The Strength of Great Apes and the Speed of Humans

by Alan Walker

Cliff Jolly developed a causal model of human origins in his paper "The Seed-Eaters," published in 1970. He was one of the first to attempt this, and the paper has since become a classic. I do not have such grand goals; instead, I seek to understand a major difference between the living great apes and humans. More than 50 years ago, Maynard Smith and Savage (1956) showed that the musculoskeletal systems of mammals can be adapted for strength at one extreme and speed at the other but not both. Great apes are adapted for strength—chimpanzees have been shown to be about four times as strong as fit young humans when normalized for body size. The corresponding speed that human limb systems gain at the expense of power is critical for effective human activities such as running, throwing, and manipulation, including tool making. The fossil record can shed light on when the change from power to speed occurred. I outline a hypothesis that suggests that the difference in muscular performance between the two species is caused by chimpanzees having many fewer small motor units than humans, which leads them, in turn, to contract more muscle fibers earlier in any particular task. I outline a histological test of this hypothesis.

I was teaching at Makerere University in Kampala when Cliff Jolly took a sabbatical in Uganda, and it was then that I observed the amazing strength of chimpanzees. I was trying to observe and record chimpanzee locomotion in Budongo Forest in northern Uganda. On one occasion, I was minding my own business while walking along a forest trail when I nearly bumped into an adult male chimpanzee that was doing the same. The frightened animal swung at a nearby tree buttress root, making a resonating booming sound. After this display, the animal raced up the trunk and proceeded to shake branches high above me. When my heart rate returned to normal, I tried to imitate the chimpanzee by banging on the buttress as hard as I could. I could produce only a laughably feeble sound. Thus it was that I came to appreciate firsthand what many people know anecdotally—that great apes are immensely strong.

Bauman (1923, 1926) showed that adult male and female chimpanzees held long in captivity were much stronger than any of several fit young football players when normalized for body mass. He had the animals (when they felt like it) and the students pull on a calibrated metal loop dynamometer. The female recorded a two-handed pull of 1,260 pounds, while the male recorded a one-handed pull of 847 pounds. The strongest student managed a one-handed pull of 210 pounds and a two-handed pull of 491 pounds. When normalized for body mass, this meant that the chimpanzees were more than four times as strong as the men. But note that Finch (1943) could not persuade several chimps at the Yale Primate Laboratory to match a single male human in pulling strength when pulling on a rope for food items—unlike students, apes cannot always be trained to behave.

Those of us who have watched great apes at close quarters are not surprised when reading Bauman's account. He asked the obvious question, "To what factors do they owe this very striking superiority?" And the related questions on his list are some of those that we ask today: Are the ape muscle fibers intrinsically stronger? Are the motor nerve impulses different? Or is it a combination of the two?

Bauman went on to speculate about the human ancestral condition, mentioning Neanderthals particularly. Bauman did not address the effect of the skeletal levers or muscle and tendon architecture, and following Smith and Savage (1956), we would add those to the list today. Also, we might ask about the importance of neuromuscular control in locomotion.

Some recent work has again raised the issue of ape versus human strength. Scholz et al. (2006) measured vertical jumping in three *Pan paniscus* and claimed that their mechanical output was higher than typical human maximum performance. They used an inverse dynamics modeling approach and concluded with the following questions concerning their

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results: Did we overestimate the mechanical output at the hip by using a rigid trunk model? Did we underestimate the animals' hip and knee extensor muscle masses? Can humans voluntarily recruit as much of their muscle mass as bonobos? Is bonobo muscle different from human muscle in that it produces higher forces per cross-sectional area? These researchers are also thinking along the lines outlined by Bauman, but they favor the last possibility to explain their results.

Body Mass Distribution, Moment Arms, and Muscle Architectural Differences

Between 60% and 80% of a primate's body mass is used for locomotion (Grand 1977). Humans have small torsos relative to their limbs and especially to their legs. The forelimb muscle mass of the great apes that is required for climbing is also a particularly obvious difference from that of humans.

