Length dependence of active force production in skeletal muscle

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Rassier, D. E., B. R. MacIntosh, and W. Herzog. Length dependence of active force production in skeletal muscle. J. Appl. Physiol. 86(5): 1445–1457, 1999.—The sliding filament and cross-bridge theories of muscle contraction provide discrete predictions of the tetanic force-length relationship of skeletal muscle that have been tested experimentally. The active force generated by a maximally activated single fiber (with sarcomere length control) is maximal when the filament overlap is optimized and is proportionally decreased when overlap is diminished. The force-length relationship is a static property of skeletal muscle and, therefore, it does not predict the consequences of dynamic contractions. Changes in sarcomere length during muscle contraction result in modulation of the active force that is not necessarily predicted by the cross-bridge theory. The results of in vivo studies of the force-length relationship suggest that muscles that operate on the ascending limb of the force-length relationship typically function in stretch-shortening cycle contractions, and muscles that operate on the descending limb typically function in shorten-stretch cycle contractions. The joint moments produced by a muscle depend on the moment arm and the sarcomere length of the muscle. Moment arm magnitude also affects the excursion (length change) of a muscle for a given change in joint angle, and the number of sarcomeres arranged in series within a muscle fiber determines the sarcomere length change associated with a given excursion.

force-length relationship; sarcomere length; isometric contraction; sliding filament theory; cross-bridge theory; moment-angle relationship; force-calcium relationship

The fact that passive force, peak single-twitch force, and peak tetanic isometric force depend on muscle length has been well described over 100 years ago by Blix in a series of three scientific papers (6–8). Blix performed experiments on frog striated muscles and found for the adductors of the thigh and the gastrocnemius that isometric force increased with increasing lengths, reached a plateau, and then decreased. Based on these correct observations and the available structural information, Blix (8) proposed a mechanism of force production and shortening that was based on the shortening of longitudinally arranged “protein filaments” (Protoplasmastäbchen or Inotagmen).

The length dependence of skeletal muscle force production may be conveniently divided into two broad categories: maximal and submaximal contractions. For maximal contractions, the length dependence of force is typically referred to as the force-length relationship. Force-length relationships are obtained during isometric contractions; i.e., either the total muscle length, the fiber length, or the sarcomere length (depending on the preparation) is kept constant during the experiment. With few exceptions (28), the force values in force-length experiments are the maximal steady-state forces that are reached during isometric contractions. Submaximal contractions are all contractions for which maximal activation is not achieved. For submaximal contractions, we will not use the term “force-length relationship” but will talk about the length dependence of force production.

This review is divided into four parts. In the first part, the theoretical background for the force-length relationship is given based on the cross-bridge theory. In the second and third parts, the force-length relationship and the length dependence of force production are discussed for single-fiber and for entire muscle preparations, respectively. In the fourth part, some functional
implications of the force-length property for in vivo force and moment production are described. Throughout the review, length dependence of force production refers to active force production, except when specifically indicated. Active force is defined as the rise in force observed on activation of a muscle and is associated with cross-bridge interactions between myosin and actin. This restriction to active force production should not be interpreted as a value judgment on the importance of active vs. passive muscular forces. It merely reflects the interest of the authors in active rather than passive muscle properties.

**PART 1: THEORETICAL BACKGROUND OF THE FORCE-LENGTH RELATIONSHIP**

The force-length relationship of sarcomeres may be explained, to a large degree, by the sliding filament (41, 44) and the cross-bridge theories (40, 43). The sliding filament theory assumes that length changes in sarcomeres, fibers, and muscles are accomplished by relative sliding of the essentially inextensible myofilaments, actin and myosin, within a sarcomere (Fig. 1). [Recent evidence suggests that actin and, possibly, the myosin filaments are not as inextensible as originally assumed (27, 37, 46, 52, 84); however, myofilament compliance may safely be ignored in the following considerations]. The cross-bridge theory suggests that the relative sliding of actin and myosin is caused by independent force generators; i.e., the cross bridges. Of particular importance for the derivation of the sarcomere force-length relationship are the following assumptions underlying the cross-bridge theory.

1) The cross bridges are uniformly distributed along the length of the myosin filament.
2) The actin filament has uniformly distributed attachment sites for the cross bridges.
3) The attachment/detachment probability of a cross bridge only depends on its location relative to an actin attachment site and is independent of its prior history or its neighboring cross bridges (40).
4) Each cross bridge exerts, on average, the same amount of force as any other cross bridge.

