Estimate of muscle-shortening rate during locomotion

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LINDSTEDT, STAN L., HANS HOPPELER, KATHLEEN M. BARD, AND HARLEY A. THRONSON, JR. *Estimate of muscle-shortening rate during locomotion.* Am. J. Physiol. 249 (Regulatory Integrative Comp. Physiol. 18): R699–R703, 1985.—All skeletal muscle can produce roughly the same maximal cross-sectional force; however, the power (energy·time^{-1}) required to develop and maintain that force increases with increasing contraction velocity. Thus the rate of muscle tension development may be of primary importance in setting the energy demand of contracting muscle. We have estimated the rate of muscle shortening during terrestrial locomotion in mammals as a function of body mass. The rate of muscle shortening of the knee extensors is much faster in small than large mammals, scaling in proportion to the −0.23 power of mass. This exponent suggests a constant body size-independent relation among skeletal muscle: O_2 consumption, mitochondria content, myosin ATPase activity, and in vivo shortening velocity.

allometry; muscle energetics; knee extensors; oxygen consumption; mitochondria

SKELETAL MUSCLE is the most abundant single tissue type among vertebrates, and it may also be the most plastic, both morphologically and physiologically. At rest, the O_2 consumption of skeletal muscle is among the lowest of all tissues; yet its maximum rate of O_2 consumption can be the highest. During maximum sustained aerobic activity >90% of the O_2 consumed by the lungs is utilized by mitochondria in the working skeletal muscle.

Morphologically, skeletal muscle is equally plastic, responding to the type of demands placed on it by structural shifts in both metabolic and contractile properties. The densities of capillaries and mitochondria within skeletal muscle adjust rapidly to the specific demand placed on the muscle. For instance, as few as 15 h endurance training in humans may result in a 30% increase in volume density of mitochondria and number of capillaries (17). If endurance-type stimulation persists over a longer period of time, the contractile proteins will eventually shift to a greater predominance of slower contracting filaments. These shifts in muscle fiber type have been reported in response to either chronic electrical stimulation of the muscle (26) or even daily endurance training (21). Thus dramatic shifts in muscle structure and function occur acutely, in response to the intensity of the demand, and chronically, in response to the nature of the demand.

Skeletal muscle also shows body size-dependent differences; there are a suite of characteristics that are apparent only if examined across a broad range of body sizes (19, 24). These comparative features of skeletal muscle are apparently linked to, and hence may be dependent fucntions of, body size. Although skeletal muscle tissue from all mammals is seemingly identical in both structure and mechanism of contraction, this seemingly identical tissue must perform in a number of fundamentally different ways in shrews than in elephants. These size-imposed differences in muscle function may have significant consequences in the energetic cost of muscle contraction and therefore locomotion.

The relation between body size and the energetics of locomotion has been the subject of intense investigation, especially by Taylor and his co-workers. For instance, in running mammals the external (weight-specific) work performed does not vary systematically with body size (13, 31); however, the energy required to perform that work does (14); small animals expend much more energy for a given force production than large animals. Additionally, irrespective of body size, as the work output increases (or more correctly, as the mass supported by the muscle increases), proportionally more energy is used by the animal. Hence, the increment increase (i.e., the slope of the relation between energy input and work output) is highly body size dependent (31). At all loads and speeds, small animals require much more energy than large animals for the same force production. As a consequence, the energetic cost of locomotion (energy·mass^{-1}·distance^{-1}), and hence efficiency of locomotion, is highly body size dependent in mammals, birds, and lizards (10). There seems to be an unavoidable energetic penalty incurred with small body size, the physiological source of which is uncertain.

Might this pattern be explained by the obligate scaling of muscle contraction velocity? The intrinsic rate of muscle shortening has been speculated to increase with decreasing body size in mammals (16). Indeed, the in vitro rate of muscle shortening in both extensor digitorum longus and soleus scales inversely to body mass in mammals from mice to dogs (data from Ref. 28). In spite

of its rate of tension development, all skeletal muscle can generate nearly the same force per unit cross-sectional area (16, 22). Thus the rate of tension development has been implicated in setting the energy requirement of contracting muscle. Experimental verification is difficult, but all available data suggest that the energy required to produce and maintain muscular force increases with increasing shortening velocity (11, 27). It has thus been suggested that the rate of cross-bridge cycling between actin and the myosin heads may ultimately set the cost of muscular contraction in direct proportion to the intrinsic rate of muscle shortening (14, 20, 31). In this report we attempt to test this hypothesis by estimating the rate of muscle shortening during locomotion in quadrupedal terrestrial mammals.

