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# Poliomyelitis and Skeletal Asymmetry in Gombe Chimpanzees

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ABSTRACT. Skeletons of free-ranging chimpanzees from Gombe National Park, Tanzania allow assessment of the effects of long-term, unilateral upper limb paralysis due to the infectious viral disease, poliomyelitis. Comparison of left and right upper limb bone weights, lengths, joint areas, and diaphysial diameters between two adult females with long-term, partial paralysis and a group of unaffected adult Gombe chimpanzees show that the disease caused considerable asymmetries in the skeleton. Detailed analyses of mineral content and diaphysial cross-sectional geometry of the humerus in the affected females show individual differences. The analysis extends to consideration of bone-muscle relationships and peripheral and central nervous system involvement. For each individual, sex, estimated age at death, and behavioral information during life are known from field studies. The impact of the disease on survival and reproductive outcome of the affected individuals is discussed.

Key Words: Skeletal biology; Poliomyelitis; Gombe chimpanzees.

## INTRODUCTION

Free-ranging chimpanzees from Gombe National Park, Tanzania expressed limb, trunk, or neck paralyses in 1966. Two individuals disappeared. Field observations of ten others showed that the extent and duration of constraints on movement and weight-bearing capabilities, as well as individual age at the onset of paralysis, affected chimpanzees in different ways. Outcomes included earlier-than-expected death, morbidity associated with musculoskeletal disuse, and biosocial influences on survival and well-being, mating, and rearing of offspring (GOODALL, 1971, 1983, 1986; TELEKI et al., 1976; BAUER, 1977; ZIHLMAN et al., 1990).

The cause of the paralyses was poliomyelitis (MORBECK et al., in press), an infectious, potentially neurotropic disease of human and nonhuman primates produced by strains of poliovirus. A diagnosis of paralytic poliomyelitis is supported in three ways: (1) known characteristics of the disease and observed clinical symptoms (HUCKSTEP, 1975; GOODALL, 1971, 1983, 1986; BAUER, 1977); (2) reported poliomyelitis cases in the local human population (GOODALL, 1971, 1983, 1986; TELEKI et al., 1976; HUCKSTEP, 1975); and (3) specificity of host organism immune response to poliovirus and lack of new cases after researchers gave Sabin live virus oral poliovaccine (OPV) to chimpanzees (SABIN, 1985; GOODALL, 1983, 1986, pers. comm.).

This paper examines the impact of paralytic poliomyelitis and partial forelimb paralysis on the skeletons and biosocial and reproductive life histories of two Gombe chimpanzee females, *Gilka* and *Madam Bee*. *Gilka*'s right forelimb became partially paralyzed when she was about 7 years old. Since *Gilka* still was young, fusion of joint epiphyses and skeletal

growth probably was incomplete. In contrast, *Madam Bee* was reproductively and presumably also skeletally mature when her left forelimb first was paralyzed. Both of these chimpanzees lived with extensive forelimb paralysis for many years after contracting paralytic poliomyelitis.

The Gombe chimpanzee skeletons provide a natural experiment for explaining variation in size, shape, weight, mineral content, and internal structure of bones. Field observations of the same individuals document lifetime behaviors (GOODALL, 1968, 1971, 1983, 1986, 1990 and cited references). The skeletons of *Gilka* and *Madam Bee* are compared to those of Gombe chimpanzee adults that were not affected by long-term paralysis as well as to each other. From these analyses we interpret observed variation in bone morphology, in particular, as related to skeletal asymmetry. The combination of skeletal data and field observations allows us to assess the impact of the paralytic disease on survival and reproduction.

## MATERIALS AND METHODS

### FIELD OBSERVATIONS

Gombe chimpanzees were habituated to human observers during the 1960's by establishing a research camp, selective provisioning, and following individuals. Behaviors and environmental contexts are recorded either by ad libitum or quantitative methods. Details of social interactions and physical observations, including state of health, reproductive status, mating, and rearing of offspring have been documented by GOODALL and colleagues for almost three decades (GOODALL, 1986 and cited references).

### FIELD OBSERVATIONS OF FORELIMB MOVEMENT AND WEIGHT-BEARING

*Gilka* and *Madam Bee* each modified their behaviors to accomodate partial upper limb paralysis. This was apparent, for example, in locomotion and food-getting, manipulation and grooming, and carrying and caring for infants.

*Gilka*'s physical growth and development and social interactions were observed during almost the entire course of her life (GOODALL, 1986; ZIHLMAN et al., 1990). In 1966 when *Gilka* was in late childhood/early adolescence, her infant brother died several days after limb paralyses first were observed. *Gilka* groomed him and tried to play with him after his death (GOODALL, 1971). Her right forelimb later became partly paralyzed.

When first paralyzed, *Gilka* walked tripedally. After three months of disuse, she again could walk quadrupedally. But, she never regained full wrist movement and could not use her thumb in locomotion and manipulation (GOODALL, 1986; ZIHLMAN et al., 1990). Behavioral observations continued until she died at 19 years old in 1979.

Details of *Madam Bee*'s life history are less well known (GOODALL, 1986). When GOODALL began her study in the early 1960's, *Madam Bee* was an adult and already had a daughter. *Madam Bee*'s left forelimb became partially paralyzed in 1966. As with *Gilka*, pre- and postparalysis behaviors were documented in Gombe field records. Until her death in 1975, *Madam Bee* exhibited limited shoulder and finger mobility. She usually walked tripedally or, for example, when supporting an infant, bipedally.

## SKELETAL SERIES

Field researchers, when possible, retrieve cadavers after chimpanzees die in Gombe National Park. They clean skeletons via use of endemic beetle larvae and subsequent washing of bones (SUMNER et al., 1989; ZIHLMAN et al., 1990). Twenty-three skeletons, most of known age and sex, represent all life stages.

Since *Gilka* and *Madam Bee* each exhibited partial upper limb paralysis, the present study compares their forelimb bones to many aspects of forelimb osteology that were not affected by paralytic poliomyelitis in other adult Gombe chimpanzees.[1)] Six females and four males serve as a comparative, control group (Table 1). Adults are defined by presence of third molars and fully developed joints, all with some epiphysial fusion (MORBECK & ZIHLMAN, 1989). Known sex, chronological age estimates, and information about life history events are from field studies (GOODALL, 1986).

