	Kasekela	7/2/1956*	5/16/1987	30.87*	complete	MN
Miff	f	Kasekela	7/2/1956*	5/28/1987	30.9*	complete	MN
Atlas	m	Kasekela	7/3/1959*	1/14/1999	31.3*	complete	Gombe
Rix	m	Kasekela	7/2/1935*	11/19/1968	33.39*	complete	Dar es Salaam
Beethoven?	m	Kasekela	7/4/1959*	12/15/2002	33.45*	cranium only	Gombe
Humphrey	m	Kasekela	7/2/1946*	6/5/1981	34.93*	cranium only	MN
Melissa	f	Kasekela	7/2/1950*	10/24/1986	36.31*	complete	MN

Chimpanzee (? Tentative ID)	Sex	Community	Birth Date (*estimate)	Death Date	Age at death (years) (*estimate)	Completeness of remains	Location
Hugo	m	Kasekela	7/2/1936*	2/1/1975	38.58*	complete	MN
Cusano	m	Mitumba	7/2/1956*	6/11/1996	39.94*	nearly complete (post burial damage)	Gombe
Goblin	m	Kasekela	9/6/1964	8/24/2004	39.96	Complete	Gombe
Patti	f	Kasekela	7/2/1961	10/3/2005	44.25	Complete	Gombe
Flo	f	Kasekela	7/2/1919*	8/22/1972	53.14*	complete	MN
Bwavi male	m	Kalande	?	1994 or 1995	40+?*	Complete	Gombe
Unknown male	m	?	?	?	adult	cranium only	Gombe
Old Female	f	?	?	1974?	old adult	complete	MN

The bodies of dead chimpanzees were recovered whenever possible for the purpose of skeletal preservation and for analysis of soft tissue for pathological lesions (K. Terio, unpublished data). Skeletons of chimpanzees who died prior to 1987 were cleaned according to protocols laid out elsewhere (Zihlman *et al.*, 1990). Skeletons of chimpanzees who died after 1987 were processed in a different way. Following necropsy (K. Terio, unpublished data), the bodies were buried in a permeable bag and exhumed after at least one year. Skeletons were then carefully cleaned using water and a soft brush and allowed to dry thoroughly.

Because some chimpanzees at Gombe are infected with a strain of the Simian Immunodeficiency Virus (SIVcpz) (Keele *et al.*, 2009; Santiago *et al.*, 2002), an extra step was added to the cleaning procedure of chimpanzess known to be infected in order to reduce the risk of possible transmission of the virus to skeletal researchers. Infected chimpanzees were briefly (2 minutes) rinsed in a mild bleach solution after initial cleaning, and then subsequently soaked in water to remove bleach residue.

Each skeleton was then inventoried and stored in mosquito net bags (to reduce the risk of insect damage to the bones in storage) in a secure location at Gombe National Park.

Dominance rank data for this study were generously made available by Dr. Anne Pusey and have their basis in long-term behavioral research. Rank data are presented in Table 1.3 and are collated from Goodall (1986), Foster *et al.* (2009), Murray *et al.* (2007; 2006), and unpublished data from Gombe Stream Research Center (GSRC). Because rank often changes over the course of a chimpanzee's life, highest achieved rank and rank at death were both considered whenever possible in an attempt to encompass rank

variation over the course of a chimpanzee's life. Maternal rank at the time of a chimpanzee's birth is also included in the table, but it was not possible to include this variable in statistical analyses due to small sample size (see Chapter 4).

Female dominance ranks are usually categorized as high ranking versus middle ranking versus low ranking (e.g. Pusey *et al.*, 2005). Male chimpanzees may be given ordinal ranks (1 – n adult males in the community) (Bygott, 1979; Goodall, 1986), but here these were collapsed into categories similar to the females' for the purpose of the statistical analyses undertaken in this study (in order to avoid having to divide the sample). Males ranked 1-4 are considered high ranking, 5-8 are considered middle ranking, and ranks 9 and above are low ranking. These categories were adjusted twice to account for the number of males in a group. Vincent was ranked 3rd of 3 adult male chimpanzees at the time of his death, and is therefore considered low ranking. Similarly, Jomeo's numerical rank during the year of his death could be considered either high or middle ranking because of the relatively small number of adult males. Jomeo's relative rank (Jameson *et al.*, 1999) places him in the top third of adult male ranks, however, so he is counted as high ranking at time of death.

Summary

Modern primates such as chimpanzees provide a comparative framework for improved understanding of primate evolution. Studying the skeletons of wild chimpanzees with known life histories allows us to explore the relationship between behavior, soft tissue, and the skeleton, thus facilitating the interpretation of skeletons without known life histories, such as fossil primates and museum skeletal collections. I

examine the potential influences of age, sex, and dominance rank on skeletal signs of health and stress. Chapter 2 reports on skeletal trauma and pathology and their relationship to dominance rank. Chapter 3 is a trauma case study that examines the ways skeletal analysis can improve our understanding of infanticide in chimpanzees. Chapter 4 is a study on enamel hypoplasia severity in the adult canine and its relationship to dominance rank. Chapter 5 compares levels of fluctuating asymmetry between the 1st and 2nd molars.

Table 1.3: Dominance rank data for the Gombe chimpanzee skeletal collection

Name	Sex	Mother	Age	Highest Rank	Rank at death	Mother's rank at baby's birth	Citation
Andromeda	F	Aphro	0.73	3	3	2	(based on age) Pusey et al., 2005; Deus Mjungu, unpublished data
Atlas	M	Athena	31.3	1	2	2	Wroblewski *et al*., 2009; GSRC unpublished data; Goodall, 1986
Beethoven	M	x	33.45	1	1	x	Wroblewski et al., 2009; GSRC unpublished data
Bwavi male	M	x	40+	x	x	x	unknown individual
Charlie	M	x	25.87	1	1	x	Bygott, 1979; Goodall, 1986
Cusano	M	x	40.95	1	1	x	GSRC unpublished data
Ebony	M	Eva	8.39	3	3	x	(based on age) Pusey et al., 2005
Echo	F	x	22.5	x	x	x	data unavailable
Flint	M	Flo	8.55	3	3	1	(based on age) Pusey et al., 2005; Goodall, 1986
Flo	F	x	53.14	1	1	x	Goodall, 1986
Fred	M	Fifi	0.9	3	3	1	(based on age) Pusey et al., 2005
GA baby 3	M	Gaia	0	3	3	x	(based on age) Pusey et al., 2005

Name	Sex	Mother	Age	Highest Rank	Rank at death	Mother's rank at baby's birth	Citation
Galahad	M	Gremlin	11.4	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Getty	M	Gremlin	3.89	3	3	3	based on age Pusey et al 2005; Pusey et al 97
Gilka	F	Olly	18.87	3	3	2	Goodall, 1986; Pusey et al., 1997
Goblin	M	Melissa	39.96	1	2	2	Wroblewski et al., 2009; GSRC unpublished data
Groucho	M	Melissa	1.28	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Gyre	M	Melissa	0.81	3	3	2	based on age Pusey et al 2005; Pusey et al 97
Hugo	M	x	38.58	x	3	x	Goodall, 1986
Humphrey	M	x	34.93	1	2	x	Goodall, 1986
Jackson	M	Jiffy	10.41	3	3	3	(based on age) Pusey et al., 2005
Jomeo	M	Vodka	30.87	1	1	x	Wroblewski et al., 2009; GSRC unpublished data
Kidevu	F	x	25.59	x	x	x	data unavailable
MacDee	M	Jessica	13.45	3	3	x	(based on age) Pusey et al., 2005
Madam B	F	x	28.22	x	x	x	data unavailable

Name	Sex	Mother	Age	Highest Rank	Rank at death	Mother's rank at baby's birth	Citation
Mel	M	Miff	10.71	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Melissa	F	x	36.31	2	2	x	Pusey et al., 1997; Goodall, 1986
Michaelmas	M	Miff	12.99	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Miff	F	Marina	30.9	2	2	2	Goodall, 1986; Pusey et al., 1997
ML Baby 2	F	Melissa	0.01	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Pallas	F	x	27.2	2	3	x	Pusey et al., 1997
Passion	F	x	30.61	1	1	x	Pusey et al., 1997
Patti	F	x	44.25	1	1	x	Murray et al., 2006
PI Baby 1	M	Patti	0.02	3	3	2	(based on age) Pusey et al., 2005; Pusey et al., 1997
Plato	M	Pallas	2.61	3	3	3	(based on age) Pusey et al., 2005; Pusey et al., 1997
Rejea	F	Rafiki	0.38	3	3	x	(based on age) Pusey et al., 2005
Rix	M	x	27.38	x	x	x	data unavailable
Satan	M	Sprout	29.83	1	1	x	Wroblewski et al., 2009; GSRC unpublished data

Name	Sex	Mother	Age	Highest Rank	Rank at death	Mother's rank at baby's birth	Citation
Sherehe	F	Sandi	15.78	2	2	2	GSRC unpublished data
Sugar	F	Caramel	10.91	3	3	x	(based on age) Pusey et al., 2005
Tumaini	M	x			x	x	data unavailable
Vincent	M	x	28.47	1	3	x	GSRC unpublished data
Winkle	F	x	28.9	2	2	x	Pusey et al., 1997
Yolanda	F	x	24.35	x	3	x	GSRC unpublished data

Chapter 2

The Relationship of Dominance Rank to Skeletal Trauma and Pathology in the Gombe Chimpanzees

SUMMARY

Skeletal trauma and pathology studies can provide information on sources of morbidity and mortality at the species level as well as document how selection pressures may vary within a single species. I studied trauma and pathology in 39 chimpanzee skeletons from Gombe National Park, Tanzania, through macroscopic evaluation of injury, arthropathy, bone loss pathologies, bone formation pathologies, and congenital variations. The influences of sex, age, and dominance rank on trauma and pathology incidence were considered using regression analysis. Age was significantly correlated with trauma and pathology incidence, but sex was not. The relationship between age and trauma incidence, however, may be more complex than has been previously described. There is some evidence to suggest that change in rank is correlated with arthropathy and trauma incidences. While the Gombe chimpanzee skeletal collection is the largest assemblage of wild chimpanzee skeletons with known life histories, sample size limits some analyses. The number of chimpanzees whose rank changed between the time of their highest achieved rank and rank at death, for example, is small. This restricts the explanatory power of inferences about the influence of change in rank on trauma and pathology incidence. Continued preservation and study of skeletal material from wild primates with known life histories may enable future research to address some of these concerns.

INTRODUCTION

Evolutionary mechanisms such as natural selection act largely at the level of the individual. The study of individual life histories is therefore of particular interest when considering the selective pressures that act upon an organism. The Gombe chimpanzees provide a unique opportunity to explore these mechanisms, both through the vast amounts of behavioral data collected over the course of 50 years as well as how these life histories impact the hard tissues. The study of the relationship between behavior and the hard tissues is of special relevance for a complete picture of skeletal biology, because it is only through the study of skeletons with known life histories that it is possible to interpret unknown skeletal material with any accuracy.

In addition to providing a more complete life history for a particular organism, individual-level analysis can also make species-level trends apparent, especially over relatively long periods of time. This makes the Gombe skeletal collection especially relevant as it includes chimpanzees of a wide variety of ages with diverse causes of death who have died over the course of half a century. Because chimpanzees are so long lived, it is only after considerable time has passed that it is possible to more fully understand their life histories, the selective pressures that act on individual chimpanzees, and the common sources of morbidity and mortality of the species. Skeletal evidence gives us information about which sources of morbidity and mortality are reflected by the hard tissues. This is relevant for interpreting the fossil record, determining which illnesses / injuries are likely to appear on the skeleton, and helping identify cause of death for each chimpanzee. In addition, continued re-examination of a growing skeletal collection can

aid in the revelation of long-term trends that would otherwise not be apparent. The larger skeletal sample from Gombe presented here allows for statistical analyses not possible with earlier, smaller sample sizes.

We are indeed fortunate to be able to study the complete lives – from birth to death – of chimpanzees from Gombe. Former alpha-male Goblin is a prime example of this phenomenon. First observed as an infant just a few days old, the entire course of his life has been carefully recorded and analyzed. I am pleased to present an analysis of Goblin's skeleton here for the first time, along with the skeletons of 38 other chimpanzees from Gombe National Park. Eleven of the chimpanzees discussed here have been presented previously, and have been described in a wide range of useful research on morphological and size variation, trauma, and pathology (see Chapter 1). This study's primary purpose is not to negate previous studies, but rather to engage the sample in a quantitative way that was not previously possible due to a smaller sample size, and to evaluate whether that smaller sample can be considered representative of the larger collection. Inter-species comparisons will further illuminate variation within and between species and provide a better understanding of the selective pressures confronting different species of primates as well as how these pressures might be reflected by the skeleton.

It is my hope that this research will stimulate further inquiry into this valuable skeletal collection, improve its curation, and inspire other primate researchers to preserve the skeletons of their study animals whenever possible.

I examine the relationship between dominance rank, age, sex, and incidence of trauma and pathology. Dominance rank plays an important role in the social lives of

chimpanzees. It affects access to resources (Murray *et al.*, 2006; Pusey *et al.*, 2005), inter-individual relationships (Foster *et al.*, 2009; Goodall, 1986), and reproductive success (Constable *et al.*, 2001; Pusey *et al.*, 1997; Wroblewski *et al.*, 2009), among other variables. As such, dominance rank can be considered a useful proxy for measuring fitness. How chimpanzee fitness may be affected by trauma and pathology is relevant for better understanding the selective pressures confronting chimpanzees.

The long term behavioral research at Gombe as well as studies of other wild primate skeletons allow for reasonable predictions concerning the relationship between rank and incidence of trauma and pathology. Males who achieved high rank, because of the agonistic encounters necessary for securing and maintaining high rank (Muller and Wrangham, 2004; Thompson *et al.*, 2009), should have the highest frequencies of trauma. The lowest ranking males may be attacked less than males who are in more active competition, as low ranking males would be considered less of a threat (Honess and Marin, 2006). Low ranking females, particularly peripheral or immigrant females, should also have a high incidence of trauma because of elevated levels of intra-sexual aggression (Kahlenberg *et al.*, 2008). Skeletal pathology, as a generalized sign of stress, may have a similar pattern, with high ranking males and low ranking females showing the highest frequencies of skeletal pathology.

Previous studies of wild primate skeletal material (Jurmain, 1997; Jurmain and Kilgore, 1998; Latimer, 1993; Lovell, 1990) have found a correlation between incidence of trauma and pathology and age, and I expect those trends to hold true for this enlarged sample from Gombe, as a longer life span means a chimpanzee has more time to accumulate injuries and illnesses. Degenerative pathologies such as premortem tooth loss

and arthropathy should be closely correlated with age, and are likely to be less influenced by dominance rank. Most studies of primate skeletons focus on adults, sometimes including older sub-adults. As this sample includes a much wider variety of age categories (from young infant to old adult), the correlation between age and trauma incidence may not be comparable to previous findings based on samples more restricted in age categories. This is especially relevant in light of the injuries observed during chimpanzee infanticides (Arcadi and Wrangham, 1999) and the inclusion of 4 infanticide victims in this sample.

MATERIALS AND METHODS

Forty-eight chimpanzees are represented in the Gombe chimpanzee skeletal collection; 39 are presented here. Of the 39 chimpanzees discussed below, 28 are complete or nearly complete skeletons. Most of the chimpanzees have known life histories, and many of those who were born after 1960 include complete life histories from birth until death. Table 1.2 summarizes the complete Gombe skeletal sample, including location of curation (Gombe National Park for chimpanzees who died in 1987 or later or the University of Minnesota for those who died in 1987 or earlier).

The bodies of dead chimpanzees are recovered whenever possible for the purpose of skeletal preservation and analysis of soft tissue for pathological lesions (K. Terio, unpublished data). Skeletons of chimpanzees who died prior to 1987 were cleaned according to protocols laid out elsewhere (Zihlman *et al.*, 1990). Skeletons of chimpanzees who died after 1987 were processed in a different way. Following necropsy (K. Terio, unpublished data), the bodies were buried in a permeable bag and exhumed

after at least one year. Skeletons were then carefully cleaned using water and a soft brush and allowed to thoroughly dry.

Because some chimpanzees at Gombe are infected with a strain of the Simian Immunodeficiency Virus (SIVcpz) (Keele *et al.*, 2009; Santiago *et al.*, 2002), an extra step was added to the cleaning procedure in order to reduce the risk of possible transmission of the virus to skeletal researchers. Infected chimpanzees were briefly (2 minutes) rinsed in a mild bleach solution after initial cleaning, and then subsequently soaked in water to remove bleach residue.

Each skeleton was then inventoried and stored in mosquito net bags (to reduce the risk of insect damage to the bones in storage) in a secure location at Gombe National Park.

Each skeletal element was macroscopically examined under natural light for signs of trauma and pathology. Traumata and pathologies were scored according to the system outlined by Carter *et al.* (2008) in a study of chimpanzee skeletons from Kibale National Park, Uganda. Table 2.1 outlines the categories used in this study. To the five categories outlined by Carter and colleagues, I added a sixth category: "indeterminate / combined" for those cases when it was unclear what process was at work or for cases where multiple factors accounted for the pathology. (These cases make up a very small number (under 1%) of the observed pathologies.) This particular scoring system has the advantage of allowing for direct comparison between the Kibale and Gombe skeletal samples. It also provides a structured way to assess pathology and examine trends within the sample without the need for differential diagnoses in each case (which will inherently be more or less accurate / precise depending on the nature and specificity of the pathology in

question). Because joint disease can result in both bone loss and bone formation, instances of arthropathy were only scored in this category and not also scored as bone loss or bone formation instances. Criteria for determining the category of trauma or pathology followed protocols laid out in bone disease manuals (Aufderheide and Rodriguez-Martín, 1998; Buikstra and Ubelaker, 1994; Ortner, 2003; Resnick, 1988; Rogers and Waldron, 1995). Trauma and pathology was assessed as being mild, moderate, or severe based on the degree to which the injury or illness is hypothesized to have interfered with normal activity, or is known from behavioral records to have interfered. Degenerative joint disease was also classified according to severity. Mild degenerative joint disease was diagnosed for joint surfaces exhibiting increased porosity, but without significant trabecular exposure (under 2mm) or osteophyte formation (one osteophyte 2mm in length or less). Moderate degenerative joint disease included trabecular exposure and eburnation of the joint surface in an area more than 2mm in diameter, with lipping around the margin of the articular surface. Severe degenerative joint disease is evident in the form of major remodeling of the joint surface, including morphological change to the articular surface, or pitting / trabecular exposure across the majority of the articular surface. Either or both articular surface(s) within a joint (e.g. the distal femur and proximal tibia make up the knee) could be affected in order for a joint to be considered affected by degenerative joint disease.

Dental eruption and epiphyseal fusion were documented using categories laid out in Tables 2.2 and 2.3. Vertebral degeneration is assessed after Zukowski and Falsetti (2009) (Table 2.4). For simplicity, only data on the vertebral body are included here in anticipation of a more extensive future study focusing on vertebral degeneration.

Table 2.1: Scoring system for pathology and trauma (after Carter et al. (2008)).

pathology / trauma	**elements affected**	**extent of affected area**	**severity**	**degree of healing**
1- trauma	e.g. humerus	measured in cm	1: mild; 2: moderate; 3: severe	1: perimortem; 2: moderate; 3: advanced
2- arthropathy				
3- bone loss				
4- bone formation				
5 - developmental abnormality 6 – Bone formation and loss combination / indeterminate				

Table 2.2: Dental eruption scoring system

Score	**Description**
1	the tooth is not visible. No perforation of the alveolar bone is evident
2	alveolar bone is perforated, but the forming tooth is still inside the crypt (tooth is visible inside crypt by the naked eye)
3	less than half of the tooth is erupted
4	half or more of the tooth is erupted
5	the tooth is in full occlusion
6	premortem loss, with at least some resorption of alveolar bone (primarily for adult teeth)
7	missing (postmortem, or damage)
8	missing (congenital)

Table 2.3: Scoring system for post-cranial skeletal maturation

Score	**Description**
1	epiphysis has not begun to fuse (totally unattached to the diaphysis)
2	the epiphysis is fused to the diaphysis around half or less of the circumference of the shaft
3	the epiphysis is fused around more than half of the circumference of the shaft
4	the epiphysis is fused around the entire shaft, but a line or gap is still visible along some portion of the epiphysis
5	the epiphyseal line is completely obliterated

Table 2.4: Scoring system for vertebral body degeneration, after Zukowski (2009)

Stage 0	No evidence of osteophytosis; smooth rim with no scalloping or osteophytic points (no reactive bone activity)
Stage 1	Minor evidence of osteophytosis; one or two small osteophytic points (less than 2mm in length and width) but no larger osteophytes that protrude above the rim and/or the beginnings of arthritic lipping but no horizontally-projecting lipping
Stage 2	More developed osteophytosis; three or more small osteophytic points or larger osteophytes and/or horizontally-projecting arthritic lipping at least 3mm in length or the fusion of multiple osteophytic points that protrude
Stage 3	Arthritic lipping/fused osteophytes that extend out either superiorly or inferiorly at least 3mm in height (towards the center of the vertebral body or the adjacent vertebra)
Stage 4	Either partial or complete fusion of arthritic lipping/fused osteophytes between adjacent vertebrae

Many of the data on trauma and pathology (Jurmain, 1989; Jurmain, 1997; Jurmain, 2000; Jurmain and Kilgore, 1998; Kilgore, 1989) as well as skeletal and dental maturation (Zihlman *et al.*, 2004b; Zihlman *et al.*, 1990) have been previously published for the chimpanzees who died before 1987, but I summarize them again using a slightly

finer-scale scoring system for consistency with the expanded sample. Ages and sexes are known from the behavioral literature (e.g. Goodall, 1986) when positive identification of a chimpanzee at the time of death was possible. Unknown chimpanzees, skeletons of uncertain identification, and chimpanzees with estimated ages are indicated in Table 1.2.

Rank data are presented in Table 1.3 and are collated from Goodall (1986), Foster *et al.* (2009), Murray *et al.* (2007; 2006), and unpublished data from Gombe Stream Research Center (GSRC). Because rank often changes over the course of a chimpanzee's life, highest achieved rank and rank at death were both included in an attempt to encompass rank variation over the course of the chimpanzee's life.

Female dominance ranks are usually categorized as high ranking versus middle ranking versus low ranking (e.g. Pusey *et al.*, 2005). Male chimpanzees may be given ordinal ranks (1 – n adult males in the community) (Bygott, 1979; Goodall, 1986), but these were collapsed into categories similar to the females' for the purpose of the statistical analyses undertaken in this study (in order to avoid having to divide the sample). Males ranked 1-4 are considered high ranking, 5-8 are considered middle ranking, and ranks 9 and above are low ranking. These categories were altered twice to account for the number of males in a group. Vincent was ranked 3rd of 3 adult male chimpanzees at the time of his death, and is therefore considered low ranking. Similarly, Jomeo's numerical rank during the year of his death could be considered either high or middle ranking because of the relatively small number of adult males. Jomeo's relative rank (Jameson *et al.*, 1999) places him in the top third of adult male ranks, however, so he is counted as high ranking at time of death.

Because many chimpanzees are missing a few smaller bones (usually from the hands / feet), trauma and pathology incidence is expressed as a percentage of observable bones affected by trauma or pathology, in order to control for the differences in number of preserved skeletal elements. T-tests checked for differences in trauma and pathology frequency between males and females, and adults and sub-adults, and were carried out in R version 2.11.0 (www.r-project.org). I employed Welch's t-test because it does not require an assumption of equal variances in the two samples, a possible concern with small sample sizes (Welch, 1947).

I analyzed the relationship between trauma and pathology incidence, dominance rank, age, and sex using the statistical software Arc (Cook and Weisberg, 1999). I considered trauma incidence, arthropathy incidence, and pathology incidence and their correlation with age, sex, high rank, death rank, a variable that considers the relationship between age and rank, and factors that describe a change in rank between highest achieved rank and rank at death. I used scatterplot matrices to visually evaluate potential relationships between each variable, eliminated co-varying variables, and tested each of the variables separately. Because response data expressed as percents or counts are usually in need of a variance-stabilizing transformation, I applied the arc-sine square-root transformation to the responses (*Ibid*.)

I included only complete or nearly complete skeletons in the statistical analyses. Chimpanzees represented by only a cranium or for whom data on rank are not available were excluded from statistical analyses (Satan was also excluded due to low skeletal element representation). Their cases are discussed qualitatively.

Trauma

I included 30 chimpanzees (16 females, 14 males), most with known age, sex, and dominance ranks (Table 2.5) in the analysis of these variables' effects on trauma incidence. Trauma incidence is the percent of observable bones affected by trauma. I used a t-test to examine sex differences in trauma incidence, and also analyzed trauma by location to further explore possible sex differences in trauma pattern.

Degenerative Joint Disease

Only chimpanzees with well developed, observable bony joint surfaces were included in the analysis of degenerative joint disease. This included 22 chimpanzees (12 females, 10 males) from the age of 8.2 to over 50 years of age (Table 2.6). Degenerative joint disease incidence is the percent of observable joint surfaces affected by moderate or severe degenerative joint disease / arthropathy.

Pathology (non-arthropathy)

I included 30 chimpanzees (16 females, 14 males), most with known age, sex, and dominance ranks (Table 2.7) in the analysis of these variables' effects on pathology incidence. Pathology incidence is the percent of observable bones affected by bone formation or bone loss pathologies or congenital abnormalities.

Table 2.5: The trauma analysis sample

Chimpanzee	Age	Sex	Observed bones	Number with trauma	Trauma incidence	HighRank	DeathRank
Andromeda	0.73	f	132	52	0.00	3.00	3.00
Atlas	31.30	m	215	5	0.62	3.00	3.00
Charlie	25.87	m	183	3	0.39	3.00	3.00
Cusano	39.94	m	154	7	0.02	2.00	2.00
Ebony	8.19	m	206	2	0.05	1.00	1.00
Echo	34.00	f	210	4	0.01	3.00	3.00
Flo	53.14	f	188	9	0.00	2.00	2.00
Fred	0.90	m	141	0	0.06	2.00	2.00
Gaia's infant	0.00	m	125	0	0.01	2.00	2.00
Galahad	11.84	m	170	0	0.01	2.00	2.00
Gilka	18.87	f	177	2	0.00	2.00	3.00
Goblin	39.96	m	207	10	0.03	1.00	1.00
Hugo	38.58	m	183	8	0.02	1.00	1.00
Infanticide, November	0.67	f	134	0	0.36	3.00	3.00
Infanticide, October	0.75	m	76	47	0.01	2.00	2.00
Jackson	10.41	m	199	0	0.00	3.00	3.00
Jomeo	30.87	m	198	4	0.01	?	3.00
Kidevu	25.59	f	205	0	0.02	1.00	2.00
Madam Bee	28.22	f	197	12	0.02	1.00	1.00
Mel	10.71	m	203	3	0.05	1.00	1.00
Melissa	36.31	f	207	3	0.01	3.00	3.00
Miff	30.90	f	201	1	0.00	3.00	3.00
Pallas	27.20	f	165	0	0.00	3.00	3.00
Passion	30.61	f	225	6	0.00	3.00	3.00
Patti	44.25	f	214	5	0.05	1.00	2.00
Rejea	0.27	f	106	38	0.04	?	3.00
Sherehe	15.78	f	215	1	0.00	3.00	3.00
Sugar	10.91	f	178	0	0.02	1.00	1.00
Vincent	28.47	m	195	60	0.02	3.00	3.00
Yolanda	24.00	f	216	3	0.31	1.00	1.00

Table 2.6: Degenerative Joint Disease sample

Specimen	Sex	Age	Observed joint surfaces	Joint surfaces affected by moderate or severe DJD	%	highrank	deathrank
Atlas	m	31.3	166	3	0.02	1	2
Charlie	m	25.9	135	0	0	1	1
Cusano	m	39.9	107	3	0.03	1	1
Ebony	m	8.2	159	0	0	3	3
Echo	f	34.0	169	3	0.02	2	2
Flo	f	53.1	136	29	0.21	1	1
Galahad	m	11.8	141	0	0	3	3
Gilka	f	18.9	131	7	0.05	3	3
Goblin	m	40.0	156	20	0.13	1	2
Hugo	m	38.6	129	19	0.15	?	3
Jackson	m	10.4	153	0	0	3	3
Jomeo	m	30.9	147	4	0.03	1	1
Kidevu?	f	25.6	155	0	0	2	2
Madam Bee	f	28.2	143	1	0.01	2	2
Mel	m	10.7	158	0	0	3	3
Melissa	f	36.3	160	6	0.03	2	2
Miff	f	30.9	158	6	0.04	2	2
Pallas	f	27.2	114	21	0.18	2	3
Passion	f	30.6	180	8	0.04	1	1
Patti	f	44.3	185	11	0.06	1	1
Sherehe	f	15.78	165	0	0	2	2
Sugar	f	10.9	131	0	0	3	3
Vincent	m	28.5	149	3	0.02	1	1
Yolanda	f	24.0	177	5	0.03	?	3

Table 2.7: Pathology sample

Specimen	Age	sex	Observed bones	Bones with pathology	%	highrank	deathrank
Andromeda	0.7	f	132	0	0	3	3
Atlas	31.3	m	215	26	0.121	1	2
Charlie	25.9	m	183	12	0.066	1	1
Cusano	39.9	m	154	15	0.097	1	1
Ebony	8.2	m	206	7	0.034	3	3
Echo	34.0	f	210	22	0.105	2	2
Flo	53.1	f	188	50	0.266	1	1
Fred	0.9	m	141	12	0.085	3	3
Gaia's infant	0.0	m	125	4	0.032	3	3
Galahad	11.8	m	170	11	0.065	3	3
Gilka	18.9	f	177	23	0.13	3	3
Goblin	40.0	m	207	34	0.164	1	2
Hugo	38.6	m	183	32	0.175	?	3
Infanticide, November	0.67	f	134	0	0	3	3
Infanticide, October	0.75	m	76	0	0	3	3
Jackson	10.4	m	199	8	0.04	3	3
Jomeo	30.9	m	198	24	0.121	1	1
Kidevu	25.6	f	205	8	0.039	2	2
Madam Bee	28.2	f	197	8	0.04	2	2
Mel	10.7	m	203	12	0.059	3	3
Melissa	36.3	f	207	11	0.053	2	2
Miff	30.9	f	201	17	0.084	2	2
Pallas	27.2	f	165	10	0.06	2	3
Passion	30.6	f	225	26	0.116	1	1
Patti	44.3	f	214	10	0.047	1	1
Rejea	0.3	f	106	2	0.019	3	3
Sherehe	15.78	f	215	12	0.056	2	2
Sugar	10.9	f	178	6	0.034	3	3
Vincent	28.5	m	195	32	0.164	1	1
Yolanda	24.0	f	216	22	0.102	?	3

RESULTS

Individual Cases

The narratives below are meant to provide an overall picture of the traumata and pathologies accumulated on each chimpanzee's skeleton at the time of death rather than complete descriptions of each affected element.

Gaia's Infant (2008) (male - 1 week?)

Gaia, aged 15 years in 2008, gave birth to twin sons in July of that year. Both infants were seized a few days after their birth by Gremlin, their maternal grandmother, who has previously been observed to take and (attempt to) care for Gaia's infant (Godot in 2006). Both of the infants born in 2008 died. The smaller of the infants had always looked feeble, was a poor clinger, and may have died within a few days or even hours after birth as the soft tissues were already heavily autolyzed at the time of necropsy. Gremlin eventually dropped the body while climbing a tree, and Gaia re-claimed her already dead infant and carried him around for a few more days. Eventually she abandoned the body and it was recovered by field observers. The brother's body was, unfortunately, never recovered (Gombe Stream Research Center (GSRC), unpublished data).

The infant's skeleton is at the developmental stage expected for a chimpanzee neonate. The frontal bones and mandible are completely unfused. The post-cranial skeleton is also unfused, with no development of secondary ossification centers. No teeth are erupted. The crowns of 13 deciduous teeth in the process of forming were recovered.

No significant soft tissue lesions were noted (Karen Terio, unpublished data); though a few skeletal lesions are of potential significance.

Both parietals exhibit increased porosity, with perforating foramina near the sagittal suture. This seems to be outside the range of what is usual even for early bone development, though it might be discounted if not for the pathologic appearance of the maxillae. Both palatal surfaces of the maxillae lack the outer table of bone, and the trabeculae are enlarged, forming the characteristic "hair-on-end" appearance that is commonly associated with various kinds of anemia (Moseley, 1974) (Figure 2.1). What is of particular interest in this case is the lack of soft tissue lesions, and that though autolysis was advanced, large or severe lesions would have been observable if present (Karen Terio, unpublished data). This suggests that the skeletal manifestations are part of the early stages of an anemic disease, and it is possible that the anemic condition contributed to his death.

Figure 2.1: Palatal view of maxilla, Gaia's infant (2008)

Rejea (female - 3 months)

A female infant from the Mitumba community, Rejea was snatched from her mother, Rafiki, killed, and partly consumed by males from the Kasekela community in 1993 (Wilson *et al.*, 2004). Her frontal bone and mandible are fused while the occipital and temporal bones are unfused. Her deciduous incisors and first premolars are in full occlusion.

Her skeleton shows damage patterns characteristic of chimpanzee consumption of primates (per Pobiner *et al.*, 2007), including compression fractures to the cranial vault, puncture wounds in the frontal bone, step fractures of the long bones (Figure 2.2), frayed long bone ends, and incipient fractures on the ribs.

Figure 2.2: Rejea's partially consumed upper extremities

November Infanticide (unknown) (female - 8 months?)

This unidentified female infant was an infanticide victim in November of 1975. Her mother, unfamiliar to Gombe researchers, was attacked by Sherry and Jomeo. The mother dropper her infant as she fled up a tree. Eventually, Figan ended up in possession with the infant, and he displayed with her for several minutes, flailing her against tree branches. Eventually she was abandoned without being eaten, but was carried around for two more hours by both Freud (then aged 4 years) and then by Jomeo before finally being abandoned. She was still alive when recovered by the research staff, but died that night from her wounds. That she was not observed to be either bitten nor consumed (Goodall, 1977) is a partial explanation for the lesser amount of trauma to her skeleton compared to the other infanticide victims presented here.

The skeleton is nearly complete, lacking only some distal phalanges and some carpals and tarsals (lack of the latter is likely due to incomplete ossification). Age of this chimpanzee at death is estimated to be approximately 8 months due to comparability in size and dental development to Andromeda. None of the secondary ossification centers are present. The mandible and frontal bones are completely fused, and the deciduous incisors and 1st premolars are all in occlusion. The 2nd deciduous premolars have begun to erupt but are not in full occlusion (because Andromeda's 2nd deciduous premolars are in full occlusion, this infant's age is estimate to be slightly less than Andromeda's).

The right scapula, ilium, raidus, both ulnae, 3 left ribs, and 1 proximal manual phalanx all show possible incipient fractures, but the timing of this damage is unclear. The ribs are cracked but not separated, and they resemble the damage pattern seen on

other chimpanzee infanticide victims (Figure 2.3). The other elements may display bending deformities due to postmortem change (Figure 2.4).

Figure 2.3: Ribs of an unknown infant with likely perimortem trauma

Figure 2.4: Right scapula of an unknown infant with possible postmortem damage

October Infanticide (unknown) (male - 9 months?)

This unidentified male infant was killed by Figan, Jomeo, and Satan in October 1975. Researchers observed him being displayed with and partially consumed. Part of his brain, the contents of the abdominal and pelvic cavities, both legs, and one hand had been consumed when researchers recovered the abandoned body (Goodall, 1977). Skeletal damage is consistent with consumption patterns associated with chimpanzee infanticide victims (See Chapter 3 for further discussion). His age at death is approximately 9 months based on skeletal size and dental development, which is comparable to Andromeda. The deciduous incisors and premolars are all erupted, while the frontal bone and mandible are both fused. Long bones are assumed to all be unfused, though none of the long bones is complete. Incipient fractures on the ribs, and frayed ends or step fractures to the long bones are consistent with consumption. Puncture wounds in his frontal bone (Figure 2.5) and left parietal are consistent with the head wounds observed shortly after the infant's death (*Ibid*.)

Figure 2.5: Puncture wound superior to the right orbit in a three-month old male infanticide victim

Andromeda (female - 9 months)

Andromeda is another female infanticide victim from the Mitumba community. Her mother, Aphro, emigrated to the Mitumba community from Kasekela in December 1988. Andromeda's maternal uncle, Atlas, is also part of the Gombe skeletal collection (both Atlas and Aphro are Athena's offspring) (GSRC).

Andromeda's death in 2005 was not directly observed, though males from Kasekela – including Atlas - were followed into the Mitumba community's territory and field observers heard sounds consistent with an aggressive inter-community encounter. Andromeda was not consumed, but was re-claimed by Mitumba males subsequent to (or not long before) her death. Her body was observed to be used in displays, carried, and groomed before it was eventually abandoned and recovered by researchers (M.L. Wilson, unpublished data).