Cross bridges are small and have a limited attachment range. Therefore, cross-bridge attachments only occur in the actin-myosin overlap zone of a sarcomere (Fig. 1). Together with the assumptions underlying the cross-bridge theory, this limitation implies that the maximal isometric force of a sarcomere is linearly related to the amount of actin-myosin overlap (Fig. 2).

In frog striated muscle, actin and myosin lengths are ~0.95 and 1.6 µm, respectively; the Z-line width (Figs. 1 and 2) is ~0.1 µm, and the central part of the myosin filament (~0.2 µm) does not contain cross bridges (45, 85). With these geometrical assumptions, it is obvious that maximal overlap between actin and myosin occurs at a sarcomere length of ~2.0 µm [i.e., twice the thin filament length (1.9 µm) plus the Z-line width (0.1 µm)]; therefore, sarcomere force should be maximal at a sarcomere length of 2.0 µm. A reduction in actin-myosin overlap caused by extension of the sarcomere by 0.2 µm does not decrease the force potential, because the midzone of the myosin filament does not contain cross bridges. Extending the sarcomere beyond 2.2 µm is associated with a linear decrease in the number of potential actin-myosin interactions, and active force production should become zero at a sarcomere length of ~3.6 µm (Fig. 2) [twice the thin filament length (1.9 µm) + the thick filament length (1.6 µm) + the width of the Z line (0.1 µm)], where no overlap of the myofilaments occurs.

The cross-bridge theory predicts well the results of the experimental sarcomere force-length relationship for the plateau region (2.0–2.2 µm) and the descending
limb (2.2–3.6 µm). On the ascending limb of the force-length relationship (i.e., sarcomere lengths <2.0 µm), active isometric sarcomere force decreases with decreasing length (Fig. 2). There are two conceptual possibilities why force decreases in this region of the force-length relationship: 1) active force production may be reduced and/or 2) internal forces may oppose the active forces. Active force production may be reduced because of an increasing lateral distance between the actin and myosin filaments at short length effecting reduced probability of cross-bridge interaction (26). Interference of cross-bridge interaction may occur with double overlap of the thin filaments (28). Also, below sarcomere length of 1.7 µm, proper cross-bridge attachments may be limited by deformation of the myosin filament.

Internal forces that may oppose active forces include forces associated with deforming actin and myosin filaments. Particularly myosin deformation, which occurs when the Z lines of the sarcomere push against the ends of the myosin filament at a sarcomere length of ~1.7 µm, appears to cause a decrease in force (28) (Fig. 2). Furthermore, when frog sarcomeres are shortened below optimal length and are not constrained, they tend to extend to ~2.1 µm (72). The magnitude of this restoring force is not known, but it presumably would subtract from the active force. It has been suggested that the restoring force is minimal in the sarcomere length range from 1.65 to 2.0 µm, since maximal velocity of fiber shortening is not affected (48). Last, fluid and osmotic pressures may be elevated in shortened muscle fibers and may contribute to oppose the active sarcomere forces, as suggested by Sato (77) and Gordon et al. (28).

The above theoretical relationship between sarcomere length and force was derived for myofilament lengths found in frog striated muscles. Actin filament lengths vary appreciably across species (33, 85), and this variation should influence the shape of the sarcomere force-length relationship. For example, the theoretical plateau region and descending limb of cat and human skeletal muscles are shifted to the right of that for frog (Fig. 3). The shift corresponds to twice the difference in actin filament lengths between the species. Human and frog actin filament lengths are ~1.27 and 0.95 µm, respectively; therefore, the shift in the theoretical plateau and descending limb region for human and frog muscles is ~2 × (1.27 – 0.95) = 0.64 µm. The ascending limb region for human and cat sarcomeres has not been determined and cannot be derived without further assumptions. Sarcomere force-length relationships of mammalian skeletal muscle are rare; however, an excellent relationship was derived experimentally by ter Keurs et al. (83) for the rat extensor digitorum muscle.

The force-length property of muscle is a static property; static in the sense that one experimental trial gives one data point. This basic fact is often neglected in theoretical models of muscle, presumably because the force-length relationship is typically represented with continuous lines (e.g., Fig. 2) rather than by isolated data points (Fig. 4) (Fig. 2; Ref. 28), and mathematical descriptions of the force-length relationship are given as continuous functions (89), implying that the relationship is continuous in time and length. However, this implication is not correct.