Zihlman (1992) gives comparisons between the amount of forelimb to hind limb mass in chimpanzees and humans. She found that chimpanzees had 16% of their total body weight in their forelimbs versus 24% in the hind limbs. The corresponding values for humans that she took from the literature were 9% and 38%, respectively. So chimpanzees have nearly double the relative amount of forelimb muscle mass that humans have, and humans have about half again as much hind limb muscle mass as chimpanzees. Thorpe et al. (1999) provide a comprehensive quantitative study of muscle cross-sectional areas, moment arms, and fascicle lengths based on three chimpanzees, and they compared the data with that from humans. Following that study, Carlson (2006) dissected two chimpanzee cadavers, and he also concluded that their muscle architecture differed from that of humans. These last two studies quantify the differences between chimpanzees and humans and show that similar studies of gorilla and orangutan cadavers would be useful. In studies such as these, the physiological cross-sectional area was calculated by examination of the fiber direction in all types of muscle, including pennate ones. Detailed studies of the pennation angles for all chimpanzee and human limb muscles have not been published, but the recent finding that the architecture of pennate muscles allows variable gearing as a mechanism to modulate muscle performance in variable tasks (Azizi, Brainerd, and Roberts 2008) suggests that such studies would be worthwhile.

Thorpe et al. (1999) showed that both forelimb and hind limb muscle fascicle lengths were shorter in humans than in chimpanzees. They point out that animals that need greater joint mobility, as in climbing, also need longer muscle fascicles. The shorter fascicles of humans prevent large joint excursions, but their muscle architecture instead makes for large moments at joints. Joint anatomy (mechanical advantage) is important when measuring noncyclic forces, but it is less important for power because the effects of lever anatomy on forces are counteracted by opposite changes in speed. Scholz et al. (2006) thought that their bonobos were producing more force per cross-sectional area than humans, but this is extremely unlikely, because it has been shown that the maximum net force output by all sorts of motors (and not just biological ones) is surprisingly similar to force output scaling as $(\text{motor mass})^{0.67}$ (Marden 2005). Residual variation is related to things such as isoform composition of contractile proteins and fractional area of myofibrils, but this is of minor importance. It seems clear that myosin isoforms are major determinants of functional differences in muscle fibers, but there are many unresolved issues having to do with aging and fiber variation, so the study of isoforms has not yet brought about any uncontroversial synthesis (Bottinelli 2001). Further, to my knowledge, no isoform studies have been done on chimpanzee limb musculature, and to study all chimpanzee and human limb muscles histochemically would be a huge, expensive, and probably unnecessary task.

The Control of Limb Muscles

The anatomical basis for this section can be found in any comprehensive anatomy text (see, e.g., Standring 2004). The control over limb muscles is through acetylcholine secretion at the motor end plates of the motor neurons. Motor units are the functional contractile entities—a single alpha motor neuron and all of its innervated muscle fibers. The size of these can vary considerably from as few as six to more than 2,000 fibers per nerve, and those fibers innervated by a single nerve can be spread unevenly through a whole muscle and even between muscles (Burke and Tsairis 1973). So the control of single muscles is complex, and to my knowledge, there are no comparative studies of relative motor units in apes and humans. But differences in end plate distribution could contribute to differences in muscle function. The Henneman size principle (Henneman, Somjen, and Carpenter 1965; Milner-Brown, Stein, and Yemm 1978; Henneman 1985; Cope and Pinter 1995) is an empirical rule that says that in a given muscle action, the smaller motor units (those with few fibers per nerve) contract first and that increased muscle force involves the successive, orderly recruitment of larger and larger units based on their increasing force output. This serves to make sure that the muscle fibers most susceptible to fatigue are recruited first and also to aid in fine control of muscle force, whatever force the task calls for.