Andromeda's skeleton is mostly complete, though it displays both peri- and postmortem damage. Her deciduous incisors and premolars are all in full occlusion, and the deciduous canines are less than half erupted.

Even though she was not at all consumed, Andromeda still shows some skeletal damage similar to that of Rejea, including step fractured long bones, insipient rib fractures, and compression fractures to the cranial vault. Because she was not consumed, Andromeda displays much lower levels of bone fragmentation than Rejea, and better bone survivorship (see Chapter 3). Andromeda's skeleton also displays surface weathering that is superficially similar in appearance to digested bone. This is likely due to a longer than desirable burial period before her skeleton was cleaned. Photos of

Andromeda's skeleton and further discussion of skeletal trauma to chimpanzee infanticide victims are available in Chapter 3.

Fred (male - 10.8 months)

Fifi's infant Fred died during the sarcoptic mange outbreak at Gombe in 1997. Mange is a contagious disease spread by parasitic mites; symptoms recorded in affected chimpanzees included hair loss, itchiness, weight loss and lethargy. Younger and older chimpanzees were more likely that prime-aged animals to succumb (Williams *et al.*, 2008). Genetic testing revealed that Frodo was Fred's father. Since Frodo is also Fred's half brother (Constable *et al.*, 2001), lack of genetic diversity, in addition to his increased vulnerability as an infant, may have contributed to Fred's cause of death, though certainly the possibility of decreased milk production during his mother's illness could be a factor compounding Fred's illness.

Fred's skeleton is complete, though secondary ossification centers are not yet developed. His deciduous incisors and premolars are all erupted, though one of the maxillay second premolars is not yet in full occlusion. The deciduous canines are less than half erupted. Fred also exhibits a metopic suture in the glabellar region (it does not extend superiorly) (Figure 2.10).

Pathologic lesions on Fred's skeleton involve abnormal bone formation. This includes periostitis on 4 metapodials and 3 manual intermediate phalanges, which, as a general sign of infection (Larsen, 1998), is consistent with his cause of death. His temporal glenoid fossae are also abnormally rough, perhaps indicative of malocclusion of

the temporal-mandibular joint. As Fred was still nursing at the time of his death, mandibular joint problems are not likely to have been a serious problem.

Figure 2.6: Fred's frontal bone, with partial metopic suture still visible

Ebony (male - 8.2 years)

An adolescent male from the Mitumba community, Ebony died in 2005. He was one of Eva's sons, and the half brother of Edgar, one of two adult males currently in residence at Mitumba (GSRC, unpublished data). Ebony's body was discovered shortly after an incursion by the Kasekela males into the Mitumba community's territory, and it showed soft tissue wounds consistent with chimpanzee attack (M.L. Wilson, unpublished data).

The skeleton is complete, with well developed secondary ossification centers. None of the epiphyses are fused. The inferior pubic ramus is fused to the ischium, and the 2nd through 5th sacral vertebrae have begun to fuse (the 1st and 2nd remain separate).

The deciduous canine is still present, and the 3rd molars are unerupted. The mandibular 1st premolars are over half erupted, and the rest of the adult dentition is in full occlusion.

As a young chimpanzee, Ebony shows few signs of skeletal pathology or trauma. Most notably, his skeleton shows no signs of perimortem trauma. Two proximal manual phalanges show fractures in the process of healing. The femoral and tibial surfaces of both knees, as well as the distal articulations of the tibiae (Figure 2.7), have possible subchondral bone cysts: a lesion whose etiology is likely to follow one of two trajectories, both of which relate to bone development. A subchondral bone cyst may be epiphyseal cartilage that becomes "trapped" in the ossifying epiphysis (Thompson, 2007), or may develop as a result of synovial fluid leaking through damaged articular cartilage and then becoming trapped in the epiphysis once the bone is completely ossified (Yochum and Rowe, 2005). This kind of cyst may also be a sequel to degenerative joint disease in some species (Weisbrode, 2007), though this seems unlikely in Ebony's case due to his young age. Subchondral cysts are unlikely to be a source of inconvenience, and usually resolve with age (Thompson, 2007). Laminar deficiency is evident in the 3rd and 4th sacral vertebrae (Figure 2.8); the discussion section elaborates on the possible implications of laminar deficiency, which is evident in 7 of the chimpanzees in the Gombe skeletal collection.

Figure 2.7: Ebony's distal tibia, with possible sub-chondral bone cyst

Figure 2.8: Ebony's unfused sacrum, showing laminar deficienty in the 3rd and 4th segments

Jackson (male - 10.4 years)

Jackson's mother, Jiffy, was first seen in the Kasekela community in 1975, when she was approximately 10 years old. Jiffy gave birth in 1988, but the infant only lived 6 months. Jackson was born in 1989, and Jiffy has no other offspring (GSRC, unpublished data). Bacterial pneumonia is the suspected cause of death for Jackson, whose body was recovered in 2000 (Williams *et al.*, 2008).

The inferior pubic ramus is fused to the ischium, and all other epiphyses are unfused, though secondary ossification centers are well developed. The permanent incisors and 1st and 2nd molars are in full occlusion. The 3rd molar is not erupted. The deciduous canines and premolars are still in situ and their adult counterparts are unerupted.

Jackson shows no signs of skeletal trauma, but several pathologies. The distal articulation of the left tibia displays a small (approximately .5mm in diameter) lesion that may have a similar etiology as the proposed sub-chondral cysts described for Ebony (see above). The distal articulations of both ulnae display a bone-loss pathology of unknown etiology, and the right clavicle exhibits a lesion related to pathological bone loss on its inferior, lateral surface. A manual intermediate phalanx shows abnormal bone formation consistent with mild periostitis. Given the cause of death, skeletal signs of infection are not surprising.

Both tali have extensive proliferative lesions on the superior, distal articular aspect. The new bone formed is waxy in appearance, though also very porous (Figure 2.9). Jackson also displays laminar deficiency in the 2nd sacral vertebra (Figure 2.10).

Figure 2.9: Jackson's talus, with proliferative lesion

Figure 2.10: Jackson's sacrum, note deficiency in lamina of 2nd vertebra

Mel (male - 10.7 years)

Mel's mother, Miff, died in 1987, leaving him orphaned. Miff is also part of the Gombe skeletal collection. Though only 3 years old at the time, Mel survived his mother's death (GSRC, unpublished data). This is an age at which a young chimpanzee often dies if his mother dies (Goodall, 1986), but Mel lived another 7 years. His body was discovered in 1994 sporting soft tissue injuries consistent with either a leopard predation attempt or chimpanzee attack (Williams *et al.*, 2008). The fact that he was not

at all consumed suggests that a leopard attack is less likely (Wilson *et al.*, 2004). The skeleton is complete and shows no signs of perimortem trauma.

The epiphyses are well developed, but unfused. The inferior pubic ramus has fused to the ischium, and the acetabuluar ossification center between the three bones of the os coxa is in the early stages of unification. Most of the adult dentition is in full occlusion, exceptions being the unerupted permanent 3rd molars and the maxillary deciduous canines, which are still *in situ.* The mandibular deciduous canines have been shed, and the right permanent mandibular canine is beginning to erupt. The left canine has not started erupting, nor is there a patent alveolus. A bulge in the mandibular corpus seems to indicate that the canine is, in fact, developing, but a radiograph would be necessary to confirm. The mandibular incisors are crowded (Figure 2.11), suggesting a possible discordance between mandibular versus dental development (see Zihlman *et al.*, 1990, for commentary related to Flint)

Two phalanges (one manual and one pedal) show evidence for well healed fractures. His maxilla shows an area of reactive bone near prosthion (Figure 2.12). The anterior maxillary dentition, which was recovered when the skeleton was exhumed, does not show signs of breakage. The reactive bone in this case more closely resembles an extended area for an erupting tooth. That the incisor would have already been in full occlusion (as was its left pair) and the lack of abscess drainage sites or carious lesions on the teeth themselves also suggest a diagnosis other than periodontal disease or trauma. The left maxillary 1st molar does show evidence for periodontal disease, as the bone on the palatal side shows a smooth-edged erosion that widens the alveolus (Figure 2.13).

Three carpals, both distal femora, and both articular surfaces of the right tibia display round lesions approximately 2-3mm in diameter that may be evidence for sub-chondral bone cysts (see *Ebony* for more detail). The right 5th metatarsal shows proliferative bone consistent with periostitis.

Mel exhibits laminar deficiency in the 3rd and 4th sacral vertebrae (Figure 2.14). The sacral vertebrae are all unfused.

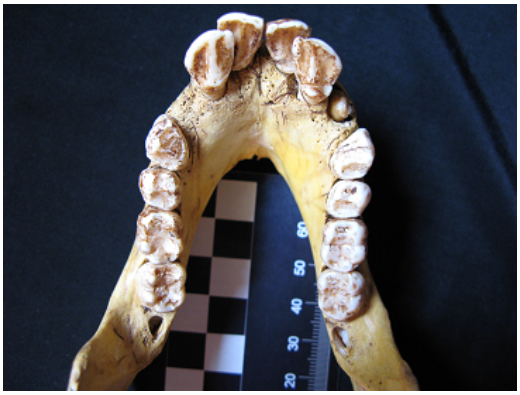


Figure 2.11: Mel's crowded mandibular incisors

Figure 2.12: Mel's maxilla, with area of reactive bone near alveolus for the right central incisor

Figure 2.13: Alveolar erosion adjacent to Mel's left maxillary 1st molar

Figure 2.14: Laminary deficiencry in S3-4 in Mel's sacrum

Sugar (female - 10.9 years)

Sugar is one of Caramel's offspring; Sugar's half sister, Candy, and her offspring still live in the Kasekela community (GSRC, unpublished data). Sugar died of a respiratory infection in 1987 (Williams *et al.*, 2008). Her complete skeleton is well preserved and shows few signs of pathology or trauma. The maxilla and other facial bones have not fused with the neurocranium. The epiphyses are mostly unfused and secondary ossification centers are well formed. The inferior pubic ramus has fused to the ischium, and the distal epiphysis of the left humerus is in the early stages of fusion. The adult canines and 3rd molars are the only unerupted permanent dentition. The maxillary deciduous canines are still in occlusion, but the permanent mandibular canines are beginning to erupt. The mandibular incisors are very slightly crowded.

A lesion on the distal articulation of a pedal proximal phalanx (4.3 x .4 mm) shows abnormal bone resorption; its etiology is unknown. It is not morphologically consistent with a sub-chondral bone cyst. Interesting congenital variations include patent olecranon foramina on both humeri (Figure 2.15) and asymmetrical transverse foramina on the 1st cervical vertebra (Figure 2.16). Sugar displays laminar deficiency in the 3rd and 4th sacral vertebrae (Figure 2.17).

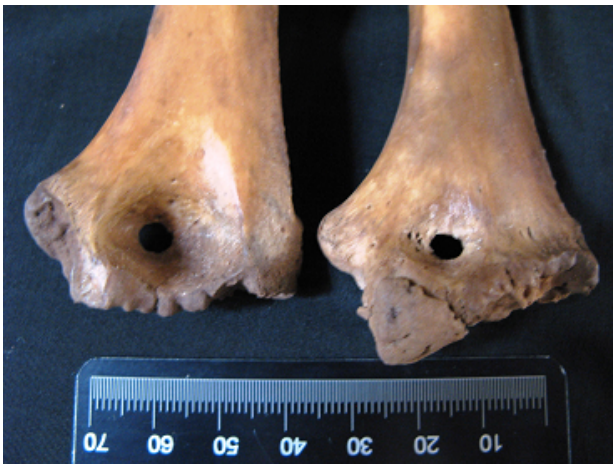


Figure 2.15: Sugar's humeri with olecranon foramina

Figure 2.16: Asymetrical transverse foramina in Sugar's 1st cervical vertebra

Figure 2.17: Laminar deficiency in Sugar's sacrum

Galahad (male - 11.8 years)

Galahad was one of Gremlin's offspring, and Melissa's grandson. He is survived by his mother as well as several maternal siblings: sister Gaia, the twin sisters Golden and Glitter, brother Gimli, and a new brother born in 2009 (GSRC, unpublished data). Galahad is suspected to have died of pneumonia in 2000 (Williams *et al.*, 2008). Besides the adult canines, which are less than half erupted, only the maxillary incisors and the mandibular lateral incisors are preserved, and dental eruptions are estimated from the alveoli. The 3rd molar crowns were likely well formed at the time of death, but it is unlikely they had begun to erupt (Zihlman *et al.*, 2004b). The rest of the adult dentition (excepting the canines) was in full occlusion at death. The secondary ossification centers are well formed but largely unfused. The distal humerus is less than half fused, as is the right medial epicondyle. The inferior pubic ramus is fused to the ischium.

Sub-chondral bone cysts affect several articular surfaces in the hands as well as the left talus. The articular surface of the left patella is abnormally porous and has a shiny appearance. Its appearance is not consistent with the eburnation characteristic of degenerative joint disease, but is indicative of some other kind of proliferative lesion (Figure 2.18). The vertebral end of a right rib shows an abnormal articular morphology. The head and tubercle are both fan shaped, and the articular surfaces are irregular. It is possible this is a dislocation deformity subsequent to trauma, though none of the thoracic vertebrae exhibit a corresponding pathology. Skeletal evidence for similar injuries is evident on Kidevu and Passion. A dent on the frontal bone near bregma, measuring approximately 13 x 3 mm, may be evidence of well-healed cranial trauma (Carter *et al.*, 2008) (Figure 2.19). This would have been inflicted during infancy, and is now well

healed. The 6th cervical vertebra is congenitally missing the transverse foramina (Figure 2.20). In addition, laminary deficiency in the 3rd, 4th, and 5th sacral vertebrae is present (Figure 2.21).

Figure 2.18: Galahad's patella with proliferative bone on articular surface

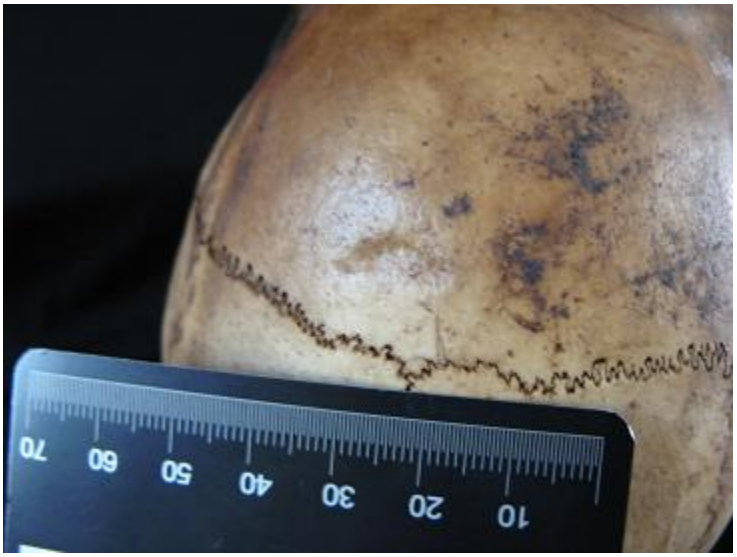


Figure 2.19: Well-healed cranial trauma near bregma, Galahad

Figure 2.20: Galahad's cervical vertebrae, note congenital absence of transverse foramina on C6 (far right)

Figure 2.21: Galahad's sacrum

Sherehe (female - 15.78 years)

Sparrow, born in 1960, is the oldest chimpanzee known to researchers currently at Gombe. She has 4 surviving offspring, including Sandi (b. 1973), the mother of Sherehe (GSRC, unpublished data). In 2006, Sherehe was found with extremely limited mobility and she died soon after. Her infant son, Shangaa, died not long after his mother. Her cause of death was not immediately known and disease or a fall from height were both considered possible causes of death. Soft tissue pathological analysis was not possible in this case due to the circumstances of death, though gross examination revealed a ruptured gall bladder (K. Terio, unpublished data). The skeleton does not reveal any evidence of perimortem trauma, though several pathologies consistent with infection (unknown etiology) are presented here.

Epiphyses are well formed and many are completely fused. The acromion, medial scapular margin, medial clavicle, and iliac crest are all unfused. The proximal humerus is less than half fused. The distal radius, distal femur, proximal tibia, proximal fibula, and sacrum are at least half fused. Fully fused elements where a clear epiphyseal line is still visible include the femoral head and distal tibia. The other epiphyses are fully fused with no visible epiphyseal line. The complete, adult dentition is in full occlusion and no dental pathologies are evident.

Some of the major synovial joints (hip, knee, elbow) show very early signs of degenerative joint disease, primarily in the form of excessive porosity of the joint surface. The fingers and toes also show minimal involvement. The left temporal glenoid fossa and both mandibular condyles show signs of degenerative joint disease, possibly indicating malocclusion of the temporal-mandibular joint.

Possible sub-chondral bone cysts were observed on the distal articulation of the left humerus (Figure 2.22), the proximal articulations of two metapodials as well as the right 1st cuneiform show the same pathology.

Both 13th ribs are covered with proliferative bone indicating a localized infection (Figure 2.23). The associated vertebra is not affected. Sharp-edged pieces of bone with irregular surfaces of unknown provenance were recovered with the skeleton (Figure 2.24), and an unidentified broken fragment somewhat resembling a tooth was also recovered (Figure 2.25). Future analysis of this possible tooth fragment may reveal more information. Laminar deficiency in the 2nd through 5th sacral vertebrae is present (Figure 2.26). Sherehe's cause of death remains unknown, but her skeleton shows evidence of infection, and not trauma.

Figure 2.22: Sub-chondral bone cyst on distal articulation of Sherehe's left humerus

Figure 2.23: Sherehe's rib with reactive bone over entire surface

Figure 2.24: Unidentified, sharp pieces of bone recovered with Sherehe's skeleton


Figure 2.25: Unidentified tooth fragment recovered with Sherehe's skeleton

Figure 2.26: Sherehe's sacrum

Gilka (female - 18.9 years)

During her life, Gilka suffered from many ailments, including polio in 1966 and a fungal infection that caused her face to swell beginning in 1968. She had at least 2 infants stolen from her and eaten by Passion and Pom in 1974 and 1976. Gilka never fully recovered from this second attack, and died in 1979 (Goodall, 1986). Her primary cause of death is considered a "wasting disease," usually attributed to gastro-intestinal disorder (Williams *et al.*, 2008).

Gilka's skeleton is complete, with well formed epiphyses, most of which are fully fused. The acromion process, medial margin of the scapula, and medial clavicle are still unfused. The iliac crest is more than half fused, and the epiphyseal line is still evident on the proximal humerus, proximal radius, femoral head, and sacrum. The complete adult dentition is in full occlusion, though the teeth are more worn than is usual for a chimpanzee her age (Kilgore, 1989).

Jurmain (1989) has described likely healed fractures to two of Gilka's metatarsals, and erosional lesions on 5 of her phalanges attributable to a bacterial infection. For at least two years before her death, open sores were visible on Gilka's hands and it is unclear whether this infection is of similar etiology as the fungal infection that affected her face (Godall, 1986; *Ibid*.). The sores on her hands somewhat limited Gilka's mobility during life, as did the fungal infection, which seemed to interfere with her vision, as she was observed to bump into trees when her brow ridge was very swollen (Goodall, 1986)

Gilka also exhibits mild degenerative joint disease in several major joints, mild-to-moderate degenerative joint disease on both articular surfaces of all 5 of her left metacarpals, and a moderate degree of degeneration on her right capitate and right distal humerus. Her right ulna and humerus are noticeably of a decreased diameter compared to the left, a result of her bout with polio. She never regained the full use of her forelimb (Jurmain, 1989; Morbeck, 1987; Morbeck *et al.*, 1991). This certainly affected her ability to protect her infants from Passion and Pom (Goodall, 1986), and favoring the left limb over the less useful right may have resulted in the relatively advanced degenerative joint disease for a chimpanzee her age evident in her left hand.

It is possible that Gilka's maxillary incisors were broken; they exhibit a higher degree of wear than the mandibular incisors, and the pulp cavity of both maxillary central incisors is exposed. A corresponding abscess drainage site is evident near her nasal aperture (Figure 2.27) (see also Kilgore, 1989).

Her right humerus exhibits a patent olecranon foramen, and the left navicular has only the articulations for the cuneiforms, lacking the tubercle.

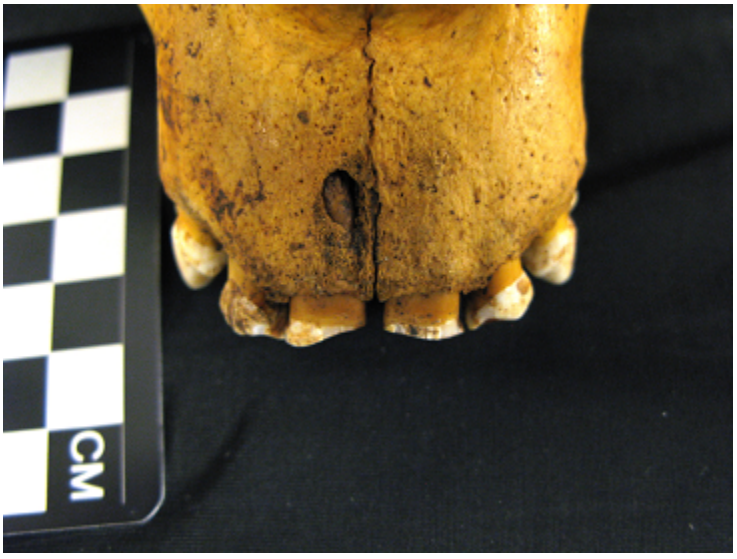


Figure 2.27: Gilka: abscess drainage site associated with maxillary incisor

Yolanda (female - 24 years)

Yolanda was first observed in the Kasekela community in 1997 (GSRC, unpublished data). She died in 2008, 3 years after contracting SIVcpz. Histopathological analysis of tissue samples taken at necropsy were consistent with end-stage AIDS in humans (Keele *et al.*, 2009). Her skeleton exhibits some pathologies that may be considered usual for an adult female chimpanzee, none of which are likely to have significantly affected her daily life.

Most of the epiphyses are fused. The acromion and medial clavicle remain entirely unfused. The iliac crest is over half fused, but the secondary ossification center is still separate in some places. The medial margin of the scapula, distal radius, femoral head, and proximal fibula still show a clear epiphyseal line even though the epiphysis is fully fused. The epiphyseal line is completely obliterated in all other cases. The complete adult dentition is in full occlusion and shows no signs of pathology.

Three ribs exhibit an abnormal contour that may be indicative of well healed fractures. No bony callus remains, so further assessment should rely on radiographs. These ribs (both 12th ribs and another from the right side) also show a moderate level of degeneration of the vertebral articulation; this line of evidence supports the hypothesis of some kind of trauma in this case.

Both hip and knee joints exhibit early stages of degenerative joint disease consisting in increased porosity of the articular bone. The right talus is also involved. The proximal ulnar articulation also displays mild to moderate degenerative joint disease in the form of osteophytic growth and proliferative bone on the articular surface. The 4th lumbar and 1st sacral vertebrae exhibit mild osteophytic lipping.

Five of the carpals and tarsals display abnormal, smooth foramina on an articular surface consistent with a sub-chondral bone cyst. The 3rd and 4th lumbar vertebrae also feature irregular foramina on the anterior vertebral bodies, likely to accommodate larger than average blood vessels.

The shafts of both femora and the left ulna exhibit reactive bone characteristic of periostitis. Both humeri have osteophytes beginning to bridge the bicipital groove, indicative of partial ossification of the transverse humeral ligament. An intermediate pedal phalanx exhibits a callus of smooth-looking bone near the distal articulation. Its morphology is consistent with benign tumors more commonly found on the cranium (Figure 2.28). Lastly, two pedal proximal phalanges have unusual nutrient foramen morphology.

Figure 2.28: Yolanda's intermediate pedal phalanx with bone formation pathology

Unknown male (cranium only) (20-30 years?)

The cranium of an unknown male was recovered in the Mitumba community's territory (GSRC, unpublished data). The recovery date is not recorded. This cranium shows no signs of trauma or pathology. The adult dentition was fully erupted at the time of death, though this is estimated from the alveolar morphology as only the 2nd molars remain. There is no evidence for premortem tooth loss.

Kidevu (identity tentative) (female - 25.6 years)

The estimated age of this skeleton coincides with the estimated age of Kidevu at the time she disappeared, though the body was not positively identified. Kidevu was first observed in the Kasekela community in 1979, when she was estimated to be 12 years old. She disappeared in 1992 (GSRC, unpublished data).

The skeleton exhibits some surface abrasion and bleaching from post-depositional damage but is otherwise in good condition and nearly complete, lacking only some hand and foot bones.

Most of the epiphyses are fused. The medial clavicle is still unfused, and the lateral clavicle was unobservable due to post-depositional damage. Her developmental stage is consistent with it being fully fused. The medial scapular margin and the iliac crest are both more than half fused, and, though fully fused, the sacrum still shows clear fusion sites between the individual vertebrae. The complete adult dentition is in full occlusion.

Kidevu exhibits mild degenerative joint disease in her wrists and ankles in the form of abnormally increased porosity of articular surfaces. The hips, knees, and elbows (proximal ulnae) as well as the vertebral articulations of 4 ribs are also involved. Not all ribs had observable vertebral articulations. Both temporal glenoid fossae show degenerated joint surfaces, but the mandibular condyles were unobservable due to post-depositional damage.

The left acetabulum has a diffuse, vascularized lesion unassociated with the articular surface. Bone formation pathologies include a discrete callus approximately 3mm in diameter near the distal articulation of an intermediate manual phalanx, and uneven palmar muscles attachment sites on 2 proximal manual phalanges, a possible indication of a muscle injury (Jurmain, 1989). The posterior aspect of the manubrium shows an area of proliferative bone; the original extent and nature of this lesion cannot be determined due to post- depositional damage.

The left 13th rib exhibits a bifurcated vertebral articulation, indicating extensive remodeling of the costal head and tubercle (Figure 2.29) possibly associated with a dislocation injury, though as the corresponding vertebra is not involved, any diagnosis

remains tentative. A 4th molar is in full occlusion in the left maxilla (Figure 2.30). A developing alveolus occupies the corresponding place on the left mandible.

Figure 2.29: Possible dislocation injury on Kidevu's 13th rib

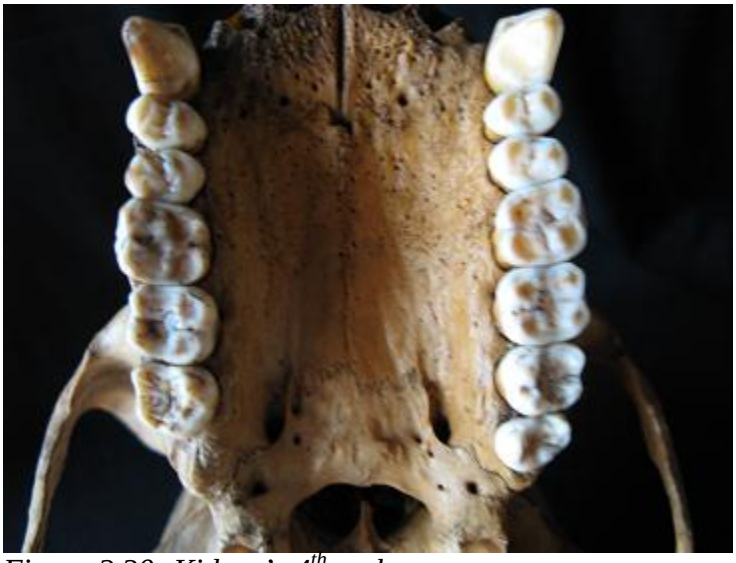


Figure 2.30: Kidevu's 4th molar

Charlie (male - 25.9 years)

Charlie was the alpha male of the break-away Kahama community, and died in 1977 during the lethal attacks by the Kasekela males that resulted in the extinction of the Kahama community. The presumed attack on Charlie was not directly observed. His body was found lying in Kahama stream, a few days after fishermen heard the sounds of fierce chimpanzee fighting in that area. His body had wounds on the head, neck, rump, scrotum, legs, arms, hands, and feet: a pattern of injuries consistent with chimpanzee attack (Goodall, 1986).

Charlie's skeleton is complete, and the epiphyses are entirely fused, except for the medial clavicle, iliac crest, and sacrum, which are all more than half fused. A clear epiphyseal line is still evident on the acromion, femoral head, and greater trochanter. The adult dentition is in full occlusion, and shows little wear (Kilgore, 1989).

Unhealed puncture wounds to the right side his frontal bone (Jurmain, 1989) and right maxilla (Figure 2.31) are further evidence of the manner of Charlie's death. Charlie exhibits very early stages of degenerative joint disease in the hip, elbow, and ankle. There are 4 small lesions consistent with subchondral bone cysts evident on wrist and ankle bones in addition to the large lytic lesion on the distal end of his right tibia that is likely related to some kind of bacterial or fungal infection (Jurmain, 1989).

Figure 2.31: Perimortem bite wound on Charlie's right maxilla

Pallas (female - 27.2 years)

A lower-ranking female whose range was in the middle of the Kasekela community's territory (Goodall, 1986), Pallas died in 1982 of a "wasting disease" (Williams *et al.*, 2008), leaving behind no surviving offspring (Goodall, 1986). Her skeleton is complete and well preserved. The epiphyses are fully fused, with the exception of the acromion, whose end would have included a great deal of cartilage at the time of death (Figure 2.32). As a similar morphology is seen on Flo, it may be that this is a result of age or shoulder use rather than delayed development. Unfused or partially fused acromion processes have been recorded in humans who vigorously use their shoulders, such as archers and oarsmen (e.g. Stirland, 2001). Similar repetitive shoulder motions may have caused this partial acromial fusion in some of the chimpanzees from Gombe, but more investigation of inter-individual variation in locomotion is necessary to determine why some chimpanzees (mostly females) exhibit this feature and others do not.

All of Pallas's epiphyses are completely fused other than the sacrum, which is more than half fused. The remains of the epiphyseal lines are still evident on the

proximal humerus and proximal radius. The complete adult dentition is in full occlusion, and exhibits moderate wear (Kilgore, 1989). Pallas does not exhibit any severe skeletal trauma, but there is evidence for severe muscle pulls or minor dislocations (Jurmain, 1989). There is no skeletal evidence for the severe attack by multiple adult males that she suffered in 1973 in which she sustained a severe gash to her mouth (Goodall, 1986). Pallas exhibits mild or moderate degenerative joint disease in most of her synovial joints.

Figure 2.32: Pallas's acromion process

Madam Bee (female - 28.2 years)

Madam Bee became part of the Kahama community when it split off from Kasekela, though her daughter Little Bee remained in the Kasekela community's range. Madam Bee was killed by males from Kasekela, and was the victim of a series of attacks towards the end of her life. She sustained a deep gash in one leg after an attack in September 1974, and survived another attack without serious wounds in February 1975.

She received a bad wound to her thigh in May 1975, and also fell from a tree while grappling with her assailants. There was at least one unobserved attack in September 1975; Madam Bee was seen with several wounds, the worst of which were on her head and shoulder. The fatal attack occurred a few days after this sighting. Figan, Satan, Jomeo, and Sherry all attacked Madam Bee, dragging, hitting, kicking, stamping on, and pounding her until she could barely move. She eventually dragged herself away and died five days later. Close to her time of death, Madam Bee was observed with severe wounds on her left ankle, right knee, left wrist, right hand, several wounds on her back, and the hallux of her right foot was almost completely detached (Goodall, 1986).

Madam Bee's skeleton lacks the long bones of her lower extremities. The acromion (see notes in section on Pallas), medial margin of the scapula, and iliac crest are not yet fully fused. The sacrum still shows obvious epiphyseal lines. The rest of the observable epiphyses are fully fused with obliterated epiphyseal lines. The complete adult dentition is in full occlusion, and shows moderate to severe wear (Kilgore, 1989).

The most dramatic of her traumatic lesions is the non-union fracture to her right ulna, which was only partially healed at the time of death (Jurmain, 1989). It is possible she sustained this injury in one of the attacks leading up to her death; the fall from the tree seems a likely candidate for the cause of this injury. A partly healed traumatic lesion to her left ischial tuberosity is also evident (Figure 2.33), and is likely a result of an attack by chimpanzees (Jurmain, 1989). The morphology of the lesion suggests it may have been a puncture from a bite wound. There are also several examples of possible perimortem traumata that are consistent with the injuries observed on her body just prior to her death. This includes an unhealed fracture to the left scaphoid, damage to the

medial margin of the left scapula, fraying / crenulation damage to two proximal manual phalanges as well as similar damage to the distal end of the left first metatarsal (Figure 2.34). This damage is consistent with wounds inflicted by biting or chewing, which has been observed in cases of infanticide (Arcadi and Wrangham, 1999, and see Chapter 3) as well as prey consumption (Pobiner *et al.*, 2007). Unhealed fractures to both proximal shafts of the ulnae and a severe fracture to the left proximal radius (Figure 2.35) may also be evidence for perimortem trauma.

The shoulder, elbow, and wrist are affected by mild degenerative joint disease, as are the vertebral articulations of 8 ribs. The right 4th metatarsal shows an abscess drainage site, possibly a sequel to trauma.

Figure 2.33: Partially healed trauma to Madam Bee's left ischial tuberosity

Figure 2.34: Perimortem trauma to Madam Bee's left first metatarsal


Figure 2.35: Unhealed fracture to Madam Bee's left radius

Vincent (male - 28.5 years)

Vincent was the alpha male of the Mitumba community until he was badly injured in a fall from a tree. He survived multiple serious traumata, and spent a larger proportion of his time alone than prior to his injury. He re-joined the community after 3 months, and was killed by the other two adult males, Rudi and Edgar, in December 2004 (M.L. Wilson, unpublished data). Vincent is known to have been SIV+ since 2002 (Keele *et al.*, 2009).

The epiphyseal lines on the medial clavicle, iliac crest, and sacrum are still visible, though the elements are completely fused. The rest of the secondary ossification centers are fused and have obliterated lines. The complete adult dentition was in full occlusion during Vincent's prime, but substantial premortem tooth loss is noted, likely related to the fall (see below).

Vincent's skeleton exhibits many examples of premortem trauma. Six ribs exhibit irregular contours and slight calluses indicating relatively well healed fractures that predate his dramatic fall. Three additional ribs exhibit less well healed fractures (Figure 2.36). Both lateral and the left central maxillary incisors are chipped on the incisal surface. The pulp cavity of the right maxillary canine is exposed. The chipped surfaces of the incisors are smooth – the damaged surface exhibits wear. The right canine is much less worn; suggesting that exposure of the pulp cavity may have been traumatic in nature rather than through normal wear, especially as the left maxillary canine is in good condition. Three abscess drainage sites associated with the right canine are evident on the maxilla (Figure 2.37). The right zygomatic arch displays a partially healed non-union fracture (Figure 2.38). The mandible exhibits the premortem loss of the left 1st molar through the right lateral incisor (Figure 2.39). The alveoli are mostly closed, though a fragment of the broken left canine remains in situ. The left mental foramen is enlarged, probably due to abscess drainage. The left canine fragment and the only moderate wear on the surviving dentition support the hypothesis that these teeth were lost due to injury rather than periodontal disease. (Though some carious lesions are recorded, see below.) The 5th sacral vertebra is deviated compared to the rest of the sacrum, and reactive bone indicates a healing fracture (Figure 2.40).

Figure 2.36: Vincent's partially healed rib

Figure 2.37: Abscess drainage sites, Vincent's right maxilla

Figure 2.38: Partially healed non-union fracture to Vincent's right zygomatic arch

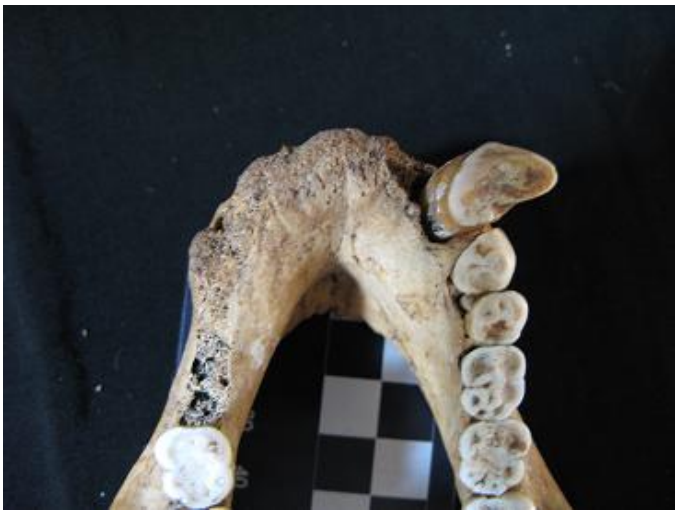


Figure 2.39: Occlusal view of Vincent's mandible with extensive premortem tooth loss

Figure 2.40: Vincent's deviated 5th sacral vertebra

The left ischium exhibits a non-union fracture separating the ischial tuberosity from the rest of the os coxa (Figure 2.41). Other partly healed fractures include the body of the 1st lumbar vertebra (Figure 2.42), the distal left fibula and tibia, and the left and right calcanei (Figure 2.43). New, waxy-looking bone laid down near the right internal acoustic meatus (Figure 2.44) may be related to cranial trauma as a result of the fall. The severity of Vincent's injuries, particularly the fracture to the ischial tuberosity, are likely to have significantly affected his mobility.