Considering the force-length relationship as a continuous, dynamic property has led to the notion of instability in sarcomere length on the descending limb of the force-length curve. The negative slope of the descending limb represents the behavior of a softening material, an unstable behavior. However, the notion of instability may be incorrect, because a fiber that shortens or elongates shows a positive stiffness. For example, Edman et al. (14) showed that a fiber that is stretched on the descending limb of the force-length relationship will become stronger as it is stretched, rather than weaker, as one should expect based on the force-length relationship (Fig. 5). However, and even more importantly, Edman et al. (14, 15) showed that the force enhancement observed during the stretch did not disappear, but some of the force enhancement was maintained for a long time (4.2 s). This additional force following the stretch did not only cause a force enhancement relative to the isometric force at the stretched length but also produced an isometric, steady-state force following the stretch that was larger than the isometric force preceding the stretch. This latter result, if transferable to the sarcomere level, would ensure unconditional stability of sarcomere length on the

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**Fig. 3.** Sarcomere force-length relationship in frog, cat, and human skeletal muscles.

**Fig. 4.** Actual data points of a sarcomere force-length relationship obtained around the plateau region of frog skeletal muscle. Error bars indicate ±1 SE. (Adapted from Gordon et al. [28].)
**PART 2: SINGLE-FIBER EXPERIMENTS**

To test predictions of the cross-bridge theory associated with the force-length relationship, strict criteria must be adhered to: the sarcomere length must be isometric, and activation of the muscle must be maximal. When muscle contracts under conditions that do not comply with these conditions, muscle length still has an effect on the magnitude of the contractile response, but the predictions of the cross-bridge theory will not necessarily be confirmed. In this section, the force-length properties of single skeletal muscle fibers are described, and the length dependence of the contractile response is considered when these strict criteria (isometric sarcomeres and maximal activation) are not present.

**Fig. 5.** Actual data of force enhancement and positive sarcomere stiffness on descending limb of the force-length relationship for frog skeletal muscle. Relationship between force (stress) and sarcomere length; the black and the corresponding solid lines represent isometric force-length relationship for single fibers from semitendinosus of *Rana temporaria*. A: ■ and the corresponding line represent steady-state force at 4.3 s after completion of 0.2 µm/sarcomere stretches at constant speed. Dashed lines connecting the isometric force-length curve with steady-state force values following stretch show the origin and the end of stretch. Note that dashed lines do not show the actual force-length trace followed during stretch. B: stretches of varying lengths (different symbols), beginning at 2 different initial lengths are illustrated. Temperature: 1.49°C, cross-sectional area of fiber: 10.312 × 10⁻³ mm². [Adapted from Edman et al. (14, 15)].

Force-length relationship. To ensure isometric conditions of sarcomeres while the muscle fiber force-length properties are studied, feedback control of sarcomere length is necessary. This can be approximated by control of a segment of the fiber length. Gordon et al. (28) used segment length control in their classic study of the force-length properties of isolated fibers of frog skeletal muscle. These authors used a spot follower device and servo control to maintain a constant distance between two markers on a short segment of the muscle fiber. Even with segment length control, there is some uncertainty that sarcomere length remains constant throughout the tetanic contraction. Redistribution of sarcomere length was thought to occur after the rapid rise of tension, and this redistribution of sarcomere length was coincident with a slow rise in tension referred to as tension creep.

Gordon et al. (28) were the first to describe the force-length properties of skeletal muscle sarcomeres. They reported that peak active tension during the rapid rise of tension was constant across a sarcomere length range from 2.0 to 2.2 µm. At progressively longer sarcomere lengths, active tension decreased linearly, reaching zero at 3.6 µm (Fig. 2). This segment of the force-length relationship is referred to as the descending limb. When sarcomere length was <2.0 µm, developed tension was less than the peak value, decreasing linearly to a sarcomere length of ~1.7 µm and decreasing more steeply at even shorter sarcomere lengths (ascending limb). These observations are consistent with predictions based on the cross-bridge and sliding filament theories of muscle contraction.

