

Relationships Between Range of Motion, L_0 , and Passive Force in Five Strap-Like Muscles of the Feline Hind Limb

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ABSTRACT The relationships between range of motion, optimal length for force production (l_0), and passive force provide useful insights into the structure and function of muscles but are unknown for most individual muscles. We measured these values and examined their relationships in five strap-like muscles of the cat hind limb: caudofemoralis, semitendinosus, sartorius anterior, tenuissimus, and biceps femoris anterior. The range of motion relative to l_0 was found to vary significantly between different muscles and even between different specimens of the same muscle. The passive force-length (FL) curve was found to be correlated with both l_0 and l_{max} (maximal *in situ* muscle length) but was correlated more strongly with l_{max} . The mean passive force produced by these muscles at l_{max} was less than 7% of estimated maximal isometric force, suggesting that passive force may not be important in these muscles during normal activation patterns. The variance in passive FL curves between specimens of the same muscle was found to be significantly lower when length was scaled by l_{max} as opposed to l_0 . These results suggest that l_{max} may provide a more useful scaling factor for generic models of muscle. However, the passive length-tension properties of mammalian muscle appear to reflect a complex mix of structures at both the myofibril and connective tissue levels that may differ depending on muscle-fiber architecture and perhaps on the history of trophic influences on a particular specimen. © 1996 Wiley-Liss, Inc.

Despite the vast amounts of specific muscle morphometric data currently available, it is still unclear how universal certain relationships are across different muscles. In particular, the relationships between passive force, the *in situ* range of motion (ROM), and l_0 (length at which maximal isometric tetanic tension occurs) have not been well established, nor is there even agreement on the relative importance of myofibrils vs. connective tissues to these relationships in whole muscle. Much of the research that has been done on cat hind limb muscles has focused on a select few muscles with a simple architecture—the pinnate triceps surae (Rack and Westbury, '69; Goslow et al., '73; Morgan, '77; Scott et al., '96; others). To expand our knowledge to include muscles of a different architecture, this study examined the relationships between ROM, l_0 , and passive force in five strap-like muscles of the cat hind limb.

Knowing the relationship between the ROM and l_0 is important for developing a model of muscle. Because l_0 is a cornerstone

of many models (Zajac, '89; Brown et al., '96), identifying the ROM (relative to l_0) over which a model might be used is important for the design and validation of a model. Models also need to incorporate scaling factors for variables such as length and force if they are to be generic. L_0 is often used as a scaling factor because it is believed to be a species-specific constant of the active component of the force-length (FL) curve at the sarcomere level (Otten, '87; Herzog et al., '92). The FL curve, however, is comprised of both an active component and a passive component. One goal of this study was to determine whether l_0 is also an appropriate scaling factor for the passive FL curve.

Passive force arises in muscles when they are stretched beyond a nominal slack length (l_{slack}). The nature of this passive elasticity in muscle has been investigated many times

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and has produced many conflicting results (Ramsey and Street, '40; Hill, '50; Magid and Law, '85; Horowitz et al., '86; Horowitz, '92). Connective tissues, sarcolemma, and sarcomere myofilaments have all been suggested as being responsible for passive elasticity, but in whole muscle it still remains unclear which factor dominates.

The situation is much simpler in skinned single fibers, which also have an inherent passive elasticity. Of the previously stated three possible contributors to passive force, elasticity of the myofilaments within the sarcomeres must be responsible for elasticity in individual skinned fibers by process of elimination (Magid and Law, '85; Horowitz et al., '86; Horowitz, '92). Such myofilament properties seem likely to be genetically specified and perhaps closely related to other myofilament properties such as l_0 . There is evidence that in intact single fibers of frog sartorius, the sarcolemma can affect passive elasticity but only at relatively long sarcomere lengths (Rapoport, '73; Magid et al., '84). These results explain single fiber passive elasticity but may not be useful for explaining whole muscle passive elasticity and its possible variation in different muscles and species. If the elasticity inherent in the connective tissue matrix surrounding the muscle fibers contributes substantially to whole muscle passive force, then this opens a very different class of genetic and trophic mechanisms whereby the mechanical properties of whole muscles might become tuned to their conditions of use. In particular, such mechanisms might scale according to sarcomere properties other than l_0 , such as l_{max} , the maximal length that the sarcomeres experience at the anatomical limits of motion of the musculoskeletal system. The last goal of this study was to examine the relationships between the passive FL curves and both l_0 and l_{max} and to make inferences about the relative importance of various sources of passive elasticity in whole muscle.