Perimortem, unhealed traumata also abound on this skeleton. Many of the wrist and ankle bones are incomplete or punctured. In particular, the left calcaneus and talus are incomplete, the left distal fibula is missing, and the left distal tibia is punctured. Several metapodials have exposed trabecular bone on the proximal articulations. Unlike in degenerative joint disease, the margins of these lesions are sharp, indicating a possible traumatic origin. The left scapular blade, right iliac crest, and the spinous process of the 4th lumbar vertebra show un-healed fractures. Two of the ribs may show perimortem step

fractures, as inferred from the direction of the break (towards the concavity of the rib), though this is difficult to separate from postmortem damage to the rib cage during necropsy to access the thoracic cavity (these fractures would most likely be towards the convexity of the rib). The left maxilla shows an un-healed puncture wound (Figure 2.45). When chimpanzees attack, the face, hands, feet of their targets are often bitten, and the attackers may jump on or strike the back of the target (targets often assume a crouching position in an attempt to protect themselves) (Goodall, 1986). Vincent's perimortem traumata are consistent with these recorded behaviors.

Figure 2.41: Partially healed non-union fracture to Vincent's left ischial tuberosity

Figure 2.42: Partly healed fracture to Vincent's 1st lumbar vertebra

Figure 2.43: Partly healed fracture to Vincent's calcaneus


Figure 2.44: Bone formation pathology associated with Vincent's internal acoustic meatus

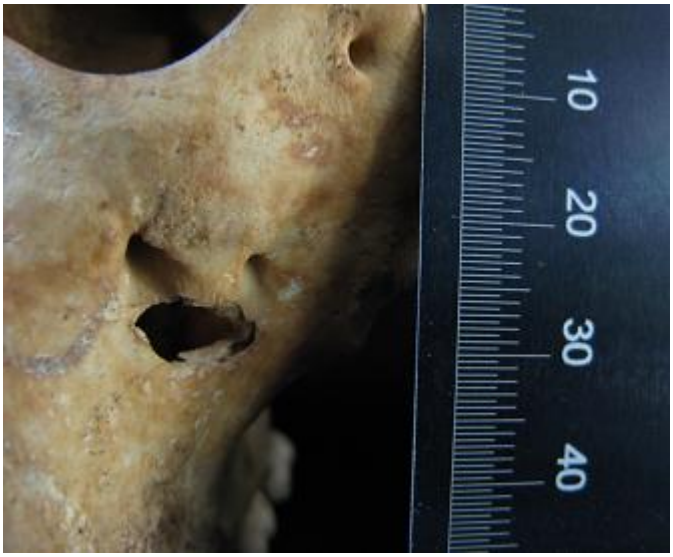


Figure 2.45: Unhealed bite wound to Vincent's left maxilla

Mild to moderate degenerative joint disease is evident on the distal phalanges of both halluces and pollices, the acromion processes, the sternal articulations of the clavicles, the distal humeri, the proximal ulnae, the scapular glenoid fossa, the acetabula, 4 metacarpals, and the vertebral articulations of 13 ribs (including 4 with partially healed fractures). Ten vertebral bodies are affected by mild osteophytic lipping.

Interproximal caries, most of which are only moderately invasive, are evident on the right maxillary 1st premolar through the left lateral incisor. The 1st premolar has more extensive involvement than the other teeth. Relative to the right temporal line, the left temporal line is deviated inferiorly on its posterior aspect. Both coronoid processes (the insertion point for the temporalis muscle) show osteophytic growths, which may indicate partial ossification of the muscle's tendon. This may be related to the mechanical alterations to mastication that likely accompanied the sudden loss of several teeth. The

right femur shows extensive remodeling of the lesser trochanter, possibly related to relying more heavily on this limb while recovering from the severe fracture to the left ischium, which, as the origin for the hamstrings, would have made use of this muscle group very difficult.

Other bone formation pathologies include periostitis on the right tibia and an intermediate manual phalanx. Two other manual phalanges show uneven palmar muscle attachments, and a third intermediate manual phalanx exhibits a callus of unknown etiology (~ 3 x 4 mm). Both bicipital grooves have bridging osteophytes indicating partial ossification of the transverse humeral ligament. Lastly, one metacarpal exhibits both bone formation and bone loss related to the same pathology: a pus drainage site approximately 3mm in diameter accompanied by surrounding remodeled bone.

Winkle (cranium only – identity tentative) (female - 28.9 years)

Recovered from the Kasekela community's range in 1988, this cranium's discovery coincided with the disappearance of adult female Winkle (GSRC, unpublished data). Only the neurocranium remains; most of the facial bones were broken post mortem, though portions of both orbits survive. The right orbit shows evidence of postmortem rodent gnawing on its medial aspect.

Both parietals and the occipital show multiple bone loss pathologies in the form of small, regular divots with smooth margins (Figure 2.46). Their morphology is consistent with one or more well-healed cranial traumata such as bite wounds or depression fractures (Carter *et al.*, 2008). Remodeling is extensive, making more detailed diagnosis difficult. Radiographic analysis may be helpful in the future.

Figure 2.46: Possible healed cranial trauma in Winkle

Satan (male - 29.8 years)

A large male from the Kasekela community, Satan was less successful in climbing the dominance hierarchy than his impressive size might suggest (Goodall, 1986). He is assumed to have died in 1987 of a respiratory infection (Williams *et al.*, 2008). Satan was one of Sprout's offspring, a matriline at Gombe that has now died out (GSRC, unpublished data). Only a portion of his skeleton was found subsequent to his disappearance: the mandible, ossa coxae, both humeri, and the left ulna, whose distal end is missing due to post mortem damage. The obseravable joint surfaces are affected by very mild degenerative joint disease.

Tumaini (male - 30 years)

Tumaini, an adult male from the Kalande community, was poached in 2002, and his body was found partially burned and with multiple machete wounds (GSRC, unpublished data). Unhealed skeletal traumata reflect his cause of death. Most of the axial skeleton (ribs and vertebrae), many hand and foot bones, and the left lower extremity are not preserved, and are likely related to the manner of his death (and subsequent partial recovery of possibly scattered remains).

Most secondary ossificaition centers are fully fused. The iliac crest is still unfused, and several epiphyses still have a visible line on the fused epiphysis: the medial scapular margin, proximal humerus, proximal radius, distal radius, distal ulna, femoral head, proximal tibia, and sacrum. The complete adult dentition is in full occlusion, with no premortem tooth loss.

Perimortem traumata include spiral fractures to both clavicles and damage to the scapular blades and right ilium (Figure 2.47). The 3 preserved rib fragments also exhibit unhealed spiral fractures. Several of the anterior teeth are calcined (Figure 2.48).

Premortem traumata include a pedal proximal phalanx with a missing distal end (Figure 2.49). Reactive bone indicates that some remodeling occurred between the time of this injury and death, even though the margins of the break are still relatively sharp.

Three metapodials are in the process of healing and exhibit large, mid-shaft calluses that dramatically alter the normal contour of the bone (Figure 2.50). These injuries may have limited Tumaini's mobility to some extent when they were less well healed. A manual proximal phalanx is similarly affected, though its degree of healing is not as extensive (Figure 2.51).

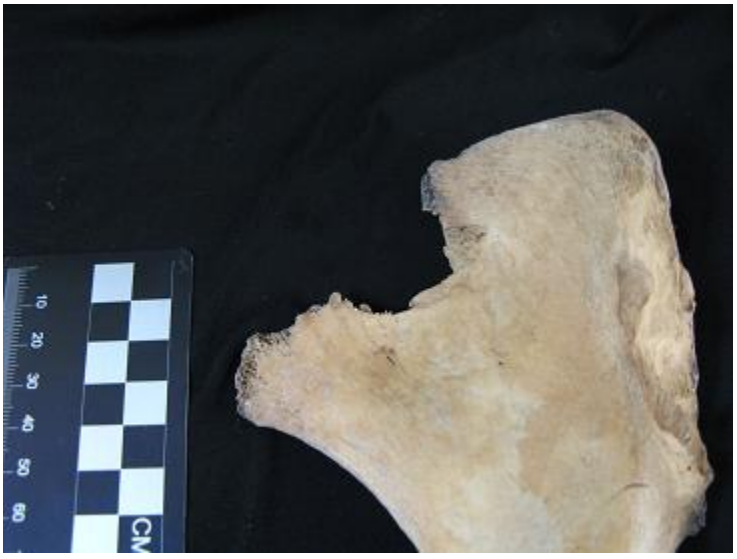


Figure 2.47: Perimortem trauma to Tumaini's right ilium

Figure 2.48: Example of Tumaini's calcined incisor

Figure 2.49: Pedal proximal phalanx missing the distal end – premortem trauma in Tumaini

Figure 2.50: Tumaini's metacarpal with healed fracture, lateral view

Figure 2.51: Partly healed proximal manual phalanx from Tumaini, palmar view

The elbow, wrist, ankle, hip, and knee demonstrate mild degenerative joint disease in the form of increased porosity of articular surfaces. The 1st sacral vertebra exhibits mild osteophytic lipping. The only other surviving vertebra, the 4th lumbar, does

not. Bone loss pathologies include notches on the distal aspect of both maxillary canines (Figure 2.52), likely resulting from wear rather than carious lesions due to their morphology and the notches' disassociation with other tooth surfaces. The left maxilla has an abscess drainage site related to the canine.

The left maxilla also has a bone formation site that may be an osteoma (Figure 2.53), though its morphology is not the usual "button" shape associated with this pathology. The maxilla is also very asymmetrical, with the left side more bulbous than the right. Whether the asymmetry is related to the possible osteoma, the abscess, or healed trauma is unclear; radiographic analysis in future may be informative.

Alveolar erosion is evident near the mandibular left 1st premolar as well as the maxillary canines, and the maxillary left 2nd premolar is chipped. Erosive lesions are present on both mandibular condyles (Figure 2.54); the mandibular fossae are not involved.

The left temporal line is positioned more laterally in its posterior aspect than the right (Figure 2.55). Periostitis is evident on the right tibia and an intermediate manual phalanx. Proliferative lesions on a metatarsal and the right ulna are also likely to have resulted from periosteal response. Two manual proximal phalanges have asymmetrical palmar muscle attachments, and one toe has fused intermediate and distal phalanges, possibly as a result of trauma.

Figure 2.52: Distal notch on Tumaini's maxillary canine

Figure 2.53: Tumaini's left maxilla with bone formation pathology

Figure 2.54: Erosive lesion on Tumaini's mandibular condyles


Figure 2.55: Tumaini's uneven temporal lines

Passion (female - 30.6 years)

Passion, a high-ranking female from Kasekela, is most famous for killing and eating several young infants in her own community. With her daughter Pom's assistance, Passion may have killed up to 10 infants between 1974 and 1977. Three of these infanticides were observed (Goodall, 1986). Passion died in 1986 of a "wasting disease" (Williams *et al.*, 2008).

Her skeleton is mostly complete. All of the epiphyses are fused except for the medial margin of the scapula. The lines of the fused epiphyses are still evident on the iliac crest and distal fibula. The complete adult dentition is in full occlusion, and displays moderate wear. Loss of alveolar bone indicating periodontal disease is evident in the posterior dentition (Kilgore, 1989). The maxillary right 1^{st} molar was chipped on the buccal aspect prior to death as indicated by the wear on the break surface.

Uneven muscle attachments and reactive bone near attachment sites on 3 manual phalanges are indicative of severe muscle pulls or strains. A traumatic dislocation of both 13^{th} ribs is indicated by the remodeling of both rib heads and the corresponding vertebra (Figure 2.56) (Jurmain, 1989). Several major synovial joints are affected by very mild degenerative joint disease, and moderate progression is noted on the right capitate, both acetabula, both proximal radii, the right distal radius, and the left proximal ulna. Passion also has a patent olecranon foramen on her right humerus.

Figure 2.56: Passion, rib dislocation injury resulting in remodeling of costal tubercle

Jomeo (male - 30.9 years)

A male of impressive size, Jomeo lost interest in acquiring high dominance rank during adolescence, an unusual occurrence for a male chimpanzee. Jomeo was noted to have sustained severe wounds at approximately 9 years of age, and it may be that whatever experience yielded these wounds resulted in this lack of interest (Goodall, 1986). Satan is often credited with being the largest male chimpanzee observed at Gombe, and data on body mass collected during their lives indicate Satan and Jomeo were similar in size (Pusey *et al.*, 2005), but Jomeo's skeleton is markedly larger; the length of Jomeo's right os coxa is 20mm greater than Satan's.

Jomeo's skeleton is complete, and the epiphyses are all fully fused. The epiphyseal line is still visible on the medial clavicle. The 4th lumbar vertebra has fused to the sacrum. The complete adult dentition is in full occlusion, and exhibits moderate wear.

The upper left canine was damaged some time during life (Figure 2.57), and an abscess was observed to swell and finally burst, though swelling persisted for 3 more weeks after the abscess began draining (Goodall, 1986). Goodall (1986) speculates that this abscess affected an upper molar, but the extensive damage to the canine indicates that this was, in fact, the tooth affected. Little wear on the broken edges of the tooth indicates the possibility that this occurred not very long before death, but the lack of an abscess drainage site associated with this severely damaged tooth makes this less likely. Premortem loss of the mandibular premolars on the left side may be associated with this injury. Though the alveoli for these teeth are well healed, the enlarged left mental foramen, a common pus drainage site for the mandibular dentition, supports the traumatic tooth loss hypothesis.

A left rib, distal phalanx, and the distal shaft of the right ulna (Figure 2.58) all show calluses indicative of well healed fractures. Moderate degenerative joint disease affects both elbows (Figure 2.59), a possible sequel to the ulnar injury. Osteophytes on the anterior surface of the left patella indicate partial ossification of the quadriceps tendon in this region, possibly due to a severe muscle pull or strain. The transverse processes on the 1st lumbar vertebra are incompletely fused (Figure 2.60).

Figure 2.57: Jomeo's damaged left maxillary canine


Figure 2.58: Healed fracture, Jomeo's distal right ulna

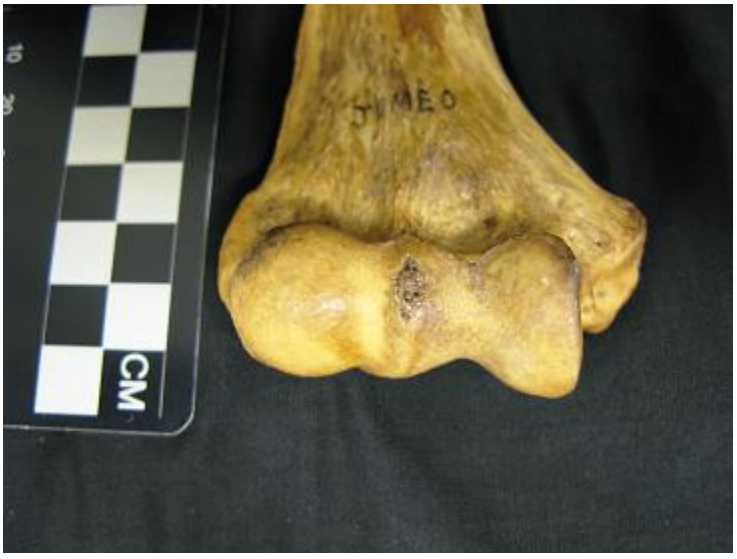


Figure 2.59: Moderate degenerative joint disease on Jomeo's left distal humerus (ulna also affected)

Figure 2.60: Incompletely fused transverse process on Jomeo's 1st lumbar vertebra

Miff (female - 30.9 years)

Miff was one of Marina's offspring, and the only one to survive into later adulthood. Miff gave birth to 5 offspring, but only one has survived to adulthood. Moeza, born in 1969, emigrated to the Mitumba community in 1983. Mel and Michaelmas both died as adolescents, and are part of the Gombe skeletal collection (Goodall, 1986; GSRC, unpublished data). Miff died in 1987 of a respiratory infection (Williams *et al.*, 2008).

Her skeleton is complete, and most of the epiphyses are fully fused. An epiphyseal line is still evident on the medial clavicle and proximal humerus. The iliac crest and sacrum are more than half fused. The acromion process remains unfused, but see the section on Pallas for comments on this phenomenon. The complete adult dentition is in full occlusion and exhibits mild to moderate wear. Several teeth were lost prior to death: the upper left 1st premolar, all of the upper left molars, and the lower left canine (Figure 2.61). The right mandibular molars are more worn than the left, likely due to the fact that the upper left molars were missing.

The left deltoid tuberosity exhibits severe remodeling and extra bony growth, indicating likely trauma to the deltoid muscle (Figure 2.62). It is not clear whether this injury is a result of the attack on Miff by multiple males in 1977. During this attack, her son, Michaelmas, was seized and displayed with (Goodall, 1986), resulting in a hip dislocation injury (Jurmain, 1989).

Two manual phalanges exhibit calluses consistent with well-healed fractures. Mild degenerative joint disease affects many of the joints of the hands and feet as well as

the vertebral articulations of 8 ribs. Both elbows, one manual proximal phalanx, and both mandibular fossae (Figure 2.63) are affected by moderate degenerative joint disease.

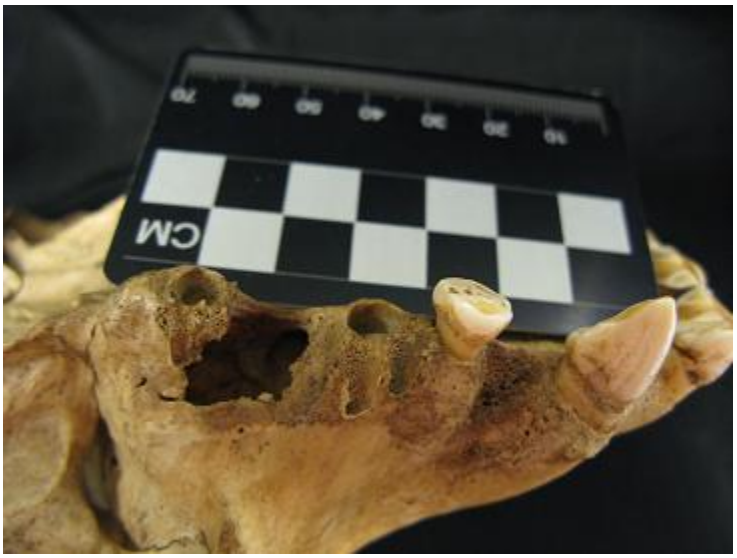


Figure 2.61: Miff's maxillary teeth, showing extensive premortem tooth loss

Figure 2.62: Healed injury to Miff's left detoid tuberosity

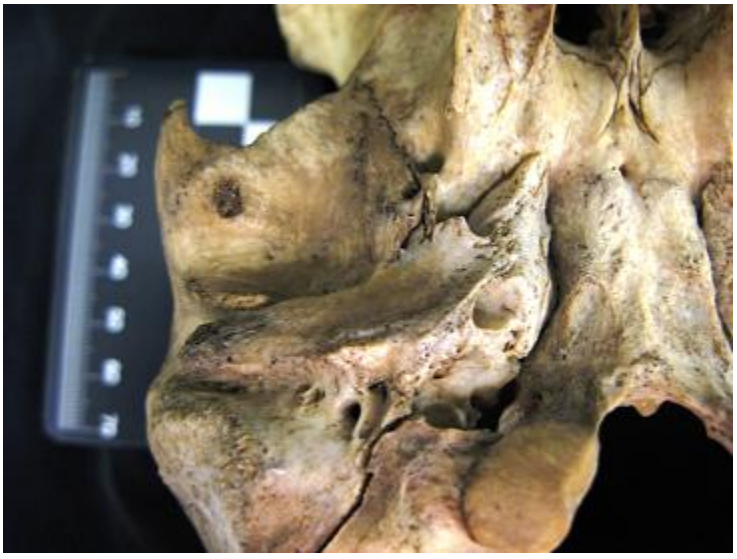


Figure 2.63: Moderate degenerative joint disease on Miff's mandibular fossa

Atlas (male - 31.3 years)

Atlas (born in 1967) is one of Athena's offspring. His half brother Apollo (born in 1979) still lives in the Kasekela community, and his half sister sister Aphro (born in 1973) is a member of the Mitumba community (GSRC, unpublished data). Atlas's death in 1999 is attributed to a "wasting disease" that is a relatively common cause of death for chimpanzees at Gombe, and is likely related to intestinal parasites in this case (Williams *et al.*, 2008).

His skeleton is complete and well preserved, with most secondary ossification centers fully fused. The medial clavicle remains unfused, and the iliac crest and sacrum are more than half fused. Fully fused epiphyses that still show a clear epiphyseal line include the proximal humerus, proximal radius, and proximal ulna. The complete adult dentition is in full occlusion.

Observed traumata include a chipped maxillary right lateral incisor, and 3 ribs with abnormal contours suggesting well healed fractures. Nearly all of the joints exhibit degenerative joint disease in various stages. Most examples are mild, but moderately affected joints include the shoulder, hip, and digits. Three metapodials show severe degeneration of a joint surface (Figure 2.64). Two cervical and 1 thoracic vertebra exhibit mild osteophytic lipping. The left 4th metatarsal and its associated proximal phalanx do not retain their bony articulation; the articular ends of these bones are severely eroded (Figure 2.65). The extensive damage suggests this lesion may have a traumatic origin; bites to the feet that sever or nearly sever toes have been previously observed for chimpanzees at Gombe (e.g. Leakey and Madam Bee: Goodall, 1986).

Bone loss pathologies include notching of the acromion processes (see Pallas for more details), and lesions on the articular surfaces of 7 carpals. Alveolar erosion is associated with the maxillary canines and interproximal caries are evident between the upper right 1st pre-morlar and canine, and the upper left canine and 2nd premolar. The upper left 1st premolar was lost premortem (Figure 2.66).

Figure 2.64: Severe degenerative joint disease on two of Atlas's metacarpals

Figure 2.65: Severely remodeled distal articulation of Atlas's left 4th metarsal


Figure 2.66: Atlas's maxillary teeth, left lateral view

Two proximal and one intermediate manual phalanges have asymmetrical palmar muscle attachment sites, and two manual intermediate phalanges show proliferative bone consistent with periostitis. The first cervical vertebra has asymmetrical transverse foramina (Figure 2.67).

Figure 2.67: Atlas's atlas, with asymmetrical transverse foramina

Rix (male - 33.4 years)

In 1968, Rix fell approximately 100 feet from a tree and was killed instantly (Williams *et al.*, 2008). Rix is currently an articulated skeleton housed at the University of Dar es Salaam, and the curation of his skeleton makes certain pathological assessments impossible. Rix is excluded from any of the analyses on degenerative joint disease (including vertebral degeneration), as these surfaces were unobservable without damaging the skeleton's mountings. As a valuable part of the University's comparative collection, the skeleton was observed in its articulated state.

The 3rd or 4th thoracic vertebra is largely reconstructed for the purposes of articulation, but whether damage to this vertebra is due to perimortem trauma or postmortem damage is difficult to determine. His skeleton shows no other signs of perimortem trauma.

Moderately severe inter-proximal caries are evident on the maxillary incisors, between the upper left 2nd premolar and 2nd molar, and between the lower left 1st and 2nd

molars. The root of the upper right central incisor is exposed, though it is unclear whether this is postmortem damage or true dental pathology.

A smooth-edged hole in the left infraspinous fossa measuring approximately 15mm in length was observed (Figure 2.68). No reactive bone around the hole was observed, but the thickness of the scapular body as well as the lack of cracking or other damage around the hole makes postmortem damage unlikely in this case.

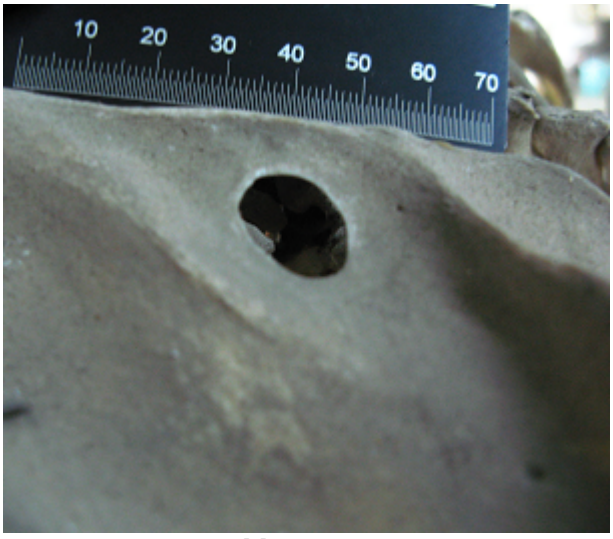


Figure 2.68: Possible premortem trauma to Rix's scapula

Beethoven (cranium only – identity tentative) (male - 33.5 years)

The recovery of this cranium from the Kasekela community's range coincided with the disappearance of adult male Beethoven in 2002 (GSRC, unpublished data; Williams *et al.*, 2008). The cranium shows few signs of trauma and pathology. Only the 3rd molars remain in situ, though the remaining alveoli are consistent with the adult

dentition being in full occlusion. Resorbtion of the alveoli for the left central incisor and left 1st molar indicate premortem loss of these teeth (Figure 2.69).

Figure 2.69: Beethoven's maxillary teeth

Echo (female - 34 years)

Echo emigrated to the Kasekela community from the southern group at Kalande in 2004, and has two surviving offspring. Unused to human observation during her early life, she was not observed frequently (GRSC, unpublished data). Echo was SIV+ from at least 2003, when her fecal samples were first tested (Keele *et al.*, 2009). In 2006, Echo died of complications from a massive spinal cord injury (T5) that will be described elsewhere (K. Terio, unpublished data). A section of the thoracic vertebral column was removed *en bloc* during necropsy for analysis and as such is not included in the skeletal

analysis (it is a preserved soft tissue specimen). Echo was unable to use her lower extremities at all during the last period of her life and dragged herself around using only her forelimbs. Nutritional deficit may have been a contributing cause of death, and constant abrasion with the forest substrate may have contributed to the development of abdominal abscesses noted before her death (*Ibid.*).

Most of the epiphyses are fully fused, though the medial clavicle is less than half fused and the medial scapular margin as well as the iliac crest still show prominent epiphyseal lines. The complete adult dentition is in full occlusion.

Several traumata in addition to the spinal column injury are evident. These include a well-healed fracture to a manual proximal phalanx, and osteophytic growth associated with palmar muscle attachment sites on two metapodials and a pedal proximal phalanx, possibly associated with trauma to these muscles. Eight of the ribs show an abnormal contour, suggesting possible well-healed fractures, though no bone callus is evident. Future radiographic assessment would likely help with diagnosis.

Most of the major synovial joints as well as many hand and foot bones display mild to moderate degenerative joint disease, primarily in the form of increased porosity of the articular surfaces. Some areas show concentrations of more porous articular bone; these would have likely developed into more severe articular lesions if Echo had survived to older adulthood. Ten of 17 observable vertebral bodies exhibit mild osteophytic lipping.

The left and right tibiae as well as an intermediate manual phalanx show signs of reactive bone on the periosteal surface (periostitis), a generalized sign of infection. The left talus has smooth, osteophyte-like growths unassociated with the articular surface

(Figure 2.70). The bicipital groove of the left humerus has "bridging" osteophytes indicative of partial ossification of the transverse humeral ligament (Figure 2.71). The right clavicle has osteophytes on its acromial articulation, and the superior angle of the right scapula also exhibits bone growth that is osteophytic in appearance (Figure 2.72). The right olecranon process displays an area of proliferative bone, though this lesion is diffuse. These particular bone formation pathologies may be related to the fore-limb-reliant locomotion upon which Echo was forced to depend after her spinal cord injury.

The most dramatic of the proliferative lesions occur on the pubic bones, and are likely to have been associated with the abdominal abscesses mentioned earlier. The right pubis in particular shows severe bone involvement, the lesion being more than 2 cm in diameter with an extensive drainage site (Figure 2.73).

Congenital variations include unusually long transverse processes on the 1st lumbar vertebra (Figure 2.74), and laminar deficiency in the 1st through 5th sacral vertebrae (Figure 2.75).

Figure 2.70: Echo's left talus with bone growth pathology

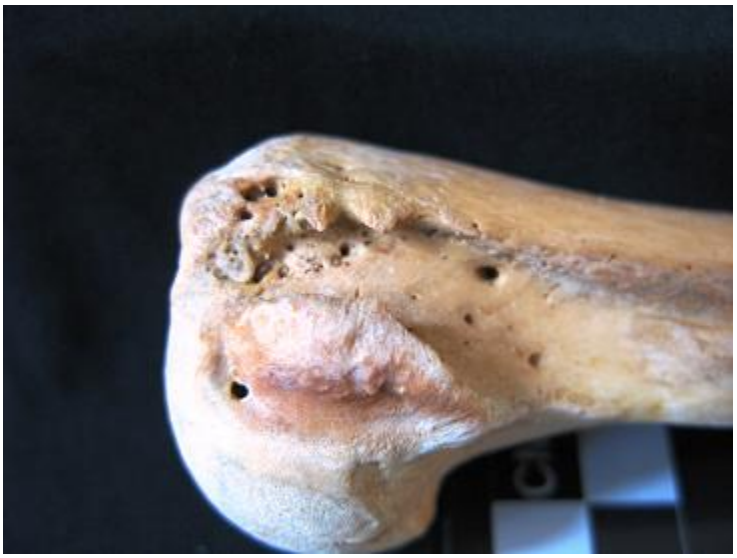


Figure 2.71: Partial ossification of Echo's transverse humeral ligament

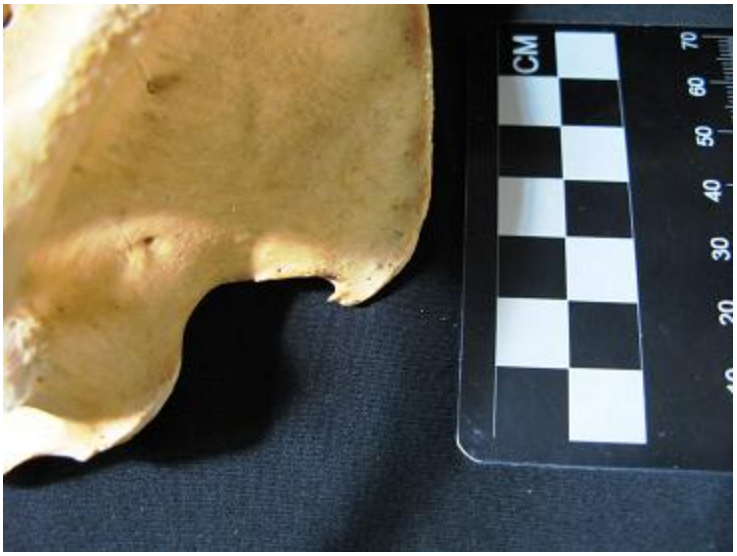


Figure 2.72: Osteophytic growth on the superior angle of Echo's right scapula

Figure 2.73: Lesion on Echo's right pubis, likely associated with abdominal abscesses

Figure 2.74: Unusually long transverse processes in Echo's 1st lumbar vertebra

Figure 2.75: Laminar deficiency in Echo's sacrum

Humphrey (male - 34.9 years)

Humphrey was already an adult male when research at Gombe began in 1960. He is thought to be related to Mr. McGregor and to Melissa; the close social bond between Humphrey and McGregor as well as their physical resemblance suggests that they were brothers. A very aggressive alpha male when in his prime (Goodall, 1986), Humphrey died in 1981 of unknown causes (Williams *et al.*, 2008). Only the cranium is preserved. Most of the dentition is moderately worn, though the canines are severely worn, with the pulp cavities exposed. There are associated abscess drainage sites with both canines (Figure 2.76) as well as the right central incisor (Kilgore, 1989). The abscess Humphrey developed in his right ear in 1964 (Goodall, 1986) has left no gross morphological sign on the skeleton.

Figure 2.76: Abscess drainage site near Humphrey's maxillary canine

Melissa (female - 36.3 years)

A high ranking Kasekela female, Melissa gave birth to many offspring, and though only two of them reached adulthood, both Goblin and Gremlin had relatively high reproductive success and rank. Gremlin (born in 1970) is still a member of the Kasekela community, and Goblin is part of the Gombe skeletal collection. Melissa gave birth to twins in 1977, Gimble and Gyre, neither of whom lived past their first year. Gremlin and Gaia, Melissa's granddaughter, have also given birth to twins, though only Gremlin's twins (Golden and Glitter, born in 1998) have survived (GSRC, unpublished data).

Melissa died in 1986 of a "wasting disease" (Williams *et al.*, 2008). She was affected by the polio epidemic, and though she regained the use of her arms, her neck remained crooked on the left side for the rest of her life (Goodall, 1986).

Her skeleton is complete, with fully fused epiphyses. The sole exception is the left acromion process, which is still patent though more than half fused (Figure 2.77). (See notes on this phenomenon in the section about Pallas.) An epiphyseal line is still visible on her proximal humerus, proximal radius, femoral head, and proximal fibula. The complete adult dentition is in full occlusion, and is moderately worn.

The left 1st metacarpal has an angular deformity consistent with a well-healed fracture. Another partially-healed fracture is evident on an intermediate manual phalanx. Uneven plantar muscle attachments on a proximal pedal phalanx suggest a muscle injury resulting in bony remodeling. The hips, knees, shoulders, hands, and feet all show early signs of degenerative joint disease. Three ribs, the left calcaneus, and both proximal ulnae are moderately involved. Reactive bone on the right transverse processes of the 4th

lumbar and 1st sacral vertebrae indicate that these elements were beginning to fuse (Figure 2.78).

Figure 2.77: Melissa's left acromion process is incompletely fused

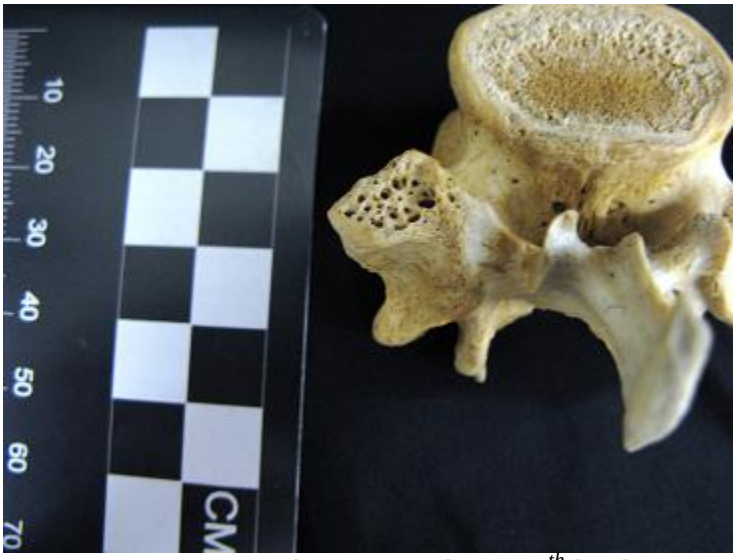


Figure 2.78: Reactive bone on Melissa's 4th lumbar vertebra (inferior view shown) is a likely indicator that it was beginning to fuse to S1

Hugo (male - 38.6 years)

A high ranking male known for his "leadership abilities" (Goodall, 1986: 69), Hugo died in 1975 of a respiratory infection (Williams *et al.*, 2008; *ibid*.), in the same month as his close companion Mike. Hugo was noticeably emaciated towards the end of his life (Goodall, 1986). His skeleton is mostly complete: neither femur is preserved. The epiphyses are all fully fused, and epiphyseal lines are visible only on the distal ulna and distal fibula.

All of his adult dentition is presumed to have been in occlusion at one point, but the mandibular posterior teeth were all lost premortem. Only the roots of the canines remain, and the maxillay canines and right mandibular canine each have an associated abscess drainage site. Most of the maxillary posterior dentition is moderately worn (Kilgore, 1989). The maxillary first premolars are likely to have been lost shortly before death; the alveoli are patent but reactive bone is evident.

Hugo has at least 8 healed fractures including one to his right zygomatic arch, made evident by the altered countour of this feature (Figure 2.79) and the compression fracture to his right calcaneus that was likely the result of a fall (Jurmain, 1989). Most of the synovial joints are affected by degenerative joint disease. The right calcaneus, elbows, ankles, and left mandibular condyle are moderately affected. The distal end of 2 metacarpals, the proximal end of one metatarsal, and the left mandibular fossa (Figure 2.80) are severely affected. One manual intermediate phalanx has uneven muscle attachments suggestive of injury to the associated soft tissue, and 6 intermediate manual phalanges have reactive bone consistent with mild periostitis. Both olecranon foramina are patent.