Edman and Reggiani (17) observed a force-length relationship that was slightly different from that reported by Gordon et al. (28). They observed a sigmoidal decline in developed tension on the descending limb of the force-length relationship and suggested that this sigmoidal rather than linear decrease in force was consistent with small sarcomere length nonuniformities within the length-controlled segment. They argued that such a distribution of sarcomere lengths might diminish the magnitude (width) of the plateau region of the force-length relationship and may permit some force development in the fiber at a mean sarcomere length that is >3.6 µm.

Although there are minor discrepancies in the results of studies that have used a segment length-control approach, the general findings are the same and they support the cross-bridge theory of muscle contraction (16, 17, 29, 31, 47).

Fixed-end muscle contractions. In circumstances where sarcomere or segment length-control is not used, the term “fixed-end contraction” is appropriate. In this type of experiment, “isometric” refers to the muscle (fiber) tendon unit, since only the distance between the fixed ends remains constant. In addition to changes in sarcomere length during the slow change in developed tension (creep), there can be rapid adjustments of sarcomere length due to stretching of compliant structures in series with the fiber. Under these circumstances, there is still a length dependence of developed
tension. Some investigators have reported a shape of the apparent force-length relationship quite similar to the force-length relationship described above (48, 82). However, others have reported differences in the shape of the length-dependence of force in fixed-end contractions, compared with the shape found with length-controlled contractions. The plateau of the relationship is more curved and extends to longer sarcomere lengths in fixed-end experiments than in length-controlled experiments. This extended plateau results in greater forces at any given average sarcomere length on the descending limb than those obtained by using length-controlled experiments (31, 82), giving a length dependence of active force that is situated to the right, relative to the length-controlled force-length relationship (Fig. 6). The difference in the descending limb between theoretical and experimental studies has been a focus of criticism of the sliding filament theory by Pollack (68–70) and ter Keurs et al. (82). However, it is recognized that nonuniform sarcomere lengths may account for this difference and it is not appropriate to discard the cross-bridge theory based on the results of experiments with fixed-end contractions.

Hill (38) was the first to propose that, when a muscle is stimulated to contract at a long length, redistribution of segment lengths occurs, and he suggested that this redistribution was responsible for the slow phase of rise in force tension (creep) during a tetanic contraction. This idea was advanced by Gordon et al. (28, 29), who proposed more specifically that tension creep was caused by a redistribution of sarcomere lengths during a tetanus, and that this redistribution would cause an increase in sarcomere length nonuniformity. In a series of experiments, Edman and Reggiani (16, 17) provided evidence that sarcomere length is not generally uniform along the length of an isolated muscle fiber, neither at rest nor during activation. At rest, in highly stretched fibers, sarcomere lengths are shorter near the ends compared with the middle of fibers (9, 42, 49). During contraction at long lengths, sarcomeres situated near the ends of the fibers were found to shorten, whereas sarcomeres in central parts of the fiber were found to be stretched (16, 17). The important question is, How can nonuniformity of sarcomere length permit greater force development on the descending limb?

Assuming that each sarcomere follows a force-length relationship according to the cross-bridge theory and that sarcomere lengths are nonuniform, then long sarcomeres will be weaker than short sarcomeres on the descending limb of the force-length relationship. Therefore, on activation, long (weak) sarcomeres should shorten, and short sarcomeres should shorten. Because the ends of the fiber are fixed, the average velocity of sarcomere length change must be zero. Considering the simplest case (2 sarcomeres in series), the velocity of lengthening of one sarcomere will be equal to the velocity of shortening of the other one, and the force transmitted across the sarcomeres will be equal. Considering that the slope of the eccentric side of the force-velocity relationship is steeper than that of the concentric side means that the force transmitted across these two sarcomeres in series will be much closer to the isometric force of the shorter (stronger, shortening) sarcomere, than to the force for the "average" isometric sarcomere length (63, 64). Therefore, the force at any average sarcomere length beyond the sarcomere length of optimal overlap of the myofilaments will be greater for fixed-end contractions than the force predicted by cross-bridge theory, for that (average) sarcomere length.

This explanation of the enhanced force on the descending limb of the length-tension relationship of skeletal muscle requires that individual sarcomeres will continue to change length, resulting in increased inhomogeneity (16) and increased isometric tension during a tetanic contraction. This increasing tension is thought to be the tension creep that has commonly been observed. It has been predicted that when individual sarcomeres shorten to a length less than the optimum (to the ascending limb of the force-length relationship), they will eventually reach a length where the isometric force of the sarcomere is equal to the force transmitted across the sarcomeres, and shortening will stop (64).