ANALYSIS AND RESULTS

We have estimated the rate of muscle shortening of the extensors of the knee (the quadriceps group) by combining a number of physiological and anatomic measurements. To make comparisons, all physiological measurements (and therefore our final estimates) were made for the physiologically equivalent speed at the trot-gallop transition (see Ref. 15). Calder (8) has observed that this speed is not physiologically equivalent in small and large species; rather it represents a mechanically equivalent speed.

Because all the needed morphological and physiological data are unavailable for the same individual animals, we have formed a series of allometric equations. Using these equations we describe a large number of variables (Y) as a function of body mass (M) in the familiar power-law equation

$$Y = a\mathrm{M}^b \qquad (1)$$

To estimate the intrinsic rate of muscle shortening it is necessary to measure (or determine) total muscle length, the length it shortens per stride, and the time over which the length change occurs. It is the purpose of this paper to calculate these in the mammalian hindlimb (specifically for the knee extensors) at the trot-gallop transition.

Geometry of knee. The length that the knee extensors must change is a function of the angle of the knee. Hence, we first had to determine some geometric features of the knee, specifically the dimensions of the condyles of the femur (see Fig. 1). To determine these features we measured the femurs of 30 species of mammals. Even though not all species were explicitly cursorial, we did exclude animals that would be regarded primarily as climbers or diggers. Measurements were made on adult specimens only (epiphyses were fused). Whenever possible, species' means were calculated and plotted as a function of mean adult body mass, which was taken from other sources. For those mammals with weight dimorphism, separate means were calculated for males and females. In mammals spanning over five orders of magnitude in body mass, from shrews to bison, the condyle diameter (d_c = $s + d$ in Fig. 1, where s is the radius of the circular portion of the femur condyles and d is equal to s plus the distance due to the eccentricity of the patellar surface of the condyle) scales in a regular and very predictable relation to body mass. As might be expected, d_c scales with the same exponent as other bone diameters; we find d_c (mm) is roughly three times larger than the least diameter of the tibia shaft[1] (Fig. 2)

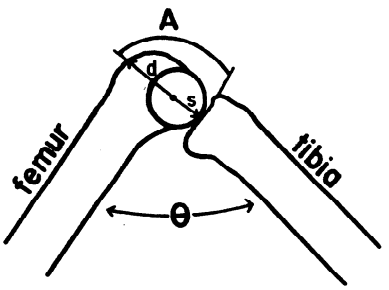

FIG. 1. Diagrammatic representation of mammalian knee. Angle of knee joint (θ) determines length of the arc (A) across knee and therefore change in length of knee extensors. Condyle of the femur has diameter equal to $d + s$.

$$d_c = 10.3\mathrm{M}^{0.38} \qquad n = 36;\ r^2 = 0.96 \qquad (2)$$

We did not measure d and s separately in all femurs; we did measure them in the smallest and largest bones and a few between. We consistently found that $d = 2s$ regardless of body size. Consequently, s is very nearly equal to the least diameter of the tibia. Referring to Fig. 1, we can estimate the length of arcuate segment A as the fraction of a circumference $2\pi \cdot$ radius $\cdot$ fraction of a full circle or

$$A = 2\pi\left(d + \frac{s}{\sin\theta}\right)\left(\frac{1}{2} - \frac{\theta}{360}\right) \qquad (3)$$

which assumes that the rotation of the knee occurs about a point of contact between the femur and the tibia. To make the dependence on the angle of flexion (θ) more apparent, we can reduce *Eq. 3* to

$$A(\theta) = 2\pi s\left(\frac{d}{s} + \frac{1}{\sin\theta}\right)\left(\frac{1}{2} - \frac{\theta}{360}\right) \qquad (4)$$

If we assume that $\theta = 170°$ when the knee is fully extended in all mammals (this has been measured in dogs, see Ref. 12), then when extended (A_e)

$$A_e = 2\pi s\left(\frac{d}{s} + \frac{1}{\sin 170}\right)\left(\frac{1}{2} - \frac{170}{360}\right) = 2\pi s(0.216) \qquad (5)$$

Substituting *Eq. 2* into *Eq. 5*, we can solve for A_e (in mm) as a function of body mass

$$A_e = 6.8\pi\mathrm{M}^{0.38} \cdot (0.216) \qquad (6)$$

Similarly, when the knee is flexed, A_f can also be solved as a function of knee angle (θ)

$$A_f(\theta) = 6.8\pi\mathrm{M}^{0.38}\left(2 + \frac{1}{\sin\theta}\right)\left(\frac{1}{2} - \frac{\theta}{360}\right) \qquad (7)$$

[1] We measured tibia diameter as its least geometric mean diameter (d_t, in mm) in 18 species of mammals (see *Eq. 12*). $d_t = 4.0\ \mathrm{M}^{0.38}$; $n = 18$; $r^2 = 0.99$.