MATERIALS AND METHODS

All data were collected from ten recently sacrificed (nonrigored) cats of either sex (2.5–4.4 kg) at the end of acute experiments that did not involve hind limb musculature. Five parallel-fibered muscles were examined in this study: sartorius anterior (SA), semitendinosus (ST), caudofemoralis (CF), biceps femoris anterior (BF), and tenuissimus (TN). Because all of these muscles have minimal

aponeuroses, muscle belly length provides an accurate indication of contractile component length, although not necessarily the individual fiber lengths (Loeb et al., '87). The anterior portion of BF was defined as per Chanaud et al. ('91). All five muscles were studied in six animals, while only some of the muscles were studied in the remaining four animals.

Immediately after sacrificing each animal with an IV overdose of pentobarbital, the muscles to be examined were exposed just enough to allow the *in situ* anatomical range of motions to be measured (muscle belly length). Both l_{min} (minimal *in situ* muscle length) and l_{max} were determined by moving the hip and knee joints to those positions that produced the minimum and maximum possible *in situ* lengths. Because the muscles were often slack and buckled when the joints were in a position to measure l_{min} , the tendons were pulled taut to mimic a contraction. l_{min} was then measured as the distance between the two tendons—that is, the length of the muscle belly if it were actually contracting. The muscles were then further dissected one at a time to prepare for measurements of passive force. All passive force measurements were recorded on a calibrated physiograph attached to a force transducer (NARCO Biosystems myograph F2000, Houston, TX). Length measurements (muscle belly) were recorded at zero passive force, and length increments were applied in steps by a ratchet (5.0 cm range of motion). Forces were recorded at the various lengths during the time interval 2–4 s after each incremental stretch, after which length was increased for the next measurement. Because force tended to decline approximately 5% during the 2–4 s measurement interval, the force used for this study was an average of the force over the 2–4 s interval. All passive force measurements were completed within 3 h of sacrificing the animals.

The experimental setup for each muscle was similar in that each muscle was separated from all other tissues except at the origin and insertion. One end of the muscle was then immobilized, while the other end was cut and clamped to a force transducer suspended directly above the muscle so that force was measured in a vertical line. For SA, the cat was placed on its back and the ilium (SA origin) immobilized by pressing down on the ipsilateral innominate bone to make it flush with the table top. The patella (SA

insertion), which was separated from all other tissues, was clamped to the force transducer for force measurement. For ST, the ischium (ST origin) was immobilized in the same manner as the ilium for SA, and the insertion tendon was clamped at the tendon-aponeurosis interface so that no tendon was included in the preparation. The two in-series heads of ST were treated together as if there were no intermediate inscription. For CF, the cat was placed on its belly and the caudal vertebrae (CF origin) immobilized by pressing them down on top of the pelvis. The CF insertion tendon was clamped to the force transducer at the tendon-aponeurosis interface. For BF (with the cat on its belly) the tibia (BF insertion) was immobilized by pressing it against the table top and the portion of the ischium from which BF originated clamped into the force transducer. Finally, with the cat still on its belly, the caudal vertebrae (TN origin) were secured as per CF and TN's insertion tendon clamped at the tendon-aponeurosis border.

After collection of the passive force data, the muscles were removed, aponeurosis lengths measured, and the muscles weighed. The animal was then left to rigor overnight, lying on its belly with hindlimbs splayed to each side. Rigored contralateral muscle belly lengths were measured and tissue samples from various parts of each muscle removed for measuring sarcomere length. The number of sarcomeres within 50 graticule units on our microscope (58 μm) was counted from at least twelve different samples for each individual muscle.