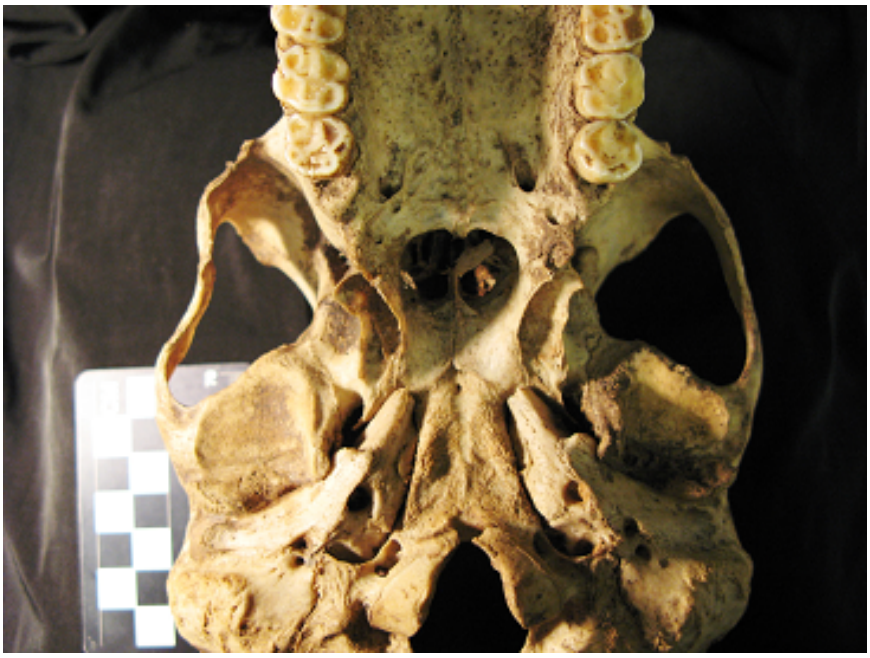


Figure 2.79: Altered contour of Hugo's right zygomatic arch

Figure 2.80: Severe degenerative joint disease on Hugo's left mandibular fossa

Cusano (male - 39.9 years)

Former alpha male of the Mitumba community, Cusano died in June 1996 at nearly 40 years of age, and his skeleton was not exhumed until August 2005 (GSRC, unpublished data). Postmortem damage to the skeleton is extensive. All of the bones have extremely friable surfaces and elements of the axial skeleton (ribs and vertebrae) are mostly fragmentary. Both scapulae are fragmentary, and only the right radius survives. Both femora survived, though the right is fragmentary, and only the left tibia is present. No humeri, ulnae, or fibulae were preserved. The cranium and mandible are some of the better preserved elements, though the mandible exhibits a post-burial break near the symphysis.

The adult dentition is in full occlusion, with no premortem tooth loss, and all observable epiphyses are fused.

The left zygomatic arch is of irregular contour (Figure 2.81) and the temporal-zygomatic suture is obliterated, suggesting a healed fracture. It is similar to Hugo's zygomatic injury. The left temporal glenoid fossa also shows signs of degeneration, whereas the right is well preserved and not pathological. This supports the hypothesis that some trauma occurred to the left side of the face during life. Possible tooth breakage may also be associated with this injury. The maxillary teeth with possible premortem breakage are: the right canine, right lateral incisor, left central incisor, left canine, and left 1st premolar. The left mandibular canine may have also been broken premortem. The described damage to these teeth may be due to wear rather than breakage, but 3 of the 4 maxillary incisors are affected while the right central incisor is not, which supports the tooth breakage hypothesis in this case. Other supporting evidence for tooth breakage

comes in the form of the 5 maxillary abscesses ranging in size from approximately 1.5-3.7 cm in diameter and the mandibular abscess associated with the left canine.

Interproximal caries are observed on the upper right 1st premolar, the upper right central incisor, the lower left 1st molar, and all of the mandibular incisors.

Some joint surfaces were well enough preserved to evaluate degenerative joint disease. The let hip, knee, and ankle show mild degenerative joint disease, including increased porosity of the joint surface and some localized porotic lesions. Four metapodials and the right calcaneus also show signs of mild degenerative joint disease. One cervical vertebra exhibits mild osteophytic lipping of the body, and the 4th lumbar vertebra is completely fused to the 1st sacral vertebra, though no osteophytic lipping is evident on either element and the disc space between the two is still patent.

Figure 2.81: Healed trauma to Cusano's left zygomatic arch

Goblin (male - 40 years)

Goblin is one of Melissa's offspring. Tenacious in his quest for dominance rank, Goblin eventually achieved alpha status in the Kasekela community. Goblin was severely attacked by other chimpanzees twice. The first time was in November 1979, shortly after his initial rise to the alpha position. He sustained a severe laceration to his groin during a gang attack that occurred in association with a hunting episode. The wound bled for an hour. This injury restricted his mobility, and, starting one week after the injury, leaked pus for 2 weeks. Goblin's total recovery time from this injury was 5 weeks, after which he re-asserted himself in the dominance hierarchy and reclaimed the alpha position (Goodall, 1986). Goblin was alpha male for approximately ten years before being severely injured by Wilkie, the number two male, in September 1989. During their fight, both males fell out of a tree. Goblin bit Wilkie on the face, but in turn received bite wounds on his right wrist, left foot, several fingers and toes, and scrotum. The scrotal injuries were the most severe, and became infected. It is likely that Goblin's life was saved by human veterinary intervention in this case (Goodall, 1992). Goblin finally succumbed to intestinal parasites many years later (Williams *et al.*, 2008). The skeleton is complete and well preserved, exhibiting several traumata and pathologies that are of interest.

All secondary ossification centers and epiphyses are fully fused, with no lingering observable epiphyseal lines. The complete adult dentition was in full occlusion during Goblin's prime, though several dental pathologies of interest are discussed below.

Goblin exhibits relatively little evidence for healed trauma, consistent with the fact that his known major injuries primarily affected soft tissues. The 1st lumbar vertebra

is missing the left transverse process, and the reactive bone at this site suggests this is a traumatic loss rather than a congenital variation. Well healed fractures with relatively smooth calluses are evident on the left 5th metatarsal, 8 ribs (4 pairs), an intermediate manual phalanx, and an intermediate pedal phalanx.

Nearly all the joints are involved in degenerative joint disease, ranging in severity from mild to advanced. The knees, ankles, hips, temporal-mandibular joints (temporal bone and mandible), 3 pedal phalanges and 2 sesamoids are moderately affected, while 3 pedal phalanges are severely affected. All of the cervical vertebral bodies show mild to moderate osteophytic lipping, as do 9 of 13 thoracic vertebrae, and 3 lumbar vertebrae. The 4th lumbar and 1st sacral vertebrae are fused.

The teeth are well worn, with dentin exposure over the most of the occlusal surface (Figure 2.82). The upper left 1st premolar was lost premortem. Interproximal caries are evident on the anterior maxillary dentition as well as the right 1st premolar, the mandibular left premolars, left canine, left incisors, right lateral incisor, and right premolars. The mental foramina are both enlarged and show osteophytic growth within (Figure 2.83). The maxillary canines and central incisors also have associated abscess drainage points (Figure 2.84). The anterior maxillary dentition has advanced pulp cavity exposure, especially for the left canine, whose alveolus is so eroded the root was only held in place by soft tissue during life. The crown of the left maxillary canine is entirely gone (Figure 2.85); damage to this canine was noted when Goblin was anesthetized in 1989 (GSRC, unpublished data). Pulp cavity exposure is much more limited on the anterior mandibular dentition, involving only a small exposure at the tip of the left

canine. This suggests the possibility that the maxillary dentition may have been broken rather than just worn.

Osteophytic growths partially bridging the bicipital groove indicate partial ossification of the transverse humeral ligament. Lastly, some unidentified bony fragments were recovered with Goblin's skeleton and are likely the abnormal ossification of soft tissue (Figure 2.86).

Figure 2.82: Goblin's maxillary(left) and mandibular teeth

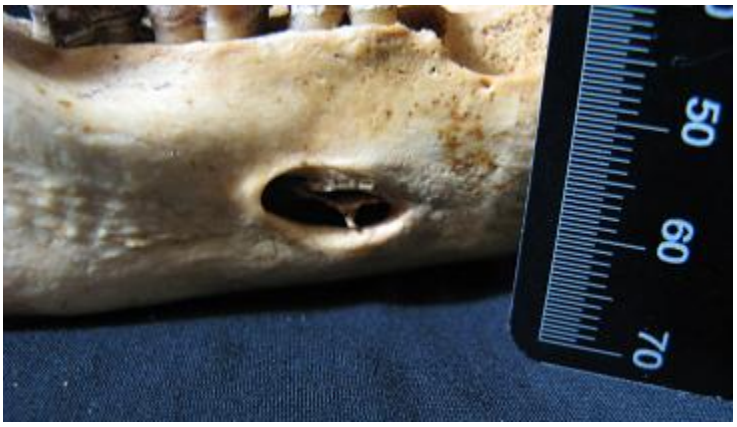


Figure 2.83: Goblin's right mental foramen

Figure 2.84: Abscess associated with Goblin's maxillary incisors

Figure 2.85: Goblin's left maxillary canine

Figure 2.86: Unprovenanced bony fragments recovered with Goblin's skeleton, likely ossified soft tissue

Patti (female - 44.3 years)

A high ranking female from Kasekela, Patti has 3 surviving offspring, including her son Titan (born in 1994), now eagerly climbing the male dominance hierarchy (GSRC, unpublished data). Patti was killed by male chimpanzees from the Kalande community in 2005 (Williams *et al.*, 2008) while on a consortship with Frodo (GSRC, unpublished data), Titan's father (Constable *et al.*, 2001). Patti's skeleton is complete and well preserved. Secondary ossification centers are all fused. The fully fused medial margin of the scapula still has a visible epiphyseal line. The complete adult dentition is in full occlusion.

Few examples of trauma are observable on Patti's skeleton. A puncture in the right talus (Figure 2.87) is the only example of perimortem trauma. Impression at necropsy was that extensive soft tissue trauma had occurred (K. Terio, unpublished data). Well healed fractures on 2 ribs and a manual intermediate phalanx are evident. The maxillary right 2nd premolar is chipped.

Most joints are affected by degenerative joint disease, and only mild progression is evident in most cases. Seven total vertebrae are mildly affected by osteophytic lipping. Moderate severity is observable on the distal femora (Figure 2.88), proximal and distal ulnae, 2 manual intermediate phalanges and 3 metatarsals.

The dentition is overall in excellent condition considering Patti's age, with only moderate occlusal wear. The upper left 1st molar was lost premortem (Figure 2.89). The mandible exhibits moderate periodontal disease; both mental foramina are enlarged and alveolar erosion is evident in association with the posterior dentition on the left side.

Both ulnae and one rib exhibit mild periostitis. Another rib has a smooth-looking callus on its anterior aspect, probably not associated with a fracture, as the rest of the rib's contour is regular (Figure 2.90).

Figure 2.87: Perimortem puncture wound to Patti's right talus

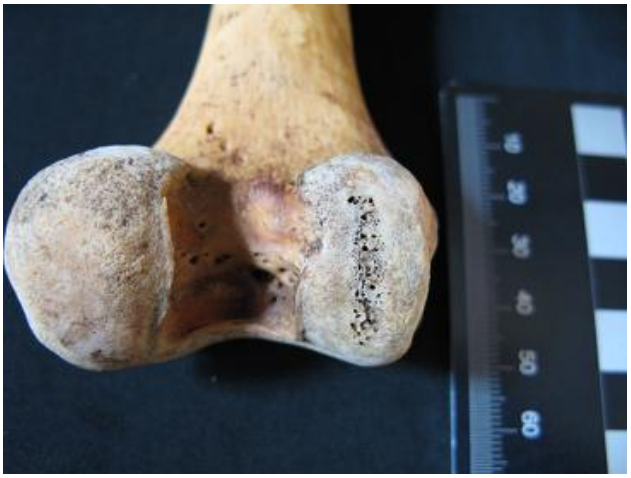


Figure 2.88: Moderate degenerative joint disease on Patti's distal femur

Firue 2.89: Patti's maxillary teeth

Figure 2.90: Callus on anterior aspect of Patti's rib

Bwavi male (unknown) (45 years?)

This unknown male from the Kalande community wandered into the Kasekela range towards the end of his life in 1994 or 1995. Field observers remember this particular chimpanzee (though not the exact year his case was noticed). He was clearly ill, did not eat, and died soon after first being observed (GSRC, unpublished data). His teeth (Figure 2.91) are not as worn as Goblin's, but are more worn than Patti's and his skeleton displays a remarkable number of pathologies, particularly severe degenerative joint disease.

All secondary ossification centers are fully fused, though epiphyseal lines are still visible on the sacrum. The complete adult dentition is in full occlusion and no premortem tooth loss is evident.

All of the joint surfaces in the skeleton, as well as the vertebral bodies, are severely affected by degenerative joint disease. Significant remodeling of articular surfaces is common, as well as extensive pitting, eburnation, and osteophytic growth at the margins of articular surfaces (Figure 2.92). It is likely that this condition limited his mobility. Both femora exhibit osteophytic growth on the trochanters, as does the left ischial tuberosity. In addition, 6 ribs exhibit well healed fractures, as do both 5th metatarsals. A partially-healed, non-union fracture of the right coracoid process is evident (Figure 2.93). The left 4th and 5th metacarpals are fused together proximally, possibly as a result of healing trauma (Figure 2.94), as are an intermediate and a distal pedal phalanges.

The mandibular 1st molars are extremely worn, with most of the crown eliminated. The roots alone remain on the buccal aspect. Alveolar resoprtion is associated with both of these teeth (Figure 2.95).

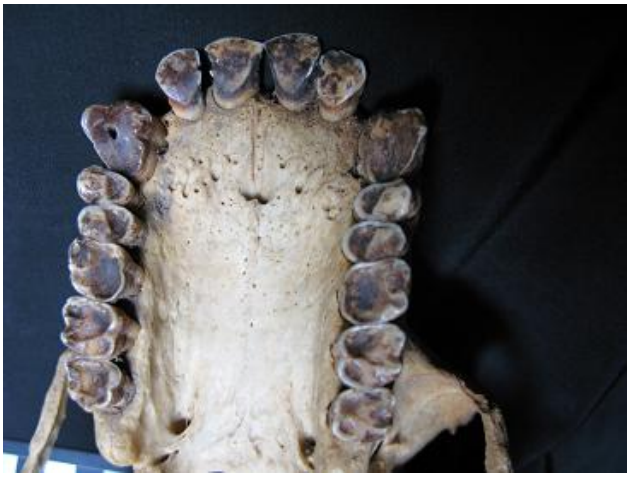


Figure 2.91: Bwavi male's maxillary teeth

Figure 2.92: An example of extensive, severe degenerative joint disease: Bwavi male's proximal humerus

Figure 2.93: Non-union fracture of Bwavi male's right coracoid process

Figure 2.94: Bwavi male's left 4th and 5th metacarpals are fused, likely a sequel to trauma

Figure 2.95: Severe wear on Bwavi male's mandibular 1st molar

Old Female (50 years plus)

The body of an elderly female chimpanzee was discovered in August 1973 by Y. Selemani (Richard Wrangham, personal communication; Goodall, 1986). She had probably been dead at least one day. Community membership is attributed to the unhabituated Kalande community as she was not an identified chimpanzee (Wrangham *et al.*, 2006). Wounds on her body and her body's position are consistent with intra-specific violence as a cause of death. Her body was discovered when males from the Kahama community made a convoluted detour in order to find it. Their knowledge of the body's location implies that they had prior knowledge of the location and had perhaps been involved in, or at least witnessed, the assault (Richard Wrangham, personal communication; Goodall, 1986).

Her skeleton is mostly complete, but poorly preserved; bone surfaces are very friable. The degree of dental attrition is similar to Flo's (Figure 2.96); they were probably of similar age at the time of death.

A non-union fracture to the right mandibular corpus was still in the process of callus formation at the time of death. A well-healed, but severe, fracture to the distal shaft of the right humerus is also evident. The right humerus was distinctly shorter than, but also broader than, the left (Jurmain, 1989). Her teeth are severely worn, and 9 teeth were lost premortem (Kilgore, 1989).

Moderate or severe degenerative joint disease is apparent on both distal femora, both glenoid fossae, the left proximal ulna, the right distal humerus, the proximal end of a metatarsal, the left acetabulum, and the right proximal tibia. Old female is excluded from the analysis on degenerative joint disease due to the poor preservation of her skeleton.

Figure 2.96: Old Female's mandibular dentition

Flo (female - 53.1 years – possibly older)

A high ranking female from Kasekela, 3 of Flo's offspring survived to adulthood and were also high ranking. Her son Figan became alpha male, assisted by the simultaneous displays of his half brother Faben. Because of his crippling during the polio epidemic, Faben never achieved high rank on his own, but his support of his brother was crucial to Figan's rise to power (Goodall, 1986). Flo's younger sons Flint (1964-1972) and Flame (1968-1969) are also part of the Gombe skeletal collection. Flo's daughter Fifi (1928-2004) birthed 7 offspring who survived past infancy. Furaha and Fred died as infants, and Fred is part of the Gombe skeletal collection. Flo's surviving grandchildren via Fifi are Freud (born in 1971), Frodo (born in 1976), Fanni (born in 1981), Flossi (born in 1985), Faustino (born in 1989), Ferdinand (born in 1992), and Flirt (born in 1998). As researchers are currently observing Fanni, Flossi, and Flirt's offspring mature, it is evident that the F family is one of the most successful at Gombe (GSRC, unpublished data).

Flo died in 1972, and her death resulted in the death of infant Flame as well as the death of her still-dependent juvenile son Flint. Her primary cause of death may be old age; she also lost much weight towards the end of her life (Goodall, 1986), a symptom consistent with the "wasting disease" attributable to gastro-intestinal disorder (Williams *et al.*, 2008).

Flo's skeleton is mostly complete, lacking only a few hand and foot bones. Most of the epiphyses are fused, though the acromion processes are not (see notes in section on Pallas for further discussion), and the medial margin of the scapula and iliac crest are

more than half fused, but a gap is still evident between the primary and secondary ossification centers.

Flo's teeth are severely worn, and 9 were lost premortem (Kilgore, 1989). Healed fractures are evident in the right clavicle, right distal shaft of the ulna, and both 5th metatarsals (Jurmain, 1989). Degenerative joint disease affects nearly all of the synovial joints. The wrists, ankles, elbows, hips, feet, shoulders, knees, and 3 ribs are moderately or severely affected (Figure 2.97).

Fiure 2.97: Flo's proximal tibia with moderate degenerative joint disease

Overall pattern

Nearly every chimpanzee in the Gombe skeletal collection is affected by some kind of pathology or trauma. This high rate is comparable with results from the previously-studied portion (Jurmain, 1989) of the Gombe skeletal collection as well as the skeletal collection from the Kanyawara community at Kibale National Park in Uganda (Carter *et al.*, 2008).

Of the 1,752 incidences of trauma or pathology recorded in this sample, arthropathy is the most common. Most of these instances are mild. Including only moderate or severe arthropathy, there are 1,056 incidences of trauma or pathology. Of these, 247 (23%) are elements affected by trauma, 282 (27%) are affected by moderate or severe arthropathy, 283 (27%) are affected by bone resorption pathologies, 222 (21%) are affected by bone deposition pathologies, 31 (2%) by congenital variation of abnormalities, and 2 (0.2%) by a combination of resorptive and depositional pathologies.

Trauma

The cranium was the skeletal element most affected by trauma, with 21 of the 37 observed crania show evidence for either pre- or perimortem trauma (~57%). The scapula is the second most affected element, but it is a distant second at only 15%. The number of observed skeletal elements affected by trauma are summarized in Table 2.8.

Table 2.8: Incidences of trauma by skeletal element

Skeletal Element	Number Observed	Number with Trauma	Proportion
cranium	37	21	0.568
scapula	65	10	0.154
humerus	65	9	0.138
ulna	62	8	0.129
mandible	34	4	0.118
tibia	60	7	0.117
femur	63	7	0.111
rib	792	87	0.11
os coxa	67	7	0.104
fibula	57	5	0.088
clavicle	64	4	0.062
metapodials	585	30	0.051
teeth	920	41	0.045
radius	62	2	0.032
podials	730	19	0.026
phalanges	1341	33	0.025
vertebra (C,T,L)	733	8	0.011
sacral vertebra	150	1	0.007
sternum	29	0	0
hyoid	20	0	0
patella	48	0	0

Sex Differences

The mean percent of bones affected by trauma for males is 4.2% and for females is 6.5%. Welch's t-test does not provide sufficient evidence that the male and female means are different ($t = .59$, $df = 24.3$, p-value = 0.56).

Jurmain and Kilgore (1998) demonstrated differences in the distribution of injuries in male versus female chimpanzees, with males exhibiting more cranio-facial trauma and females more trauma to the long bones. A similar pattern is true for this study. I counted the number of injuries to different regions of the body on male versus female chimpanzees; counts are listed both including Andromeda, Rejea, Infanticide

Victim, and Vincent as well as excluding these influential cases. These data are summarized in Table 2.9. I performed a z-score test for the different between population proportions to see if males or females were more commonly affected by skeletal trauma. In the full sample (Table 2.9b), there are 290 instances of trauma to skeletons of known sex, 142 (0.490) are from males, and 148 (0.510) are from females. The reduced sample (Table 2.9a) contains 119 instances of trauma, with 66 (0.555) from males and 53 (0.445) from females. Neither z-score test demonstrated a significant difference in overall trauma rates between males and females; this is unsurprising given the results of the t-test.

Table 2.9a: Distribution of traumata by body part in males versus females (excludes Andromeda, Rejea, Vincent).

MALES

Body Region	Number of Injuries	Proportion
Head and face	5	0.076
Fore limb (with hand)	12	0.182
Thorax/pelvis	15	0.227
Hind limb (with foot)	9	0.136
Teeth	25	0.379
Total	66	

FEMALES

Body Region	Number of Injuries	Proportion
Head and face	2	0.038
Fore limb (with hand)	25	0.472
Thorax/pelvis	10	0.189
Hind limb (with foot)	10	0.189
Teeth	6	0.113
Total	53	

Table 2.9b: Distribution of traumata by body part in males versus females (full sample).

MALES

Body Region	Number of Injuries	Proportion
Head and face	10	0.07
Fore limb (with hand)	26	0.183
Thorax/pelvis	41	0.289
Hind limb (with foot)	31	0.218
Teeth	34	0.239
Total	142	

FEMALES

Body Region	Number of Injuries	Proportion
Head and face	16	0.108
Fore limb (with hand)	51	0.345
Thorax/pelvis	54	0.365
Hind limb (with foot)	21	0.142
Teeth	6	0.041
Total	148	

The number of injuries to different regions of the body do not meet the sample size requirements required for a statistical test, but qualitative discussion is still of interest. The reduced sample, which excludes Andromeda, Rejea, the infanticide victims from 1975, and Vincent, matches previous research on this topic. Males are more commonly affected by cranio-facial trauma than females (0.076 versus 0.038), and also have more instances of thoracic / pelvic trauma (0.227 versus 0.189) as well as trauma to the teeth (chipped / broken teeth or teeth lost due to a traumatic event) (0.379 versus 0.113). Females have more trauma to both the limbs, though the difference is much more

dramatic in the forelimb (0.472 versus 0.182) compared to the hindlimb (0.189 versus 0.136).

The pattern of male-female difference in location of injuries changes if Andromeda, Rejea, the 1975 infanticide victims, and Vincent are included in the sample. Andromeda and Rejea especially drive up the frequencies of female injuries to most areas of the body. Notably, cranio-facial (0.07 versus 0.108) and thoraco-pelvic (0.289 versus 0.365) injuries are more common in females than males. Hind limb injuries are more common in males (0.218 versus 0.142). Forelimb injuries are still more common in females in this sample (0.345 versus 0.183), and tooth injuries are still more common in males (0.239 versus 0.041). Differences between the two samples likely result because 3 of the 4 infanticide victims were females, and the high number of injuries sustained by Andromeda and Rejea due to display behavior and consumption. Results from the reduced sample support previous findings that male chimpanzees are more likely to experience cranio-facial trauma than female chimpanzees, but that female chimpanzees are more likely to experience trauma to the limbs than males (Jurmain and Kilgore, 1998). Expansion of the sample to include more infanticide victims in future could make the full samples more comparable.

Regression Model

Visual inspection of the scatterplot matrix showed that the rank variables covary with each other; highest achieved rank and rank at death also covary with age. In the full trauma sample, most of the predictors were not significantly correlated with trauma incidence ($R^2 \leq 0.065$, $p \geq 0.17$). Only a change in rank from high ranking to low

ranking was significantly correlated, though the correlation was not strong ($R^2 = 0.15$, $p = 0.04$). This is based on a single data point, and is one of the influential cases discussed below (Vincent).

Of the non-significant variables, age comes the closest to having a significant correlation with trauma incidence ($R^2 = 0.065$, $p = 0.17$), but age would be negatively correlated with trauma incidence in this sample. This is unexpected given previous research on chimpanzee skeletons that demonstrated a positive correlation between age and trauma incidence (Jurmain, 1997). Of interest are 4 influential cases (Figure 2.98): Rejea, Andromeda, the male infanticide victim form 1975, and Vincent, all of whom show much higher than average trauma incidences. In each of these cases, trauma incidence is an order of magnitude higher than in the other chimpanzees in this sample (see Table 2.5). It is possible that the full sample does not represent a linear relationship between age and trauma incidence.

If the influential cases are removed, a much more conventional picture emerges. Age is positively correlated with trauma incidence ($R^2 = 0.57$, $p = 0.0$) (Figure 2.99). Death rank and high rank are also significantly positively correlated, ($R^2 \geq 0.34$, $p \leq 0.002$), but these variables covary ($R^2 = 0.84$), and variation in age accounts for most of the variation in either of these rank variables ($R^2 = 0.73$, $p = 0.0$ for high rank). The factors describing change in rank and sex were not significantly correlated with trauma incidence ($R^2 \leq 0.09$, $p \geq 0.14$). The variable describing the interaction between age and rank was significantly correlated with trauma incidence ($R^2 = 0.16$, $p = 0.05$), though its predictive power is low. Of the variables tested in the reduced sample, age best explains variation in trauma incidence, which is consistent with previous findings (Jurmain, 1997).

Inclusion of the infanticide victims, 3 of whom have high levels of perimortem trauma, has great influence on the interpretation of these data, which is discussed in more detail below.

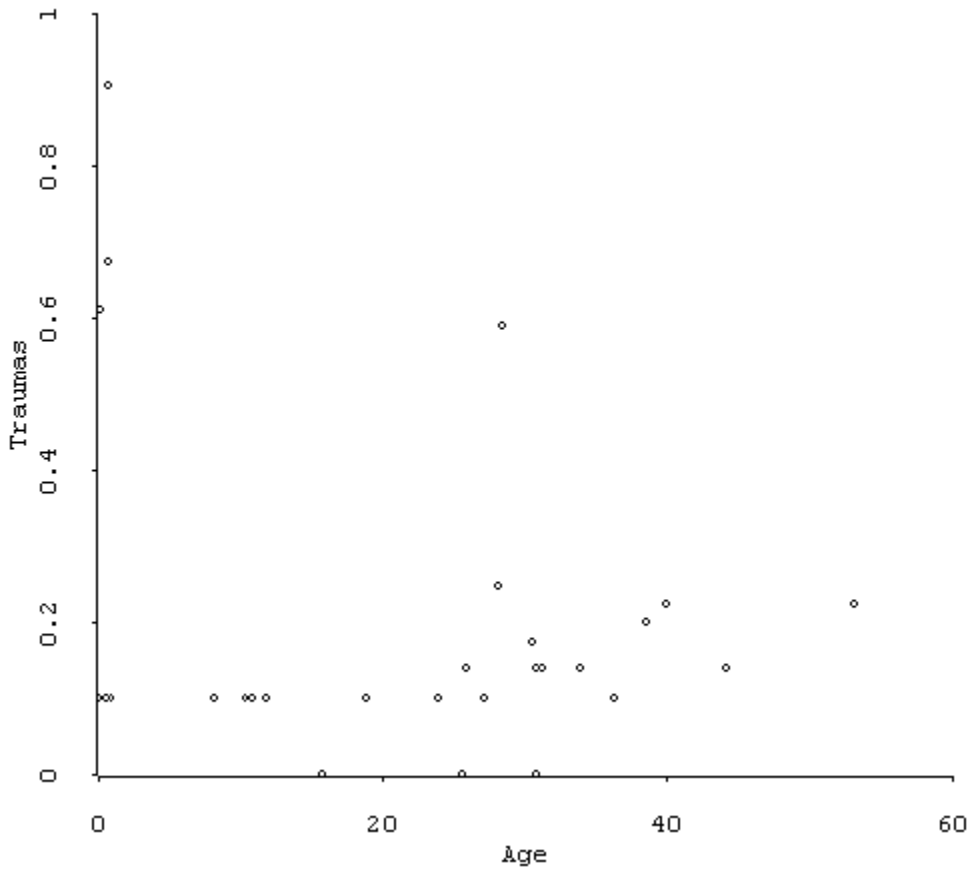


Figure 2.98: A plot of age versus trauma incidence (transformed data); there is no clear linear relationship between the two variables.

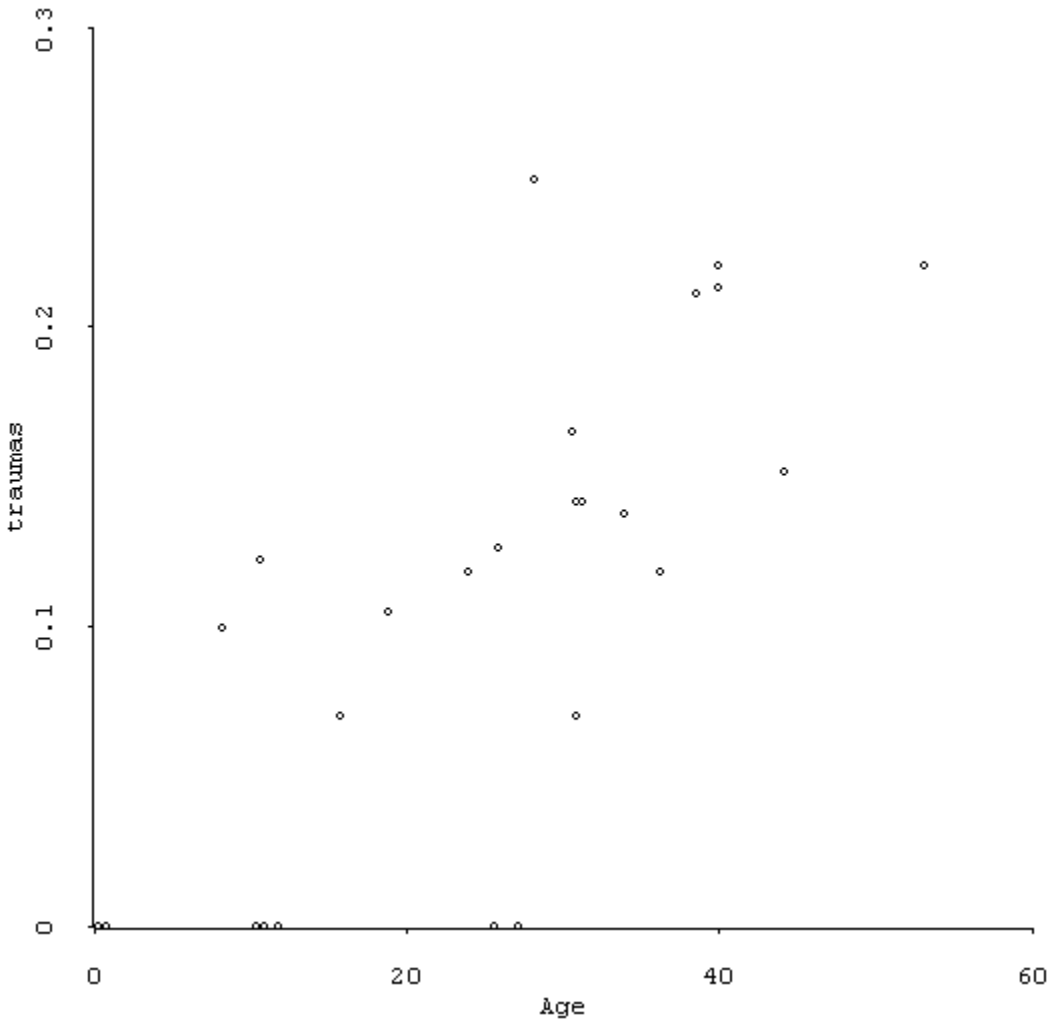


Figure 2.99: Age plotted against trauma incidence (transformed data) in the reduced sample. A clear positive correlation between age and trauma incidence is evident.

Arthropathy

The elbow is the joint most commonly affected by moderate or severe degenerative joint disease, followed by the hip and knee. Each joint surface may be unequally affected (e.g. the proximal femur was not affected as often as the acetabulum). These data are summarized in Table 2.10.

Sex Differences

The mean percent of bones affected by arthropathy for females is 5.1% and for males is 3.4%. Welch's t-test does not provide sufficient evidence that the male and female means are different ($t = 0.69$, $df = 21.96$, p-value = 0.5).

Regression Model

Welch's two sample t-test determined that the mean percent of joints affected by arthropathy differs between adults and sub-adults ($t = 3.81$ $df = 18$, $p = 0.001$). Because sub-adults have a mean arthropathy incidence of 0%, sub-adults were excluded from the models.

As expected from the results of the t-test, sex was not significantly correlated with arthropathy incidence ($R^2 = 0.00003$, $p = 0.98$). The variables high rank and death rank covary, but neither was significantly correlated with arthropathy incidence ($R^2 \leq 0.05$, $p \geq 0.37$). Age also covaries with high rank because subadults are always low ranking. The variable describing the combined effects of age and high rank was not, however, significant ($R^2 = 0.07$, $p = 0.32$). The factor describing a change in rank from high ranking to middle ranking was not significant ($R^2 = 0.03$, $p = 0.48$).

Age was significantly correlated with arthropathy incidence ($R^2 = 0.38$, $p = 0.005$) (Figure 2.100). This is consistent with previous findings on the relationship between arthropathy and age in chimpanzee skeletons. The factor describing a change in rank from middle ranking to low ranking was also significant ($R^2 = 0.22$, $p = 0.06$) (it did not covary with age), but only 1 chimpanzee (Pallas) fits this description, so this result, while suggestive, should be treated with caution (Figure 2.101).

Table 2.10: Joint surfaces affected by degenerative joint disease.