Muscle shortening during locomotion. During locomotion, at the trot-gallop transition, the angles of limb excursion scale to body mass. According to the model of elastic similarity, those angles should scale as length divided by diameter or in proportion to $M^{-1/8}$. In fact, McMahon (25) measured the excursion angle of the entire hindlimb and found it varied as $M^{-0.1}$, and Biewener (6) found a similar same exponential scaling of the flexed knee angle also at the trot-gallop transition. These observations agree with predictions for proximal leg joints proposed independently by Alexander et al. (3). Thus *Eq. 6* can be further modified to include body size dependence of $\Delta\theta$.

Twice during each stride the knee is flexed and extended. When the foot is off the ground the knee is maximally flexed and must be extended prior to landing. Likewise, during the support phase the knee is flexed when the foot is beneath the pelvis (this prevents large shifts in gravitational potential energy, since the center of mass would have to be raised if the knee were not flexed then) and is maximally extended just as the foot leaves the ground (12). During that portion of the support phase when the knee is extended, the knee extensors provide thrust as they actively shorten, performing work. We have estimated the rate of muscle shortening during this portion of the support phase, though it must be proportional when the foot is off the ground, since each event is a constant fraction of a single-stride duration. Hence, the rate of muscle shortening can be estimated by calculating the time over which the knee angle changes and solving for the change in muscle length.

At the trot-gallop transition, the duration of a single stride (in s) is body size dependent and equal to $0.223M^{0.14}$ (15). However, the duty factor (the fractional duration of the support phase) is independent of body mass and therefore a constant fraction of the stride. In a variety of mammals, spanning a size range from 20 g to 3,500 kg, Biewener (6) found that the duty factor of the hindlimb at the trot-gallop transition is equal to 0.42 with relatively little variance. Again, as the knee is extended during the last half of the support phase the time of extension (t_e, in s) must scale as: $t_e = 0.21 \cdot 0.223M^{0.14} = 0.047M^{0.14}$.

The change of length of the knee extensors per stride (Δl, in mm) and their shortening velocity may now be calculated from *Eqs. 6* and *7*

$$\Delta l = A_f - A_e \tag{8}$$

$$= 6.8\pi M^{0.38}\{[2 + l/\sin(170 - 60M^{-0.1})] [0.5 - (170 - 60M^{-0.1})/360] - 0.216\} \tag{9}$$

Equation 9 can be simplified by solving and regressing calculated values

$$\Delta l \simeq 8.43\ M^{0.26} \tag{10}$$

Therefore the muscle-shortening velocity ($\Delta l \cdot t_e^{-1}$) must be

$$\Delta l \cdot t_e^{-1} = 8.43M^{0.26}/0.047M^{0.14} = 179M^{0.12} \tag{11}$$

Finally, we can estimate the rate of shortening by dividing muscle-shortening velocity by muscle length.

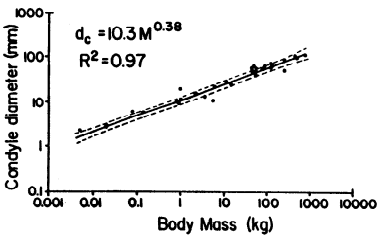

FIG. 2. Condyle diameter of femur (d_c) is shown as function of body mass in 30 species of mammals from shrew to bison. *Dashed lines,* 95% confidence interval.

For this purpose we have made the assumption that muscle length scales in direct proportion to femur length. There have been several published values expressing femur length as a function of body mass in mammals. Among several orders of mammals and across a broad range of body sizes, femur length (l_f) scales as

$$l_f = 63M^{0.36} \qquad n = 39\ (2) \tag{12}$$

$$l_f = 60M^{0.31} \qquad n = 32;\ r^2 = 0.96\ (6) \tag{13}$$

Our data, excluding climbers and diggers (extending from 5 g to 800 kg), fall between these values (Fig. 2)

$$l_f = 61M^{0.345} \qquad n = 18\ \text{(species)};\ r^2 = 0.97 \tag{14}$$

By dividing *Eq. 10* by femur length the estimated rate of muscle shortening ($\Delta l \cdot t_e^{-1} \cdot l_f^{-1}$) will thus vary between $M^{0.19}$ and $M^{0.24}$. Using our measurements (*Eq. 14*), we calculate the rate of muscle shortening (in s^{-1}) as

$$\Delta l \cdot t_e^{-1} \cdot l_f^{-1} = 179M^{0.12}/61M^{0.35} = 2.93M^{-0.23} \tag{15}$$