To estimate fascicle lengths, the average aponeurosis length ($\frac{1}{2}$ insertion length + $\frac{1}{2}$ origin length) was subtracted from the muscle belly lengths. It was assumed that changes in aponeurosis length due to compliance were

negligible. L_0 was estimated by the following method: the highest and lowest sarcomere counts for each muscle were dropped and the mean (\pm standard deviation) calculated from the remaining counts; the mean (\pm standard deviation) rigored sarcomere length was calculated from the previous sarcomere counts; the fascicle length (l_0) corresponding to an optimal sarcomere length of 2.4 μm (approximate center of the isometric active FL plateau [Herzog et al., '92]) was then extrapolated from the mean rigored sarcomere length and rigored fascicle length. Physiological cross-sectional areas (PCSA) were calculated using equation 1 (pennation angle effects were assumed negligible because of the small pennation angles involved).

$$\text{PCSA} = \frac{\text{muscle mass}}{\text{density} \times \text{fascicle length}}$$

$$\text{density} = 1.06 \text{ g/cm}^3 \text{ (Mendez and Keyes, '60)} \quad (1)$$

$$\text{fascicle length} = l_0.$$

RESULTS

The morphological data collected in this study are summarized in Table 1. The measurements of the *in situ* l_{\min} and l_{\max} are shown in Figure 1 with all fascicle lengths scaled by l_0 . The uncertainty in l_0 , arising from the averaging of sarcomere counts, is illustrated in Figure 1 by use of a bar (two standard deviations long) centered on the mean length. A simple analysis of variance demonstrated that some of the l_{\max} means (CF = 1.11 l_0 , ST = 1.43 l_0 , SA = 1.41 l_0 , BF = 1.19 l_0 , TN = 1.27 l_0) were significantly different from each other ($P < .001$). An *a posteriori* comparison between all possible pairs of means using Tukey's Honestly Significant Difference procedure found the fol-

TABLE 1. Morphological data of five hind limb muscles¹

	CF (N = 9)	ST (N = 9)	SA (N = 9)	TN (N = 7)	BF (N = 9)
l_{\min} (mm)	40 \pm 7	45 \pm 13	54 \pm 7	113 \pm 12	54 \pm 5
l_{\max} (mm)	66 \pm 6	117 \pm 10	128 \pm 7	182 \pm 10	96 \pm 8
l_0 (mm)	60 \pm 4	82 \pm 9	92 \pm 9	145 \pm 14	81 \pm 9
$l_{F=2}$ (mm)	72 \pm 7	112 \pm 12	122 \pm 11	190 \pm 7	105 \pm 9
$l_{F=2}$ (l_0)	1.22 \pm .12	1.38 \pm .20	1.35 \pm .16	1.32 \pm .10	1.30 \pm .10
$l_{F=2}$ (l_{\max})	1.09 \pm .07	.96 \pm .06	.96 \pm .07	1.04 \pm .05	1.10 \pm .09
Apon. (mm)	11 \pm 3	22 \pm 3	20 \pm 6	—	32 \pm 3
Mass (g)	2.4 \pm .8	7.9 \pm 2.4	5.9 \pm 1.6	.31 \pm .08	25.8 \pm 5.8
PCSA (cm ²)	.38 \pm .13	.92 \pm .28	.60 \pm .13	.020 \pm .004	2.97 \pm .45
Normalized variance (var./mean ²)					
$l_{F=2}$ (mm)	0.0094	0.0106	0.0075	0.0014	0.008
$l_{F=2}$ (l_0)	0.0091	0.0218	0.0143	0.0061	0.0055
$l_{F=2}$ (l_{\max})	0.0037	0.0045	0.0054	0.0021	0.0067

¹All lengths are fascicle lengths, apon. is the average of the insertion and origin aponeuroses, and all data are listed as mean \pm standard deviation.