Another potential explanation for the enhanced isometric force on the descending limb exists. It is known that stretch of a muscle fiber results in a permanent enhancement of active force above that predicted for an isometric contraction at the new length (14, 15) and that shortening has the opposite effect (13). This effect of stretch has been referred to as stretch-induced force enhancement (14, 15). Although these force modulations may also be attributed to inhomogeneity of sarcomere lengths (18, 47, 62), these modulations could be a property of individual sarcomeres.

Submaximal contractions. The shape of the force-length relationship and the length-dependence of force development in fixed-end contractions, as described above, was determined in single muscle fibers that
were maximally activated (tetanic contractions). When
the stimulation is at a subfusion frequency, activation
is submaximal, and the shape of the length-dependence
of force development is not the same as for maximal
contractions (3, 71). In particular, during submaximal
levels of stimulation, peak active tension occurs at a
length longer than the length associated with maximal
overlap of the myofilaments (79).

During submaximal contractions, greater force is
obtained when the number of available cross bridges is
reduced by an increase in muscle length. This property
of muscle appears to be associated with enhanced
activation at long muscle lengths and has been referred
to as length-dependent activation (16, 53, 78). The term
“activation” has been used in many different ways in
the skeletal muscle literature, for example, to indicate
the relative frequency of stimulation or frequency of
membrane action potentials of a muscle or motor nerve
(5); to reflect the intracellular levels of free Ca²⁺ (4); or
to reflect the proportion of troponin C with Ca²⁺ bound
(2), among others. In general, this term is used to
indicate a change of state from inactive to active and is
often coupled with an implication of relative amplitude
(“level of activation”). For this reason, the term “muscle
activation” is defined here as the proportion of troponin
C with Ca²⁺ bound.

The isometric developed tension in a submaximally
activated, single skinned skeletal muscle fiber is deter-
mined by the following: average overlap of the myofil-
aments, free Ca²⁺ concentration, and the force-Ca²⁺
relationship (Fig. 7). Factors affecting Ca²⁺-binding
kinetics alter the proportion of troponin C that is
occupied by Ca²⁺ at a given free Ca²⁺ concentration. In
an intact cell, the frequency and magnitude of Ca²⁺
oscillations (10) will affect the extent of Ca²⁺ binding to
troponin C. Therefore, the level of activation depends
on the time history of Ca²⁺ transients. However, the
kinetics of Ca²⁺ binding to troponin C and of cross-
bridge interaction have a damping effect on the relation-
ship between intracellular Ca²⁺ concentration and ac-
tive force, since oscillations in force at a given frequency
of stimulation are smaller than oscillations in free Ca²⁺
concentration. The force developed at a given average
free Ca²⁺ concentration and a given magnitude of
myofilament overlap depends on the proportion of Ca²⁺
binding sites that are occupied and on the kinetics of
cross-bridge interaction. The kinetics of cross-bridge
interaction can be considered, in the simplest case, to
be governed by two rate constants (f and g; the rates of
attachment and detachment of cross bridges, respecti-
vely). For the case in which there is a large constant
number of identical attachment possibilities [i.e., cross
bridges with identical x-values (40) at each instant in
time], the proportion of attached cross bridges (n) will
be a function of time exclusively; i.e.

\[
\frac{dn}{dt} = (1 - n)f(x) - ng(x)
\]

For the specific state of dynamic equilibrium, when
\(dn/dt = 0\), the value for the proportion of attached cross
bridges, \(n_{eq}\), becomes

\[
\frac{n_{eq}}{f(x)} = \frac{f(x)}{f(x) + g(x)}
\]

Attempts to measure Ca²⁺ binding to troponin during
skeletal muscle activation have failed to demonstrate a
length dependence of Ca²⁺ affinity of troponin (25, 67,
86, 87). Furthermore, the increase in force at long
lengths during submaximal stimulation has been ob-
served in skinned fiber preparations (20), so it is not
de dependent on an enhancement of the free Ca²⁺ con-
centration. Furthermore, Balnave and Allen (3) have shown
that, for the mouse flexor digitorum brevis muscle,
average free Ca²⁺ concentration is independent of
length for a given low frequency of stimulation.