Joint Surface	Number observed	Moderately / severely affected	Proportion	Mildly affected	Proportion	Total affected	Proportion
proximal ulna	50	16	0.32	16	0.32	32	0.64
distal humerus	50	14	0.28	16	0.32	30	0.6
acetabulum	53	12	0.23	19	0.36	31	0.58
distal femur	43	8	0.19	17	0.4	25	0.58
proximal tibia	48	9	0.19	16	0.33	25	0.52
proximal femur	47	8	0.17	23	0.49	31	0.7
1st sacral vert	23	3	0.13	3	0.13	6	0.26
glenoid fossa	51	6	0.12	13	0.26	19	0.37
mandibular fossa	68	7	0.1	7	0.1	14	0.21
distal fibula	45	4	0.09	12	0.27	16	0.36
podials	685	60	0.09	100	0.15	160	0.23
proximal humerus	50	4	0.08	23	0.46	27	0.54
proximal radius	51	4	0.08	18	0.35	22	0.43
acromion	52	4	0.08	2	0.04	6	0.12
distal tibia	46	3	0.07	20	0.43	23	0.5
lumbar vert.	97	6	0.06	42	0.43	48	0.49
distal ulna	51	3	0.06	15	0.29	18	0.35
ribs	620	40	0.06	155	0.25	195	0.31
lateral clavicle	39	2	0.05	8	0.21	10	0.26
medial clavicle	41	2	0.05	7	0.17	9	0.22
mandibular condyle	56	3	0.05	2	0.04	5	0.09
proximal fibula	45	2	0.04	16	0.36	18	0.4
distal radius	50	2	0.04	14	0.28	16	0.32

Joint Surface	Number observed	Moderately / severely affected	Proportion	Mildly affected	Proportion	Total affected	Proportion
patella	48	2	0.04	12	0.25	14	0.29
metapodials	962	38	0.04	141	0.15	179	0.19
thoracic vert.	311	6	0.02	78	0.25	84	0.27
cervical vert.	139	3	0.02	26	0.19	29	0.21
phalanges	1938	34	0.02	113	0.06	147	0.08
manubrium	22	0	0	1	0.05	1	0.05

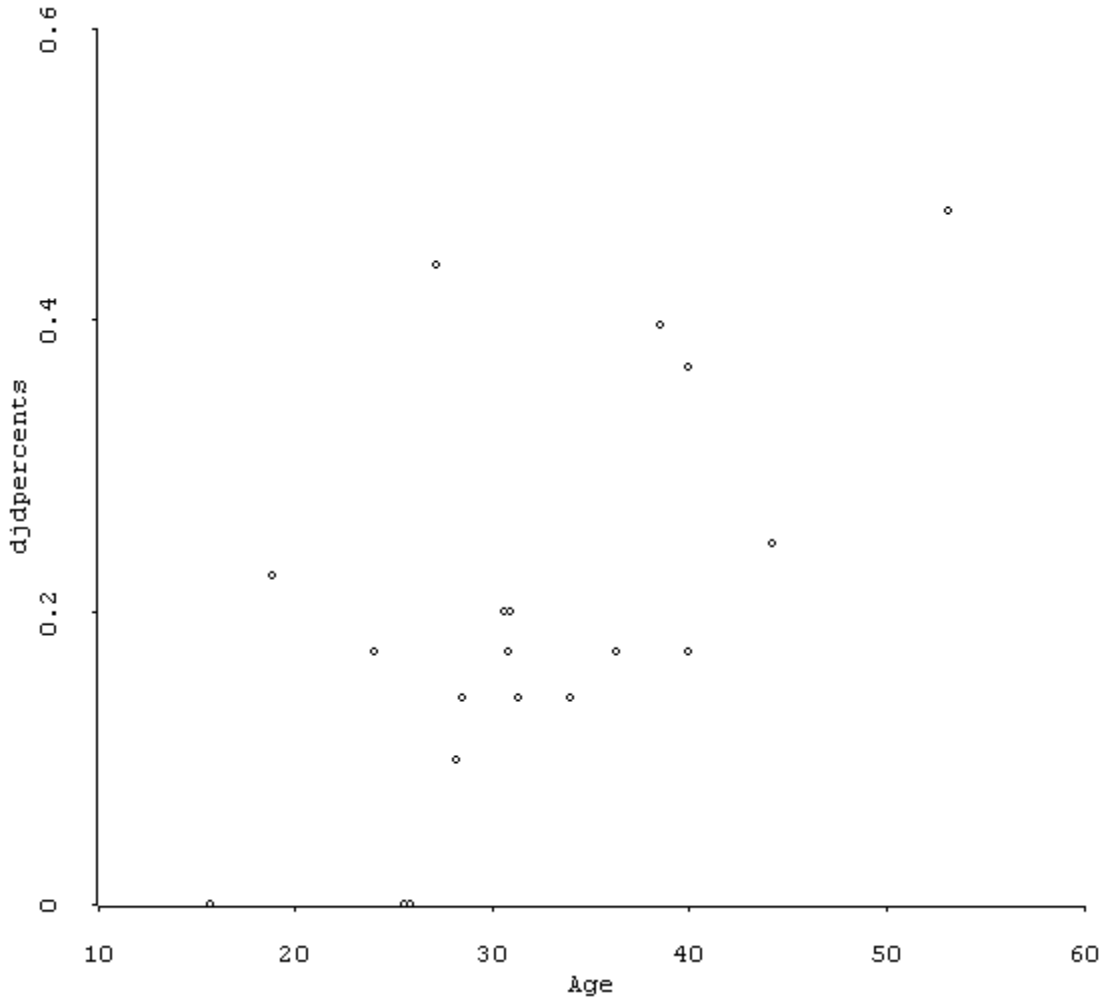


Figure 2.100: A plot of age and arthropathy incidence ("djdpercents") indicated a positive correlation between these variables. (Graph depicts transformed data.)

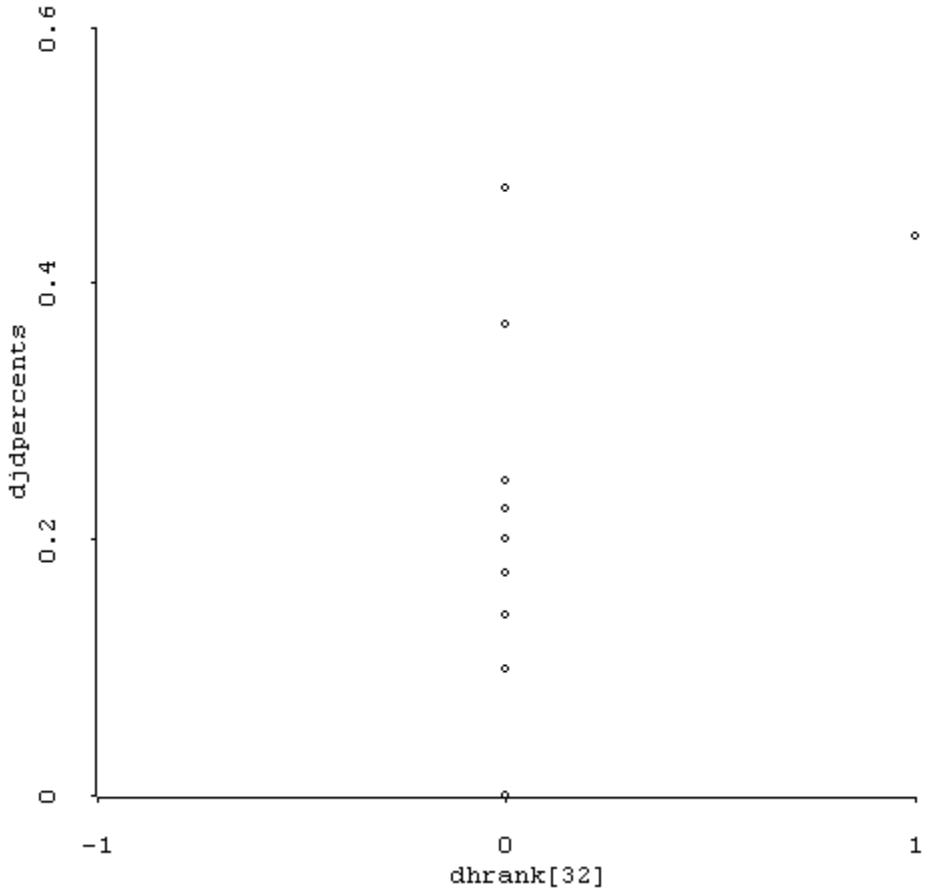


Figure 2.101: A plot of the factor describing a change from middle to low ranking ("dhrank32") versus arthropathy incidence ("djdpercents"), with transformed data. While a correlation may exist, it is based on only one specimen and should therefore be treated with caution.

Pathology

The cranium was also severely affected by pathology, including bone resoprtion or deposition pathologies and congenital abnormalities. Eleven crania exhibit several instances of pathology, especially periodontal disease, and sixteen chimpanzees have one or more dental pathologies, including premortem tooth loss. Nearly 60% of the mandibles were affected by pathology, also reflecting the prevalence of periodontal disease. Skeletal elements affected by bone deposition or resorption pathologies are summarized in Table 2.11. Laminar deficiency is the most common congenital abnormality recorded for this sample, affecting 6 chimpanzees. Another common variation is asymmetrical transverse foramina in the cervical vertebrae.

Sex Differences

The mean percent of bones affected by bone formation or bone loss pathologies for females is 7.7% and for males is 9.4%. Welch's t-test does not provide sufficient evidence that the male and female means are different ($t = -0.8$, $df = 25.83$, p-value = 0.43).

Table 2.11: Skeletal elements affected by bone loss or bone formation pathologies, or congenital abnormalities.

Skeletal Element	Number Observed	Number with Pathology	%
cranium*	37	39	1.054
mandible	34	20	0.588
humerus	65	23	0.354
ulna	62	14	0.226
tibia	60	13	0.217
os coxa	67	14	0.209
femur	63	12	0.19
scapula	65	10	0.154
clavicle	64	8	0.125
sacral vertebra	150	17	0.113
teeth	920	91	0.099
phalanges	1341	116	0.087
patella	48	4	0.083
fibula	57	4	0.07
sternum	29	2	0.069
metapodials	585	39	0.067
rib	792	50	0.063
podials	730	40	0.055
radius	62	3	0.048
vertebra (C,T,L)	733	21	0.029
hyoid	20	0	0

*Many crania are affected by multiple pathologies.

Regression Model

As expected from the results of the t-test, sex was not significantly correlated with pathology incidence ($R^2 = 0.03$, $p = 0.35$). None of the factors describing a change in rank were significant ($R^2 \leq 0.09$, $p \geq 0.12$). High rank and death rank were both significant predictors ($R^2 \geq 0.32$, $p \leq 0.002$), but they covaried ($R^2 = 0.87$, $p = 0.0$). Both rank variables also covaried with age ($R^2 = 0.75$, $p = 0.0$ for high rank). The variable describing the interaction between age and rank was significant ($R^2 = 0.32$, $p = 0.002$), but its predictive power was less than age alone, which was the best single predictor tested ($R^2 = 0.57$, $p = 0.0$). Age is therefore the only variable with a significant correlation with pathology incidence (Figure 2.102); other significant correlations are accounted for by age alone.

There was a significant difference in pathology incidence between adults and sub-adults ($t = 4.56$, $df = 27.47$, $p = 0.0$). Only age was a significant predictor for both the full sample and the adults-only sample ($R^2 = 0.21$, $p = 0.05$ for adults-only sample), and the correlation coefficient is higher for the full sample ($R^2 = 0.57$, $p = 0.0$). I therefore kept the sub-adults and adults in the same sample.

In another demonstration of covariation between rank variables and age, none of the rank variables significantly correlated with pathology incidence in the adults-only sample ($R^2 \leq 0.14$, $p \geq 0.13$). Figure 2.102 demonstrates that there is not a different trend in pathology incidence for adults versus sub-adults.

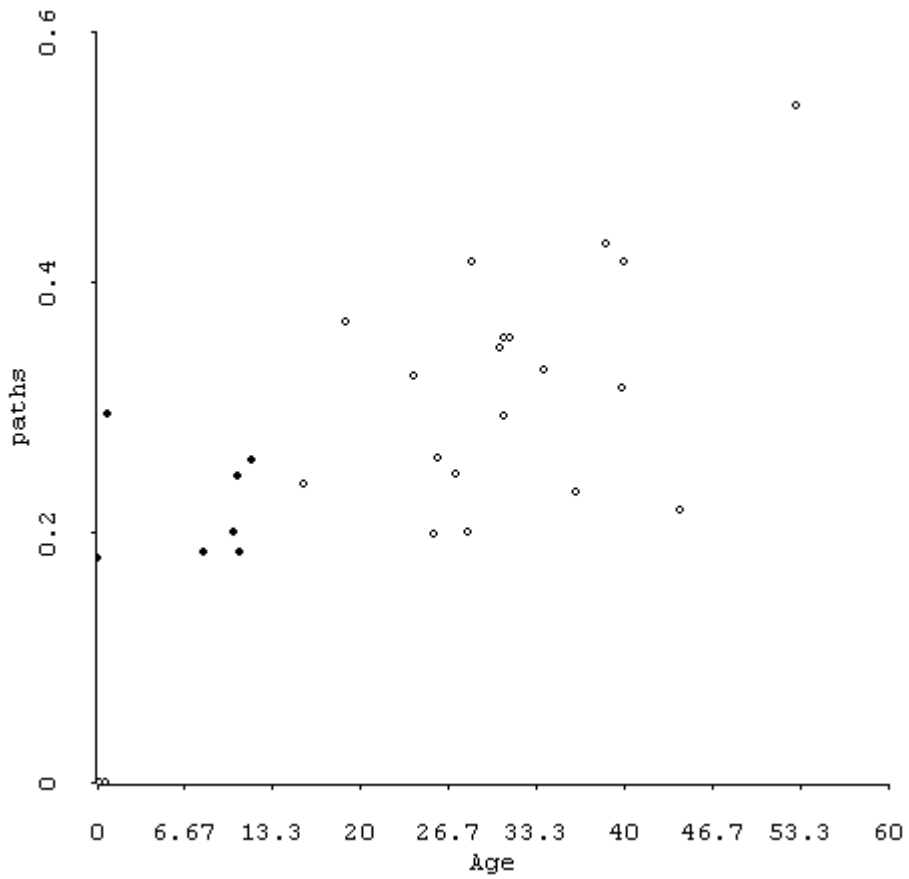


Figure 2.102: A plot of age and pathology incidence ("paths") indicates a positive correlation between these variables. Dark dots indicated sub-adults. (Graph depicts transformed data.)

DISCUSSION

In the regression models, dominance rank was not usually a significant predictor. There is some evidence that change in dominance rank is more significant than rank itself, but an increased number of chimpanzees whose rank changed during life is necessary to further evaluate this hypothesis. Only 4 chimpanzees changed rank between their highest achieved rank and rank at death (Atlas, Goblin, Vincent, and Pallas). Age was a significant predictor of incidences of trauma, arthropathy, and other pathologies. This is broadly consistent with previous analyses of primate skeletal material that have hypothesized that longer-lived apes have more time to accumulate traumata and pathologies (Jurmain, 1989; Jurmain, 1997; Lovell, 1990), though some influential cases in the trauma sample merit deeper consideration.

Trauma

Sex was not significantly correlated with trauma incidence, nor was the difference between males and females in the mean number of incidences of trauma significant. I expected males, especially high ranking males, to exhibit higher rates of trauma than other demographic groups. The results may be limited by sample size, but must be affected by the high levels of soft tissue trauma that leave no trace on the skeleton. Skeletal evidence for perimortem trauma can be scarce in terms of number of skeletal elements affected, even in chimpanzees whose cause of death is known to be conspecific aggression. Ebony, for example, does not have any examples of perimortem trauma, nor does Goblin have any skeletal signs of the severe thigh laceration that nearly killed him. Causes of death reflected by the skeleton are discussed in more detail below.

Differences in distribution of injuries between the sexes were, however, evident. Males were more affected by cranio-facial trauma, and females more affected by trauma to the limbs. This is consistent with previous findings on chimpanzee skeletons from Gombe and museum collections (Jurmain and Kilgore, 1998).

Dominance rank was significantly correlated with trauma incidence in the reduced sample, but it covaries with age, and age variation accounts for all of the variation in dominance rank in this sample. A change in dominance rank from high to low ranking was significantly correlated with trauma incidence. This should be treated with caution, as it is based on a single influential case: Vincent. It is relevant, however, that Vincent's injuries due to his fall directly contributed to his loss of rank.

In the reduced sample, age was the most important variable accounting for trauma incidence, but age was not significantly correlated with trauma incidence in the full sample. The influential cases in the full sample include 3 infanticide victims with severe perimortem trauma. Infanticide is a rare, but important, event among chimpanzees (Arcadi and Wrangham, 1999). Traditionally accumulated museum skeletal collections (wild shot animals (e.g. Harman, 2005)) would not include infanticide victims, and younger animals are often excluded from comparison with adults because the number of skeletal elements is not the same (e.g. Jurmain, 1997). Expressing trauma incidence as a percent of observable bones affected mitigates these concerns.

Including chimpanzees of all ages, particularly the infanticide victims, may change some of our impressions about the relationship between age and trauma incidence. In the full sample, age is not positively correlated with trauma incidence; the correlation may be negative, or even non-linear. Because conspecific aggression is a

significant source of mortality for chimpanzees of many ages (Williams *et al.*, 2008), and infanticide has been observed at most long-term chimpanzee study sites (Arcadi, 2000), discarding the cases of Andromeda, Rejea, and the male infant killed in 1975 as outliers would be misleading. The Gombe skeletal collection provides evidence that skeletal traumata do accumulate throughout the lifetime of adult chimpanzees, but also that young chimpanzees can be even more severely affected by fatal skeletal trauma than adults.

Arthropathy

Sub-adults were much less affected by degenerative joint disease than adults. This difference was so extreme that sub-adults were removed from the regression model.

In addition to age, a change in dominance rank from middle to low ranking was significantly correlated with arthropathy incidence. (The change in rank factor did not covary with age ($R^2 = 0.017$, $p = 0.6$.)) The sample only includes 1 chimpanzee whose rank changed between her highest achieved rank and rank at death: Pallas. The association between change in rank and arthropathy incidence is not strong because of the extremely restricted sample size, but it does suggest that physical ability is correlated with dominance rank in chimpanzees.

Other chimpanzees who changed rank include Atlas, Vincent, and Goblin, all of whom were high ranking males during their prime. Atlas and Goblin subsequently became middle ranking during the latter part of their lives, and Vincent became low ranking after his fall (GSRC). While these changes in rank did not have a statistical correlation with arthropathy incidence, a larger sample might yield different results.

Males who are past prime age are not generally able to remain the alpha male, though some are able to remain high ranking. The chimpanzee with the highest arthropathy incidence is Flo, who despite her age and feeble condition towards the end of her life retained her high rank (possibly because of her association with high-ranking family members) (Goodall, 1986). If the relationship between degenerative joint disease and rank described here may be applied to a larger sample, this raises questions about the direction of causality between loss of rank and arthropathy incidence. Do chimpanzees who lose rank wear out their joints faster than rank-maintaining conspecifics? Are worn out joints more likely to cause a loss in rank than joints in better condition relative to age? For male chimpanzees, I suspect that age and senescence prevent the vigorous competition necessary to maintain high rank, but the process is likely to be more subtle in females. Discerning the relationship between skeletal pathologies and changes in dominance rank requires a larger sample of chimpanzees who experienced a loss in rank.

Other pathologies

Age was significantly correlated with pathology incidence, as were rank variables, but age variation accounted for all of the variation due to rank. Neither sex nor changes in dominance rank were correlated with pathology rates. As the data for arthropathy hint, perhaps a larger sample of chimpanzees with known ranks – particularly animals who experienced a change in rank – would better represent the relationship between skeletal pathologies and dominance rank.

Vertebral laminar deficiency was a fairly common pathology in this study, affecting 7 chimpanzees (4 males, 3 females), 5 of whom were subadults at the time of

death. Whether this constitutes a neural tube defect or variation in skeletal growth is relevant for assessing the health status of these chimpanzees.

Non-union of the neural arch is not necessarily a neural tube defect (Mulhern *et al.*, 2009), and because incidence decreases with age (Sutow and Pryde, 1956), it is possible that delayed union of the two halves of the neural arch may be part of normal variation in skeletal maturation (Resnick, 1988; Turkel, 1989). In some instances, the neural arch is likely to be completed by cartilage and fibrous tissue (Resnick, 1988).

In order to be considered an actual case of spina bifida occulta, constituting a neural tube defect, the vertebral canal should be enlarged and the edges of the lamina should be pushed outward or distended posteriorly (Aufderheide and Rodriguez-Martín, 1998; Barnes, 1994; Mulhern *et al.*, 2009).

In humans, the neural arches of the sacral vertebrae usually fuse between 2-6 years of age (Schaefer *et al.*, 2008). Definitive ages of fusion for this feature in wild chimpanzees have not been published, but it is likely the ages are at least somewhat comparable. Ebony (8 years), Jackson (10.4 years), Mel (10.7 years), Sugar (10.9 years), and Galahad (11.8 years) all display lack of fusion of at least one neural arch in the sacrum. Because the laminar deficiencies in the subadults are all minor in that the vertebral canal is the normal size, the gap is usually 3mm or less, the edges of the disconnected lamina are not distended, and a partial spinous process may sometimes be formed, these are unlikely to be cases of spina bifida occulta. Instead, this represents diversity in skeletal maturation in the Gombe chimpanzees. Of possible interest is that Sugar seems to be skeletally immature for her chronological age in that her facial

skeleton is not fused to the neurocranium (the facial skeletons of the other sub-adults with laminar deficiencies are fused to the neurocranium).

Sherehe (15.8 years) and Echo (34 years) were both skeletally and dentally mature adults at the time of death. Each female had also given birth to at least one infant before she died (GSRC unpublished data), confirming their adult status. Sherehe's case is ambiguous in terms of diagnosis. The 2nd through 5th sacral vertebrae have incomplete lamina, but it is not clear whether the vertebral canal is enlarged. The 2nd vertebra may have a partial spinous process, but it is clear that 2-5 do not (Figure 2.26). Her adult status makes a diagnosis of spina bifida occulta more likely for Sherehe than for the sub-adults, but not all of the criteria for the disease are met.

Echo's sacrum meets nearly all of the bony criteria for spina bifida occulta (Figure 2.75). All of the sacral vertebrae lack a completed neural arch, and the gap approaches half a centimeter. There are no partial spinous processes, and the edges of the gap have a slight posterior flare, making the vertebral canal larger and wider than it would be otherwise.

Spina bifida is a neural tube defect that can have both environmental and genetic causes (Ferembach, 1963; Hol *et al.*, 1995; Larsen *et al.*, 2001). Insufficient folic acid in early embryonic development is most often the cause considered for the severe forms of spina bifida, which may result in paralysis or death (e.g. Cech and Burau, 2010). Spina bifida occulta is likely to differ in its causes from the much more severe spina bifida cystica, in that it is possible that spina bifida occulta is more likely to be caused by genetic predisposition (Hol *et al.*, 1995). The cause of Echo's spina bifida occulta is unknown, though the lack of widespread spina bifida occulta in the skeletons at Gombe

suggests that lack of B-complex vitamins may be less likely for these free-ranging chimpanzees.

Comparison with Kibale

Comparison of skeletal pathology and trauma between chimpanzee research sites has the potential to inform us about possible differences in sources of morbidity and mortality. Study of the Gombe skeletal collection as well as skeletons from Kibale National Park in Uganda (n = 20 chimpanzees) shows that chimpanzees in both study sites experience high levels of pathology and trauma (Carter *et al.*, 2008). Eleven of the 12 chimpanzees from Kibale with post-cranial skeletons are affected by trauma (92%) (I*bid.*). Of the 34 complete skeletons from Gombe presented here, 28 are affected by trauma (82%). The number of chimpanzees affected by pathology in the Kibale sample also approaches 100% (I*bid.*). (A more specific rate describing chimpanzees affected by trauma is not possible to ascertain from the published data as some pathologies are listed categorically and it is unclear whether a single chimpanzee appears in more than one category.) Thirty-two of 34 (92%) complete skeletons from Gombe exhibit some kind of pathology (bone loss, bone formation, arthropathy, or congenital variation).

The cranium is one of the most affected skeletal elements in both skeletal collections, with 56.8 % of crania at Gombe affected, and 42.1% at Kibale. The hand is more affected by trauma at Kibale (87.5% of skeletons) than at Gombe (35.3% of skeletons). The much higher rate of snare injuries at Kibale may be one reason this difference exists. Only 20% of the skeletons from Kibale are affected by trauma to the foot (data on Kibale from Carter *et al.*, 2008), and in this case, the rate at Gombe is

higher, with 44.1% of chimpanzees affected. These data suggest that hands and feet are not equally affected by snares.

The elbow, hip, and knee are the joints most commonly affected by degenerative joint disease in the Gombe skeletal collection. The shoulder and temporal-mandibular joint are also highly affected. This is similar to the Kibale sample (I*bid*.), though the order of which are most / least affected differs, likely due to the smaller Kibale sample. The sternoclavicluar joint is highly affected by degenerative joint disease at Kibale (2 / 10 or 20% of skeletons) (*Ibid*.), but is affected about half as frequently at Gombe: of the 22 chimpanzees with observable manubria and medial clavicles, 2 clavicles are affected (~9%), but none of the manubria. This marked difference in the rate that the sternoclavicular joint is affected by degenerative joint disease may be due to the smaller sample currently available from Kibale. Other contributing factors may include terrain and / or vegetation density differences between the two parks.

Complete comparison of differences in rates between Kibale and Gombe is not currently possible from the published literature, as the Kibale report includes complete joints (e.g. "hip") rather than joint surfaces (e.g. "acetabulum" and "femoral head"), and does not indicate whether both joint surfaces need be affected in order for the entire joint to be scored as affected. Future more detailed analyses on a larger skeletal sample may help to further describe possible differences and similarities in pathology rates between the two populations.

It is clear from the Gombe and Kibale skeletal collections that we can expect chimpanzees to show high levels of skeletal pathology and trauma, reflecting sources of morbidity and mortality such as falls from height and inter-individual aggression.

Comparison between these two sites allows us to hypothesize that these are species-level selective pressures. Differences in trauma patterns, for example the greater incidence of hand trauma at Kibale, tell us about stressors that may be specific to the local environment (e.g. snares).

Causes of death visible on the skeleton

Of the 39 chimpanzees in the Gombe skeletal collection, only 11 (28%) show signs of their cause of death on the skeleton. Most skeletons do not provide evidence of the cause of death (24 / 34 complete skeletons = 70.5%; 24 / 39 chimpanzees in any skeletal condition = 61.5%), or provide tentative evidence (4 / 39 = 10%). Table 2.12 summarizes the causes of death for each chimpanzee and whether the skeleton shows evidence for the cause of death. Thirty-four chimpanzees have a known cause of death. Of these, 11 (32%) show skeletal evidence of their cause of death. Twenty (59%) do not, and 3 show tentative evidence (8%). Some of the most common causes of death for chimpanzees at Gombe, including respiratory infection and intestinal parasites or "wasting disease" (Williams *et al.*, 2008) do not leave signs on the skeleton.

Nearly all of the 11 chimpanzees (9 / 11 = 82%) with skeletal evidence for cause of death died as a result of traumatic injury. Eight of these chimpanzees were killed by other chimpanzees and one (Echo) died as a result of complications from a paralyzing spinal cord injury. Flo and the Bwavi male are considered to have died of old age. The Bwavi male's extremely severe arthritis affecting all of his synovial joints is evidence for his age, and Flo's extensive dental attrition is evidence of her advanced age. While older males like Goblin and Hugo show either extensive wear or significant tooth loss, only

Old Female shows a degree of premortem tooth loss comparable to Flo. Age may be a secondary cause of death in Old Female's case, but the primary cause of death is chimpanzee attack.

Though most of the skeletons that show signs of the cause of death are due to chimpanzee attack, the variation in skeletal evidence for perimortem trauma from chimpanzees varies widely. On one extreme is Rejea, an infanticide victim who was both killed and partially consumed by chimpanzees; the extensive perimortem trauma inflicted by chimpanzees is described in detail in Chapter 3. Madam Bee, though she was not eaten, also shows several perimortem wounds, including the loss of her hallux. Vincent is intermediate, with two puncture wounds evident on his facial skeleton and other possible perimortem fractures of ribs and the ilium. Charlie also shows puncture wounds on his face, but no other perimortem trauma. Often, injuries from chimpanzee attack may primarily affect soft tissues. Patti, for example, displays only a small puncture in the talus, but much soft tissue trauma was noted during necropsy. Neither Old Female, Mel, nor Ebony show signs of perimortem trauma despite having been killed by chimps.

These results indicate that causes of death, even violent ones, are likely to be underrepresented by the skeleton.

Table 2.12: Causes of death summaries, indicating whether cause of death is evident on skeleton

Individual	Age at death (yrs)	SIV status	Cause of Death	Skeletal Signs of Death?
Rix	33.4	unknown	fall	maybe - damaged vertebra
Goblin	40.0	negative	wasting disease	maybe - abnormal ossifications
Gaia's infant	0.0	negative	neonatal death, possible lack of maternal care	maybe - anemia?
Sherehe	15.78	negative	unknown - injury possible	maybe - infection?
Yolanda	24.0	positive	AIDS	no
Jackson	10.4	negative	bacterial pneumonia	no
Unknown male	~30	negative	cran only	no
Ebony	8.2	negative	killed by chimps	no
Old Female	old adult	unknown	killed by chimps	no
Fred	0.9	negative	mange	no
Galahad	11.8	negative	pneumonia	no
Infant	0.3	unknown	possible infanticide	no
Mel	10.7	negative	possibly killed by chimps	no
Sugar	10.9	negative	respiratory infection	no
Satan	29.8	unknown	respiratory infection	no
Jomeo	30.9	unknown	respiratory infection	no
Miff	30.9	unknown	respiratory infection	no
Hugo	38.6	unknown	respiratory infection	no
Kidevu?	25.6	negative	unknown	no
Winkle?	28.9	negative	unknown	no
Humphrey	34.9	unknown	unknown	no
Cusano	39.9	negative	unknown	no
Gilka	18.9	unknown	wasting disease	no
Pallas	27.2	unknown	wasting disease	no
Passion	30.6	unknown	wasting disease	no

Individual	Age at death (yrs)	SIV status	Cause of Death	Skeletal Signs of Death?
Atlas	31.3	negative	wasting disease	no
Beethoven?	33.5	negative	wasting disease	no
Melissa	36.3	unknown	wasting disease	no
Charlie	25.9	unknown	attacked by chimps	yes - puncture wounds on face
Madam Bee	28.2	unknown	attacked by chimps	yes - multiple perimortem traumata
Vincent	28.5	positive	attacked by chimps	yes - multiple perimortem traumata
Echo	34.0	positive	complications after spinal cord injury	yes - vertebral fracture
Rejea	0.3	negative	infanticide	yes - damage from consumption
infanticide victim	0.3	unknown	infanticide	yes - damage from consumption
Andromeda	0.7	negative	infanticide	yes - puncture wound on cranial vault
Patti	44.3	negative	killed by chimps	yes - puncture in talus
Bwavi male	40+?	negative	old	yes - severe arthritis
Tumaini	~30	negative	poached	yes - multiple perimortem traumata
Flo	53.1	unknown	old	yes - tooth loss

Comparison with the hominin fossil record

Comparison of rates of trauma and pathology between modern and ancient groups includes several inherent difficulties, including the fragmentary nature of the fossil record (Campillo, 2006), and taphonomic changes that may have occurred during the course of a long burial (Pérez, 2006). Fossil sites infrequently represent an actual, living population or organisms that lived approximately contemporaneously (let alone interacted with each other). Because of the nature of fossil assemblages, pathological evaluation is usually only possible for isolated case studies (e.g. Campillo, 2006) rather than for a group or population. The Gombe chimpanzee collection, on the other hand, is comprised of many complete or nearly complete skeletons, all of whom are roughly contemporary, and many of whom directly interacted with one another during life.

Some paleopathological case studies have been presented in a way that does not make the frequency of pathologies clear. A study on periodontal disease in South African australopith fossils indicates that at least 5 specimens of *Australopithecus africanus* and *Paranthropus robustus* have skeletal signs of moderate to severe periodontal disease, but does not state how many specimens were evaluated (Ripamonti and Petit, 1991), and thus inferences about the frequency of periodontal disease are not possible from this publication. Periodontal disease, tooth wear, tooth loss and tooth breakage have the potential to affect an organism's ability to secure sufficient nourishment, and dental health is therefore an important measure of overall health (Lovell, 1991). Dental pathologies are common occurrences in the Gombe skeletal collection (Kilgore, 1989; This study), and are also likely to have affected ancient populations (e.g. Lebel and Trinkaus, 2002). Further study on the dental remains of

animals with known ages and life histories has great potential for illustrating how impaired masticatory function may affect survivorship and fitness, diet hardness at different life history stages, and, because edentulous animals are usually considered old adults (e.g. Rightmire *et al.*, 2008; Tappen, 2005), the evolution of life history stages and life span in the hominoid lineage.

Despite obstacles, some comparisons with qualitative studies are possible. A reported compression fracture to a talus attributed to *A. africanus* from Sterkfontein (Fisk and Macho, 1992) bears a resemblance to the calcaneal fractures seen in Hugo and Vincent. Hugo's healed calcaneal fracture is attributed to a fall from height (Jurmain, 1989), and Vincent is known to have survived a fall from a tree (GSRC, unpublished data). The talar fracture at Sterkfontein is attributed to a similar cause, and is held up as evidence that *A. africanus* spent at least some time in trees (Fisk and Macho, 1992). While Fisk and Macho (1992) wonder if this kind of injury could result from a loss of arboreal adaptations in australopiths, the presence of similar injuries in arboreal chimpanzees does not support this hypothesis.

A few fossil sites have yielded samples of hominin bones that can be considered as a population. The site of this nature on which the most paleopathological work has been done is Sima de los Huesos at Atapuerca, Spain. The skeletal remains from Sima de los Huesos represent at least 33 hominins. As we might expect from two different species so separate in time, place, and behavior, there are important differences in the patterns of trauma and pathology between the hominins from Sima de los Huesos and the Gombe chimpanzees. The most profound difference between this site and the Gombe chimpanzees is that among the 1200 postcranial skeletal elements recovered from Sima

de los Huesos, none exhibit evidence for trauma (Pérez *et al.*, 1997). This is in stark contrast to the high rates of skeletal trauma seen in chimpanzees from both Gombe and Kibale.

There may be some evidence for cranial trauma, in the form of circular indentations observed on the outer table of the cranial vault of 8 hominins. Whether, in the case of the specimens from Sima de los Huesos, these pathologies are indeed due to trauma or to some kind of scalp-related infection is unclear (Pérez *et al.*, 1997), but similar pathologies are evident on some of the crania from Gombe, including Winkle and Galahad. If these pathologies do have the same etiology, the incidence is higher for the hominins at Sima de los Huesos than for the chimpanzees at Gombe. In the case of the hominin fossils, there is speculation that if the lesions are traumatic in nature their cause could be blunt force trauma from the stone roof of a cave (Pérez, 1991). As the Gombe chimpanzees are not cave dwelling, it is not to be expected that they would be at risk for a similar source blunt force trauma.

Two hominins from Sima de los Huesos are affected by cranial osteomas (Pérez *et al.*, 1997), while this is not a secure diagnosis for any of the Gombe chimpanzees. Eight crania from Sima de los Huesos are reported to have some kind of pathology (Pérez *et al.*, 1997), and if we use the minimum number of individuals represented by the skeletal assemblage at Sima de los Huesos, 33, the frequency of hominins affected by cranial pathology is 24.2%. For purposes of comparison, because many specimens from Sima de los Huesos include only the cranial vault, I consider only pathologies of the frontal, temporal, parietal, and occipital bones from Gombe. Nine of 37 cranial vaults

from Gombe (24.3%) are affected by pathology; this is very similar to the rate from Sima de los Huesos.

The rate of temporal-mandibular joint arthropathy differs dramatically between Sima de los Huesos and Gombe. The rate of mandibular fossae affected by arthropathy at Sima de los Huesos is 70% (Pérez *et al.*, 1997), but only 3 / 56, or 5%, of mandibular fossae from Gombe are affected. Sima de los Huesos has a much higher attrition rate for this joint than other contemporary and other recent human populations, indicating high levels of stress to the temporal-mandibular joint (I*bid.*). This may indicate extra-masticatory use of the dentition and suggests that, although only one dental abscess is reported from Sima de los Huesos, the actual frequency of dental pathology is likely to be obscured by under-representation of dental and periodontal remains. Nineteen chimpanzees (48.7% of 39 chimpanzees with dental remains) from Gombe exhibit some kind of dental pathology, ranging from periodontal disease, caries, premortem tooth loss, and tooth breakage.

CONCLUSIONS

As the longest continuously-studied population of wild chimpanzees, the Gombe skeletal collection offers an unparalleled opportunity to understand how sources of morbidity and mortality are reflected on the skeleton. The high levels of trauma reported here are consistent with previous studies and reflect the risks of inter-individual aggression as well as arboreality. Causes of death are underrepresented by the skeletal evidence, and many common sources of mortality in chimpanzees (e.g. respiratory illness, soft tissue injury in agonistic attacks(Williams *et al.*, 2008)) may not leave any

skeletal evidence at all. This study has also revealed questions that are unanswerable with the current skeletal sample, but may be answerable in future as the collection continues to grow. The effects of change in rank on skeletal trauma and pathology incidences are of particular interest when exploring the relationship between chimpanzee behavior and the signature it leaves on the skeleton.

Skeletal analysis is able to complete the picture of an organism's life history, may contribute to knowledge on cause of death, and, especially as skeletal samples from other primate study sites are accumulated, will allow for comparison of rates of trauma and pathology across study sites in order to contrast selective pressures reflected by the skeleton which may differ geographically, temporally, and between species.

Chapter 3

A Taphonomic Study of Two Chimpanzee Infanticide Victims from Gombe National Park, Tanzania

SUMMARY

Infanticide in chimpanzees is a complex phenomenon; behavior varies considerably among cases. It is similar in some respects to monkey hunting and in others to conspecific aggression directed at non-infant chimpanzees. Whether infanticide more closely resembles one or the other may provide further insight into how infants are regarded by chimpanzees as well as chimpanzee aggression. I used taphonomic and regression analyses in an attempt to discover how closely infanticide resembles hunting versus conspecific aggression.

The regression analysis uses previously published data on infanticides to assess whether the number of adult males present, time spent consuming, infant's age, infant's sex, or infant's group status predicted degree of consumption. Only time spent consuming was significantly correlated.