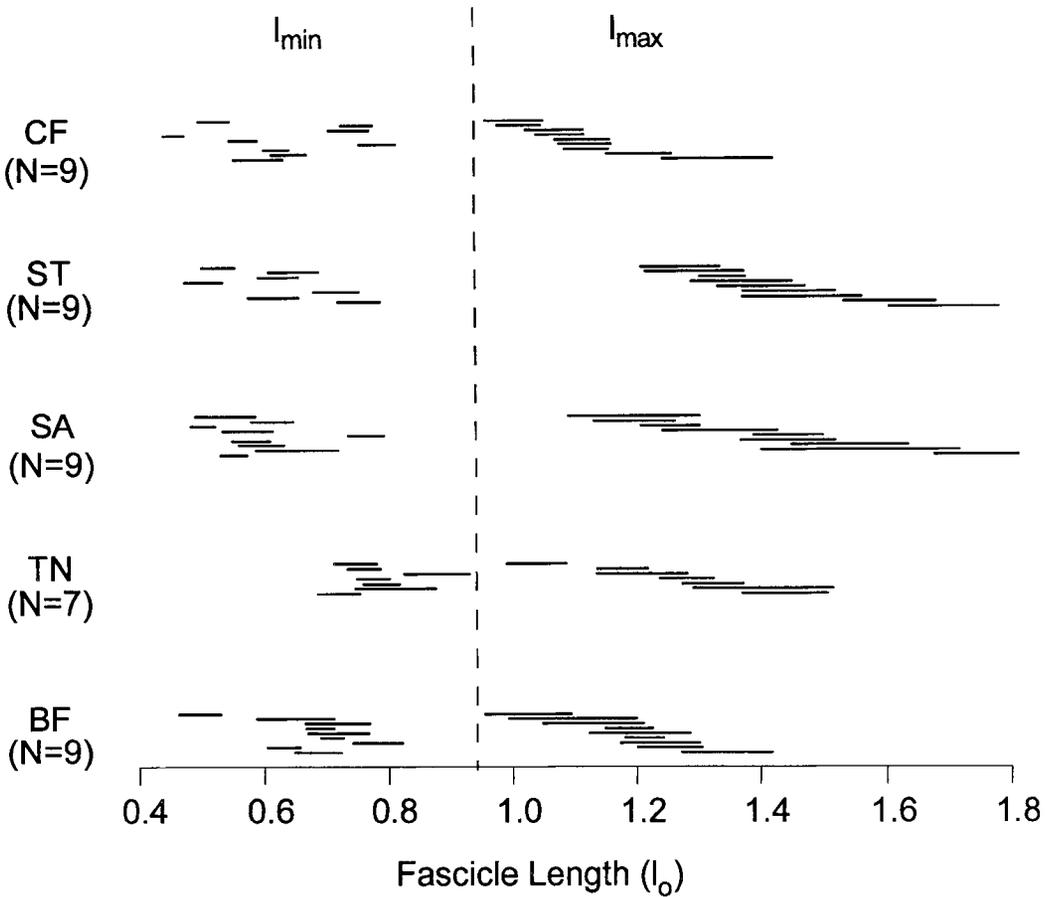


Fig. 1. l_{\min} and l_{\max} (fascicle lengths) for all 43 muscles normalized by l_0 . (Bar indicates uncertainty in measurement of l_{\min} or l_{\max} arising from the averaging of sarcomere counts and is two standard deviations centered on the mean.)

lowing four pairs of means significantly different ($P < .05$): CF-ST, CF-SA, BF-ST, BF-SA.

Figure 2 shows plots of the passive tension (scaled to PCSA) vs. fascicle length (scaled to l_0) for all 43 muscles studied. The range of measured passive forces was then plotted in Figure 3A along with similar data from cat soleus (SOL) (replotted from Scott et al., '96) and frog ST (replotted from Magid and Law, '85). L_0 for frog muscle was taken to be 2.1 μm (approximate center of the isometric active FL plateau [Herzog et al., '92]).

Figure 3B shows the same data in Figure 3A but plotted with fascicle length scaled by l_{\max} instead of l_0 (frog ST l_{\max} estimated as 3.5 μm as per Lieber and Boakes ['88]). A comparison between the two graphs suggests that passive force is better correlated with l_{\max} . As

a first approximation for examining the correlation between the passive FL curve and l_{\max} , the length at which passive force = 2.0 N/cm^2 ($l_{F=2}$) was used as a length measure for each passive FL curve (see Fig. 4 for SA example). $l_{F=2}$ was used instead of l_{slack} because it is easier to determine more accurately than l_{slack} due to the differences in slope at the two lengths (Brown et al., '96).

With all muscles lumped together in a single group (including SOL and frog ST), significant correlations of $r = .85$ ($P < .001$) and $r = .35$ ($P < .02$) were found between $l_{F=2}$ and l_{\max} (both lengths normalized by l_0) and $l_{F=2}$ and l_0 (both lengths normalized by l_{\max}), respectively. The correlation between $l_{F=2}$ and l_{\max} was found to be significantly greater than the one between $l_{F=2}$ and l_0 ($z = 4.35$,