Studies using skinned fiber preparations have shown
that when free Ca²⁺ concentration is submaximal there
is more relative active force developed at long than at
short sarcomere lengths. This result implies that there
is a leftward shift in the force-Ca²⁺ relationship when
the muscle is stretched to lengths beyond optimum (19,
20, 59, 61, 80). This property of skeletal muscle is
illustrated in Fig. 7. The force-Ca²⁺ relationship has
been used as an indicator of Ca²⁺ sensitivity (59, 79). A
leftward shift of the force-Ca²⁺ relationship represents
increased sensitivity, and a rightward shift represents
decreased sensitivity. At long sarcomere lengths, the
Ca²⁺ sensitivity is increased, but activation is not
affected. Therefore, the increased Ca²⁺ sensitivity at
long sarcomere lengths is likely caused by changes in
cross-bridge kinetics. It is believed that changes in
intermyofilament spacing, as a consequence of changes
in muscle length, may be responsible for these altered
cross-bridge kinetics and, therefore, length-dependent
changes in Ca²⁺ sensitivity (24, 25, 65, 67, 87).
It has been shown that intermyofilament spacing decreases as skeletal muscle is stretched (60, 74, 75). Using skinned fibers of rabbit soleus, Godt and Maughan (26) confirmed that compression of the myofilament lattice at short sarcomere length with high-molecular-weight dextran resulted in enhanced Ca\(^{2+}\) sensitivity (leftward shift in the force-Ca\(^{2+}\) relationship). These results have been confirmed by other investigators (12, 81, 87). Because compression of the lattice can enhance Ca\(^{2+}\) sensitivity, and the myofilament lattice is compressed at long lengths, it is reasonable to assume that lattice compression is responsible for the length-dependent increase in Ca\(^{2+}\) sensitivity.

The specific conditions within the myofilament space that affect length dependence of Ca\(^{2+}\) sensitivity are unknown. One explanation that has been offered is that proximity of the myofilaments enhances the probability of cross-bridge attachment, \(f(x)\). If rate of detachment, \(g(x)\), remains unaffected by intermyofilament spacing, an increase in \(f(x)\) would effectively result in a greater number of cross bridges being engaged during a contraction and thus enhance the amount of tension produced.

**PART 3: ISOLATED WHOLE MUSCLE**

It is difficult to use the segment or sarcomere length control approach to the study of the force-length properties of whole isolated or semi-isolated muscles. Therefore, we will not discuss segment-controlled experiments in this section, and the results of experiments with whole muscle will be described as the length-dependence of active force.

Rack and Westbury (71) found an extended plateau in the length dependence of active force for tetanic contractions in cat soleus muscles. The developed force did not start to decrease significantly until a muscle length corresponded to an average resting sarcomere length of \(\approx 3.0\ \mu m\). The extended plateau of the length-dependence of active force could be caused by elastic elements in series with the muscle fibers that allow for sarcomere shortening to a length much less than the resting sarcomere length.

Close (11) reported a length dependence of force in whole muscle, similar to the force-length relationship observed by those using segment control in single fibers (17, 28, 31); i.e., the force decreased linearly with decreasing filament overlap on the descending limb of the force-length relationship. Close (11) minimized the effect of series elastic elements that may have permitted better agreement between the active and passive sarcomere lengths than had been obtained by others (57).

Submaximal contractions. As in studies with single cells, studies with whole muscles revealed that the length dependence of force varies with different levels of muscle stimulation. The results of these studies showed that the optimal length for twitch contractions (11, 71, 76) and submaximally activated muscle (76) was shifted to longer muscle lengths than would be predicted according to the cross-bridge theory. Presumably, the same mechanism responsible for this shift in single fibers can explain this shift in whole muscles.

**PART 4: FUNCTIONAL IMPLICATIONS**

In the previous parts of this review, we were concerned with the force-length properties and the length dependence of force production in isolated skeletal muscles, fibers, or sarcomeres. In reality, muscles are attached (typically) to bones, and they cross one or more joints at which they function to produce movements. The forces exerted by muscles create a moment about the joint(s).