Hence, at the trot-gallop transition the rate of shortening of the knee extensors is body size dependent, being roughly 25 times faster in a shrew than an elephant.[2]

DISCUSSION

Is it possible to relate the rate of muscle shortening to the shortening rates of individual muscle fibers or even sarcomeres? To do so, we must make two assumptions. First, there is no body size dependence in the viscoelastic elements of the muscle-tendon system. Hence, any deviation between the intrinsic and extrinsic rates of shortening should not be body size dependent. This should be tested experimentally. Second, fibers of pinnate muscles will shorten more rapidly than the muscle itself. This difference is minimized when the angle of pinnation is small, as in the knee extensors (4). Hence, we suggest that there is a constant relationship between the estimated rates of muscle shortening and the intrinsic rate

[2] We have also made an estimate of rate of shortening of the hamstrings or knee flexors. To make this estimate we measured the in-lever length (l_i, in mm), i.e., the length of the tibia from its proximal end to the mean tendon attachment point, and found: $l_i = 3.2\ M^{0.40}$; $n = 36$; $r^2 = 0.96$. The change in muscle length (Δl) was then solved, once more as a function of knee angle. $\Delta l(\theta) = (l_i + s)(\cos\theta_f - \cos\theta_e) \propto M^{0.24}$. Again dividing by femur length, shortening rate should scale as $M^{-0.21}$. In addition, although it is only an estimate, it is unlikely that the final exponent could vary much from this value. We suspect that during locomotion the shortening rate (or time course of isotonic force production) of all locomotory muscles scales near $M^{-1/5}$ to $M^{-1/4}$, parallel to weight-specific maximum O_2 consumption.

of cross-bridge cycling in mammalian muscle.

Muscle shortening and energy demand. Skeletal muscle mitochondria must resynthesize ATP at a rate that supplies the muscles' peak aerobic demands. These demands are not static but vary with training, body size, and inactivity, for example. The structural plasticity of skeletal muscle permits changes in the mitochondrial density (mitochondrial volume per volume muscle fiber), ensuring a dynamic match between ATP supply and demand (18). Among African ungulates, the mean volume of skeletal muscle mitochondria is strongly body size dependent, scaling close to $M^{-0.20}$ (24).

The rate of ATP synthesis must be tuned to its demand by the contracting myofilaments. The enzymes that regulate rates of ATP cleavage are the calcium-activated ATPases both in the sarcoplasmic reticulum and on the myosin heads. About 70% of the ATP is utilized by the myosin ATPase for cross-bridge cycling and the remaining 30% by the sarcoplasmic reticulum for Ca^{2+} sequestering (27). We feel this ratio must be independent of contraction velocity (since contraction and relaxation times must be coupled). Myosin ATPase activity has been quantified in at least two interspecific studies. Delcayre and Swynghedaw (9) compared the heart myosin ATPase from several different species. Additionally, Syrovy and Gutmann (28) presented data from several species comparing myosin ATPase from both fast (extensor digitorum longus) and slow (soleus) muscles. We used the above data to express myosin ATPase activities as a function of body size and found it to scale between $M^{-0.23}$ and $M^{-0.31}$ (Fig. 3). These data support the suggestion of Barany (5) that myosin ATPase activity is a nearly constant function of the intrinsic speed of muscle shortening; i.e., the activity of myosin ATPase, although it varies greatly among species and muscles of the same species, scales parallel to the calculated rate of muscle shortening.

The rate of cross-bridge cycling between actin and myosin has been implicated in setting the energy demands of contracting skeletal muscle in mammals; the results of this study support this hypothesis. There is a parallel, and one is tempted to speculate causal, relationship among O_2 consumption, the activity of myosin ATPase, the volume density of ATP-producing mitochondria, and the calculated rate of muscle shortening (Fig. 4).

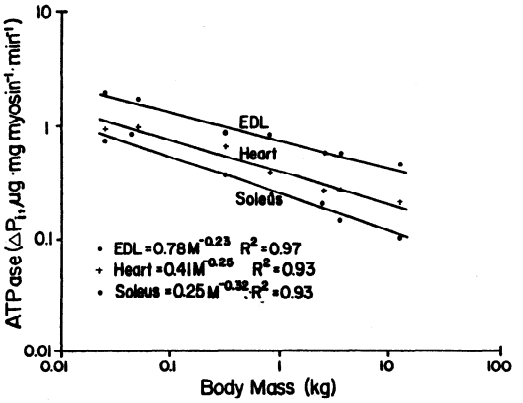

FIG. 3. Calcium-activated myosin ATPase activity is shown as function of body mass in 3 different muscles. Body masses have been estimated for mice, hamsters, rats, guinea pigs, rabbits, cats, and dogs (data from Ref. 28). EDL, extensor digitorum longus.