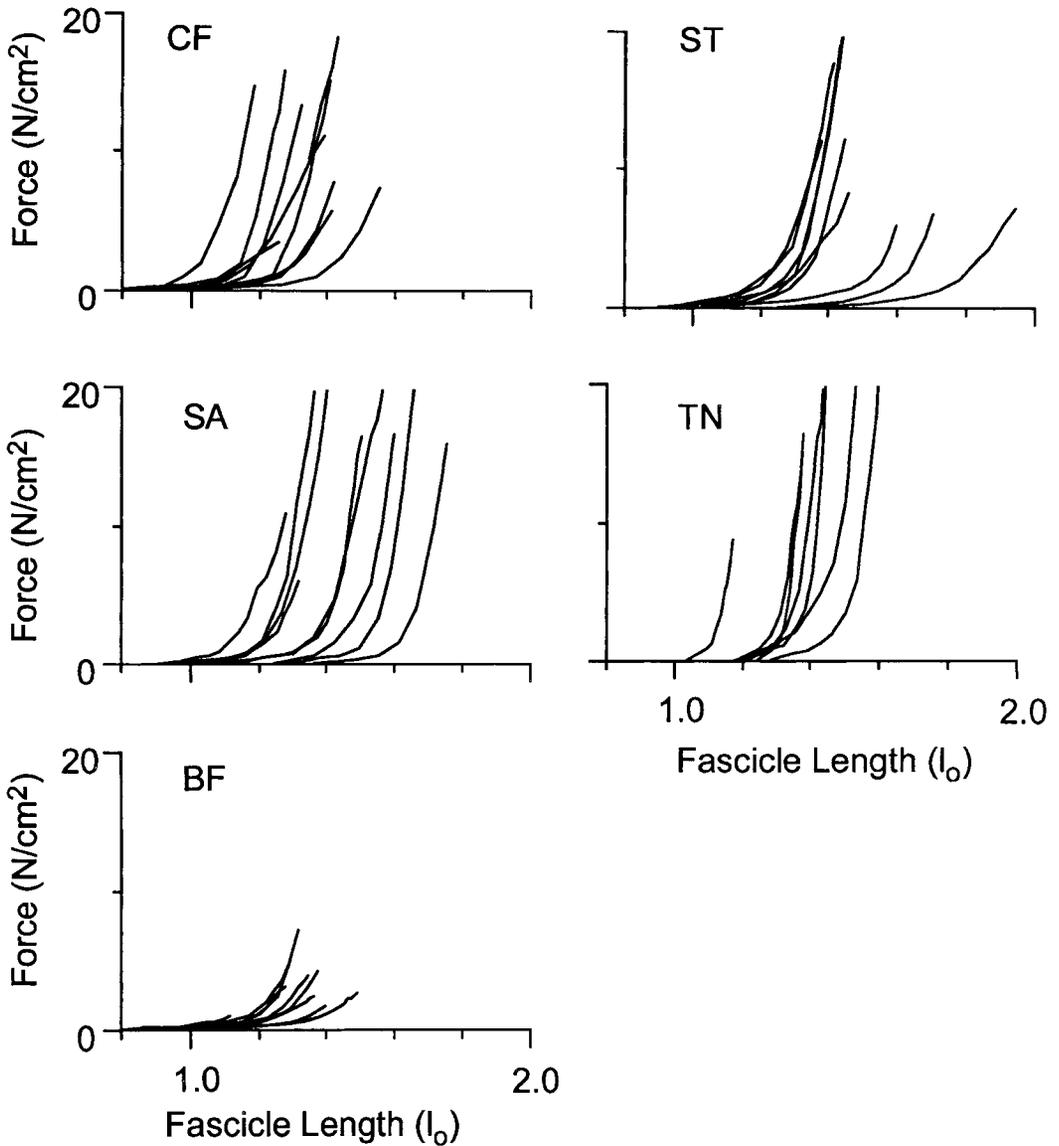


Fig. 2. Passive FL curves for all 43 muscles examined in this study. Forces are normalized to PCSA and fascicle lengths to l_0 (as estimated from sarcomere lengths).

$P < .001$). The values of $l_{F=2}$ are listed in Table 1 using three different units: millimeters, l_0 , and l_{max} . These three scaling units were compared by examining the normalized variances ($\text{var.}/\text{mean}^2$) within each muscle group (listed in Table 1). The average normalized variances for all muscles lumped together were 0.0077 for $l_{F=2}$ (mm), 0.0116 for $l_{F=2}$ (l_0), and 0.0046 for $l_{F=2}$ (l_{max}). F-test comparisons of these average normalized vari-

ances showed that scaling by l_{max} produced significantly less variance than scaling by either millimeters ($F = 1.67$, $P < .05$) or by l_0 ($F = 2.52$, $P < .01$).