There is also the upstream control of motor neurons to consider. There are well-known cases of "hysterical strength," where people suffering seizures exhibit considerably more muscle power than normal. There are also many anecdotes about people in very stressful situations being able to do things that would normally be considered impossible—lifting cars off trapped people, for instance. Add to this the effect of severe electric shock, where people are often thrown violently by their own extreme muscle contraction, and it is clear that we do not contract all our muscle fibers at once. So there might be a degree of cerebral inhibition in people that prevents them from damaging their muscular system that is not present, or not present to the same degree, in great apes. I

do not know of any experimental evidence to support or refute this idea, although cortical inhibition of motor impulses has been experimentally demonstrated in animal models and with magnetic inhibition in people.

Fossil Evidence

Long-bone robustness has been examined in fossil hominins (e.g., Ruff et al. 1993; Ruff, McHenry, and Thackeray 1999) because bone responds to the mechanical stresses placed on it during life, and there is a clear reduction in long-bone strength late in human evolution. Ruff (1987) showed that chimpanzees have stronger average femoral strength than modern humans, and Ruff, McHenry, and Thackeray (1999) showed that some early hominins (*Australopithecus* and *Homo habilis*) had femora that were as strong as those of chimpanzees. Comparison of the reduction in diaphyseal strength to that seen today in humans suggests that it began in early *Homo erectus* by about 1.6 million years ago, at about the time that major trophic and locomotor shifts occurred in our genus (Shipman and Walker 1989; Bramble and Lieberman 2004). Robusticity continued in an exponential decline from then until today (Ruff et al. 1993). Variation in bone strength is to be expected given the great differences in modern human lifestyles, but cultural development and technological advances must be major contributing factors in the reduction in skeletal loads during development as well as later in life in people over the past few thousand years.

A Hypothesis about Ape Strength

The control of the muscles involved in locomotion comes about through the corticospinal tracts acting on motor neurons in the spinal cord. The motor nerves are themselves made of a variable number of motor units such that some muscles have more innervation and others less. MacLarnon (1993, 1996) showed that chimpanzees have much smaller amounts of gray matter in their spinal cords than do humans. This is not surprising, because the central nervous system has precise mapping of its constituent parts that results in brain weight scaling to spinal cord weight in most mammals with a slope very close to 1. Also, chimpanzees have much smaller brains than humans for their body size. MacLarnon (1996) used a mixed sample of extant primates and found that the 95% confidence interval for the regression slope of brain weight to spinal cord weight was 1.03–1.34 ($r = 0.96$, $P < .001$). Her data included animals with very different body plans and types of locomotion.

Figure 1 shows MacLarnon's (1993) figure 15.5*A* and 15.5*B* spinal cord data for chimpanzees and humans but replotted to the same scale, showing the much greater cross-sectional area of the spinal cord in humans. In fact, chimpanzees have only about one-fifth of the cross-sectional area of neuron-containing gray matter that humans have, despite having roughly three-quarters the body mass. Some of these neurons

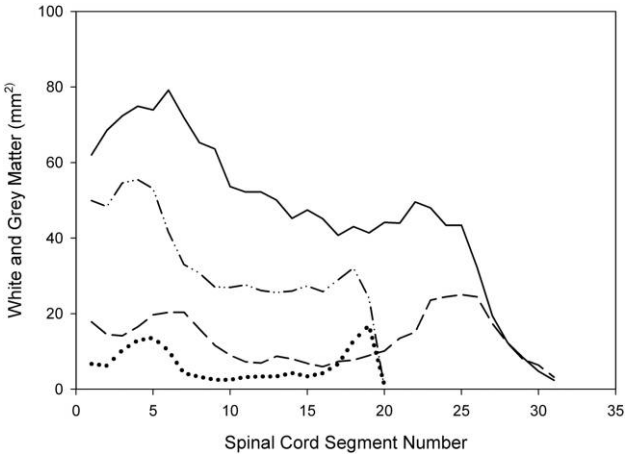

Figure 1. White matter and gray matter areas (mm^2) at measured levels of the spinal cords of chimpanzees and humans. Data from MacLarnon (1993). Solid line = human white matter. Dashed line = human gray matter. Dashed-dotted line = chimpanzee white matter. Dotted line = chimpanzee gray matter.

are interneurons that connect the sensory ganglion neurons to motor and other neurons in the cord, but those in the ventral half of the gray matter are mostly motor neurons for skeletal muscle. There are motor neurons in the intermediolateral cell column that are visceromotor to smooth muscle and glands, but the cervical and lumbosacral enlargements of the cord that correspond to increases in nerves supplying the arm and leg muscles are clearly seen as peaks on the graphs showing the dominance of these motor nerves.