**Table 1.** Gombe adult chimpanzees, sample size and composition.

| | Age at death[1)] | Life stage[2)] |
|---|---|---|
| Females | | |
| *Gilka*[3)] | 19 | Mature |
| *Pallas* | 27 | Mature |
| *Madam Bee*[3)] | 28 | Mature |
| *Passion* | 31 | Mature |
| *Miff* | 31 | Mature |
| *Melissa*[4)] | 36 | Old age |
| *Flo*[4)] | 43 | Old age |
| *Old Female*[4)] | >40 | Old age |
| Males | | |
| *MacDee*[4)] | 15 | Young |
| *Charlie* | 26 | Prime |
| *Jomeo* | 31 | Middle age |
| *Hugo* | 39 | Old age |

1) Estimates from field observations (GOODALL, 1986, 1987a, b); 2) defined in GOODALL (1986); 3) *Gilka* and *Madam Bee* each exhibited long-term partial paralysis of one forelimb; 4) see Footnote 1.

1) No obvious diseases that affect external bone size and shape are evident in the comparative sample. Two older females, *Flo* and *Old Female*, display apparent age-related mineral loss and endosteal resorption within the femur (SUMNER et al., 1989). *Old Female* also shows low BMC values similar to those of *Gilka* and *Madam Bee* at the 35% site of the humerus (unpubl. data). These females, therefore, are included only in comparisons of bone lengths and joint areas.

Another female, *Melissa*, experienced temporary upper limb paralysis and limited shoulder and neck mobility as a prime adult. She regained forelimb movement and weight transference capabilities as documented in normal quadrupedal walking (GOODALL, 1983, 1986, 1987b). However, neck movements were restricted during the remainder of her life. Right-left side differences used here and those analyzed for a larger set of measurements are less than 4% (unpubl. data). As with the older females, only external measurements of the forelimb elements are used in the comparative sample.

*MacDee*, a young adult male, lost use of his forelimbs and was unable to travel and get food. He was put to death for humane reasons by researchers shortly after the onset of paralysis (GOODALL, 1983, 1986). Like normal adult males, differences of left and right upper limb bone lengths, most areas, and weights are less than 3%. Thus, it is likely that he died before extensive bone remodeling could have taken place. External measurements are included here in the pooled-sex sample.

## SKELETAL MEASUREMENTS

Table 2 lists measurements taken on left and right upper limb bones. These include: (1) weights (g); (2) linear dimensions (mm); (3) joint areas measured from latex templates using a Zeiss MOP Image Analyzer ($mm^2$); (4) BMC, bone mineral content (g/cm) calculated from single photon absorptiometry (SPA) data using a Lunar Radiation System with $I^{125}$ source; and (5) cross-sectional area dimensions including; MA, medullary area, CA, cortical area (compact bone), and TA, or total subperiosteal area, that is, MA + CA ($mm^2$) determined from computed tomographic (CT) scans using a Siemens Somatom 2 whole body scanner, 125 kVp, 2 mm slice thickness, and reconstructed images in digital form.

Linear diameters, BMC, TA, MA, and CA are determined for the humerus of *Gilka* and *Madam Bee* at five equidistant sites along the diaphysis. SPA and CT data collection use

**Table 2.** Skeletal measurements.

| | |
|---|---|
| Weights[1] | |
| Scapula | |
| Humerus | |
| Radius | |
| Ulna | |
| Lengths[1] | |
| Clavicle: | length |
| Scapula[2]: | length |
| | breadth |
| Humerus: | length |
| | diaphysis, length |
| | diaphysis, 80% location: AP, ML diameters |
| | diaphysis, 65% location: AP, ML diameters |
| | diaphysis, 50% location: AP, ML diameters |
| | diaphysis, 35% location: AP, ML diameters |
| | diaphysis, 20% location: AP, ML diameters |
| Radius: | length |
| | diaphysis, length |
| | proximal aspect, maximum radial head diameter |
| | distal aspect, ML diameter |
| | diaphysis, 50% location: AP, ML diameters |
| Ulna: | length |
| | diaphysis, length |
| | diaphysis, 50% location: AP, ML diameters |
| Joint areas | |
| Shoulder: | scapula, glenoid fossa |
| | humerus, humeral head |
| Elbow: | humerus, capitulum |
| | trochlea |
| | radius, radial head |
| | ulna, trochlear notch |
| | radial notch |
| Wrist: | radius, radial fossa |
| Bone mineral content and cross-sectional area[3] | |
| Determined at five equidistant scan sites along the humeral diaphysis (80%, 65%, 50%, 35%, 20%) | |
| | Mineral content |
| | Areas: total subperiosteal area |
| | medullary area |
| | cortical area |

1) Standard linear and weight measurements of left and right sides are used; 2) scapular length and breadth are defined by SHEA (1986); 3) see text for discussion of bone mineral content values, and joint surface and cross-sectional area dimensions. AP: anteroposterior; ML: mediolateral.

standardized techniques. SPA equipment produces a collimated stream of photons which are passed through the shaft of the humerus surrounded with a tissue equivalent. Bone density is reflected in the amount of reduction of the photon stream; this is used to calculate BMC. CT images reconstruct the distribution of bone tissue in cross-section. They are produced using calculations based on the distribution of X-ray attenuation in a series of volume elements in a "slice" of bone. Procedures follow methodology presented in SUMNER (1984), MORBECK and ZIHLMAN (1989), SUMNER et al. (1989), ZIHLMAN et al. (1990), and cited references.

### DATA ANALYSES

Two analytical approaches examine forelimb skeletal anatomy of *Gilka*, *Madam Bee*, and other adult Gombe chimpanzees. First, general characteristics of upper limb bone asymmetry are investigated in two Gombe chimpanzee subsamples, one that includes six unaffected, adult females and a second sample that includes these females combined with four unaffected, adult males. Data are used to assess the degree of bilateral variation in Gombe chimpanzees that were not affected by long-term paralysis. Second, the nature of the bilateral asymmetries of the forelimb bones of *Gilka* and *Madam Bee* are investigated in greater detail. Comparisons include: (1) weight, linear, and joint area dimensions, cross-sectional geometric data from CT, and bone mineral data from SPA of the humeri; and (2) weight, linear, and joint area dimensions of the radius and ulna.