DISCUSSION

The physiological range of lengths over which muscle fibers can extend is important in defining which part of the FL curve needs to be best understood. The results shown in

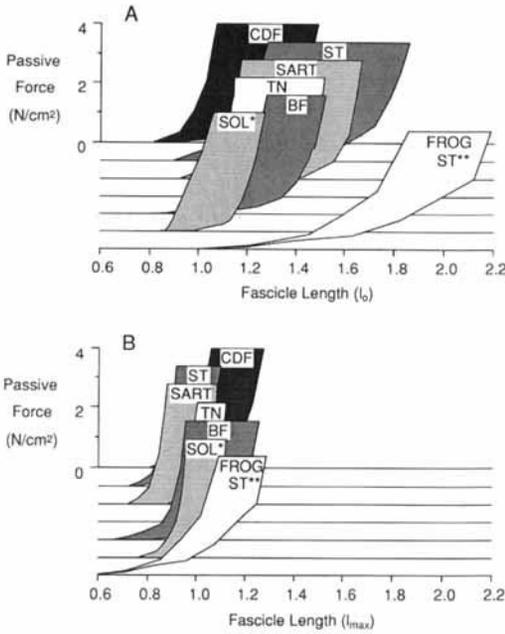


Fig. 3. Range of passive forces observed in this study compared to cat soleus whole muscle and frog semitendinosus whole muscle (SOL* (N = 5) from Scott et al. ('96), Frog ST** (N = 4) from Magid and Law ('85)). Forces scaled by PCSA. **A:** Fascicle lengths scaled by l_0 . **B:** Fascicle lengths scaled by l_{max} .

Figure 1 suggest that the location of l_0 within the ROM is variable. Muscles such as SOL and CF can be active on a large portion of the ascending limb of the FL curve and only a small portion of the descending limb ($< 1.11 l_0$). Other muscles such as SA have ROMs that extend well beyond l_0 (up to $1.41 l_0$) and are known to be active at these long lengths (Hoffer et al., '87). SA is similar in this re-

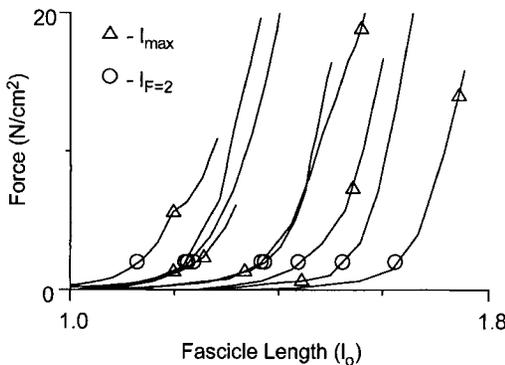


Fig. 4. Example of $l_{F=2}$ (O) and l_{max} (Δ) for SA passive FL curves.

spect to both tibialis anterior (TA) and extensor digitorum longus (EDL) that are active over $0.85-1.35 l_0$ and $0.75-1.25 l_0$, respectively (estimated by normalizing data from Goslow et al ['77] data with fascicle lengths from Sacks and Roy ['82]). Studies such as those of Scott et al. ('96), which systematically examined the active and passive FL and FV curves within the physiological ROM for cat SOL, thus need to be extended for those muscles that are active over a larger ROM.

An important finding of this study for modeling force production in muscles is that $l_{F=2}$ (and hence passive force) is more closely associated with l_{max} than l_0 . Although it is generally agreed that l_0 is the appropriate scaling factor for muscle length to produce generic FL curves (Zajac, '89; Brown et al., '96), l_{slack} and l_0 are often used interchangeably (Zajac, '89; Hawkins and Hull, '91; others). Data from this study (along with those of Woittiez et al. ['83] and Scott et al. ['96]) demonstrate that l_{slack} and l_0 cannot necessarily be used interchangeably. Furthermore, although there is a significant correlation between l_0 and $l_{F=2}$, $l_{F=2}$ is more tightly correlated with l_{max} across different muscles than it is with l_0 . Even within different specimens of the same muscle, scaling the passive FL curve by l_0 results in a significantly larger variability in $l_{F=2}$ than scaling by l_{max} . Use of generic passive FL curves scaled to l_0 risks large inaccuracies that need to be considered by potential modellers.