It follows that if there is much less gray matter relative to muscle mass in chimpanzees, we can hypothesize that there must be fewer motor neurons to control the locomotor muscles. There must also be relatively few small motor units and more large ones, and that is the reason apes seem so strong relative to modern humans. To put it another way, modern humans have a much greater range of motor unit sizes over their muscle mass, and this allows us to recruit muscles for more complex but less forceful tasks. It may also be that under normal circumstances, humans have greater cortical or spinal inhibition of motor neurons so that the largest motor units are recruited only under extraordinary conditions.

To map out the complex distribution of axons to the muscles would be a very difficult task, as the axons in a single unit are dispersed through and across muscles. However, the motor units are patterned in such a way that although they vary in functional properties—such as twitch force, contractile speed, axonal conduction velocity, fatigue resistance, recruitment thresholds, firing rates, and firing patterns—the properties, together with the corresponding morphological characteristics—such as soma size, axon diameter, and muscle fiber size—are interrelated (Sale 1987). The smallest motor units have the smallest twitch force, the slowest contraction speed, the slowest conduction velocity, the greatest resistance to fatigue, the lowest recruitment thresholds, and the lowest

minimum and maximum firing rates but also have the smallest soma size, axon diameter, and muscle fiber size. The converse applies to the largest motor units (Sale 1987).

It is fortunate that the diameter of the motor axons is itself related to the number of muscle fibers that it innervates (Sale 1987; Burt 1993) because it means that a statistical analysis of the distributions of axon diameters in each spinal nerve can be used as a proxy for the distribution of motor unit sizes in those nerves. The motor nerves themselves travel in the ventral nerve root (see fig. 2). There are no sensory fibers in the ventral root of most vertebrates, and so the motor axon diameters can be measured and counted in sections of the ventral root. There is, however, one confusing factor, and that is that the ventral roots also contain preganglionic motor fibers of the sympathetic autonomic nerves. These, however, can be accounted for, because their axons have to synapse in sympathetic chain ganglia outside the spinal cord, and in order for these nerves to reach a ganglion, they are carried away from the spinal nerve in the white communicating ramus at each spinal nerve level. A few sensory sympathetic fibers run in the spinal nerves, and the following test can account for these because they are often unmyelinated (Standring 2004).

A Test of the Hypothesis

Histological sections of each consecutive ventral nerve root can be made from preparations of spinal cords from adult chimpanzees and humans. We can then measure the distribution of axon diameters in those nerves as a proxy for motor