The degree of asymmetry is determined using per cent differences of left and right sides, calculated for each individual in the comparative series as follows: [(left side value − right side value)/left side value] × 100. For *Gilka* and *Madam Bee*, asymmetry is determined as follows: [(unaffected side value − affected side value)/unaffected side value] × 100. Therefore, for *Gilka*, with an affected right forelimb, asymmetry is calculated as: [(left side value − right side value)/left side value] × 100. The left forelimb was paralyzed in *Madam Bee*. Thus, asymmetry is determined as: [(right side value − left side value)/right side value] × 100.

Descriptive statistics include means, medians (since samples are small), minimum-maximum values, and standard deviations for variables for unaffected adults.[2)] Statistical significance of bilateral differences in unaffected adults is assessed via paired *t*-tests using left-right forelimb values. Data are analyzed using the Statistical Package for the Social Sciences (NIE et al., 1975; NORUŠIS, 1984).

## RESULTS

Unaffected Gombe chimpanzee females and the mixed-sex sample of these females and normal males show little bilateral variation in upper limb bone weights, scapular and long bone lengths, and joint areas (Tables 3 & 4). In contrast, forelimb bones of *Gilka* and *Madam Bee* each exhibit greater left-right side differences when compared to unaffected Gombe adults (Table 4). Furthermore, bones of both left and right upper limbs of *Gilka*

2) In addition, data have been normalized as follows: left and right weights, lengths, and joint areas are divided by the sum of respective upper limb dimensions for each side and humeral diameters are divided by diaphysial length. Analyses of these "size-adjusted" values are not presented since results are similar to those reported for raw data.

**Table 3.** Bilateral variation expressed as per cent differences in forelimb bones of pooled-sex sample of unaffected females and males.*

| Variable | *N* | Median | Mean | Minimum to maximum | S.D. |
|---|---|---|---|---|---|
| Weights | | | | | |
| Scapula | 8 | −0.5 | 0 | −6.0 to 6.0 | 4.0 |
| Humerus | 10 | 0 | 0 | −8.0 to 6.0 | 4.0 |
| Ulna | 8 | −3.5 | −4.0 | −11.0 to 2.0 | 5.0 |
| Radius | 9 | −1.0 | −1.0 | −7.0 to 4.0 | 4.0 |
| Lengths | | | | | |
| Clavicle | 9 | 0 | 0.8 | −2.5 to 4.4 | 2.3 |
| Scapula, length | 8 | 1.0 | 1.0 | −5.0 to 6.0 | 4.0 |
| Scapula, breadth | 9 | 2.0 | 2.0* | −2.0 to 7.0 | 3.0 |
| Humerus | 10 | 0.5 | 1.0 | 0 to 5.0 | 2.0 |
| Ulna | 8 | 0.5 | 1.0 | 0 to 2.0 | 1.0 |
| Radius | 9 | 0 | 0 | −1.0 to 1.0 | 1.0 |
| Areas | | | | | |
| Glenoid fossa | 8 | 3.0 | 3.0 | −4.0 to 9.0 | 5.0 |
| Humeral head | 9 | 0 | 0.1 | −7.0 to 5.0 | 3.8 |
| Humeral capitulum | 10 | −3.0 | −5.0 | −26.0 to 7.0 | 10.0 |
| Humeral trochlea | 10 | 0 | −1.0 | −12.0 to 7.0 | 6.0 |
| Ulnar trochlear notch | 8 | 2.0 | 2.0 | −3.0 to 7.0 | 3.0 |
| Ulnar radial notch | 9 | 11.0 | 8.0 | −17.0 to 24.0 | 13.0 |
| Radial head | 10 | 1.0 | 3.0 | −4.0 to 13.0 | 6.0 |
| Radial fossa | 8 | 1.0 | 1.0 | −13.0 to 12.0 | 8.0 |

*Per cent difference is calculated for each individual as follows: [(left value − right value)/left value] × 100. *N*: number; S.D.: standard deviation; paired *t*-test. *$p < .05$.

**Table 4.** Bilateral variation expressed, as per cent differences in forelimb bones of unaffected females and affected females, *Gilka* and *Madam Bee*.*

| Variable | *N* | Median | Mean | Minimum to maximum | S.D. | *Gilka* | *Madam Bee* |
|---|---|---|---|---|---|---|---|
| Weights | | | | | | | |
| Scapula | 5 | 0 | 0 | −6.0 to 6.0 | 5.0 | 23.4 | −19.5 |
| Humerus | 6 | −0.5 | −1.0 | −8.0 to 6.0 | 5.0 | 26.3 | 32.0 |
| Ulna | 5 | −4.0 | −4.0* | −8.0 to 1.0 | 4.0 | 15.8 | 29.4 |
| Radius | 6 | −2.0 | −2.0 | −7.0 to 4.0 | 4.0 | 8.9 | 27.3 |
| Lengths | | | | | | | |
| Clavicle | 5 | 1.0 | 1.3 | 0 to 4.0 | 1.6 | 1.5 | NA |
| Scapula, length | 5 | 1.0 | 2.0 | −2.0 to 6.0 | 4.0 | 1.7 | 2.8 |
| Scapula, breadth | 5 | 2.0 | 3.0 | −1.0 to 7.0 | 3.0 | 5.6 | 1.2 |
| Humerus | 6 | 1.0 | 1.0 | 0 to 5.0 | 2.0 | 2.1 | −0.2 |
| Ulna | 5 | 0 | 0 | 0 to 1.0 | 0 | 4.1 | 2.4 |
| Radius | 6 | −1.0 | −1.0 | −1.0 to 0 | 1.0 | 2.6 | 0.4 |
| Areas | | | | | | | |
| Glenoid fossa | 5 | 5.0 | 3.0 | −4.0 to 9.0 | 5.0 | 17.2 | −12.9 |
| Humeral head | 6 | 1.5 | −0.8 | −7.0 to 5.0 | 4.3 | 9.3 | −1.7 |
| Humeral capitulum | 6 | −8.0 | −10.0* | −26.0 to 0 | 9.0 | −3.0 | 12.9 |
| Humeral trochlea | 6 | −0.5 | −3.0 | −12.0 to 3.0 | 15.0 | 3.2 | 3.6 |
| Ulnar trochlear notch | 5 | 3.0 | 3.0 | −1.0 to 7.0 | 3.0 | 15.0 | 6.8 |
| Ulnar radial notch | 5 | 11.0 | 10.0* | 6.0 to 12.0 | 2.0 | −37.2 | −25.0 |
| Radial head | 6 | −0.5 | 2.0 | −4.0 to 13.0 | 7.0 | 7.1 | 6.4 |
| Radial fossa | 5 | 4.0 | 3.0 | −13.0 to 12.0 | 10.0 | 1.4 | 5.0 |

*See the footnotes to Table 3 for definitions. NA: not available. Per cent difference is calculated for affected females as follows: [(Unaffected value − affected value)/unaffected value] × 100.

and *Madam Bee* generally are smaller than averages recorded for other Gombe chimpanzee adults (ZIHLMAN et al., 1990; MORBECK et al., in press).