Although current theory suggests that passive force is produced primarily by C-filaments (also known as connectin or titin), the results of this study can be used to argue otherwise. Magid et al. ('84) originally suggested the C-filament as an addition to the two-filament sarcomere model partly to explain the passive length-tension relationship. Adding the C-filament to the model accounted for the passive elasticity observed in skinned muscle fibers. Furthermore, the C-filament provides a mechanism to stabilize the myosin filaments in the center of the sarcomere. Based on recordings of passive tension from both single fibers and whole muscle (frog ST), Magid and Law ('85) went on to suggest that C-filaments were, in fact, responsible for most of the passive tension recorded in whole muscle preparations of frog ST and not connective tissue as was previously assumed (Ramsey and Street, '40; Hill, '50). A potential problem with their conclusion is that the individual fibers in their study produced more

passive force than the whole muscle. Magid and Law ('85) noted this impossibility but unfortunately could not determine the cause of the artifact and so ignored it during their interpretation of the data.

An entirely separate problem with applying Magid and Law's ('85) conclusions to mammalian tissue is their preparation. Frog ST is a preferred preparation for single fiber studies precisely because it has very little intramuscular connective tissue. Extrapolating their conclusions to muscles with significant amounts of connective tissue may be unwarranted.

Table 2 lists some of the observations that need to be explained by a theory of muscle passive force. The normalized variances of $l_{F=2}$ observed from three different preparations are given in two different contexts: the normalized variance within a specific muscle (between different animals) and the normalized variance between different muscles. Let us assume that the smallest variance in the table (0.0001 for single-fiber data from different specimens of the rabbit psoas muscle) reflects largely measurement error that would be similar for the other experiments listed. The results of a χ^2 test performed on each of the variances in Table 2 showed that all of the other variances were significantly different from 0.0001 ($P < 0.001$). The additional variability in passive FL curves from skinned single fibers from different muscles of the same species as well as from any type of whole-muscle comparison (different muscles or different specimens of one muscle) would seem to reflect real structural factors rather than measurement error.

TABLE 2. Passive force variances from three different preparations¹

Study	Same muscle, different specimens	Different muscles
Cat whole muscle: CF, ST, SA, TN, BF (present study) and SOL (Scott et al., '96)	0.0107	0.0060
Rat whole muscle: semi-membranosus and medial gastrocnemius (Woittiez et al., '83)	0.0017	0.0059
Rabbit skinned single fiber: psoas (Salviati et al., '90); soleus and psoas (Horowitz, '92)	0.0001 (Salviati et al.)	0.0211 (Horowitz)

¹Normalized passive force variance was estimated by the variance of $l_{F=2}$ (l_0)—var./mean². All data are derived from fascicle (or fiber) lengths.

There are two plausible theories that can explain these observations. The first hypothesis is that passive force is produced primarily by C-filaments. For this to be true, there must be genetic and/or use-dependent trophic factors that affect C-filament strength to explain the wide variability seen in this study. To explain the studies of Salviati et al. ('90) and Woittiez et al. ('83), one must assume that the lab-raised population of rabbits used by Salviati et al. was so homogeneous in both genes and/or muscle-use patterns that no differences were observed between different specimens of the same muscle, while at the same time assuming that the lab-raised population of rats used by Woittiez et al. did not have this same level of homogeneity or that their methods resulted in substantially greater measurement error.

Alternatively, one can hypothesize that passive force is not always produced primarily by C-filaments. For this to be true, there must be genetic and/or use-dependent trophic factors, some of which affect C-filaments and some of which affect extrasarcomeric structures. Within a lab-raised population of animals, some of these trophic factors may manifest themselves homogeneously, while others may not. By assuming that those factors responsible for C-filament strength are primarily homogeneous while those factors responsible for extrasarcomeric structures are not, one can easily explain the results observed both by Salviati et al. ('90) and by Woittiez et al. ('83). We tend to favor this second hypothesis because of its simpler assumptions.