When considering the force-length characteristics of a muscle in its anatomic environment, two factors should be considered when evaluating the potential of the muscle to produce movements: the moment arm of the muscle about the target joint and the excursion of the muscle. The moment arm of the muscle determines the moment \(\vec{M}_j\) produced about a joint by muscular contraction since

\[
\vec{M}_j = \vec{r} \times \vec{F}
\]

where \(\vec{r}\) is the location vector from the center of the joint to any point on the line of action of the muscle force, \(\vec{F}\) is the muscle force (vector), and \(\times\) represents the vector cross product (Fig. 8A). For two-dimensional considerations, the above equation may be written in its scalar form as

\[
M_j = d \cdot f
\]

where \(M_j\) is the magnitude of the moment produced by the muscle about the target joint, \(d\) is the perpendicular distance (moment arm) from the joint center to the line
of action of the muscle force, and \( f \) is the magnitude of the muscle force (Fig. 8B).

The importance of the moment arm may be illustrated in an example with two muscles of identical force-length properties crossing the same joint, but their moment arms are different by a factor of two (Fig. 9). If we assume that muscle B has a moment arm twice that of muscle A, it is obvious that muscle B can exert a peak moment twice that of muscle A. However, the larger moment arm of muscle B compared with A causes a larger change in length of muscle B compared with muscle A for a given change in joint angle; therefore, muscle B has only one-half the range of active force production (in terms of joint angular displacement) compared with muscle A (Fig. 10).

Because the moment-angle relationship of a muscle about a joint is determined by the force-length properties of the muscle and the variable moment arm, the following three basic scenarios relating force and moment arm to the joint moment are possible (Fig. 11): 1) the moment arm is about constant throughout the range of joint motion; therefore, the shape of the moment-angle relation reflects the shape of the force-length relation accurately (Fig. 11A); 2) the moment arm is decreasing (increasing) as the force potential of the muscle is increasing (decreasing); therefore, the moment-angle relation is flattened compared with the force-length relation (Fig. 11B); and 3) the moment arm is increasing (decreasing) as the force potential is increasing (decreasing), therefore the moment-angle relation is an enhanced version of the force-length relation (Fig. 11C). Depending on the exact relation between the force-length property of the muscle and the corresponding moment arm, the joint angle of maximal muscle force may not correspond to the joint angle of maximal moment.

Excursion is defined as the change in length of a muscle when going through the full anatomical range of joint motion (i.e., longest to shortest in situ length). Intuitively, one might assume that a muscle is attached to the musculoskeletal system such that it operates essentially at or around its optimal length (i.e., a length at which it is strongest). However, this assumption is not correct. Most muscles whose in situ force-length properties have been determined appear to operate primarily on the ascending or descending limb of the force-length relationship, reaching the plateau toward
the end of the range of joint motion. For example, the cat soleus, gastrocnemius, and plantaris muscles have all been found to work primarily on the ascending limb of the force-length relationship (34, 71). Similarly, human gastrocnemius muscles operate primarily on the ascending limb of the force-length relationship (35). However, frog semitendinosus appears to occupy the descending limb of the force-length relationship during everyday use (56).

The question that arises is, Why do different muscles occupy different parts of the force-length relationship in vivo? The answer to this question is not known; however, it appears likely that there is some connection between the functional demands of a muscle and the region of the force-length relationship it occupies. For

Fig. 11. Force- (solid line) and moment- (dashed line) joint angle (α) relationships for a muscle with 3 different (A-C) moment arm (r)-joint angle relationships.

Fig. 12. Schematic representation of a force-fiber length relationship and a muscle undergoing a stretch-shortening (loop on left) and a shortening-stretch (loop on right) cycle.

Fig. 13. Active (a), passive (p), and total (t) force-length relationships for frog gastrocnemius, sartorius, and semitendinosus muscles [Adapted from Wilkie (88).]
example, during everyday movement tasks, muscles undergo either a stretch-shortening or a shortening-stretch cycle. In a stretch-shortening cycle, a muscle is stretched initially as it increases its force and then shortens as it decreases its force (Fig. 12). Such a function follows a “force vs. length” curve with positive slope, similar to that of the ascending limb of the force-length relationship. During a shortening-stretch cycle, a muscle shortens first as it develops force and then is stretched as its force decreases. This function follows a negative force vs. length plot, similar to the descending limb of the force-length relationship.