The taphonomic analysis compares skeletal damage to two infanticide victims from Gombe National Park, Tanzania, with damage to monkey prey skeletons reported elsewhere (Pobiner *et al.*, 2007). For each infanticide victim, I calculated the number of identified specimens (NISP), minimum number of elements (MNE), bone survivorship, and bone fragmentation. Similarities between monkey prey and chimpanzee infanticide victims include crenulated fractures to long bones, incipient fractures on ribs, and compression fractures to the cranial vault. Differences included a much higher frequency

of vertebrae and phalanges in the chimpanzee infant sample compared with monkey prey. Because it is uncertain whether infanticide completely resembles either predation or other forms of conspecific aggression, it may be useful to think of infanticide in chimpanzees as a phenomenon distinct from either.

BACKGROUND

Infanticide (e.g. Arcadi and Wrangham, 1999), hunting (e.g. Boesch, 1994), and conspecific aggression (e.g. Wilson *et al.*, 2004) have been documented at most long-term chimpanzee (*Pan troglodytes*) study sites. Behavioral observation reveals both similarities and differences between infanticide and the other two phenomena.

Chimpanzee infanticide victims are sometimes eaten (Arcadi and Wrangham, 1999), suggesting a potential similarity to monkey hunting, in which the prey are usually completely consumed (Teleki, 1973). Conversely, chimpanzee infanticide victims are not always consumed (e.g. Watts *et al.*, 2002), or may be consumed only incompletely (e.g. Goodall, 1977). Non-infant chimpanzees killed by other chimpanzees are never consumed (Goodall, 1986). This suggests that infanticide in at least some instances may be more similar to other forms of conspecific aggression than to hunting, but it raises the question of why chimpanzee infanticide victims are eaten on some occasions but not others, and why the degree of consumption varies so much more than it does in a monkey hunting context. I used previously published data on chimpanzee infanticides in a regression analysis to determine what variables might influence whether or not, and to what degree, a chimpanzee infant is consumed. I also undertook a taphonomic analysis on 2 known chimpanzee infanticide victims from Gombe National Park, Tanzania, and

compared their skeletal damage patterns to that of red colobus preyed on and consumed by chimpanzees (reported by Pobiner *et al.*, 2007) as a first step towards a better understanding of how closely chimpanzee infant consumption resembles the consumption of other primates.

If chimpanzee infanticide is more like monkey hunting than other forms of conspecific aggression, perhaps a nutritionally-driven hypothesis for chimpanzee infanticide may deserve more attention than it has previously received. Conversely, if infanticide is more similar to conspecific aggression than to monkey hunting, the status of infant chimpanzees as similar to older chimpanzees may be considered.

Why Infanticide?

Infanticide, and cannibalism associated with it, has sometimes been interpreted as maladaptive, aberrant behavior (Dagg, 1998), and in some cases may indeed be due to deep environmental disturbance or human interference (Dellatore *et al.*, 2009). Infanticide has been documented, however, for a wide variety of species and in multiple contexts that can be considered adaptive. Four primary hypotheses for the adaptive significance of infanticide have been proposed: the nutritional hypothesis (e.g. Stone and Derocher, 2007), the sexual selection hypothesis (e.g. Hrdy, 1979), the competition-elimination hypothesis (e.g. Trumbo and Valletta, 2007), and the termination of parental investment hypothesis (e.g. Soltis, 2004)

The nutritional hypothesis includes cases in which infants or sub-adults are consumed to achieve an energetic benefit, and has been proposed for polar bears (Stone and Derocher, 2007), sea lions (Wilkinson *et al.*, 2000), and assassin bugs (Thomas and

Manica, 2003), among other species. Any time an infant is consumed, a nutritional benefit is gained, but in this hypothesis, the nutritional benefit is proposed as the primary driving force in this hypothesis, in contrast to cases where an additional adaptive or fitness benefit may be secured regardless of whether the infant is consumed.

The sexual selection hypothesis proposes that males kill infants in order to enhance their reproductive success. Depending on the age of the infant, a female who loses her infant may go into estrus or become sexually receptive much sooner than if she continued to care for an infant. If the infanticidal male takes over the group, or attracts new mates who have just lost their offspring, he is more likely to father offspring than he would otherwise have been. Infanticide subsequent to male take over of a group, or change in male status within the group, has been documented in lions (Packer, 2000), alpine marmots (Coulon *et al.*, 1995), bank voles (Ylönen *et al.*, 1997), langurs (Borries, 1997; Hrdy, 1979; Moore, 1999; Vogel and Loch, 1984), proboscis monkeys (Agoramoorthy and Hsu, 2005), hamadryas baboons (Swedell and Tesfaye, 2003), patas monkeys (Enstam *et al.*, 2002), Japanese macaques (Soltis *et al.*, 2000), ursine colobus monkeys (Teichroeb and Sicotte, 2008), capuchin monkeys (Fedigan, 2003; Manson *et al.*, 2004; Valderrama *et al.*, 1990), blue monkeys (Fairgrieve, 1995), crab-eating monkeys (Bayart, 1985), and geladas (Beehner and Bergman, 2008; Mori *et al.*, 1997). Infanticide in mountain gorillas can occur after male take over (Watts, 1989) or as a means by which a male gorilla attracts females to his social group. The reproductive advantage to a female gorilla who joins the social group of the male who killed her infant may be to increase her rank in the new group, as relative arrival time in the group has a powerful influence on female rank and therefore reproductive success (Fossey, 1984).

Two cases of a female chimpanzee associating more closely with, and being more sexually receptive to, males who killed her infant have been recorded (Arcadi and Wrangham, 1999; Hamai *et al.*, 1992), though this is rare. In the context of intra-community infanticide, chimpanzee males are more likely to kill the infants of peripheral females, recent immigrants, or females whose infants are not as likely to have been sired by the males of that community. Because male chimpanzees are philopatric, infants sired by males from the same community may be likely to increase inclusive fitness (Arcadi and Wrangham, 1999; Kawanaka, 1981; Nishida and Kawanaka, 1985).

The competition-elimination hypothesis for infanticide proposes that killing the infant of a conspecific reduces future competition for resources and / or mates for the perpetrators and their offspring. This hypothesis has been proposed for various rodents (Tuomi *et al.*, 1997), reed warblers (Hansson *et al.*, 1997), badgers (Lups and Roper, 1990), brown bears (Hessing and Aumiller, 1994), bank voles (Ylönen *et al.*, 1997), some lemurs (Jolly *et al.*, 2000), and marmosets (Digby, 1995; Martins Bezerra *et al.*, 2007; Melo *et al.*, 2003; Roda and Pontes, 1998), among other species.

In chimpanzees, between-group infanticides have been proposed to decrease a neighboring community's population and expand the territory of the infanticidal males (Kutsukake and Matsusaka, 2002; Newton-Fisher, 1999; Sherrow and Amsler, 2007). Territory expansion in chimpanzees can mean better access to food resources as well as increased access to females for the males (Goodall, 1986; Sherrow and Amsler, 2007; Watts *et al.*, 2002; Williams *et al.*, 2004). Within-group infanticide, particularly those perpetrated by adult females (Goodall, 1977), has also been hypothesized to reduce

competition for food resources, especially in the central foraging ranges of high-ranking females (Goodall, 1986; Hiraiwa-Hasegawa and Hasegawa, 1994; Pusey *et al.*, 2008).

Terminating parental investment when the infant is of poor quality and therefore unlikely to survive and reproduce has also been proposed as an adaptive explanation for infanticide. In addition, parental investment termination may occur when the risks of continued investment are very high: if continued investment would mean a reduction in the fitness level of the parent, or if extrinsic factors (e.g. predation risk) mean the investment would not contribute to fitness. In these cases, the parent kills the offspring and the offspring are usually at least partially consumed (Klug and Bonsall, 2007). Among primates, behaviors attributable to the termination of parental investment hypothesis have been observed in saddle back tamarins (Herrera *et al.*, 2000) and humans (Hill and Ball, 1996; Soltis, 2004), but not in chimpanzees. A case of a mother cannibalizing her own infant, along with other members of her community, has been reported for bonobos, but the cause of the infant's death is unknown (Fowler and Hohmann, 2010). The unusual (for apes) consumption of the infant by the mother provides some support for the investment termination hypothesis.

The competition-elimination hypothesis and the sexual selection hypothesis are the most frequently cited hypotheses for cases of chimpanzee infanticide.

Comparing chimpanzee infanticide to other forms of conspecific aggression and to predation

Encounters between chimpanzees of different communities are often aggressive, though estrous females are usually an exception to this (Goodall, 1986). The males of a

chimpanzee community defend a territory, engaging in border patrols and attacking the members of other communities when the situation is numerically advantageous to the attackers (Wilson *et al.*, 2004; Wilson and Wrangham, 2003). Chimpanzees who are attacked during these inter-community disputes are sometimes killed (Goodall, 1986; Watts *et al.*, 2006; Williams *et al.*, 2008); the goal of this type of encounter seems to be incapacitation of the victim (Goodall, 1986).

This raises the question of whether infanticide is simply an extension of this kind of aggressive behavior. Perhaps it is only one form of generalized aggression towards competitors rather than a separate adaptive strategy. Whether or not an infant is the primary target or collateral damage in an attack on an adult may help to resolve this issue. Among chimpanzees, and some other primate species, infants are, in fact, observed to be the primary targets of attacks (Kutsukake and Matsusaka, 2002; Watts *et al.*, 2002). A chimpanzee mother may be relatively unharmed as her infant is pulled away from her, sustaining injuries that primarily occur as she protects her infant (Hamai *et al.*, 1992). In some cases, the attack on the mother ceases once the infant is seized (Goodall, 1977; Sherrow and Amsler, 2007) In lemurs, an infant separated from mother may be stalked before being attacked, making it clear that the infant is the target. (Jolly *et al.*, 2000).

Conversely, both in chimpanzees and other primate species (Jolly *et al.*, 2000), the mother may be the primary target, and the infant may be killed as a result of the attack on the mother (Arcadi and Wrangham, 1999; Goodall, 1977; Goodall, 1986), or it may be unclear whether the mother or the infant was the primary target (Wilson *et al.*, 2004). This may lead us to question to what degree attacks on infants resemble attacks targeted at adults and adolescents.

A primary difference between an attack on an infant compared to an older chimpanzee is body size; some behaviors commonly seen in infant killing (e.g. snatching from the mother, flailing the body) would not be possible with a larger chimpanzee. Still, attack victims of all ages are stamped upon, hit, kicked, dragged, and bitten. Infant chimpanzees who are killed are often at least partially eaten (Arcadi and Wrangham, 1999). Adult and adolescent chimpanzees killed by other chimpanzees are not eaten (Goodall, 1986; Williams *et al.*, 2008). This suggests that at least some differences may exist between killing infant chimpanzees and chimpanzees of other ages.

The hunting and consumption of monkey prey is another widespread behavior in chimpanzees, and it bears at least some superficial resemblances to conspecific attack in general and infanticide in particular, in that another organism is killed and may be consumed. In chimpanzees, predatory and conspecific aggression can have similar behaviors (Goodall, 1986), though Goodall (*Ibid*.) speculates that aggressive feelings towards prey are likely to be absent unless the prey fight back. Behavior that immediately precedes hunting in chimpanzees is characterized by a blank expression, a fixed stare, tense posture, and partial piloerection. The nature of the stare and the stare's target can usually be used by experienced observers to distinguish between a prelude to conspecific agonistic encounters versus hunting, but not always (Teleki, 1973). The context of an event (i.e. observing who the victim actually is) is used to distinguish the two types of behavior (Stanford, 1998). This may indicate that distinguishing these two types of behavior in chimpanzees will be difficult. Research in other mammals, however, particularly cats, suggests that different areas of the brain are activated and different behaviors are displayed during predation episodes versus aggressive behaviors directed at

conspecifics (Leyhausen, 1979). In humans and primates, a distinction is drawn between offensive and defensive aggression (Blanchard and Blanchard, 2003; Honess and Marin, 2006). Defensive aggression is characterized by threat gestures, activates the right frontal area of the brain, and results in elevated plasma cortisol levels. In contrast, offensive aggression is characterized by spontaneous attack, accompanied by elevated plasma testosterone as well as cortisol levels, and decreased levels of serotonin in the cerebrospinal fluid (Honess and Marin, 2006). This may be in contrast to observed behaviors in chimpanzees that are very similar during hunting and conspecific aggression (Goodall, 1986; Wrangham and Peterson, 1996)

Several questions are raised if we assume that predation on monkeys by chimpanzees is different from conspecific aggression, including whether this applies equally to chimpanzees of all ages who are killed by other chimpanzees, and what the consumption of chimpanzee infants after being killed might tell us about chimpanzee aggression, especially since chimpanzees of other ages are not consumed. I consulted the behavioral record in an attempt to answer these questions, and to better understand the phenomenon of infanticide and subsequent cannibalism in chimpanzees also compared consumption patterns between chimpanzee infants and monkey prey. I examined both skeletal and behavioral evidence to test whether there may be a distinction between infant versus adult killing in chimpanzees, and to evaluate whether there are significant differences in the killing and consumption of chimpanzee infants and non-chimpanzee primate prey.

It is my hope that the evidence presented here will inspire continued collection and preservation of skeletal material from hunting and infanticide contexts at long-term

primate research sites in order to better answer taphonomic questions and the behavioral issues they address in the future.

MATERIALS AND METHODS

I present a brief review of the literature on chimpanzee infanticide and predation, comparing and contrasting the two phenomena. In reviewing cases of chimpanzee infanticide, I evaluated whether degree of consumption is influenced by the number of adult males present at the infanticide, the amount of time spent consuming the infant, the age of the infant, the sex of the infant, and whether or not the infant belonged to the community of the infanticidal chimpanzees (infant group status). I used regression analysis with the statistical program Arc (Cook and Weisberg, 1999) to test which predictors affect degree of consumption. I expressed degree of consumption as either fully consumed, partially consumed, or not consumed; categorical data in the response are not ideal for regression analysis, so I evaluated each potential predictor separately and examined correlation coefficients.

I also present a taphonomic analysis of 2 chimpanzee infanticide victims from Gombe National Park, Tanzania, in order to compare and contrast the damage patterns observable on these skeletons with those of monkey prey consumed by chimpanzees. The primary point of comparison is Pobiner *et al.'s* (2007) analysis of the remains of red colobus prey consumed by chimpanzees at Kibale National Park, Uganda. This will be referred to as the "prey sample" and all data cited from that study may be attributed to Pobiner and colleagues. The results from the study of the chimpanzee infants are necessarily of a preliminary nature due to the small sample size, but it is to be hoped that

sufficient interest and debate may be generated by this study to encourage skeletal preservation and study whenever possible at long-term primate behavioral research sites.

I examined 2 female infanticide victims from the Mitumba Community at Gombe National Park, Tanzania. Both infants were killed by males from the Kasekela Community to the south of Mitumba. Rejea was 3 months old when killed in 1993 (Wilson *et al.*, 2004), while Andromeda was 9 months old when killed in 2005 (M.L. Wilson, unpublished data).

At Gombe, the bodies of dead animals are recovered whenever possible for necropsy and soft tissue pathology assessment, as well as skeletal preservation and study. After necropsy, chimpanzees are buried for at least one year before exhumation during the dry season. Chimpanzees are buried in permeable bags to help ensure complete recovery of all skeletal elements. After exhumation, skeletons are carefully cleaned in water with a soft brush, left to dry thoroughly, and stored in mosquito net bags to help guard against insects.

For this study, I identified the skeletal elements for the infants, and examined each element under diffuse light for surface modification using a 10x hand lens. The number of bone specimens identifiable to element and taxon (Number of Identified Specimens, or NISP) and the minimum number of complete skeletal elements represented by the assemblage (Minimum Number of Elements, or MNE), which includes both partial and complete bones, were recorded for both infants. Percentages of (%)NISP and (%)MNE were calculated: this is the percentage of the assemblage made up of a particular element (e.g. Rejea has a total of 164 identified specimens (NISP), and 25 of these are cranial fragments; the %NISP for the cranium is ≈ 16%. See results section for

complete data.). Bone survivorship, and bone fragmentation were also calculated after Pobiner *et al.* (2007) for Rejea and Andromeda separately, as well as both infants together.

Adult red colobus at Gombe weigh 9-12 kg (Stanford *et al.*, 1994a), and chimpanzees average about 1.7 kg at birth (in captivity) (Gavan, 1971). Infant chimpanzees and sub-adult colobus are therefore in the same size class (size class 1, per Brain, 1981) and it is reasonable to compare damage to their skeletons inflicted by the same species of killer.

Infanticide narratives

Rejea

Rejea's death has been described in detail elsewhere (Wilson *et al.*, 2004), but a brief overview is presented here.

Adult males from Kasekela surrounded Rafiki and her infant (Rejea) and attacked, biting Rejea on her head, ears, fingers, and toes. Rakifi attempted to protect Rejea by holding her close.

Goblin possibly defended Rafiki by kicking Apollo, Gimble, and Tubi (all adult males), who were attacking Rafiki.

Prof (adult male) killed the infant with a bite to the stomach while Rafiki was still holding Rejea. Ten minutes later Prof was able to snatch Rejea from Rafiki. Patti (adult female) and Prof then beat Rejea with their hands.

Rafiki was still being attacked 20 minutes after Rejea was bitten, but was eventually able to flee. The Kasekela males attempted to pursue, but could not find her; they then left with Rejea.

Goblin took Rejea from Apollo and began to eat Rejea. Forty-five minutes of eating ensued in which Goblin, Gimble, and Pax were observed to eat while others watched (*Ibid*.). No further details on order of consumption were described.

Necropsy data are not available for Rejea.

Andromeda

Andromeda's death has been cited in other studies (Williams *et al.*, 2008; Wrangham *et al.*, 2006), and will be thoroughly described in a forthcoming publication. An overview is presented here. Though the infanticide was not directly observed, events immediately preceding and following were directly observed (M.L. Wilson, unpublished data).

On the 13th of August, 2005, Kasekela chimpanzees heard and followed sounds of Mitumba chimpanzees. Eventually a group of Kasekela males found a group of Mitumba females, and presumably grabbed an infant (Andromeda) (this was not observed).

The Kasekela males left after calls from Mitumba males were heard by observers; by this time the Kasekela males had dropped or had the infant snatched from them by Mitumba males. Mitumba males were then seen with the dead infant. Edgar, an adult male, displayed using the body. The Mitumba chimpanzees remained agitated, engaging in buttress drumming and displaying. Edgar took Andromeda into a tree and groomed the body. He left her in the tree.

Bima (a juvenile female) came and inspected Andromeda, and then dragged her along as the group traveled. Bima fell behind the others after a few minutes and abandoned Andromeda's body; it was recovered by researchers.

Of interest is the fact that (adult male) Apollo was in the group of Kasekela males who killed Andromeda, and is (at least) the half brother of Aphro, Andromeda's mother (*Ibid.).*

Andromeda was not consumed by chimpanzees, but several peri-mortem wounds were described during necropsy. These include: a large, deep wound on the medial aspect of the left lower extremity extending all the way from the inferior, medial side of the thigh to the inguinal area, a wound on the superior aspect of the left foot, a wound on the right hand, two wounds on the lumbar area, wounds near the right eye and right ear, and on the abdominal area. In addition, the right humerus and the frontal bone were fractured.

Review of Infanticide and Hunting in Chimpanzees

A review of the literature on chimpanzee hunting and infanticide reveals both interesting similarities and differences. Because chimpanzee hunting focuses on red colobus monkeys (*Procolobus badius* per Pobiner *et al.* (2007)) as prey (Boesch and Boesch-Achermann, 2000; Goodall, 1986; Uehara *et al.*, 1992), descriptions of hunting behavior as well as the skeletal damage that results from hunting and consumption will focus on this species.

The most common method of capture for both red colobus (Stanford *et al.*, 1994a; Stanford *et al.*, 1994b; Takahata *et al.*, 1984) and chimpanzee infants (Arcadi and

Wrangham, 1999) is to be snatched from the mother, and while adult male chimpanzees are most commonly involved in the capture, adult females and near-adult males may also be involved (Arcadi and Wrangham, 1999; Stanford *et al.*, 1994a). The time spent capturing is also similar in both instances, usually taking approximately twenty minutes (Teleki, 1973). Capture usually lasts between 2-20 minutes for chimpanzee infants (Arcadi and Wrangham, 1999), and up to thirty minutes for multiple monkey kills (Stanford *et al.*, 1994b), though usually lasting twenty minutes or less for single kills (Watts and Mitani, 2002).

The method of killing both red colobus prey and chimpanzee infants is often similar, usually involving a bite to the head or abdomen (Arcadi and Wrangham, 1999; Kawanaka, 1982; Stanford *et al.*, 1994b; Takahata *et al.*, 1984; Teleki, 1973; Watts *et al.*, 2002). When consumed, this process is comparable between the two species, with some sharing and begging taking place, and the belly and/or extremities the first body parts to be eaten (Hamai *et al.*, 1992; Kawanaka, 1982; Nishida *et al.*, 1979; Takahata *et al.*, 1984; Teleki, 1973).

Monkey hunting, capture, and consumption result in great excitement for chimpanzees (e.g. Stanford, 1996; Stanford *et al.*, 1994a; Stanford *et al.*, 1994b; Teleki, 1973). Similar behaviors have been noted in some infanticide incidents, and in fact chimpanzee behavior during infanticide has been directly compared to that observed while hunting (Hamai *et al.*, 1992; Sherrow and Amsler, 2007). Other resemblances include the reaction of the mother, who has never been observed to eat her own baby in an infanticide case, though she may sometimes stay to watch consumption, but who often flees from the consumption site (Arcadi and Wrangham, 1999; Hamai *et al.*, 1992;

Nishida and Kawanaka, 1985; Stanford, 1996; Stanford *et al.*, 1994a; Stanford *et al.*, 1994b; Teleki, 1973). Lastly, the entire body, including skin, bone, and fur, may be consumed (Kawanaka, 1981; Nishida *et al.*, 1979; Norikoshi, 1982; Stanford *et al.*, 1994a; Suzuki, 1971; Takahata *et al.*, 1984; Teleki, 1973; Watts and Mitani, 2000), though this is clearly not always the case as demonstrated by this study and that of Pobiner *et al.* (2007), who report skeletal remains from 57 of 65 prey animals killed and consumed by chimpanzees at Kibale National Park in Uganda.

Marked differences between behaviors during hunting versus infanticide incidents have also been observed. While snatching the infant from the mother is a common method of capture for both phenomena, infanticide instances differ in that the mother may be severely attacked in addition to the infant – not just as a means of acquiring the infant, but also as a target. In addition, chimpanzees may cooperate in order to acquire the infant and prevent the mother from attempting to reclaim the infant during infanticide attempts, while such direct cooperation is seldom observed during hunting (Arcadi and Wrangham, 1999).

Perhaps most interesting is the wide variation in reports on consumption in infanticide compared to hunting. Both behaviors have sometimes been described as being very similar, as noted above, but can also differ significantly. Chimpanzee infants, for example, are not always consumed. This may be because the infant is simply abandoned without being consumed (Arcadi and Wrangham, 1999; Sherrow and Amsler, 2007; Watts *et al.*, 2002) or because the infant was recovered by the mother or other members of the natal group (Hamai *et al.*, 1992; Kawanaka, 1981). Monkey prey are seldom abandoned without being consumed; observed cases of abandonment include the

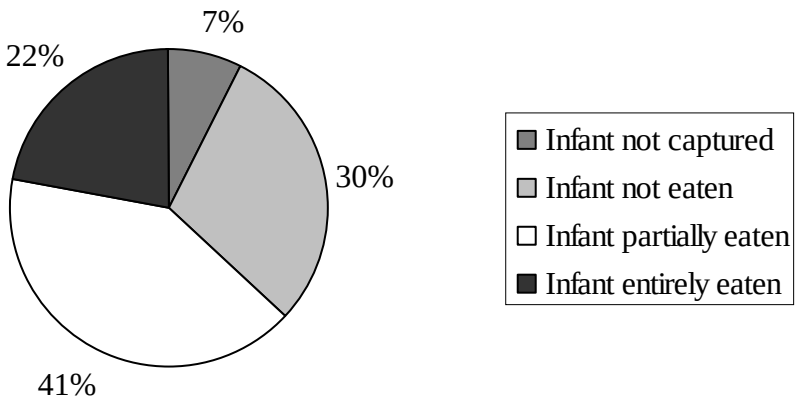
capture of non-preferred prey (infants or sub-adults are preferred as prey by most chimpanzee populations) (Nishida *et al.*, 1979) and when multiple monkeys are captured in a single hunt (Plummer and Stanford, 2000). Also, while consumption of chimpanzee infants can involve ingestion of the entire body, it is alternately described as being less complete and/or more intermittent than the consumption of monkey prey (Sherrow and Amsler, 2007; Watts and Mitani, 2000).

The completeness of monkey prey consumption is affected by the number of adult males present (Teleki, 1973); the regression analysis evaluates whether the number of adult males present influences the degree of consumption of chimpanzee infanticide victims. Table 3.1 is a summary of documented chimpanzee infanticide cases and the degree of consumption observed in each case. Figure 3.1 is a graphical summary of the 27 infanticide cases discussed here.

Table 3.1: Summary of degree of consumption of chimpanzee infanticide victims

Degree of infant chimpanzee consumption	**Number of reported cases**	**References**
Infanticide attempt not successful (infant not killed)	2	(Sherrow and Amsler, 2007; Wilson *et al.*, 2004)
Infant assumed dead and not observed to be eaten	3	(Arcadi and Wrangham, 1999; Kutsukake and Matsusaka, 2002; Sherrow and Amsler, 2007)
Infant recovered by mother / home group, but not consumed	2	(Wilson, *et al.*, in prep.) (Goodall, 1977)
Infant killed, but not eaten, and carried / groomed / "played" with by non-group males	1	(Goodall, 1977)
Killed infant abandoned without being consumed	2	(Watts *et al.*, 2002)
Infant is partially consumed	6	(Bygott, 1972; Goodall, 1977; Wilson *et al.*, 2004)
Infant at least partially consumed, but observers were not able to determine completeness	1	(Suzuki, 1971)
Infant at least partially consumed, but published report is ambiguous regarding completeness	4	(Goodall, 1977; Hamai *et al.*, 1992; Sherrow and Amsler, 2007; Wilson *et al.*, 2004)
Infant entirely consumed	6	(Hamai *et al.*, 1992; Kawanaka, 1981; Nishida and Kawanaka, 1985; Norikoshi, 1982; Watts and Mitani, 2000)

Figure 3.1: Frequency of complete consumption in 27 cases of chimpanzee infanticide



Time spent consuming monkeys versus chimpanzee infants has significant overlap and similarities in consumption pattern, but direct contrasts between monkey prey consumption and consumption of infanticide victims are frequently noted. Primate prey (non-chimpanzee) are usually consumed in three hours or less (Hamai *et al.*, 1992; Kawanaka, 1982; Nishida *et al.*, 1979), though the consumption of a baboon infant by an adult male chimpanzee at Gombe was recorded at nine hours (Goodall, 1986; Teleki, 1973).

Chimpanzee infants have been directly described as taking longer to be consumed than monkey prey (Norikoshi, 1982), and specific recorded consumption times for chimpanzee infants have commonly been reported at over three hours (Hamai *et al.*, 1992; Nishida and Kawanaka, 1985), five or six hours, and eight hours for a single chimpanzee infant (Kawanaka, 1981). In addition, display using the body of the targeted chimpanzee infant is relatively common compared to similar behavior with monkey prey,

and, contrastingly, the infant chimpanzee may also be groomed after death (Goodall, 1986; Kawanaka, 1981; Wilson *et al.*, 2004). One particular instance of infanticide from Gombe stands out as an extreme manifestation of this type of behavior. An infant from a stranger mother was killed by Kasekela males, and the body was then alternately incorporated into displays, transported, stashed, retrieved, and groomed by several adult and sub-adult males before finally being entirely discarded. This infant's body was not consumed (Goodall, 1977; Goodall, 1986).

The behavioral data published so far report both similarities and differences between hunting and infanticidal behavior in chimpanzees. Skeletal and taphonomic data have the potential to further illuminate the possible differences between chimpanzee conspecific versus predatory aggression.

RESULTS

Regression Analysis: What affects degree of consumption of chimpanzee infanticide victims?

Because consumption often accompanies infanticide, but not other conspecific killings in chimpanzees, perhaps this can inform us about possible differences between infant killing and killing chimpanzees of other ages. Completeness of consumption for non-chimpanzee primate prey is influenced by the number of adult males present (Teleki, 1973). The number of adult males present during infanticide incidences may also influence the degree of consumption of the infant. Because sub-adult red colobus are generally preferred to adults as prey (Stanford, 1998; Stanford *et al.*, 1994a), the age of the chimpanzee infanticide victim may also be relevant, with older infants being

consumed less completely or less frequently. I test whether the number of adult males present or the age of the infanticide victims predicts degree of consumption of chimpanzee infanticide victims. If either of these predictors is significant, some similarity in consumption associated with infanticide and monkey predation is indicated.

I also evaluate whether degree of consumption is influenced by the infanticide occurring within a group or between groups. The significance of this variable has the potential to inform us on adult versus infant killing in chimpanzees. If extra-group infants are consumed less frequently, this may be an indication that inter-group infanticide is similar to other inter-group killings, in that older individuals are not consumed (adult chimpanzees are very rarely killed by members of their own community; this occurs much less frequently than inter-community adult killings). If extra-group infants are consumed more frequently, inter-group infanticide might be considered more similar to hunting behavior, in which the prey is nearly always consumed.

I was not able to consider sex of the infant as a predictor for consumption completeness, because relatively few of the reported infanticide cases for chimpanzees are infants of known sex (11/28). Table 3.2 summarizes the data used for the regression analysis. For simplicity, the degree of consumption was divided into the discrete categories: completely consumed, partially consumed, not consumed, and missing data. I chose to evaluate the individual predictors at the $\alpha = 0.1$ level, because of the restricted sample available for study. Results should be treated with caution because of the high alpha level, which increases the probability of a Type I Error.

Table 3.2: Summary of the factors that might influence the degree of consumption of chimpanzee infanticide victims

Number adult & adolescent males	infant age	infant sex	degree of consumption	time observed consuming	Infant's group status	Reference
0	3 weeks	female	partial	5 hours	intra-group	Goodall 1977
0	3 weeks	female	partial	not indicated	intra-group	Goodall 1977
0	3 weeks	male	partial	5 hours	intra-group	Goodall 1977
1	2 years	male	not consumed - retained by mother	na	intra-group	Arcadi and Wrangham, 1999
2	newborn	not known	complete	2.5 ours	inter-group	Kawanaka 1981
3	3 months	female	partial	25 min	inter-group	Wilson et al. 2004
3	not known	not known	complete	not indicated	inter-group	Nishida et al., 1979
3	1.5 years	not known	partial	not indicated	inter-group	Goodall 1977
4	3 months	male	complete	over 3 (1/3 of body already consumed when observations began)	intra-group	Nishida and Kawanaka 1985
4	2.5 years	male	not eaten, abandoned	na	inter-group	Kutsukake and Matsusaka 2002
4	5-6 months	male	complete	4 hours	intra-group	Hamai et al., 1992
4	5-6 months	male	not indicated	3 hours	intra-group	Hamai et al., 1992
4	not known	not known	complete	8+ hours (overnight)	intra-group	Norikoshi 1982
4	1 year	not known	not indicated	2 hours	inter-group	Wilson et al. 2004

Number adult & adolescent males	infant age	infant sex	degree of consumption	time observed consuming	Infant's group status	Reference
4	newborn	not known	unclear	2 hours	not known	Suzuki 1971
5	1.5 years	not known	partial	1.5 hours	intra-group	Bygott 1972
5	2.5 years	not noted	partial	minutes	inter-group	Goodall 1986
6	1.5 years	female	not eaten, "rescued"	na	inter-group	Goodall 1977
6	not indicated	not indicated	partial	13 minutes	inter-group	Sherrow and Amsler 2007
7	not known	not known	not consumed - retained by mother	na	inter-group	Sherrow and Amsler 2007
7	not known	not known	not eaten, abandoned	na	inter-group	Sherrow and Amsler 2007
9	under 1 year	not known	not indicated	not indicated	inter-group	Goodall 1986
10	2 years	not known	not eaten, abandoned	na	inter-group	Watts et al., 2002
10	2 years	not known	not eaten, abandoned	na	inter-group	Watts et al., 2002
11	small, age not clear	not known	complete	2.25 hours	inter-group	Watts and Mitani 2000
17	not clear	not clear	complete	2 days	inter-group	Watts and Mitani 2000
not observed	9 months	female	not consumed - rescued by natal group	na	inter-group	M.L. Wilson, unpublished data

I examined whether the variables hours spent consuming, infant group status, infant age, and number of adult males present were correlated with degree of consumption. Only hours spent consuming had a significant correlation ($R^2 = 0.233$, $p = 0.07$). Neither infant group status nor infant age were significantly correlated with degree of consumption ($R^2 \leq 0.014$, $p \geq 0.80$). The number of adult males present approached significance ($p = 0.12$), but the correlation coefficient is low: $R^2 = 0.092$. In addition, variation in hours spent consuming accounts for the variation in the number of adult males present ($R^2 = 0.44$, $p = 0.007$). That time spent consuming is a significant predictor (even though its correlation is low) is hardly surprising, given that to completely consume anything must take longer than not to consume it at all.

Taphonomic Analysis

As might be expected from an overview of the behavioral data, damage patterns between chimpanzee infanticide victims and red colobus prey exhibit some similarities while also differing in significant ways. Pobiner *et al.* (2007) reported a monkey prey sample (referred to as the "prey sample" here) dominated by cranial bones and long bones; both of these skeletal elements are common in the chimpanzee infant sample examined here. Table 3.3 gives Minimum number of elements (MNE) and number of identified specimens (NISP) values for Rejea, Table 3.4 for Andromeda, and Table 3.5 for both infants combined.

Table 3.3: Skeletal elements for Rejea - NISP, MNE, bone survivorship, and degree of fragmentation (NISP/MNE)

Skeletal element	NISP Rejea	% total NISP Rejea	MNE Rejea	% MNE Rejea	% MNE survivorship Rejea (observed / expected MNE)	Fragmentation (NISP/MNE) Rejea
cranium	25	0.16	1	0.01	1	25
mandible	1	0.006	1	0.01	1	1
clavicle	1	0.006	1	0.01	0.5	1
scapula	1	0.006	1	0.01	0.5	1
humerus	1	0.006	1	0.01	0.5	1
Radius	2	0.012	2	0.02	1	1
Ulna	2	0.012	2	0.02	1	1
os coxa	6	0.037	2	0.02	1	3
Femur	3	0.019	2	0.02	1	1.5
Tibia	2	0.012	2	0.02	1	1
Fibula	1	0.006	1	0.01	1	1
Podials	2	0.012	2	0.02	0.04	1
metapodials	13	0.08	10	0.11	0.5	1.3
phalanges	23	0.14	23	0.24	0.41	1
Ribs	45	0.28	20	0.21	0.77	2.25
Vertebrae (all)	36	0.22	24	0.25	0.83	1.5
Total	164		95			

Table 3.4: Skeletal elements for Andromeda - NISP, MNE, bone survivorship, and degree of fragmentation (NISP/MNE)

Skeletal element	NISP Andromeda	% total NISP Andromeda	MNE Andromeda	% MNE Andromeda	% MNE survivorship Andromeda (observed / expected MNE)	Fragmentation (NISP/MNE) Andromeda
cranium	24	0.14	1	0.01	1	24
mandible	1	0.006	1	0.01	1	1
clavicle	2	0.01	2	0.02	1	1
scapula	2	0.01	2	0.02	1	1
humerus	2	0.01	2	0.02	1	1
Radius	2	0.01	2	0.02	1	1
Ulna	2	0.01	2	0.02	1	1
os coxa	6	0.04	2	0.02	1	3
Femur	2	0.01	2	0.02	1	1
Tibia	3	0.01	2	0.02	1	1.5
Fibula	2	0.01	2	0.02	1	1
Podials	0	0	0	0	0	n/a
metapodials	15	0.09	15	0.16	0.75	1
phalanges	33	0.19	22	0.23	0.39	1.5
Ribs	51	0.3	15	0.16	0.58	3.4
vertebrae (all)	23	0.14	23	0.24	0.79	1
Total	170		95			

Table 3.5: Skeletal elements for Rejea and Andromeda combined - NISP, MNE, bone survivorship, and degree of fragmentation (NISP/MNE)

skeletal element	NISP total	% total NISP total	MNE total	% MNE total	% MNE survivorship total (observed / expected MNE)	Fragmentation (NISP/MNE) total
cranium	49	0.15	2	0.01	1	25.5
mandible	2	0.006	2	0.01	1	1
clavicle	3	0.009	3	0.02	0.75	1
scapula	3	0.009	3	0.02	0.75	1
humerus	3	0.009	3	0.02	0.75	1
Radius	4	0.01	4	0.02	1	1
Ulna	4	0.01	4	0.02	1	1
os coxa	12	0.04	4	0.02	1	3
Femur	5	0.01	4	0.02	1	1.25
Tibia	5	0.01	4	0.02	1	1.25
Fibula	3	0.009	3	0.02	0.75	1
Podials	2	0.006	2	0.01	0.03	1
metapodials	28	0.08	25	0.13	0.63	1.12
phalanges	56	0.17	45	0.24	0.4	1.24
Ribs	96	0.29	35	0.18	0.67	2.74
vertebrae (all)	59	0.18	47	0.25	0.81	1.26
Total	334		190			

Crenulated edges on the breaks of long bones are also common in both samples (Figure 3.2). Few skeletal elements in either assemblage exhibit tooth marks, though some punctures are evident on the crania (Figure 3.3) in both cases. Compression fractures to the cranial vault (Figure 3.4) and incipient fractures (Figure 3.5) on ribs are also common between the samples.