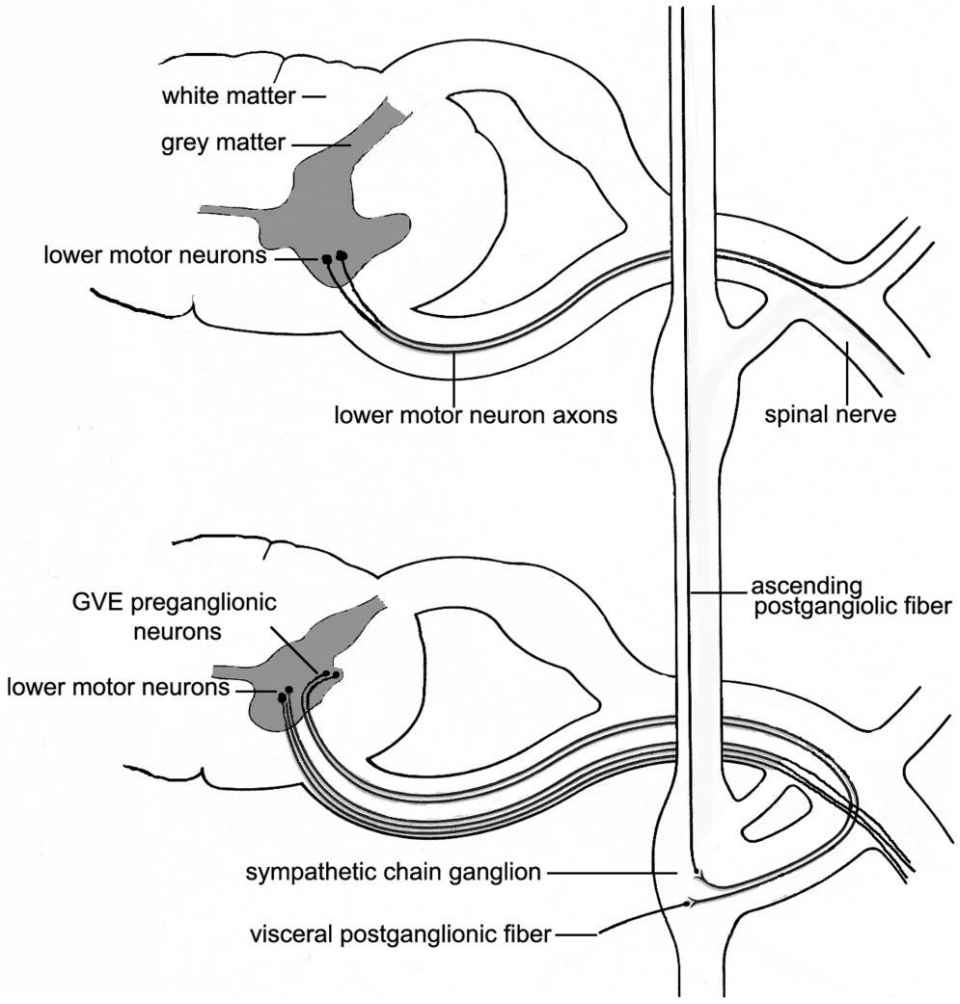

Figure 2. Schematic representation of the left sides of two adjacent spinal cord sections viewed from the front, with part of the sympathetic chain and two chain ganglia shown. Bottom right shows a white communicating ramus carrying two preganglionic visceral motor fibers. *GVE* = general visceral efferent.

unit size distribution. However, these not only contain lower motor neuron axons but also have preganglionic general visceral efferent nerve axons running in them, and these can be myelinated just like the lower motor axons. So in order to construct a proxy for the motor unit distribution, we have to subtract those axons from the spinal nerve distribution. Fortunately, the axons in the white communicating ramus are, with the exception of a small number of usually unmyelinated afferent fibers, general visceral efferent fibers, and so sections of this ramus at the same level as the spinal nerve of interest will give a distribution of myelinated autonomic fibers that can be subtracted from that of the spinal nerve. The visceral afferent fibers that are carried in the white communicating ramus are much fewer than the efferents and are variably myelinated. It is difficult to think of an easy method to account for these, but there are likely to be roughly the same number that serve the viscera of apes as those that serve the viscera of humans, so this is (probably) a complication that is not severe. If this motor unit size hypothesis is correct, then the distribution in humans, as well as having a much greater number of axons, will also show many more small axon diameters than found in chimpanzees.

Conclusions

Major differences in muscular performance between great apes and humans have been put down to differences in muscle architecture and joint lever systems due to obviously different locomotor adaptations. Whether this is sufficient to explain the differences in strength between chimpanzees and humans or whether differences in motor unit distribution are also responsible can be explored through detailed quantitative histological study of motor axon size distribution in the spinal nerves of chimpanzees and humans. The experiments that I can think of to test whether there is cerebral inhibition in humans and not in apes are probably unethical or at the least very difficult to justify and undertake.

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