### BONE WEIGHTS

Bone weights in both left and right forelimbs of *Gilka* and *Madam Bee* are lighter than minimum weights for all upper limb bones in the comparative samples (MORBECK et al., in press). In unaffected Gombe adults pairs of left and right bones are similar in weight. For example, average per cent differences range from 0 to −4.0% in both comparative groups (Tables 3 & 4). Upper limb bones of *Gilka* and *Madam Bee*, on the other hand, each show considerable side differences in bone weights (Table 4). Differences between paralyzed and nonparalyzed forelimbs are as great as 26.3% in *Gilka* and 32.0% in *Madam Bee*.

The right elements (i.e. affected side) in *Gilka* are lighter and larger differences in bone weight are evident proximally. *Madam Bee*'s left forelimb bones (i.e. affected side) also are generally lighter. The left scapula of *Madam Bee*, part of her paralyzed forelimb, however, weights more than does the right, "normal" scapula (Table 4). Compared to *Gilka*, left-right pairs in *Madam Bee* show more extreme differences in bone weight.

### BONE LENGTHS

*Gilka*'s long bone lengths are the same as or shorter than medians/means of other Gombe adults and many values (e.g. scapular dimensions and radial and ulnar lengths) lie below respective ranges of females. Similarly, all long bone lengths in *Madam Bee*, except her right clavicle, also are shorter than unaffected Gombe adult medians/means. In addition, forearm bone lengths are shorter than their respective minimum values (MORBECK et al., in press).

Average size differences between left and right linear dimensions of forelimb bones range from 0 to 2% in the pooled-sex sample of unaffected Gombe adults (Table 3). Differences in clavicle and long bone lengths in *Gilka*'s forelimb range from 1.5 to 4.1%. While left-right clavicular and scapular lengths are similar, her right scapula is small and the per cent difference in scapular breadth is greater. In *Madam Bee*, forelimb long bone length asymmetry generally is less pronounced (−0.2 to 2.8%) than observed in *Gilka* and scapular breadths are similar (Table 4).

### JOINT AREAS

Shoulder, elbow, and proximal wrist joint areas in *Gilka* and *Madam Bee*, like bone weights and lengths, are relatively small compared to other Gombe adults. Sizes of joint areas (except right radial ulnar notch area in *Gilka*) lie below medians/means for other Gombe females. The glenoid fossae and right ulnar trochlea notch in *Gilka* and both humeral trochleae and left radial fossa in *Madam Bee* are smaller than those of all other Gombe adults (MORBECK et al., in press).

In unaffected Gombe chimpanzees, there is little left-right asymmetry in glenohumeral, humero-ulnar, and proximal/distal radii joint areas (e.g. means range from 0.1 to 3.0% in the pooled-sex sample). In contrast, greater size asymmetry is evident between humeral capituli and ulnar radial notches (i.e. −5.0% and 8.0%, respectively, Table 3) with even larger differences in unaffected females (Table 4).

Joint area side differences in *Gilka* and *Madam Bee* generally are greater than those

observed in other Gombe adults (Table 4). Joint surfaces in the affected side usually are smaller than those of the nonparalyzed limb. However, respective patterns of joint area asymmetry in *Gilka* and *Madam Bee* differ when compared to each other. For instance, in *Gilka* glenohumeral joint areas (17.2%, 9.3%) and the ulnar trochlear notch in the humero-ulnar joint (15.0%) show pronounced asymmetry. The joint areas of the affected shoulder are smaller than those of the unaffected limb. In *Madam Bee* the difference between glenoid fossae (−12.9%) exceeds maximum values for other Gombe individuals while the size discrepancy between left and right humeral heads shows only a very small difference (1.7%). Furthermore, glenoid fossa area is greater in the affected shoulder joint.

In addition, compared to *Gilka*, *Madam Bee*'s humeral capituli show greater asymmetry. But her ulnar trochlear notches are more similar in size (Table 4). Like other Gombe chimpanzees, both *Gilka* and *Madam Bee* show asymmetry in ulnar radial notch joint areas, but side differences are more extreme (−37.2% and −25.0%, respectively).

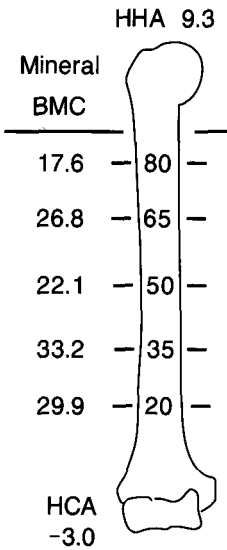
## HUMERI IN *Gilka* AND *Madam Bee*

Although maximum and diaphysial lengths of the humeri in *Gilka* and *Madam Bee* are shorter than medians/means of the comparative samples, these lengths are in the ranges of those of other Gombe adults. But, both left and right shaft sizes are relatively small. Furthermore, bone mineral content in the paralyzed limb of these females also have the lowest values recorded for Gombe chimpanzee adults (unpubl. data).

*Gilka*'s right humeral shaft, part of her paralyzed limb, is narrower than that of all other Gombe adults. Her left "normal" humerus also is small and only AP diameters of the proximal shaft lie within the range of other females. In *Madam Bee*'s left paralyzed limb, the humeral shaft is narrow, but the distal AP diameters are in the range of other adults. Unlike most bone lengths, which are shorter in *Madam Bee* compared to other Gombe females, diameters of her right, unaffected humerus are well within their range (MORBECK et al., in press). Bilateral asymmetries in BMC and cross-sectional geometry at five sites in each humeral diaphysis are summarized in Figures 1 and 2.