The scapulae and innominates show somewhat similar patterns of damage, including crenulated or frayed edges of breaks (Figure 3.6). The damage to these elements in the chimpanzee infants is, however, less extensive. While glenoid fossae in the prey sample were typically missing and possibly consumed, damage in the chimpanzee infants was restricted to the blade in the three recovered scapulae. All parts of the innominate were recovered for both of the chimpanzee infants, but only the ilium was recovered from the prey sample.

Figure 3.2: Rejea's femora, distal end missing from right, and shaft missing from left. Left femur exhibits crenulated edges.

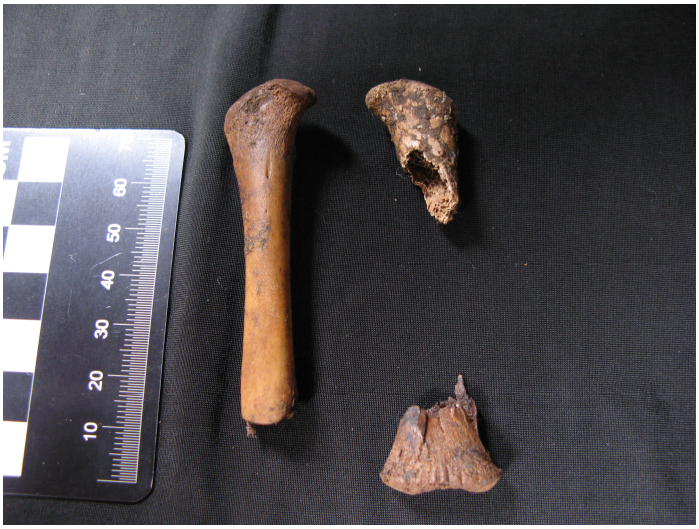


Figure 3.3: Andromeda's frontal bone, exhibiting canine puncture wounds.

Figure 3.4: Right parietal (Andromeda) with compression fracture.

Figure 3.5: Fractured ribs (Andromeda), with incipient fractures evident on the specimens second and third from the top of the image.

Figure 3.6: Rejea's left scapula, with damage to the blade. The right scapula did not survive.

Possibly one of the most drastic differences between the prey sample and the chimpanzee infant sample is that Pobiner *et al.* (2007) report no pre-caudal vertebrae from the hunted red colobus sample (n = 58 individuals, NISP = 405), while vertebrae (bodies and laminae) make up a significant portion of the chimpanzee infant sample, with a NISP of 59, or approximately 18% of the identified specimens (see Table 3.5). In addition, phalanges make up a higher %MNE in chimpanzee infants (24%) than in the prey sample (6%), and chimpanzee infant long bone ends are less commonly consumed compared to the prey sample. Overall, there is better bone survivorship (observed / expected MNE) of nearly all skeletal elements in the chimpanzee infant sample (Table 3.5) than in the prey sample, though podials make up a smaller % NISP in chimpanzee infants (n = 2) compared to the prey sample.

DISCUSSION

The relatively low number of podials recovered in the chimpanzee infant sample may reflect either developmental bias, preservation bias, or some combination of the two. Preservation bias may be of particular importance for Andromeda's skeleton, which has very friable, delicate cortical bone, suggesting that some skeletal elements maybe have been completely lost to decomposition during burial for soft tissue removal. This is probably due to the amount of time the body remained buried, which approached 4 years (3 years, 10 months). Rejea, though younger at time of death than Andromeda, exhibits less post-mortem damage to her skeleton. She was exhumed prior to 2008, but there are not specific data on exhumation timing; it is likely that Rejea was exhumed after spending less time in the ground than Andromeda.

Since Rejea was consumed for 45 minutes and Andromeda was not at all consumed, an interesting opportunity presents itself to contrast damage accrued to the skeleton during the act of consumption versus only the act of killing. Compression fractures to both crania are evident, which is consistent with the preferred kill method of infanticidal chimpanzees (a bite to the head or abdomen). Incipient rib fractures are also present in both individuals, and are possibly the result of display rather than consumption behavior in Andromeda. Both individuals also exhibit crenulated breaks on the long bones, though break type may be obscured in some cases on Andromeda due to poor preservation. A larger sample of chimpanzee infanticide victims would be useful in parsing out peri- versus postmortem damage to Andromeda's skeleton. Because she was not consumed, Andromeda shows both better bone survivorship and less bone fragmentation than Rejea.

Tappen and Wrangham (2000) report high proportions of monkey hand and foot bones in chimpanzee fecal samples (relative to other skeletal elements of prey), suggesting that these elements are often consumed. The fact that phalanges were recovered from Andromeda and Rejea, as well as the presence of numerous vertebrae or vertebrae fragments suggest less intense consumption of chimpanzee infants in these cases compared to monkey prey.

It should be noted, however, that many observations of infanticide describe the complete consumption of the infant (including skin, hair, and bones) and that the chimpanzee infant included in the Pobiner *et al.* (2007) study is represented by only 4 cranial fragments. This clearly indicates that consumption of infanticide victims is not always less intensive than consumption of monkey prey. Thus far, the skeletal samples

available match the behavioral record in terms of diversity of practice in chimpanzee infanticide. The reasons for the wide disparity in consumption patterns between infanticide episodes can perhaps be traced to the diversity of the behavioral repertoire of chimpanzee infanticide relative to other mammals.

The skeletons of two infanticide victims for Gombe who were killed in 1975 provide more insight into the variation in skeletal damage patterns. A male, killed in October 1975, was partially consumed (Goodall, 1977), and likely to be approximately 9 months old at death (see Chapter 2). A female, killed in November 1975 was not at all consumed (Goodall, 1977) and was likely to be approximately 8 months old at death (see Chapter 2). Both infants belonged to mothers who were from either the Kalande or the Kahama community (M.L. Wilson, unpublished data); neither mother was known to researchers when the infants were killed (Goodall, 1977).

The female infant shows very little damage to her skeleton, which is mostly complete for a chimpanzee of this age. Some incipient fractures of her ribs are consistent with display behavior observed near the time of the attack (*Ibid.*), but other damage to her skeleton may be post-mortem (see Chapter 2). This infant, unlike Andromeda, was not observed to be bitten, and from descriptions of the infanticides seems overall to have had her body treated more gently than Andromeda near the time of her death. Andromeda was "rescued" by males of her own community while the female infant from 1975 was not; she was groomed and carried around by members of the attacking community. If there was a struggle over possession of Andromeda's body, this would account for the greater degree of perimortem trauma to her skeleton compared to the female infant from 1975, even though neither of these infants was consumed.

The male infant from 1975 was partially consumed (Goodall, 1977), and damage to his skeleton reflects this, including fragmented ribs, ribs with incipient fractures, incomplete long bones, and puncture wounds to his cranial vault. The damage patterns are qualitatively similar to those of Rejea, who was also partially consumed. Further more intensive examination of these two skeletons would be useful.

Infanticide in primates, and in chimpanzees in particular, is a complex phenomenon. To summarize what is described above, it may sometimes seem clear that an infant is the target of the attack, but not at others. Both male and female adults may be infanticidal, and both male and female infants are killed. Infanticide victims are sometimes eaten, but not always, and the degree of consumption varies widely between cases. Consumption may closely resemble behavior observed during consumption of other vertebrate prey, and other times may be more intermittent, or accompanied by display behavior, carrying of the dead infant, or grooming the dead infant.

Another piece of this puzzle is that cannibalism in chimpanzees is only directed at infants; adults and adolescents killed by other chimpanzees have not been observed to be consumed. This may reflect a preference for eating younger primates as prey, but not all chimpanzee communities prefer immature colobus to adult colobus (Taï chimpanzees catch and eat more adults (Boesch, 1994)). Continued diligence in long-term behavioral research, accompanied by skeletal analysis of chimpanzees with known life histories, is likely to be beneficial in informing us about possible differences in predatory versus conspecific aggression in chimpanzees.

The brief regression analysis also suggests that infanticide in chimpanzees may not be exactly like either monkey hunting or other forms of conspecific aggression. None

of the variables tested here that might link infanticide to one of the other behaviors were significant. Only time spent consuming the infant had a significant correlation with degree of consumption, and this is to be expected given that engaging in any activity takes time. That the number of adult males approached significance suggests that a future, larger sample size might be valuable for further investigation of this topic, but if the number of adult males present continues to co-vary with time spent consuming, then time spent consuming would be likely to account for most of the variation in degree of consumption introduced by the number of adult males present.

The consumption by chimpanzees of chimpanzee infants and monkey prey are similar in many, but not all, ways. Damage patterns to bones are similar between both groups, but marked differences include the recovery of numerous vertebrae and phalanges from the chimpanzee infant sample compared to the prey sample. Chimpanzee infants seem to be consumed less intensively than monkey prey, at least sometimes. While chimpanzee infanticide victims are sometimes abandoned, monkeys are almost never abandoned before being eaten. Consumption of chimpanzee infants may take longer (while still being less intensive) than monkey prey. Because chimpanzee infanticide occurs for diverse reasons, it is to be expected that patterns of skeletal damage resulting from infanticide will be equally diverse. The diversity of circumstances surrounding cases of chimpanzee infanticide is reflected in the ambiguous results of the regression analysis and the pattern of bone damage in chimpanzee infanticide victims compared to monkey prey.

Chapter 4

Severity of Enamel Hypoplasia in the Gombe Chimpanzees and its Relationship to Dominance Rank

Summary

Enamel hypoplasia is an indicator of stress events that occurred during tooth formation. Dominance rank in chimpanzees is correlated with many proxies for fitness. Determining whether dominance rank can help predict incidence of enamel hypoplasia in chimpanzees is therefore of interest for evaluating the utility of enamel hypoplasia in assessing social condition, creating a better understanding of the possible implications and consequences of this skeletal marker of stress, and further illuminating the life histories of individual chimpanzees. Severity of enamel hypoplasia on the mandibular canine is evaluated for 19 chimpanzees from Gombe National Park, Tanzania, with known sexes, ages, and dominance ranks. The relationship between enamel hypoplasia severity and dominance rank is modeled using regression analysis. Simple rank values were not significantly correlated with enamel hypoplasia frequency, but some evidence suggests that change in rank may be significant. No significant differences between males and females in hypoplasia frequency were found. Mother's rank may be a better predictor for enamel hypoplasia frequency in chimpanzees, as the adult canine develops during the period of dependence on the mother. Future studies should address whether maternal condition affects skeletal manifestations of stress events such as enamel hypoplasia.

Background

Enamel defects such as hypoplasias accumulate during tooth formation and represent periods of poor nutrition and/or disease during tooth formation (e.g. El Najjar *et al.*, 1978; Goodman and Rose, 1990). Enamel defects can also reflect specific stress events such as injuries (Schwartz *et al.*, 2006). As such, defects in dental enamel provide a non-specific record of events that caused a developmental disruption during tooth crown growth. How enamel hypoplasias relate to particular events, such as episodes of illness or food unavailability, as well as to other behaviorally observable measures of health, is in the early stages of investigation. Dominance rank is an important aspect of chimpanzee's social lives, and is correlated with many proxies for fitness such as reproductive success (Constable *et al.*, 2001; Pusey *et al.*, 1997; Wroblewski *et al.*, 2009) and access to preferred foods (Murray *et al.*, 2006; Pusey *et al.*, 2005). The relationship between rank and enamel hypoplasia can therefore contribute to an improved understanding of the potential effects of physiological insults that occur during development on a chimpanzee's fitness.

Hormone analyses in chimpanzees revealed that higher ranking males have higher urinary cortisol levels, and are therefore more stressed than lower ranking males. This is likely due to the high levels of agonistic encounters associated with achieving and maintaining high rank (Muller and Wrangham, 2004). Conversely, lower ranking females, especially immigrant females, are more stressed than higher ranking females, probably because of the elevated intra-sexual competition experienced by these chimpanzees as they integrate into a new community (Kahlenberg *et al.*, 2008). In

addition, higher ranking females have better access to resourses and therefore experience less nutritional stress (Pusey *et al.*, 2005).

I aim to further explore direction of causality between rank and stress. For example, does high rank result in elevated cortisol levels, or do elevated cortisol levels contribute in some way to achieving high rank (perhaps because of an improved innate tolerance for stress)? If enamel hypoplasias, which accumulate during early development, correlate well with dominance rank achieved later in life, it may be possible to determine direction of causality. Are chimpanzees with higher levels of developmental stress more or less likely to achieve high rank? I examine this question in relationship to the frequency of enamel hypoplasia on the adult mandibular canine.

Enamel hypoplasia has long been utilized as a general indicator of physiological stress in a variety of ape skeletons (Corruccini *et al.*, 2005; El Najjar *et al.*, 1978; Guatelli-Steinberg, 2000; e.g. Hannibal and Guatelli-Steinberg, 2005; Lukacs, 2001; Skinner and Hopwood, 2004). The anterior dentition tends to be more severely affected than the molars and pre-molars, and males are often more severely affected than females, particularly in the canine (Berbesque and Doran, 2008b). Whether males actually experience more stress than females or whether the male canine is simply able to reflect more stress events because of its longer duration of growth (and therefore longer window of time in which defects can accumulate) is unclear (*Ibid*.).

In chimpanzees, the adult canines (both maxillary and mandibular) form between approximately .38 and 6.93 years (Reid *et al.*, 1998a). Male canines take, on average, a year longer to completely form, though there is significant overlap in crown formation

times between males and females, just as crown heights also overlap (Schwartz and Dean, 2001).

Using regression analysis, I tested whether:

1. Sex is a significant predictor of enamel hypoplasia frequency.
2. Dominance rank is a significant predictor of enamel hypoplasia frequency.

I also tested whether age and wear stage (after Kilgore, 1989) was a significant predictor of enamel hypoplasia frequency. Age is correlated with tooth wear (Kilgore, 1989), and labial wear on the canine has the potential to affect the ability to observe enamel hypoplasias. Because this study includes canines in a variety of mild to moderate wear stages, assessing whether age and tooth wear affected enamel hypoplasia frequency is relevant for correctly interpreting the results.

Materials and Methods

Fifty years of research at Gombe National Park in Tanzania have yielded both a tremendous amount of long term behavioral data on chimpanzees, as well as the largest wild chimpanzee skeletal collection for which both behavioral and skeletal data are available. Part of the skeletal collection, those individuals who died prior to 1987, is currently housed at the University of Minnesota, in the Evolutionary Anthropology Laboratory. The other part of the sample is curated in Gombe National Park, Tanzania, which is located on the shore of Lake Tanganyika in western Tanzania (refer to Chapter 1 for information on housing location for each skeleton).

Skeletons of chimpanzees who died prior to 1987 were cleaned according to protocols laid out elsewhere (Zihlman *et al.*, 1990). Skeletons of chimpanzees who died after 1987 were processed in a different way. Following necropsy (K. Terio, unpublished data) bodies were buried in a permeable bag and exhumed after at least one year. Skeletons were then carefully cleaned using water and a soft brush and allowed to thoroughly dry.

Because some chimpanzees at Gombe are infected with a strain of the Simian Immunodeficiency Virus (SIVcpz) (Keele *et al.*, 2009; Santiago *et al.*, 2002), an extra step was added to the cleaning procedure in order to reduce the risk of possible transmission of the virus to skeletal researchers. Chimpanzees known to be infected with SIVcpz were briefly (2 minutes) rinsed in a mild bleach solution after initial cleaning, and then subsequently soaked in water to remove bleach residue. (SIV status of individual chimpanzees has been published elsewhere (Keele *et al.*, 2009), and is summarized in Table 2.11.

Each skeleton was then inventoried and stored in mosquito net bags (to reduce the risk of insect damage to the bones in storage) in a secure location at Gombe National Park. Nineteen chimpanzees (11 females, 8 males) with an observable adult canine are included in this study (Table 4.1). Only chimpanzees with lightly to moderately worn canines are included so that lingual enamel could be observed.

Table 4.1: Chimpanzees included in this study

Chimpanzee	Age	Sex	EH count	EH index	Wear stage	High Rank	Death Rank
Atlas	31.30	2.00	8.00	0.41	3.00	1.00	2.00
Charlie	39.94	2.00	3.00	0.14	2.00	1.00	1.00
Cusano	15.78	2.00	2.00	0.14	4.00	1.00	1.00
Echo	39.96	1.00	5.00	0.40	3.00	2.00	2.00
Gilka	25.59	1.00	4.00	0.35	6.00	3.00	3.00
Goblin	28.47	2.00	2.00	0.12	7.00	1.00	2.00
Humphrey	44.25	2.00	4.00	0.32	7.00	1.00	2.00
Jomeo	28.50	2.00	8.00	0.35	4.00	1.00	1.00
Kidevu?	24.00	1.00	6.00	0.48	2.00	2.00	2.00
Madam Bee	18.87	1.00	4.00	0.28	5.00	2.00	2.00
Melissa	25.87	1.00	4.00	0.33	5.00	2.00	2.00
Miff	27.20	1.00	5.00	0.40	4.00	2.00	2.00
Pallas	28.22	1.00	0.00	0.00	5.00	2.00	3.00
Passion	29.83	1.00	2.00	0.15	6.00	1.00	1.00
Patti	30.61	1.00	4.00	0.37	4.00	1.00	1.00
Satan	30.87	2.00	10.00	0.51	3.00	1.00	1.00
Sherehe	30.90	1.00	9.00	0.58	2.00	2.00	2.00
Vincent	34.93	2.00	7.00	0.51	3.00	1.00	3.00
Yolanda	36.31	1.00	4.00	0.23	1.00	2.00	2.00

Dominance rank data for this study were generously made available by Dr. Anne Pusey and have their basis in long-term behavioral research. Rank data are presented in Table 4.1 and are collated from Goodall (1986), Foster *et al.* (2009), Murray et al. (2007; Murray *et al.*, 2006), and unpublished data from Gombe Stream Research Center (GSRC). One (1) is used to indicate high rank, 2 for middle rank, and 3 for low rank. See Chapter 1 for sources on individual rank assignment. Because rank often changes over the course of a chimpanzee's life, highest achieved rank and rank at death are both considered here in an attempt to encompass rank variation.

Female dominance ranks are usually categorized as high ranking vs. middle ranking vs. low ranking (e.g. Pusey *et al.*, 2005). Male chimpanzees may be given ordinal ranks (1 – *n* adult males in the community) (Bygott, 1979; Goodall, 1986), but

these were collapsed into categories similar to the females' for the purpose of the statistical analyses undertaken in this study (in order to avoid having to divide the sample). Males ranked 1-4 are considered high ranking, 5-8 are considered middle ranking, and ranks 9 and above are low ranking. These categories were altered twice to account for a lower than usual number of males in a group. For example, Vincent was ranked 3rd of 3 adult male chimpanzees at the time of his death, and is therefore considered low ranking. Similarly, Jomeo's numerical rank during the year of his death could be considered either high or middle ranking because of the relatively small number of adult males at that time. Jomeo's relative rank (Jameson *et al.*, 1999) places him in the top third of adult male ranks, however, so he is counted as high ranking at time of death.

I scored enamel hypoplasias on the entire dentition using the modified Defects of Dental Enamel Index (Clarkson and O'Mullane, 1989), and recorded the total number of enamel defects, their location, type, and extent after visually examining teeth with a hand lens (10x magnification) (Hannibal and Guatelli-Steinberg, 2005; Lukacs, 2001). Prevalence of hypoplasia in each individual is expressed as the number of defects visible on the mandibular canine (Berbesque and Doran, 2008b), as this tooth is the most sensitive to physiological disturbance (***Ibid.***; Lukacs, 2001). Because it is also the tooth that takes the longest to form (Reid *et al.*, 1998a), it is therefore the most likely to record stress events.

The left mandibular canine was chosen randomly as the tooth on which this study focuses. The right mandibular canine was used in cases where the left was unavailable. One individual with canines (Humphrey) is represented by only a cranium. His left maxillary canine was therefore used. (The regression model was also created excluding

Humphrey, and no significant differences were found.) I counted the number of hypoplasias on the canine using gross examination (with hand lens and fingernail palpation) as well as digital photographs of the teeth (Figure 4.1), after Berbesque and Doran (2008).

(A) (B)

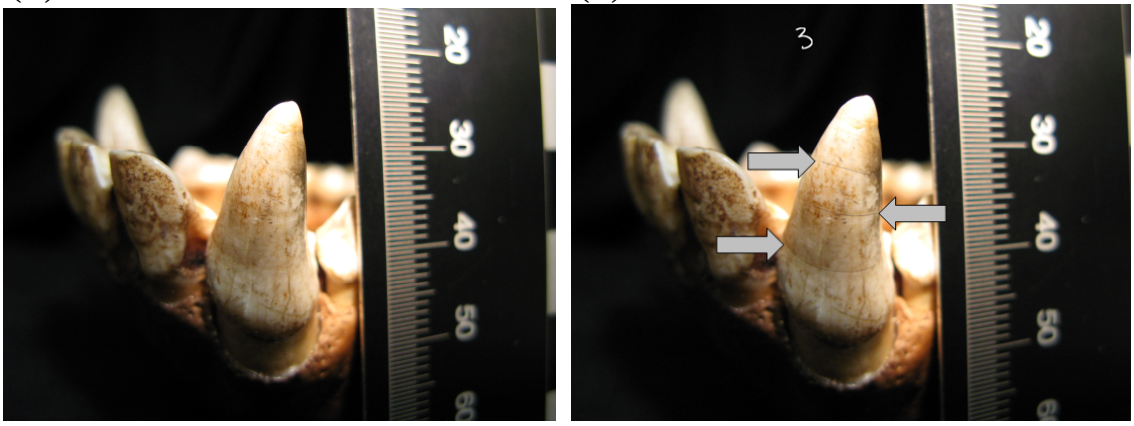


Figure 4.1: A) Charlie's left mandibular canine. B) The same canine with enamel hypoplasias indicated on the photograph.

Regression analysis models the relationship between severity of canine enamel hypoplasia and dominance rank. Both the raw counts of hypoplasias ("hypoplasia count") as well as a "hypoplasia index" were analyzed. The "hypoplasia index" is the number of observed hypoplasias on the canine divided by the crown height. Crown height was measured to the nearest .10mm using Hillson-Fitzgerald digital dental calipers. Measurements were carried out independently and repeated five times, with the mean of the five measurements used to calculate hypoplasia index.

I scored wear on the canine according to the system established for the Gombe chimpanzees by Kilgore (1989). I used Kilgore's (*Ibid*.) wear scores for Charlie, Gilka, Humphrey, Pallas, and Passion, and scored wear for the other chimpanzees included in this study myself.

I analyzed the relationship between enamel hypoplasia severity, dominance rank, age, and sex using the statistical software Arc version 1.06 (Cook and Weisberg, 1999). I considered enamel hypoplasia index and enamel hypoplasia count and their correlation with age, sex, high rank, death rank, a predictor that considers the relationship between age and rank, and factors that describe a change in rank between highest achieved rank and rank at death. I used scatter plot matrices to visually evaluate potential relationships between each variable, eliminated co-varying variables, and tested each of the variables separately. Because response data expressed as percents or counts are usually in need of a variance-stabilizing transformation, I applied the arc-sine square-root transformation to the responses (*Ibid*.). T-tests checked for differences in hypoplasia frequency between males and females, and were carried out in R version 2.11.0 (www.r-project.org). I employed Welch's t-test because it does not require an assumption of equal variances in the two samples, a possible concern with small sample sizes (Welch, 1947).

Results

Enamel hypoplasia count is negatively correlated with tooth wear stage (R^2 = 0.24, p = 0.03), indicating that when less tooth is present, enamel hypoplasias are less common. I therefore focused my analysis on the enamel hypoplasia index, which accounts for variation in crown height due to wear. Enamel hypoplasia index is not

correlated with wear stage ($R^2 = 0.14$, $p = 0.11$). Age was also uncorrelated with enamel hypoplasia index ($R^2 = 0.02$, $p = 0.61$).

Sex ($R^2 = 0.00003$, $p = 0.98$), highest achieved rank ($R^2 = 0.004$, $p = 0.79$), rank at death ($R^2 = 0.001$, $p = 0.90$), a variable describing the interaction between age and rank ($R^2 = 0.01$, $p = 0.64$), factors describing change in rank from high to middle rank ($R^2 = 0.004$, $p = 0.80$), and from high to low rank ($R^2 = 0.07$, $p = 0.29$) were not significantly correlated with enamel hypoplasia index. The factor describing change in rank from middle to low ranking was significantly correlated with enamel hypoplasia index ($R^2 = 0.46$, $p = 0.001$) (Figure 4.2). It is important to note that this trend is based on a single data point (Pallas), so while the result is suggestive, it should be treated with great caution.

A box plot produced in R (Figure 4.3) shows that the mean enamel hypoplasia index is very similar for males and females, and that the ranges overlap. Welch two-sample t-tests for the difference in two means do not indicate a significant difference between males and females in either enamel hypoplasia count ($t = -0.95$, $df = 12.06$, p-value = 0.36) or enamel hypoplasia index ($t = 0.16$, $df = 14.97$, p-value = 0.87).

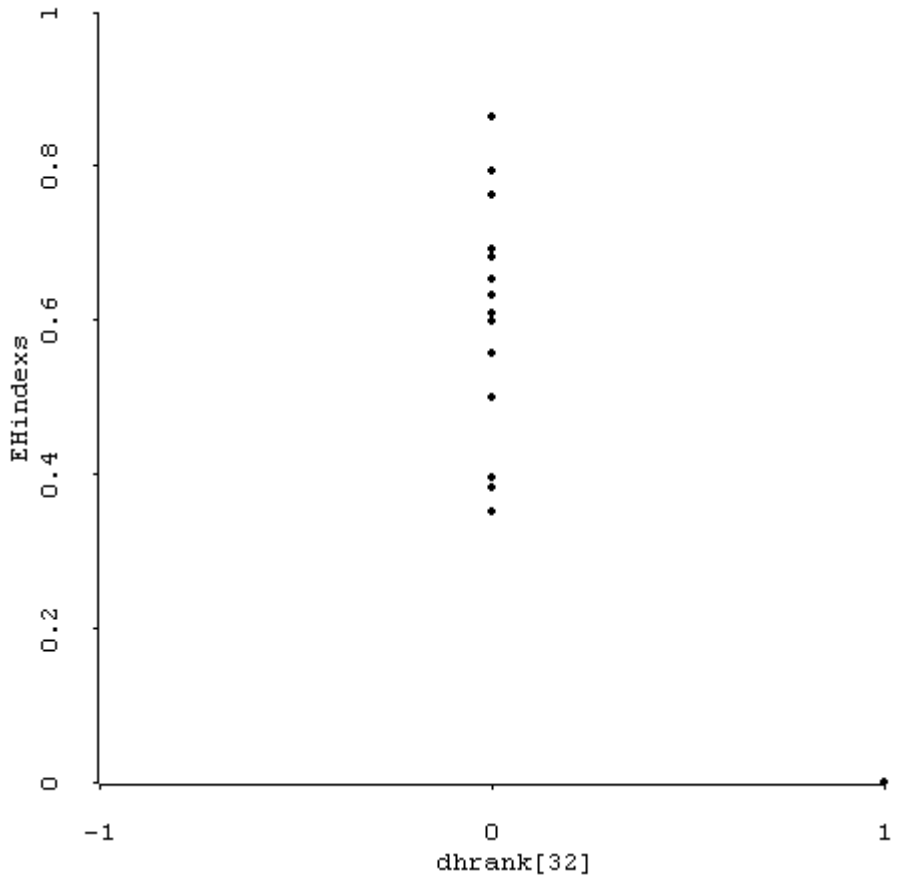


Figure 4.2: A plot showing the relationship between enamel hypoplasia index (EHindexs) (transformed data) and the factor describing a change in rank from middle to low ranking (dhrank[32]).

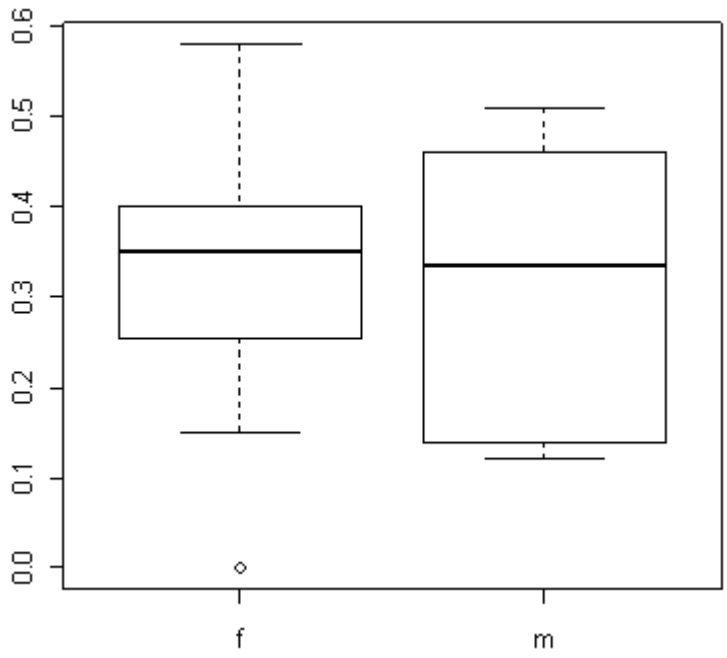


Figure 4.3: Boxplot of male versus female enamel hypoplasia indices.

Discussion

I examined the relationship between the frequency of stressful events that occurred early in chimpanzees' lives (measured by enamel hypoplasias) to the dominance ranks achieved later in life. This is useful for more fully understanding the consequences of early development on later life history as well as exploring the interaction between stress and rank in chimpanzees. I expected to find significant differences between male and female hypoplasia incidence, and that hypoplasia incidence would be correlated with adult dominance rank.

Statistical tests found no difference between males and females in the frequency of enamel hypoplasia on the mandibular canine. This was unexpected given the longer

crown formation time for the male canine (Schwartz and Dean, 2001), as well as the increased sensitivity to physiological stressors (e.g. illness) that has been frequently noted for male mammals (Guatelli-Steinberg and Lukacs, 1999). Because the sample is approximately evenly split between males and females (11 females, 8 males), the results are not likely to be affected by sampling bias. The overall sample is small, however, so these results must still be treated with caution. Future analysis with a larger sample could confirm whether the results presented here are more broadly applicable – both for male and female differences and the proposed interaction between rank and hypoplasia. At this point in time, evidence indicates that male and female chimpanzees at Gombe encounter the same levels of the kinds of stress that result in enamel hypoplasia during their early development. This is likely due to dependence on the mother during canine crown formation.

Permanent canine crown (both maxillary and mandibular teeth) formation in chimpanzees occurs between 0.38 to 6.93 years (Reid *et al.*, 1998a). Chimpanzee infants are dependent on and buffered from various potential stressors by their mothers until approximately 4 years of age, and continue to spend a large amount of time with her during the juvenile period (Goodall, 1986). The period of time when a chimpanzee is dependent on his or her mother for nutrition and comfort therefore largely coincides with the time when the adult canine forms. It would not be surprising to find that mother's rank has a significant effect on the number of stressors a young chimpanzee experiences, and therefore on the number of hypoplasias that form on the permanent canine. Too few chimpanzees (only 5 of 19) had available data on mother's rank at the time of their birth, and all of these mothers were middle ranking at the time of their infants' births (Gombe

Stream Research Center, unpublished data). I was therefore not able to consider mother's rank in this study. The exclusion of this variable helps to emphasize the continued importance of preserving and studying the skeletons of primates with known life histories, as larger sample sizes and longer term behavioral data will make more detailed analyses possible in future.

The number of stressors that result in an enamel hypoplasia is not significantly correlated with adult rank in chimpanzees. A change in rank from middle to low ranking did correlate with enamel hypoplasia index, but this is based on a single individual, Pallas. There are only 4 chimpanzees in the sample whose rank changed between highest achieved rank and rank at death: Pallas, Goblin, Atlas, and Vincent. Of interest is that none of the other rank change factors were significantly correlated with enamel hypoplasia index, and also that Pallas's enamel hypoplasia index is 0. This result is interesting as it indicates that more or less stress during early development has less of an effect on later life history than predicted. Reliance on mother during the time of canine development is likely also a reason that a chimpanzee's own highest achieved rank and rank at the time of death are poor predictors for the severity of hypoplasia on the canine. Mother's rank, as a proxy for her access to resources (Pusey *et al.*, 2005) and overall number of agonistic encounters (Kahlenberg *et al.*, 2008), may be more closely correlated with the formation of hypoplasias on her offspring's teeth. Higher ranking female chimpanzees have better access to resources and greater body weights than lower ranking females (Murray *et al.*, 2006; Pusey *et al.*, 2005), and lower ranking females experiences higher levels of agonistic encounters (Kahlenberg *et al.*, 2008). (Dominance rank data are not available for Pallas's mother, who was unknown to researchers.) Future research

may be able to address whether these observed behavioral trends result in more or fewer stress events recorded as enamel hypoplasias on offsprings' teeth. Such a study would only be possible if more chimpanzees had data on their mother's dominance rank. This is just one example of the further utility of long term behavioral research. If mother's rank is indeed correlated with the severity of enamel hypoplasia expressed in her offspring, this would constitute direct skeletal evidence for maternal condition affecting the quality of her offspring – a proxy for fitness. This opens the possibility that enamel hypoplasia itself could potentially be used to measure individual fitness.

Maternal rank was not found to correlate with cub survival in spotted hyenas, a species that, like chimpanzees, has high infant mortality and both intra- and inter-group infanticide (White, 2005). This suggests that the link between maternal rank and infant health and survivorship is probably complex. Infant health in chimpanzees is, however, likely to depend in some way on mother's health. Flo's last two offspring are good evidence of this. While her earlier offspring led relatively healthy and successful lives as the offspring of a high ranking female, Flint and Flame were both born during the later part of Flo's life (Goodall, 1986), when she was perhaps less able to feed herself due to extreme tooth wear (Kilgore, 1989) and therefore less able to adequately provide for her last offspring, Flame. Flame disappeared before he was 7 months old (Williams *et al.*, 2008), during a period of time when Flo was ill. Flint remained very dependent on his mother well past the usual age, even insisting on being carried like an infant after Flame's death. Flint died at age 8.5 years, not long after his mother finally succumbed (Goodall, 1986). Evaluation of Flint's skeleton revealed that he was smaller, had lower bone

density, and delayed dental development compared to other similarly-aged male chimpanzees (Zihlman *et al.*, 1990).

Examining maternal rank and offspring enamel hypoplasia incidence could provide quantitative skeletal evidence for a link between maternal health and status and offspring health. In addition, a larger sample of chimpanzees whose rank changed over the course of their lives could further illuminate the possible relationship between rank change and skeletal markers of stress. It is to be hoped that continued research at Gombe National Park and other long term primate research projects will enable the collection of the data necessary to address possible skeletal evidence for fitness.

Chapter 5

Fluctuating Asymmetry in the First and Second Permanent Molars of the Gombe Chimpanzees

Summary

Assessing whether developmental instability varies between life history stages has the potential to inform us about how selective pressures may change over time for an organism. Fluctuating asymmetry is one measure of developmental instability. To assess possible differences in developmental instability between the prenatal period and early infancy versus later infancy and the early juvenile period, fluctuating asymmetry was measured for the 1st and 2nd mandibular molars of chimpanzees from Gombe National Park, Tanzania. Maximum mesio-distal lengths were recorded for chimpanzees with both molars in full occlusion at the time of death, with 5 independent measurements. Tests for normality and dependence on size were carried out, and a two-way, mixed model analysis of variance (ANOVA) was used to measure fluctuating asymmetry, as well as to evaluate the possible effects of other kinds of asymmetry and measurement error. Levels of fluctuating asymmetry were significant for both teeth, but there is no evidence for a difference in fluctuating asymmetry levels between the 1st and 2nd molars. This may be due to factors such as quality of the trait evaluated, the limitations of the sample and statistical analyses, and overlap in developmental timing between the teeth rather than a real lack of difference in stress levels between early infancy and early juvenility in chimpanzees.