Also in support of the second hypothesis is a more complex argument that first requires a brief description of the importance of the C-filament in active-lengthening damage. Morgan ('90) described how a dip in the total isometric FL curve of a sarcomere could occur, depending upon the relative positions of the active and passive FL curves (see Fig. 5). Morgan argued that such a dip would produce an instability that could result in sarcomeres "popping" one at a time during active lengthening. The larger the dip, the greater the instability and therefore the greater the likelihood of popping. These pops could stretch the sarcomere to abnormally long lengths where only passive tension occurred and could sometimes break the C-filaments that normally provided the sarcomere's passive tension. A broken C-filament would lead to uncentered myosin filaments and hence to

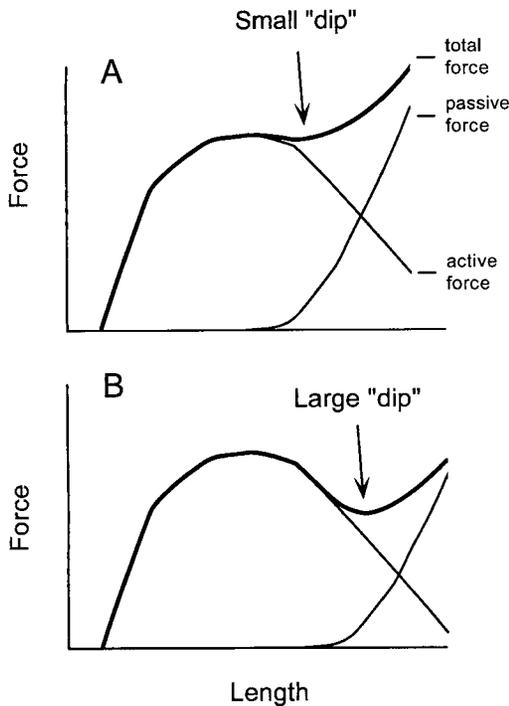


Fig. 5. Theoretical example of dip in total isometric FL curve occurring because of relative positions of active and passive FL curves. **A:** Small dip. **B:** Larger dip because passive FL curve occurs at longer lengths.

problems producing force. Visibly "popped" sarcomeres were later verified by Wood et al. ('93) and by Talbot and Morgan (in press).

By assuming temporarily that whole-muscle passive force is produced primarily by C-filaments, one can predict various muscles' susceptibility to damage based upon their passive FL curves. Because of the similarity in passive FL curves for SOL and CF (Fig. 3A), the dip in the sarcomere total FL curve for both should also be similar. Therefore, there should not be any significant difference in damage susceptibility between SOL and CF. Secondly, because the dip in SA's sarcomere FL curve tends to be much larger than CF's (Fig. 3A), one would predict that SA should be more susceptible to damage from active lengthening than CF. Data from other studies indicate that these two predictions are incorrect. Brown and Loeb ('95) observed that CF was extremely susceptible to damage, even from a single active stretch, whereas Scott et al. ('96) observed that SOL could withstand many active stretches without producing any observable damage. Although no

force data are explicitly available for SA, it is known that SA actively lengthens during the normal step cycle (Hoffer et al., '87), implying that it must have some degree of protection against active-lengthening damage. These results indicate that there is not a consistent relationship between whole-muscle passive force and susceptibility to damage from active lengthening. All of this suggests that whole-muscle passive force arises from various structures, only some of which tend to stabilize sarcomeres against active-lengthening damage.

Regardless of which structures are important for producing passive force, the high correlation of $l_{F=2}$ with l_{max} suggests that the trophic factors regulating passive force in all five hind limb muscles may be similar. The actual amount of passive force generated by these muscles at l_{max} was on average less than 7% of their maximal isometric force (based on an estimated maximal isometric force of 32 N/cm² [Scott et al., '96]). The extreme limb positions required for the various muscles to reach l_{max} were outside of the normal range of use in stereotyped behaviors such as walking or galloping, indicating that the contribution of passive force to total force is usually very small. These data are consistent with the notion that the connective tissue that probably gives rise to these passive forces grows so as to stabilize the muscle without effectively restricting its ROM.

The knowledge acquired in this study has provided insights beyond the basic morphology. We now have direct evidence that models of active force production based on data from muscles such as SOL need to be expanded to account for muscles that have different ROMs. The data presented here have also demonstrated the danger of attempting to normalize passive FL curves between muscles using l_0 . The small amount of passive force produced at l_{max} by all five muscles suggests that passive force may not play a large role in (hind limb) force production during normal use. Lastly, the data in this study suggest that C-filaments may not be primarily responsible for passive force of whole muscles in the cat hind limb. Instead, the dominant structures responsible for passive force may differ from muscle to muscle and may depend on trophic factors that remain to be identified.

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