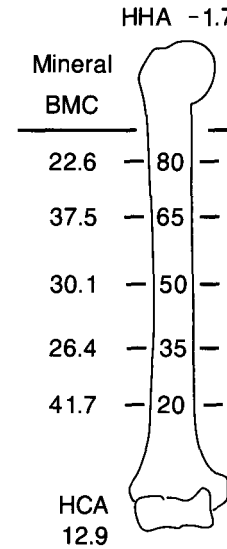
The SPA and CT data for *Gilka* indicate that the affected right humerus has 17.6% to 33.2% less BMC than the unaffected left side, while cortical area (CA) is reduced by 21.9% to 28.6% (Fig. 1). Shaft diameters and total subperiosteal area (TA) values are smaller on the affected right side, with the largest side differences found in the proximal humerus. The nonparalyzed, left medullary cavity areas (MA) also are larger than those of the affected right side. The most distal scan site, however, shows left and right MA values to be similar. Asymmetry in TA may reflect subperiosteal bone resorption, lack of normal subperiosteal expansion during growth of the affected right side, or increased subperiosteal bone formation on the unaffected ("overused") left side, or a combination of these processes. Smaller MA, most likely, represents lack of medullary expansion during growth of the paralyzed limb.

For *Madam Bee*, the affected left humerus has 22.6% to 41.7% less BMC while cortical area is diminished by 22.4% to 32.3%. Total area and diaphysial diameters also are smaller in the affected humerus. There is a proximal to distal gradient in which the asymmetry is greater proximally. Side differences in medullary cavity areas are larger in the proximal humerus. But, at the distal three scan sites asymmetry is minimal. Given that *Madam Bee* was skeletally mature at the onset of paralysis, the most likely interpretations of these data are subperiosteal resorption in the affected humerus, possibly coupled with subperiosteal bone formation in the unaffected, "normal" right humerus. In addition, at the most proximal




| Mineral BMC | | Areas TA | MA | CA | Diameters AP | ML |
|---|---|---|---|---|---|---|
| 17.6 | 80 | 28.5 | 32.0 | 21.9 | 20.0 | 12.8 |
| 26.8 | 65 | 28.8 | 29.1 | 28.2 | 20.5 | 15.8 |
| 22.1 | 50 | 27.9 | 29.8 | 24.3 | 17.5 | 16.7 |
| 33.2 | 35 | 23.8 | 21.2 | 28.6 | 11.1 | 15.8 |
| 29.9 | 20 | 11.4 | 0.7 | 26.1 | 6.3 | 11.6 |
| | | | | Maximum Length | | 2.1 |
| | | | | Diaphysial Length | | 0.6 |
| | | | | Weight | | 26.3 |

**Fig. 1.** *Gilka*: per cent differences of dimensions of left and right humeri. Per cent difference is calculated as follows: [(left, unaffected value − right, affected value)/left, unaffected value] × 100. HHA: Humeral head area; HCA: humeral capitulum area; HTA: humeral trochlea area; 80, 65, 50, 35, 20, percentage of shaft length, site locations for diaphysial measurements; BMC: bone mineral content; TA: total subperiosteal area; MA: medullary area; CA: cortical area; AP: anteroposterior; ML: mediolateral. See text for discussion.




| Mineral BMC | | Areas TA | MA | CA | Diameters AP | ML |
|---|---|---|---|---|---|---|
| 22.6 | 80 | 19.9 | 18.7 | 22.4 | 20.0 | 4.9 |
| 37.5 | 65 | 13.8 | 8.7 | 25.7 | 7.0 | 3.5 |
| 30.1 | 50 | 12.1 | 2.2 | 32.3 | 9.5 | 3.7 |
| 26.4 | 35 | 11.5 | 3.3 | 28.0 | 0 | 10.0 |
| 41.7 | 20 | 10.5 | 0.7 | 29.7 | 2.9 | 6.7 |
| | | | | Maximum Length | | -0.2 |
| | | | | Diaphysial Length | | 1.3 |
| | | | | Weight | | 32.0 |

**Fig. 2.** *Madam Bee*: per cent differences of dimensions of left and right humeri. Per cent difference is calculated as follows: [(right, unaffected value − left, affected value)/right, unaffected value] × 100. See Fig. 1 for definitions.

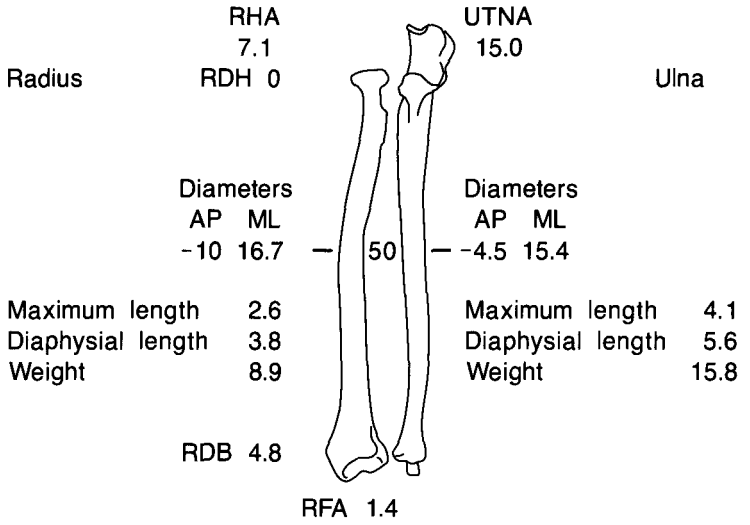



**Fig. 3.** *Gilka*: per cent differences of dimensions of left and right forearm bones. Per cent differences calculated as in Fig. 1. RHA: radial head area; UTNA: ulnar trochlear notch area; RFA: radial fossa area; RDH: radius, diameter head; RDB: radius, distal breadth; — 50, percentage of shaft length, midshaft location of diaphysial dimensions; AP: anteroposterior; ML: mediolateral.