Introduction and Background

Fluctuating asymmetry is a deviation from symmetry in a bilateral structure, and reflects the inability to buffer against physiological stress, particularly during the growth period. Thus, an individual with a high level of fluctuating asymmetry was less able to buffer against stress than a more symmetrical conspecific (Grammer et al., 2003; Guatelli-Steinberg et al., 2006; Kohn and Bennet, 1986; Moller, 2006; Perzigian, 1977; Singh and Rosen, 2001; Thornhill and Gangestad, 2006; Van Dongen and Moller, 2007; Van Valen, 1962). In ideal circumstances in which other forms of asymmetry are absent, the left and right sides would be identical, but small fluctuations in the environment of development mean that the left and right sides of a bilateral trait are rarely exact mirrors. These tiny variations between sides may occur at the level of the cell, and are not necessarily constant (Klingenberg, 2003). These fluctuations are referred to as "developmental noise" (*Ibid*.: 14). Sensitivity to developmental noise may vary through time, by body part, and / or by individual. This sensitivity, or "the tendency to produce a morphological change in response to small developmental perturbation" is referred to as an organism's level of "developmental instability" (*Ibid*.: 15). In contrast, "developmental stability" is the capacity to experience developmental perturbation without resulting in a morphological response (*Ibid*.: 16). Fluctuating asymmetry is, in the absence of other kinds of asymmetry, a measure of the degree of developmental perturbation that led to a morphological change; fluctuating asymmetry is a measure of developmental instability. Levels of fluctuating asymmetry may be used to evaluate the level of developmental instability between groups, such as differences between two populations of the same species living in different environments (Van Dongen *et al.*,

1999), the deciduous versus permanent dentition, males versus females (Guatelli-Steinberg *et al.*, 2006), or people of different time periods (Palubeckaite and Jankauskas, 2001).

Fluctuating asymmetry differs from other forms of asymmetry in that it alone is an indicator of failure to buffer against stress. It differs from directional asymmetry and antisymmetry in that these phenomena are more reflective of normal development or habitual activity. Directional asymmetry occurs when one side consistently develops larger than the other (as in the mammalian heart), while antisymmetry describes size differences that target one side, though which side is affected may vary within a population (e.g. handedness) (Van Valen, 1962). In order to evaluate fluctuating asymmetry and therefore success in buffering against stress, it is necessary to distinguish between these various sources of asymmetry as well as to assess the effects of measurement error (Palmer and Strobeck, 1986). Table 5.1 summarizes the various types of asymmetry discussed here.

Table 5.1: Summary of types of asymmetry

Type of Asymmetry	Definition	Example	Is it a measure of developmental instability?[3]	Distribution
Directional asymmetry	One side is consistently larger than the other[1]	mammalian heart[1]	No	Skewed distribution[4]
Antisymmetry	One side is larger than the other, but which side is larger may vary within a population[1]	handedness[1]	No	Bi-modal distribution[4]
Environmental asymmetry	Microenvironmental variations that influence development[2]	mechanical loading that differs between sides[2]	No	Possibly bi-modal, could possibly resemble fluctuating asymmetry depending on source of environmental variation[2]
Fluctuating asymmetry	Left-right size differences due to developmental noise[3]	random left-right size differences in teeth[3]	Yes	Normally distributed, with a mean of 0[4]

(1: Van Valen, 1962; 2: Palmer and Strobeck, 2003; 3: Klingenberg, 2003; 4: Palmer and Strobeck, 1986)

Yet another kind of asymmetry may bias observed levels of fluctuating asymmetry, and this is "environmentally induced asymmetry," or variations in microenvironment between the left and right sides that might influence development. These effects cannot be separated from developmental noise unless evidence for varying microenvironments exists. It is therefore important to choose a trait that is not likely to be influenced by high levels of directional asymmetry, mechanical loading, or significant variations in microenvironment (Nijhout and Davidowitz, 2003; Palmer and Strobeck, 2003). The dentition is a reasonable candidate for such a trait. Although directional asymmetry has been recorded in the human dentition, teeth on one side of the mouth are

not consistently larger than those on the other side. The larger side varies between tooth pairs (e.g. the right deciduous canine is larger than the left, but the left deciduous first molar is larger than the right) rather than all of the left teeth being larger than the right, or vice versa. In addition, the population in which dental directional asymmetry was recorded experienced a high level of environmental stress, and it is hypothesized that directional asymmetry in this case may be a result of a disturbance in developmental timing between the left and right sides (Guatelli-Steinberg *et al.*, 2006). Evaluating directional asymmetry in any study remains relevant in order to be sure that asymmetries are indeed due to developmental instability rather than some other aspect of development.

The dentition should also provide reasonable traits for the study of fluctuating asymmetry in a skeletal collection because teeth, unlike bones, do not remodel in response to mechanical loading. Teeth are worn down through use, but assessing teeth with intact crowns with only light to moderate wear ought to make them sufficiently comparable between individual organisms that fluctuating asymmetry can be adequately assessed. I address this issue further in the discussion section.

Statistical tests have demonstrated that the size of the trait being measured may be correlated to the degree of fluctuating asymmetry recorded. Testing for a relationship between size of the trait and the difference in size between left and right is therefore necessary in order to control for this relationship if it exists (Palmer and Strobeck, 1986).

In comparing deciduous versus permanent dentition in a single human population from 1950s South Carolina, Guatelli-Steinberg and colleagues (2006) sought to test whether levels of fluctuating asymmetry were higher for the permanent dentition than for the deciduous dentition. This hypothesis was based on the assumption that humans are

better buffered from stress during early development *in utero* and while nursing than in later development. While deciduous teeth were documented as having higher levels of fluctuating asymmetry than their permanent successors in 7 of 12 pairs, only one of these differences was statistically significant. The authors hypothesize that the overall high levels of fluctuating asymmetry in this population mean that stress levels were high for most people most of the time, thus masking any variation between sexes and life history stages (Guatelli-Steinberg *et al.*, 2006).

Determining whether levels of fluctuating asymmetry vary throughout the course of an organism's life can tell us whether stressors are the same in all life history stages; this has the potential to illuminate how selective pressures may vary between life history stages. In chimpanzees, the 1st permanent molar beings crown formation *in utero* (starting between .05 and .15 years before birth), and continues until 2.5-3 years after birth (Table 5.2). The crown development time of the 2nd permanent molar overlaps with the first, but does not start to grow until nearly two years of age (1.67-1.95 years). The crown of the 2nd molar finishes growing at around 5.5 years (Reid *et al.*, 1998b). Chimpanzee infants are dependent on and buffered from various potential stressors by their mothers until approximately 4 years of age, and continue to spend a large amount of time with her during the juvenile period (Goodall, 1986). The period of dependence on mother largely coincides with the development of the 1st and 2nd permanent molars, but comparing these two teeth can tell us whether a chimpanzee is better buffered from stress *in utero* than while nursing. In addition, since weaning usually occurs before the crown of the 2nd molar has finished growing, but after the 1st molar is done forming, it is

possible to test whether this potentially stressful event affects the level of fluctuating asymmetry in the 2nd molar compared to the 1st.

Table 5.2: Crown formation times for the 1st and 2nd permanent mandibular molars in chimpanzees (Reid et al., 1998b).

Tooth	Age that crown formation begins (years)	Age that crown formation ends (years)
1st molar	-0.05 - .15	2.5 - 3
2nd molar	1.67 – 1.95	~5.5

I expect that fluctuating asymmetry levels in the 1st molar will be lower than in the second molar because of improved buffering against stress *in utero* during the formation of the 1st molar, and decreased dependence on mother during the formation of the 2nd molar.

Materials and methods

The skeletons of chimpanzees from Gombe National Park, Tanzania, are preserved whenever possible. Skeletons of chimpanzees who died prior to 1987 were cleaned according to protocols laid out elsewhere (Zihlman *et al.*, 1990). Skeletons of chimpanzees who died after 1987 were processed in a different way. Following necropsy (K. Terio, unpublished data), the bodies were buried in a permeable bag and exhumed after at least one year. Skeletons were then carefully cleaned using water and a soft brush and allowed to thoroughly dry.

Because some chimpanzees at Gombe are infected with a strain of the Simian Immunodeficiency Virus (SIVcpz) (Keele *et al.*, 2009; Santiago *et al.*, 2002), an extra

step was added to the cleaning procedure in order to reduce the risk of possible transmission of the virus to skeletal researchers. Chimpanzees known to be infected with SIVcpz were briefly (~2 minutes) rinsed in a mild bleach solution after initial cleaning, and then subsequently soaked in water to remove bleach residue. SIV status of individual chimpanzees has been published elsewhere (Keele *et al.*, 2009), and is summarized in Table 2.11. Each skeleton was then inventoried and stored in mosquito net bags (to reduce the risk of insect damage to the bones in storage) in a secure location at Gombe National Park.

Maximum mesio-distal length for the 1st and 2nd mandibular molars was measured using a Paleo-Tech Hillson-Fitzgeral dental caliper. I took 5 independent measures of each tooth for each chimpanzee.

Chimpanzees from the Gombe skeletal collection with erupted 1st and 2nd molars at the time of death are included in this study. Only teeth with moderate or light wear (after Kilgore, 1989) are included in the sample. Table 5.3 indicates which chimpanzees are included in the 1st molar sample (n = 20), and Table 5.4 indicates which are included in the 2nd molar sample (n = 19). The two samples vary slightly due to variations in pre- and postmortem tooth loss or postmortem enamel damage. The small size of the samples restricts the statistical power of the analysis; this is addressed further in the discussion section.

Table 5.3: Chimpanzees whose 1st molars were evaluated for this study.

Chimpanzee	Sex	Age at death (years) (*estimate)
Atlas	m	31.3
Charlie	m	25.87
Cusano	m	39.94
Ebony	m	8.19
Echo	f	34
Gilka	f	18.87
Goblin	m	39.96
Jackson	m	10.41
Kidevu?	f	25.59
Mel	m	10.71
Melissa	f	36.31
Miff	f	30.9
Pallas	f	27.2
Passion	f	30.61
Patti	f	44.25
Satan	m	29.83
Sherehe	f	15.78
Sugar	f	10.91
Tumaini	m	30
Yolanda	f	24

Table 5.4: Chimpanzees whose 2nd molars were evaluated for this study.

Chimpanzee	Sex	Age at death (years) (*estimate)
Atlas	m	31.3
Charlie	m	25.87
Cusano	m	39.94
Ebony	m	8.19
Echo	f	34
Goblin	m	39.96
Jackson	m	10.41
Kidevu?	f	25.59
Madam Bee	f	28.22
Mel	m	10.71
Miff	f	30.9
Pallas	f	27.2
Passion	f	30.61
Patti	f	44.25
Sherehe	f	15.78
Sugar	f	10.91
Tumaini	m	30
Vincent	m	28.47
Yolanda	f	24

I tested for a relationship between the magnitude of fluctuating asymmetry and character size by plotting the difference between the right side (R) and left side (L) for each measurement (i) versus the right side plus the left side divided by two (a measure of trait size averaged between sides) (Palmer and Strobeck, 1986).

I then tested each of the 5 sets of measurements of each tooth for normality using the Shapiro-Wilk test in order to see if antisymmetry significantly contributed to non-directional asymmetry. If the distributions are normal, antisymmetry is not significant, and non-directional asymmetry may be assumed to be composed of fluctuating asymmetry alone (Guatelli-Steinberg *et al.*, 2006; *Ibid*.).

To assess fluctuating asymmetry for each tooth, I employed a two-way, mixed model analysis of variance (ANOVA) with sides fixed and individuals random, and then performed an F-test to determine whether the levels of fluctuating asymmetry differ between the 1st and second molars (Guatelli-Steinberg *et al.*, 2006). Statistical analyses were carried out in R, version 2.11.0 (www.r-project.org). Table 5.5 summarizes the ANOVA model presented here, and is modeled after Palmer and Strobeck (1986) and Guatelli-Steinberg *et al.* (2006).

Table 5.5: Summary of the ANOVA model for fluctuating asymmetry

Source of variation	Degrees of freedom	Expected mean square	Interpretation
Sides (S)	(S - 1)	$\sigma^2_m + M(\sigma^2_i + (A/S - 1)\,\Sigma\alpha^2)$	Directional asymmetry
Animals (A)	(A – 1)	$\sigma^2_m + M(\sigma^2_i + S\,\sigma_a{}^2)$	Non-directional asymmetry (both anti-symmetry and fluctuating asymmetry)
Measures (M) (n = 4 per side)	SA(M – 1)	σ^2_m	Measurement error

Results

Test for effects of character size

To assess whether the size of the tooth was correlated with differences in size between the left and right sides, I plotted $(R_i - L_i)$ versus $(R_i + L_i) / 2$. Character size can influence the magnitude of fluctuating asymmetry observed (Palmer and Strobeck, 1986); it is therefore important to determine whether it is an influence and to correct for it if necessary. Tooth size was not significantly correlated with left-right size differences for either the 1st molar (Figure 5.1) or the 2nd molar (Figure 5.2). This graphical assessment also aids in identifying potential outliers. Outliers can be extremely influential when attempting to detect levels of fluctuating asymmetry, and may lead to a significant result when otherwise the level of fluctuating asymmetry would not be significant (Palmer and Strobeck, 2003). I discovered one outlier in the measurements for the 1st molar (it is excluded from the graph), but because it was part of the 4th set of measurements, it was not included in the analysis (see below on tests for normality) and therefore has not influenced detection of fluctuating asymmetry.

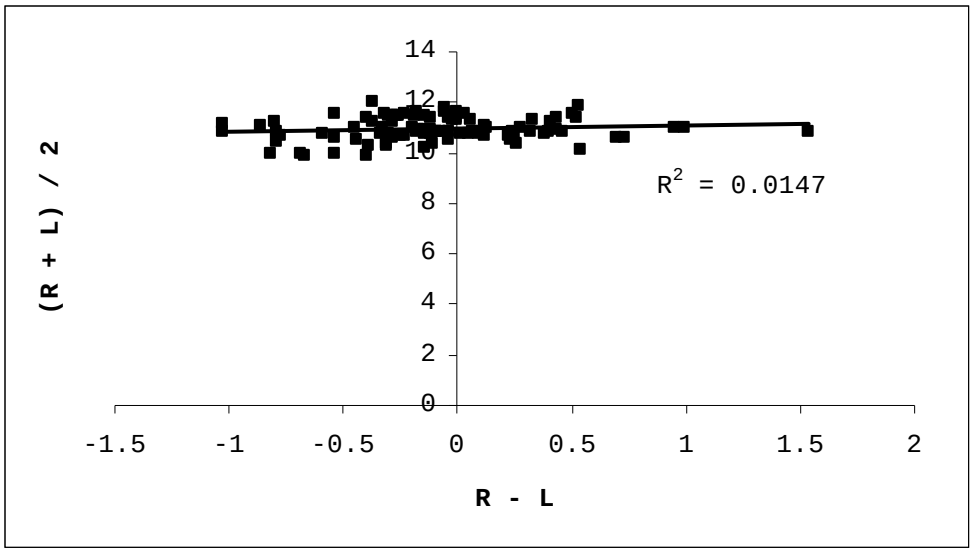


Figure 5.1: Meisio-distal length of the right 1st molar minus length of the left first molar plotted against a measure of tooth size. There is no relationship between right-left difference and tooth size for the 1st molar.

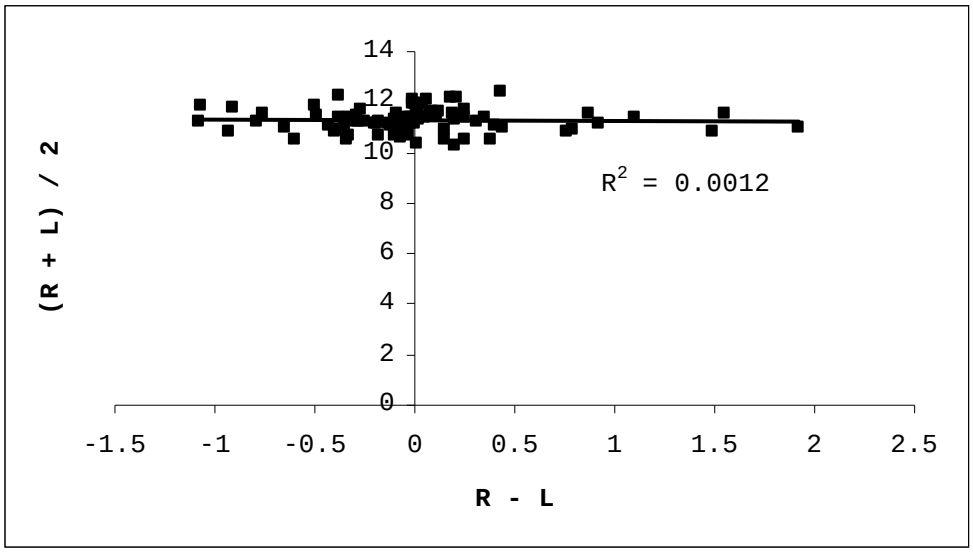


Figure 5.2: Mesio-distal enght of the right 2nd molar minus length of the left first molar plotted against a measure of tooth size. There is no relationship between right-left difference and tooth size for the 2nd molar.

Tests for normality

The null hypothesis in the Shapiro-Wilk test for normality is that the distribution of the data is normal (R version 2.11.0). The null hypothesis cannot be rejected for 4 of the 5 sets of measurements for each tooth. In each case, the anomalous set of measurements differed by at least one order of magnitude from the others. Because the majority of the sets of measurements met the requirement for normality, it is reasonable to assume that the departure from normality was due to circumstances particular to the day on which the measurements were taken (e.g. measurement error). I therefore discarded the sets of anomalous measurements for each tooth and carried out the analysis of variance on 4 sets of measurements for each tooth.

For the 1st molar, the 4th set of measurements deviated from normal ($W = 0.57$, $p = 1.15^{-6}$), but the other sets of measurements follow a normal distribution ($W \geq .94$, $p \geq 0.20$). For the 2nd molar, the 2nd set of measurements deviated from normal ($W = 0.91$, $p = 0.07$), but the other sets of measurements follow a normal distribution ($W \geq .95$, $p \geq 0.33$).

ANOVA models

The two-way, mixed model ANOVA for the 1st molar is summarized in Table 5.6, and for the 2nd molar in Table 5.7. Table 5.8 summarizes the interpretation of these models, and Table 5.5 gives the equations for each element of the ANOVA models. "Side" is the measure of directional asymmetry (Guatelli-Steinberg *et al.*, 2006). Directional asymmetry is not significant for either the 1st or the 2nd mandibular molar ($p \geq 0.4$). "Side:Animal" is the measure of non-directional asymmetry. Non-directional

asymmetry is a combination of antisymmetry and fluctuating asymmetry. Because the measurements follow a normal distribution, the level of antisymmetry is not significant and "Side:Animal" may therefore be taken as a measure of fluctuating asymmetry (Palmer and Strobeck, 1986; *Ibid*.) Levels of fluctuating asymmetry are significant for both the 1st and the 2nd molars ($p \leq 0.003$).

"Residuals" is an expression of measurement error (Guatelli-Steinberg *et al.*, 2006). When the mean square of the measurement error is subtracted from the mean square of the individual variation (Side:Animal) and divided by the number of replicate measurements, this leaves only the variance component due to nondirectional asymmetry: σ^2 (Palmer and Strobeck, 1986). Because antisymmetry is not significant in this case, σ^2 may be taken as the variance due to fluctuating asymmetry, controlled for measurement error.

For M1,

$$\sigma^2 = (0.089134 - 0.037881) / 4 = 0.0128$$

For M2,

$$\sigma^2 = (0.122646 - 0.029484) / 4 = 0.0233$$

Table 5.6: ANOVA model for the 1st molar

	Degrees of Freedom	Sum of Squares	Mean Squares	F	p-value
Side	1	0.0585	0.05823	0.66	0.428
Side:Animal	19	1.6936	0.08913	2.35	0.003
Residual	120	4.5458	0.03788		

Table 5.7: ANOVA model for the 2nd molar

	Degrees of Freedom	Sum of Squares	Mean Squares	F	p-value
Side	1	0.0523	0.05232	0.43	0.522
Side:Animal	18	2.2076	0.12265	4.16	$1.22(10^{-6})$
Residual	114	3.3612	0.02948		

Table 5.8: Summary of interpretation of the ANOVA models.

ANOVA model	**What kind of asymmetry does it represent?**	**Interpretation of results presented here**
Side	Directional Asymmetry	Not significant (p = 0.428, 0.522)
Side:Animal	Non-directional Asymmetry (Anti-symmetry + Fluctuating Asymmetry)	The measurements follow a normal distribution. Anti-symmetry therefore does not significantly contribute to Non-directional asymmetry. This line is equivalent to Fluctuating Asymmetry. Levels of fluctuating Asymmetry are significant (p = 0.003, $1.22(10^{-6})$)
Residual	Measurement Error	Accounted for by calculating the variance due to Non-directional Asymmetry: σ^2

Does fluctuating asymmetry differ between the 1st and 2nd molars?

The null hypothesis for the F-test is that the variance due to fluctuating asymmetry is equal for the 1st and 2nd molars. To calculate the p-value for this test, σ^2 and degrees of freedom for each tooth are needed. The σ^2 values have already been calculated. Degrees of freedom may be estimated using the Satterthwaite formula, as given in Palmer and Strobeck (1986: 408). Degrees of freedom for the 1st molar ≈ 6.1074, for 2nd molar ≈ 10.2919. The p-value for the F-test is 0.473; I conclude that fluctuating asymmetry levels in the 1st and 2nd molar are not significantly different

Discussion

The amount of fluctuating asymmetry in the 1st and 2nd mandibular molars of the Gombe chimpanzees is statistically significant, but does not differ statistically between the two teeth. This is contrary to expectations given the ages of development of these teeth. While the 1st molar begins crown formation before birth and is complete by 2.5 years, the 2nd molar does not begin growing until nearly 2 years, and ends at 5 years (Reid *et al.*, 1998b), which is after weaning usually occurs at approximately 4 years (Goodall, 1986). Because the 1st molar forms entirely during the period of a chimpanzee's dependence on mother, I expected developmental instability to be decreased during this time period relative to a later time period in which the infant is less buffered by mother.

Several possibilities for the lack of difference exist, the first of which is that the crown formation times of the 1st and 2nd molars overlap. It may be that this overlap is significant enough to mask any potential differences. While the 1st molar begins crown

formation *in utero*, a period of time in which developmental perturbation should be at a minimum, the 1st molar also forms during the perinatal period. It may be that both birth and weaning are sufficiently and comparably stressful events that developmental instability levels are approximately equivalent during these times. Comparison between the 1st and 3rd molars, or other tooth pairs, may be more illustrative of differences in levels of developmental instability between life history stages. The crown of the 3rd molar develops between approximately 3.6 - 7 years of age in chimpanzees (Reid *et al.*, 1998b); its formation does not overlap with the 1st molar. A future study will attempt to answer this question.

A second reason that the level of fluctuating asymmetry in the 1st and 2nd molars of the Gombe chimpanzees may be equal is that teeth are a less than ideal trait to evaluate for fluctuating asymmetry. Because teeth wear through use, their utility as a trait for measuring fluctuating asymmetry may be somewhat limited, as small variations in wear between the two sides would likely follow the same normal distribution as fluctuating asymmetry (Palmer and Strobeck, 2003). When studying skeletons, however, traits that neither respond to mechanical loading (as do the bones) nor to wear (as do the teeth) are scarce. Teeth should be preferred to bones in fluctuating asymmetry studies on skeletons because teeth do not remodel. A linear measurement from a tooth with light to moderate wear will not be as affected by wear as a trait such as occlusal surface area. In addition, the 1st molar is in occlusion longer than the second molar (beginning to erupt at ~ 4 years for the 1st molar, and ~8 years for the 2nd molar (Zihlman *et al.*, 2004b)), and therefore exhibits more wear (Kilgore, 1989). If wear is affecting assessment of fluctuating asymmetry, we would expect the 1st molar to be more asymmetric than the 2nd molar.

Because the 1st molar is more worn than the 2nd molar, however, this introduces the possibility that wear affects our ability to distinguish differences in fluctuating asymmetry between the two teeth. If the 2nd molar is indeed more asymmetric than the 1st due to developmental instability, but the 1st molar is more asymmetric due to wear, the levels of asymmetry may be equal, even though they are due to different causes. Any comparisons between the 1st and 3rd molars would be subject to the same limitations imposed by wear.

The lack of dependence of the left-right size differences and tooth size as well as the assumed insignificant effects of moderate tooth wear on the mesio-distal length of a tooth indicate that tooth wear is not likely to affect the detection of fluctuating asymmetry in this case, but testing this hypothesis would be challenging. Fluctuating asymmetry is distinguished from other kinds of asymmetry by its distribution; variance due to fluctuating asymmetry is normally distributed with a mean of zero. If another form of asymmetry is similarly distributed, as we would expect asymmetry due to wear to be, it would be difficult to separate this form of asymmetry from fluctuating asymmetry (Palmer and Strobeck, 2003).

Yet another possibility for the lack of difference in fluctuating asymmetry in the 1st and 2nd molars is that male – female differences in fluctuating asymmetry have the potential to mask other forms of variation in levels of fluctuating asymmetry. Guatelli-Steinberg and colleagues (2006) did not uncover significantly different levels of dental fluctuating asymmetry between human males and females, but sexual dimorphism is greater in chimpanzees than in humans. Male chimpanzees, because they grow for a longer period of time relative to females (Pusey *et al.*, 2005), have more time to

experience developmental instability, making it more likely that males will accumulate fluctuating asymmetry. The Gombe skeletal sample is too small to adequately test for fluctuating asymmetry in males and females separately, but this could be tested in another skeletal collection of wild chimpanzees.

Lastly, this study evaluates only one trait: mesio-distal molar length. Because of this, there is only 1 degree of freedom for the variance of "Sides" (see Tables 5.5 and 5.6); a bilateral trait can only have two sides, but two sides of multiple traits would yield increased degrees of freedom. An analysis of variance with only 1 degree of freedom has limited statistical power, and the inclusion of other traits (e.g. bucco-lingual width) might help to increase the statistical power of the analysis. Such traits would have to be selected with care in order to avoid the potentially obscuring effects of tooth wear; crown height or occlusal surface area, for example, would not be good candidates as they covary too highly with tooth wear. The overall sample size of chimpanzees evaluated is also small, which further restricts the statistical power of this study. The small sample size also means that inter-individual variation in stress levels will have a greater effect on the detection of fluctuating asymmetry. Fluctuating asymmetry is a measure of developmental instability, the ability to buffer against stress. In order for inter-individual differences in developmental instability to be meaningful, a relatively uniform level of stress should be experienced by all of the individuals in a study. This may be a reasonable assumption in a large population, but the Gombe skeletal collection is not large in this sense. It is possible that chimpanzees with low ability to buffer against stress might encounter low levels of stress, while chimpanzees with high ability to buffer against stress encounter higher levels of stress. The degree of fluctuating asymmetry

between two such individuals could potentially be similar, and in a small sample, this would confound the detection of population-level differences between life history stages.

No statistical difference between levels of fluctuating asymmetry in the 1st and 2nd mandibular molars of the Gombe chimpanzees was detected in this study. This may mean that developmental instability is similar throughout early infancy and into the early juvenile period, but enough other variables remain to make further evaluation of interest. Future studies should seek to evaluate possible differences in levels of fluctuating asymmetry in the posterior dentition between male and female chimpanzees, compare additional teeth (e.g. the 1st and 3rd molars, or deciduous versus permanent teeth), and be aware of the potential limitations imposed by tooth wear.

Chapter 6

Summary and Concluding Thoughts

During life, dominance rank plays a vital role in the lives of chimpanzees. It affects access to resources (Murray *et al.*, 2006; Pusey *et al.*, 2005), inter-individual relationships (Foster *et al.*, 2009; Goodall, 1986), and reproductive success (Constable *et al.*, 2001; Pusey *et al.*, 1997; Wroblewski *et al.*, 2009), among other variables. The effects of dominance rank on the skeleton are more subtle. Most measures of health and stress assessed here were not significantly correlated with dominance rank. This was unexpected, given the central role of rank for chimpanzees during life.

Some evidence that change in rank may be important for predicting stress as it is visible on the skeleton is an encouragement for further investigation on wild chimpanzees with known life histories. Because dominance rank is so tightly bound to measures of fitness, assessing relative skeletal health and its relationship to dominance rank has the potential to inform us about skeletons without known life histories.

Trauma and Pathology

Of all the variables tested, age best correlated with trauma and pathology incidences. Age accounted for most of the variation in dominance rank. This broadly supports other findings that chimpanzees who live longer have more time to accumulate traumata and pathologies (Jurmain, 1989; Lovell, 1991), but the relationship between age and trauma may be more complex than previously demonstrated. Including chimpanzees of a wide variety of ages and causes of death did not yield a positive correlation between

trauma incidence and age. The infanticide victims included in this sample are particularly influential. It is possible that the relationship between age and trauma is non-linear. Exclusion of the influential cases, making the data set more comparable to previous studies, does yield a positive correlation between age and trauma incidence.

Sex was not a significant predictor for trauma incidence, arthropathy incidence, or incidence of other pathologies. Sex differences in location of trauma among the Gombe chimpanzees have been previously demonstrated (Jurmain and Kilgore, 1998), and these results are generally supported here (inclusion of the infanticide cases skews the results as the sex ratio of infanticide victims is uneven). Cranial injuries are more common in males, and injuries to the extremities are more common in females.

Change from higher to lower rank was a significant predictor for arthropathy incidence and trauma incidence, but the number of chimpanzees whose rank changed from their highest achieved rank to rank at death is very small (n = 4), so this result should still be considered preliminary. It may be that change in rank is extremely important in terms of the skeleton's reflection of stress events, but the small sample masks these effects. This is one example of the utility of further study of wild primate skeletons and the continued preservation of skeletal material from primates with known life histories.

Skeletal Damage to Infanticide Victims

Observed cases of chimpanzee infanticide vary widely (Arcadi and Wrangham, 1999) and have led to discussion about the possible similarities and differences between infanticide and monkey hunting (Hamai *et al.*, 1992; Sherrow and Amsler, 2007) as well

as between infanticide and other forms of conspecific aggression (Goodall, 1977; Norikoshi, 1982). Studying infanticide in chimpanzees has the potential to inform us about the nature of chimpanzee aggression in general as well as to better understand the evolution of aggressive behaviors in the hominin lineage (e.g. Wrangham *et al.*, 2006).

The patterns of skeletal damage to chimpanzee infanticide victims bear both similarities and differences to damage inflicted on monkey prey of similar size. Degree of completeness of consumption of chimpanzee infanticide victims was not significantly correlated with the age of the infant, the infant's group status, or the number of males present. Only time spent consuming was correlated with consumption completeness. These data do not, therefore, provide evidence for infanticide being either more similar to other forms of conspecific aggression or to monkey hunting. This gives further support to the idea that infanticide in chimpanzees is complex, and may mean that infanticide is a phenomenon separate from both conspecific aggression and hunting.

The number of infanticide cases currently documented and the number of known infanticide victims whose skeletons were analyzed is small (n = 2). This indicates that continued accumulation of observations and data on chimpanzee infanticide may provide more definitive answers on the nature of this behavior in future. I plan to include additional infanticide victim skeletons from Gombe in future studies on this topic.

Enamel Hypoplasia

As a non-specific marker of physiological stress, enamel hypoplasias are a useful skeletal measure of general health (e.g. Goodman and Rose, 1990). Like trauma, arthropathy, and other pathologies, enamel hypoplasia frequency was not associated with

dominance rank or sex. Change in dominance rank from middle to low ranking may, as in the case of arthropathy incidence, be significantly correlated, but because only 1 chimpanzee experienced a rank change of this nature, this result must be treated with great caution.

Relevant to the fact that neither sex nor dominance rank were correlated with hypoplasia incidence on the canine is that the canine crown forms between approximately 0.38 – 6.93 years (Berbesque and Doran, 2008b). Chimpanzees are usually weaned near 4 years of age (Goodall, 1986), meaning that the canine mostly forms during the time when a young chimpanzee is almost entirely dependent on mother. This is likely to buffer the developing infant from potential stressors (e.g. nutritional deficit). Maternal rank may be more correlated with enamel hypoplasia frequency of her offspring, but the number of chimpanzee skeletons from Gombe with erupted adult canines whose mothers have known dominance ranks is extremely limited (n = 5), and all of these mothers were middle ranking at the time of the offsprings' births, so assessing variation in hypoplasia frequency due to maternal rank is not currently possible. In addition to the limited number of chimpanzees who experienced a change in rank, this is yet one more example of the value of continued long term behavioral research coupled with skeletal research.

Fluctuating Asymmetry

Whether stress levels vary over the course of a chimpanzee's life has the potential to illuminate how selective pressures may change during different life history stages. Fluctuating asymmetry is a measure of developmental instability, or stresses that occurred during the period of growth and development. Higher levels of asymmetry

indicate a decreased ability to buffer against stress (e.g. Thornhill and Gangestad, 2006). Examining levels of fluctuating asymmetry in teeth that developed at different times has the potential to provide evidence for varying levels of developmental instability between these two time periods. The 1st permanent mandibular molar forms from 0.05 – 0.15 years before birth until approximately 2.5 years after birth. The 2nd molar develops from approximately 1.7 – 5.6 years (Reid *et al.*, 1998b). As such, comparing these two teeth allows for assessment of levels of developmental instability in the perinatal period and early infancy versus later infancy and early juvenility (age categories afterGoodall, 1986). A two-way, mixed model analysis of variance (ANOVA) allows fluctuating asymmetry to be considered apart from other forms of asymmetry as well as measurement error. No significant difference in level of fluctuating asymmetry between the 1st and 2nd molars was found, and it may be that developmental instability is comparable throughout the formation times of these two teeth.

Several compounding factors complicate this conclusion. A limited sample size in which only a single trait was analyzed has relatively little statistical power, and future sample expansion would be helpful in further assessing the hypothesis that stress levels do no vary throughout infancy. In addition, because teeth wear with use, their ability to provide characters suitable to studies of fluctuating asymmetry may be limited (Palmer and Strobeck, 2003). Teeth are certainly preferable to bones (which remodel in response to mechanical loading), however, and other precautions were taken to minimize the potential deleterious effects of analyzing the dentition for fluctuating asymmetry. Finally, because the timing of development overlaps for the 1st and 2nd molars, they cannot be considered to represent entirely separate age categories and the same levels of

developmental instability have the potential to affect both teeth simultaneously. Future studies should consider expanding both the sample as well as number of traits examined, comparing additional teeth (e.g. the 1st to the 3rd molar), and examining other potential sources of variation (e.g. differences between males and females).

Future studies

Skeletal analysis has great contributions to make to understanding health status, sources of morbidity and mortality, and life histories – especially if we wish to use modern species as a basis for comparison to aid in the interpretation of the fossil record. Our potential to understand inter-species variation in patterns of skeletal trauma and pathology, signs of health and stress on the skeleton, and how these variables relate to both behavior and soft tissues has greatly increased with the number of long term primate behavioral research sites. Preservation and study of skeletal material from mountain gorillas at Virunga National Park, Rwanda, baboons at Amboseli National Park, Kenya, and orangutans at Tanjung Puting National Park, Borneo, in addition to chimpanzees from Gombe, the Taï Forest in Côte D'Ivoire, and Kibale National Park, Uganda, will make comprehensive comparative studies of wild primate skeletons with known life histories possible for the first time. This collaborative research will integrate data on hard tissues, soft tissues, and behavior for a variety of primate species, contributing to conservation efforts as well as our understanding of modern primates and their evolution.

Projects currently underway or planned for the near future include evaluation of skeletal trauma by Amandine Eriksen, documentation of the microanatomy of bones and teeth and skeletal and dental pathologies by Shannon McFarlin of the Karisoke mountain

gorillas. Development of local capacity for skeletal preservation and management of the collection in Rwanda is also proceeding. I plan to continue trauma and pathology research on the Gombe skeletal collection including a detailed study of vertebral degeneration, tooth wear and dental pathologies, and further studies on fluctuating asymmetry. Incorporation of the macroscopic data from Gombe with data on bone and tooth microstructure, including studies on development and stress markers such as enamel hypoplasias and arrested growth (Harris) lines, is also planned for the future. The development of comparable data sets for orangutans, baboons, and other primate species is a long term goal of this project.

The accumulation of these data sets from long-term primate research sites allows for the comparison of the integrated data between species, and improves our understanding of how selective pressures may vary for each. Comparisons within species are also possible, helping to establish what is held in common by a particular species (or what might make it a species) as well as variation within that species. This will allow us to examine the fossil record with more confidence as we become more familiar with the ways life history affects the skeleton for a wider variety of species. The availability of data on wild primate skeletons with known life histories will make new kinds of species-level comparisons possible. The availability of comparative data like that from Gombe may stimulate the creation of similar data sets on subjects like trauma and pathology for fossil species the same way that morphological and anatomical data sets are already being created and analyzed. Studying skeletons with known life histories allows us to better interpret skeletons for whom those kinds of data are not available, leading to better

interpretations of skeletons without known life histories, and a better understanding of how life history is reflected by the skeleton.

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