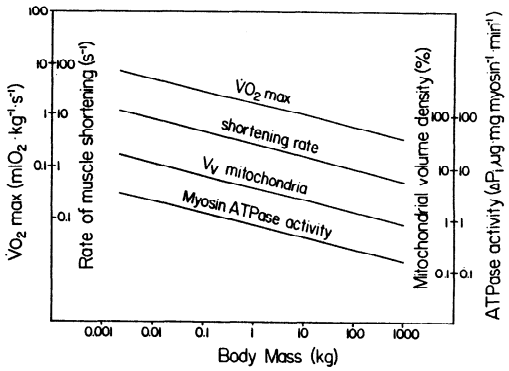

FIG. 4. Within terrestrial mammals there is constant relationship among O_2 consumption ($\dot{V}O_2$), volume density of mitochondria (Vv), myosin ATPase activity, and rate of muscle shortening (of knee extensors). Thus, although all of these measured parameters vary greatly as a function of body mass, within any given mammal their ratios remain nearly constant.

Body size and stride frequency (limb dimensions). Taylor (29) has recently analyzed stride frequency in mammals as a function of body mass. His conclusion is that a running mammal is a tuned spring system with an optimal (and therefore most efficient) stride frequency (somewhat analogous to resonating frequency). The important observation is that this frequency is solely a function of body mass; hence it is suggested to be direct physical constraint that body mass has on locomotory physiology. A second such physical constraint is the size scaling of bones. Because the strength (buckling or compressive) of support elements is directly proportional to their cross-sectional area, bones must be built relatively more robustly as body mass increases. Hence, diameters of limb bones seem to scale more predictably to body mass than to their lengths; all diameters scale near $M^{0.37}$ (2, 6). Finally, Alexander et al. (3) have likewise presented physical arguments compelling joint angles to scale near the observed $M^{0.1}$. Thus there are physical arguments that may directly set stride frequency, joint angle, and joint dimensions to body size. With these predetermined, the rate of muscle shortening and therefore the energy demand of the working muscle may indeed be inseparably linked to body mass. Consequently, in a running mammal body mass alone may explain the disparate scaling of the rate, and therefore the cost, of force production.

Body size and efficiency of locomotion. The whole-animal weight-specific cost of locomotion also decreases as body size increases. Taylor et al. (32) first reported a linear relationship between the metabolic cost of locomotion (O_2 consumption per unit mass) and running velocity in mammals. Because the relation is linear, a single number (the slope of the line) may be used to describe the species-specific cost of locomotion (energy·mass^{-1}·distance^{-1}). The relation between cost and body size has subsequently been examined in a wide variety of mammals and birds, and both classes fall on the same line. The cost of locomotion in birds and mammals (the slope, in ml $O_2 \cdot m^{-1} \cdot kg^{-1}$) scales as (30)

$$\text{cost} = 0.533M^{-0.32} \qquad n = 62;\ r^2 = 0.84 \qquad (16)$$

Although this is similar to the exponent we reported for the intrinsic rate of muscle shortening ($M^{-0.23}$, *Eq. 15*), the standard error of the cost (slope) does not include −0.23. Thus large animals seem to be gaining an added energetic advantage beyond that explained by the difference in cross-bridge cycling rates.

Elastic storage of energy during locomotion may explain the added advantage of large body size. The amount of energy that could be stored (on impact with the ground) and recovered (providing additional thrust) increases greatly as body mass increases (1, 7). Hence we propose that the difference between the apparent cost to the muscles ($M^{-0.23}$) and the overall cost to the animal ($M^{-0.32}$) could be attributed to the size-dependent scaling of the elastic potential energy storage and recovery. Thus, as body size increases, there are two apparent energetic advantages. First, the decreased rate of muscle shortening lessens the energy requirement due to slower cross-bridge cycling. Second, with greater body mass, additional potential energy can be stored and recovered with each stride.

The results of this study support earlier suggestions that muscle-shortening rates may set the energy demand of contracting muscle. In the mammalian hindlimb the shortening rate of the knee extensors must scale near $M^{-0.23}$. This exponent is similar to those describing other biological rates (23), providing further evidence for a constant linking of biological rates to body mass in mammals.

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