For the few muscles for which functional and in vivo force-length properties are known, it appears that muscles undergoing stretch-shortening cycles during normal movements operate on the ascending part (cat and skunk triceps surae, plantaris, and human gastrocnemius (30, 34, 35, 71)), and muscles undergoing shortening-stretch cycles operate on the descending part of the force-length relationship (frog semitendinosus (56)). It is also worth noting that muscles that operate on the ascending part of the force-length relationship tend to have a passive force that appears at short muscle length, whereas muscles that operate on the descending part tend to have passive forces that appear only at long muscle length (Fig. 13).

Up until now, the force-length property of skeletal muscle has been considered as a given, constant characteristic. However, muscles have a tremendous ability for adaptation. Therefore, the question arises, Do in vivo force-length properties adapt to changes in everyday requirements, changes that might be produced, for example, by chronic training or tendon transfer surgery?

In a study aimed at determining the in vivo force-length properties of human rectus femoris muscles, we made the following observations (Fig. 14): the force-length curve for normal subjects was centered around optimal length (Fig. 14A) (36), whereas the force-length curves of national caliber runners and cyclists fell on the ascending and descending limb of the force-length relationship, respectively (Fig. 14, B and C) (32). Although the sample size for the different groups was small (n = 9, 4, and 3 for normal subjects, runners, and cyclists, respectively), the force-length regression lines for the runners and cyclists were statistically (α = 0.05) different from zero, whereas those of the normal subjects were not.

There is a variety of possible explanations for why the force-length properties for the runners were different in shape from those of the cyclists; the most appealing reason is an adaptation of the number of sarcomeres arranged in series in the muscle fibers. Following this argument, one would assume that the...
number of sarcomeres in series in rectus femoris muscle fibers of runners is larger than in cyclists; therefore, for a given joint configuration, the average sarcomere length of the runners would be smaller than that of the cyclists (Fig. 15). Whether this type of adaptation really occurred in the top athletes could not be evaluated.

In an attempt to determine whether the number of sarcomeres arranged in series within muscle fibers would adapt to tendon transfer procedures, the following study was performed (50, 51). The tendon of the rabbit tibialis anterior was released from its retinacular constraint at the ankle. For the experimental animals (retinacular release), the origin to insertion distance of the tibialis anterior (and, therefore, its passive force) became smaller, but the moment arm about the ankle (and, therefore, its excursion) became larger than it was for the control animals (no surgery or sham surgery). If active or passive forces were the primary positive modulators of the number of in series sarcomeres, one would expect to see a decrease in the number of sarcomeres in the experimental compared with the control muscles; if, however, excursion was the primary positive modulator, one would expect an increase in the number of sarcomeres.

After 8 wk of retinacular release of the tibialis anterior muscles, it was found that in these growing rabbits (4–12 wk) the number of sarcomeres in series was significantly increased in the experimental compared with the control and sham-control animals (Fig. 16). Therefore, it was concluded that a (small) change in tendon path (a “tendon transfer”) that causes changes in the origin to insertion distance and moment arm of the target muscle can cause adaptation in the number of sarcomeres arranged in series in a muscle fiber. Such a change in sarcomere number also changes the shape of the length-tension curve. Specifically, a muscle with an increased number of sarcomeres in series is able to produce active force over a larger range of muscle length than a corresponding muscle with fewer sarcomeres.

Varying the type of exercise has also been shown to influence sarcomere number. Lynn and Morgan (55) studied vastus intermedius fibers of rats trained with uphill or downhill running. They found that the rats in the downhill-running group had significantly more sarcomeres arranged in series in the vastus intermedius fibers than the rats in the uphill-running group, indicating that skeletal muscle is able to adapt sarcomere number to the movement (exercise) demands.

Summarizing, it is not trivial to determine the length dependence of force of in vivo human skeletal muscles. Aside from problems associated with controlling maximal effort, voluntary contractions, and isolating the force of a single muscle from the total force of an agonist group (issues that were not addressed here), force-length properties can and will adapt to the functional requirements imposed on the muscle. In vivo, the force-length properties of muscles manifest themselves as moment-joint angle relationships that are more complex than the force-length relationships because of the nonlinearities that exist between force and moment as well as between muscle length and joint angle. These nonlinearities are directly associated with the variable moment arm of the muscle force vector about the target joint. Therefore, moment arm determination or, equivalently, the determination of the instantaneous joint axis and line of action of the muscle become of utmost importance when determining the in vivo force-length properties of muscles. However, the in vivo determination of these factors is often difficult and sometimes impossible.

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