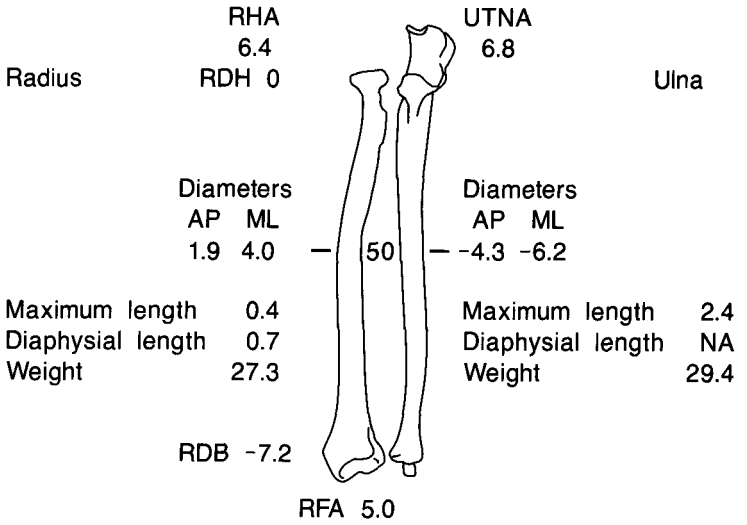



**Fig. 4.** *Madam Bee*: per cent difference of dimensions of left and right forearm bones. Per cent difference is calculated as in Fig. 2. See Fig. 3 for definitions. NA: Not available. Diaphysial length of the right ulna is difficult to measure since the bone is fractured. Location of midshaft diameters is based on maximum ulnar length.

scan site there also may have been a lack of age-related expansion of the medullary cavity in the humerus of the left paralyzed limb.

### Radii and Ulnae in *Gilka* and *Madam Bee*

Forearm bones of *Gilka* and *Madam Bee*, like the scapula and humerus, are small relative to other Gombe chimpanzees (Morbeck et al., in press). Figures 3 and 4 illustrate per cent differences between left and right weights, lengths, including midshaft diameters, and joint areas of radii and ulnae.

Midshaft ML diameters in the "normal" left forearm bones of *Gilka*, as expected, are considerably broader than in the right radius and ulna (Fig. 3). On the other hand, midshaft AP diameters of the radius and ulna of *Gilka*'s right paralyzed limb are wider than those of the nonparalyzed left side.

The midshaft region in the radius in *Madam Bee*'s left paralyzed forelimb is slightly smaller than that of the opposite side (Fig. 4). But the left ulna is larger than its nonparalyzed counterpart. The ulna in the "unaffected" right forelimb is broken distally and incompletely healed with a lack of union and possible pseudoarthrosis that developed at the fracture site (Jurmain, 1989; Zihlman et al., 1990). Ulnar remodeling probably is associated both with muscle paralysis on the left side and the dynamics of locomotor loading and response to fracture in the right forearm.

## DISCUSSION

### Skeletal Asymmetry

The forelimb skeletons of *Gilka* and *Madam Bee* display extreme, but variable, left-right asymmetry in most dimensions, whereas bilateral variation generally is not pronounced in other Gombe chimpanzees. The bones of the partially immobilized limb of each affected female show evidence of disuse osteoporosis (Morbeck et al., in press; Smith & Gilligan, 1989). Side differences may result from disturbances of bone growth and from remodeling in response to muscle paralysis as a result of paralytic poliomyelitis in the affected limb and particular muscle-bone interactions in movement and load-bearing of both paralyzed and nonparalyzed forelimbs. In contrast, hindlimbs of these individuals apparently were not affected and available bone weights, lengths, and joint areas show little left-right asymmetry (Morbeck et al., in press).

### Forelimb Disuse: Nerves, Muscles, Bones

Nerves, muscles, and bones are affected by peripheral paralytic poliomyelitis. Strains of the poliovirus produce the disease and can be transmitted among individuals.[3] Motor neurons in the anterior horn of the spinal cord of host organisms are destroyed when they serve as an environment for poliovirus replication. Motor neuron destruction leads to degeneration of its axon and motor end plate (see Bodian, 1985 for the sequence of pathohistological changes). Muscle fibers in the affected motor unit undergo spontaneous fibrillation and,

3) Muscle paralysis of limbs as observed in Gombe chimpanzees is the primary clinical symptom of peripheral paralytic poliomyelitis. This phase of the disease affects the central nervous system, peripheral nerves, muscles, and, if paralysis is continued, bone morphology. The poliovirus and the disease poliomyelitis are well-studied in chimpanzees and Old World monkeys as well as in humans (for a summary see Morbeck et al., in press.)

if not re-innervated by a neighboring motor unit axon, will atrophy. Recovery can occur, but long-term muscle paralyses or weaknesses, as observed in *Gilka* and *Madam Bee*, usually result from an overload on surviving motor neurons when these innervate many more muscle fibers than encountered in a normal motor unit (HALSTEAD & WIECHERS, 1985).

Loss of electrophysiological stimulation of muscles produces changes at many levels of biological organization. These include, for example, above-mentioned constraints on body movements and muscle strength brought about by reduction or loss of muscle fiber contractile capabilities (hypokinesia) with decrease in muscle fiber size, loss of myofibrillar contractile proteins, and biochemical changes in protein synthesis. Modification of capillary distribution and blood supply to muscles, and potential changes in endocrine regulation activities and metabolic activity also may occur. Reduction of weight-bearing abilities (hypodynamia) also may take place. Cortical bone remodeling with mechanical disuse may include a decrease in osteon density. Metabolic activity and mineral homeostasis of bone is compromised, usually with calcium loss. Loss of calcium affects bone structure and strength and influences interactions of the endocrine system, blood chemistry, and intestine and kidney function. Furthermore, the immune system also may be affected (see MUSACCHIA et al., 1988; SANDLER & VERNIKOS, 1986; CARTER, 1984; DALAKAS et al., 1984; HALSTEAD & WIECHERS, 1985; SCHNEIDER & MCDONALD, 1984; ERIKSEN et al., 1988; LANYON & RUBIN, 1985; UHTHOFF & JAWORSKI, 1978; STOUT, 1982; MAZESS & WHEDON, 1983; WHEDON, 1984; SMITH & GILLIGAN, 1989). Our focus here is on gross anatomical structure of forelimb bones, muscles, and nerves.

Size, shape, weight, mineral content, and internal structure of bone are the products of genetic instructions for physiologically-mediated processes of growth, maturation, reproduction, and aging. Appropriate nutrition and biomechanical responses to movement and load-bearing result in species-typical morphology at any given time in an individual's life history.

The details of how bone functions physiologically and remodels structurally during the life cycle and, at the same time, changes in response to a variety of mechanical loads at the molecular and cellular level are not yet completely understood (e.g. KAHN et al., 1983; PARFITT, 1984; FROST, 1988; WOZNEY et al., 1988; BLAIR et al., 1989; SMITH & GILLIGAN, 1989). However, limb bones do not develop or remodel normally under conditions in which nutrition is inadequate (HUSS-ASHMORE et al., 1982) and in which normal strains do not occur, when intermittent loads are not present due to nonfunctional muscles, and when pressure is not exerted on the periosteum, for instance, in individuals with muscle paralyses (LANYON & RUBIN, 1985).

Individualized patterns of asymmetries suggest that different muscles, or parts of muscles, and associated nerves were damaged during different life stages of the affected females. Asymmetry measured in the five sites on humeri can be matched to origins and insertions of particular muscles and their associated nerves. Differences occur, for instance, in the distal humeral diaphysis with loss or partial loss of the brachialis (musculocutaneous nerve) and medial head of triceps muscles (radialis nerve); and at the 65% site, deltoid (axillary nerve), teres major (lower subscapular nerve), coracobrachialis and brachialis muscles (musculocutaneous nerve). In addition, loss of a full range of movement of *Gilka*'s wrist and thumb relates primarily to partial paralysis of muscles innervated by the median and radial nerves.

Tracing the innervation of these muscles through the brachial plexus to their respective spinal cord roots indicates variable disfunction in lower cervical (C4 to 8) and first thoracic (T1) nerves (SWINDLER & WOOD, 1973). Neurotropic poliovirus apparently differentially

affected this portion of the spinal cords of *Gilka* and *Madam Bee*.

Skeletal analyses, therefore, can be used to identify regions of neuromuscular disfunction and effects of long-term, unilateral, partial forelimb disuse. Poliomyelitis and poliovirus activity in these individuals, however, can not be diagnosed directly from bone morphology. But long-term partial forelimb immobilization is indicated and this complements a diagnosis of poliomyelitis based on field observations and other data.

### Forelimb Disuse: Role in Individual Life History

Gombe field data allow us to link skeletal features and the poliomyelitis disease experience to observed reproductive outcomes of affected individuals. Partial loss of limb function due to paralytic poliomyelitis in *Gilka* and *Madam Bee* apparently compromised their respective abilities to rear infants. In addition, different ages at the time of onset of paralysis relative to chimpanzee life stages influenced whether these affected females made genetic contributions to the next generation.

After contracting paralytic poliomyelitis while a subadult, *Gilka* grew to reproductive maturity. She became pregnant several times but none of her infants survived. In at least two cases, adult females cooperated with each other to kill *Gilka*'s newborn infant (Goodall, 1986). *Gilka* probably could have lost her infants even if she had been healthy (Goodall, pers. comm.). But, they died, in part, as a result of locomotor and manipulatory difficulties associated with partial forelimb disfunction.

Two infants born to *Madam Bee* after she experienced paralytic poliomyelitis also died. The disease apparently affected her ability to care for an infant. But, *Madam Bee* already had two daughters when her forelimb first became partially immobilized. Later, each daughter had offspring (Goodall, 1986).

Poliovirus activity and the resulting disease, paralytic poliomyelitis, affected nerves, muscles, bones, and, ultimately, reproductive efforts of both *Gilka* and *Madam Bee* in the same way. However, the timing of the onset of disease influenced lifetime reproductive outcome. *Madam Bee* contributed to the local population gene pool at Gombe whereas *Gilka* did not.

## SUMMARY AND CONCLUSION

Patterns of bilateral variation of the forelimb skeletons of *Gilka* and *Madam Bee* are compared to those of other Gombe chimpanzees. Forelimb bones of these individuals with partially paralyzed limbs, unlike those of unaffected adults, show considerable asymmetry. Individualized skeletal responses of *Gilka* and *Madam Bee* apparently reflect differences in ages of onset of limb paralysis and differences in affected spinal cord regions, nerves, muscles, and associated adjustments of locomotor behavior. Knowledge of poliomyelitis and the nature of nerve-muscle-bone relationships allow explanations of skeletal features. Observations of the same individuals during life illustrate the impact of disease, such as paralytic poliomyelitis recorded in the skeleton, on survival and reproduction.

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## REFERENCES

BAUER, H. R., 1977. Chimpanzee bipedal locomotion in the Gombe National Park, East Africa. *Primates*, 18: 913–921.

BLAIR, H. C., S. L. TEITELBAUM, R. GHISELLI, & S. GLUCK, 1989. Osteoclast bone resorption by a polarized vacuolar proton pump. *Science*, 245: 855–857.

BODIAN, D., 1985. Motoneuron disease and recovery in experimental poliomyelitis. In: *Late Effects of Poliomyelitis*, L. S. HALSTEAD & D. O. WIECHERS (eds.). Symposia Foundation, Miami, Florida, pp. 45–55.

CARTER, D. R., 1984. Mechanical loading histories and cortical bone remodeling. *Calcif. Tiss. Int.*, 36: S19–S24.

DALAKAS, M. C., J. L. SEVER, D. L. MADDEN, N. M. PAPADOPOULOS, I. C. SHEKARCHI, P. ALBRECHT, & A. KREZLEWICZ, 1984. Late postpoliomyelitis muscular atrophy: clinical, virologic, and immunologic studies. *Rev. Infect. Dis.*, 6, Suppl. 2: S562–S567.

ERIKSEN, E. F., D. S. COLVARD, N. J. BERG, M. L. GRAHAM, K. G. MANN, T. C. SPELSBERG, & B. L. RIGGS, 1988. Evidence of estrogen receptors in normal human osteoblast-like cells. *Science*, 241: 84–86.

FROST, H. M., 1988. Vital biomechanics: proposed general concepts for skeletal adaptations to mechanical usage. *Calcif. Tiss. Int.*, 42: 145–156.

GOODALL, J. VAN LAWICK, 1968. Behaviour of free-living chimpanzees of the Gombe Stream area. *Anim. Behav. Monogr.*, 1: 163–311.

————, 1971. *In the Shadow of Man.* Houghton Mifflin Co., Boston.

GOODALL, J., 1983. Population dynamics during a 15 year period in one community of free-living chimpanzees in the Gombe National Park, Tanzania. *Z. Tierpsychol.*, 61: 1–60.

————, 1986. *The Chimpanzees of Gombe. Patterns of Behavior.* Harvard Univ. Press, Cambridge, Massachusetts.

————, 1987a. News from Gombe. Fall 1987. The Jane Goodall Institute for Wildlife Research, Education & Conservation. Tucson, Arizona.

————, 1987b. News from Gombe. Spring 1987. The Jane Goodall Institute for Wildlife Research, Education & Conservation. Tucson, Arizona.

————, 1990. *Through a Window. My Thirty Years with the Chimpanzees of Gombe.* Houghton Mifflin, Boston.

HALSTEAD, L. S. & D. O. WIECHERS (eds.), 1985. *Late Effects of Poliomyelitis.* Symposia Foundation, Miami, Florida.

HUCKSTEP, R. L., 1975. *Poliomyelitis. A Guide for Developing Countries — Including Appliances and Rehabilitation for the Disabled.* Churchill Livingstone, New York.

HUSS-ASHMORE, R., A. H. GOODMAN, & G. J. ARMELAGOS, 1982. Nutritional inference from paleopathology. In: *Advances in Archaeological Method and Theory, Vol. 5*, M. SCHIFFER (ed.), Academic Press, New York, pp. 395–473.

JURMAIN, R., 1989. Trauma, degenerative disease, and other pathologies among Gombe chimpanzees. *Amer. J. Phys. Anthropol.*, 80: 229–237.

KAHN, A. J., M. D. FALLON, & S. L. TEITELBAUM, 1983. Structure-function relationships in bone: an examination of events at the cellular level. In: *Bone and Mineral Research, Annual 2*, W. A. PECK (ed.), Elsevier Science Publ., New York, pp. 125–174.

LANYON, L. E. & C. T. RUBIN, 1985. Functional adaptation in skeletal structures. In: *Functional Vertebrate Morphology*, M. HILDEBRAND, D. M. BRAMBLE, K. F. LIEM, & D. B. WAKE (eds.), Harvard Univ. Press, Cambridge, Massachusetts, pp. 1–25.

MAZESS, R. B. & G. D. WHEDON, 1983. Immobilization and bone. *Calcif. Tiss. Int.*, 35: 265–267.

MORBECK, M. E. & A. L. ZIHLMAN, 1989. Body size and proportions in chimpanzees, with special reference to *Pan troglodytes schweinfurthii* from Gombe National Park, Tanzania. *Primates*, 30: 369–382.

————, ————, D. R. SUMNER, & A. GALLOWAY, in press. Skeletal responses to long-term paralysis in free-ranging chimpanzees. In: *Pathological Conditions of the Skeleton of Nonhuman Primates*, N. LOVELL & R. JURMAIN (eds.), Alan R. Liss Inc., New York.

MUSACCHIA, X. J., J. M. STEFFEN, & R. D. FELL, 1988. Disuse atrophy of skeletal muscle: animal models. In: *Exercise and Sport Sciences Reviews*, K. B. PANDOLF (ed.), MacMillan Publ. Co., New York, pp. 61–87.

NIE, N. H., C. N. HULL, J. G. JENKINS, K. STEINBRENNER, & D. N. BENT, 1975. *SPSS: Statistical Package for the Social Sciences.* McGraw-Hill Book Co., New York.

NORUŠIS, M. J., 1984. *SPSS/PC for the IBM PC/XT.* SPSS Inc., Chicago.

PARFITT, A. M., 1984. The cellular basis of bone remodeling: the quantum concept reexamined in light of recent advances in the cell biology of bone. *Calcif. Tiss. Int.*, 36: S37–S45.

SABIN, A. B., 1985. Oral poliovirus vaccine: history of its development and use and current challenge to eliminate poliomyelitis from the world. *J. Infect. Dis.*, 151: 420–436.

SANDLER, H. & J. VERNIKOS (eds)., 1986. *Inactivity: Physiological Effects.* Academic Press, New York.

SCHNEIDER, V. S. & J. MCDONALD, 1984. Skeletal calcium homeostasis and countermeasures to prevent disuse osteoporosis. *Calcif. Tiss. Int.*, 36: S151–154.

SHEA, B. T., 1986. Scapula form and locomotion in chimpanzee evolution. *Amer. J. Phys. Anthropol.*, 70: 475–488.

SMITH, E. L. & C. GILLIGAN, 1989. Mechanical forces and bone. In: *Bone and Mineral Research, Vol. 6*, W. A. PECK (ed.), Elsevier Science Publ., New York, pp. 139–173.

STOUT, S. D., 1982. The effects of long-term immobilization on the histomorphology of human cortical bone. *Calcif. Tiss. Int.*, 34: 337–342.

SUMNER, D. R., 1984. Size, shape and bone mineral content of the human femur in growth and aging. Ph.D. thesis, Univ. of Arizona, Tucson.

————, M. E. MORBECK, & J. J. LOBICK, 1989. Apparent age-related bone loss among adult female Gombe chimpanzees. *Amer. J. Phys. Anthropol.*, 79: 225–234.

SWINDLER, D. R. & C. D. WOOD, 1973. *An Atlas of Primate Gross Anatomy. Baboon, Chimpanzee, and Man.* Univ. of Washington Press, Seattle, Washington.

TELEKI, G., E. E. HUNT, JR., & J. H. PFIFFERLING, 1976. Demographic observations (1963–1973) on the chimpanzees of Gombe National Park, Tanzania. *J. Human Evol.*, 5: 559–598.

UHTHOFF, H. K. & Z. F. G. JAWORSKI, 1978. Bone loss in response to long-term immobilization. *J. Bone Joint Surg.*, 60-B: 420–429.

WHEDON, G. D., 1984. Disuse osteoporosis: physiological aspects. *Calcif. Tiss. Int.*, 36: S146–S150.

WOZNEY, J. M., V. ROSEN, A. J. CELESTE, L. M. MITSOCK, M. J. WHITTERS, R. W. KRIZ, R. M. HEWICK, & E. WANG, 1988. Novel regulators of bone formation: molecular clones and activities. *Science*, 242: 1528–1534.

ZIHLMAN, A. L., M. E. MORBECK, & J. GOODALL, 1990. Skeletal biology and individual life history of Gombe chimpanzees. *J. Zool. London*, 210: 37–61.

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