

Review paper

Passive extensibility of skeletal muscle: review of the literature with clinical implications

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Received 1 August 2000; accepted 3 August 2000

Abstract

The purpose of this article was to review the literature on passive extensibility of skeletal muscle with reference to its anatomic and physiologic properties, mechanisms of adaptations and clinical implications. Studies with animal muscles have shown that passive extensibility is influenced by the size (mass) and length of muscle fibers, and the amount and arrangement of the connective tissues of the muscle belly. The resistance to passive lengthening is influenced by the readily adaptable amount of muscle tissue, including the contractile proteins and the non-contractile proteins of the sarcomere cytoskeletons. The relationship of adaptable changes in the muscle tissue and in the extracellular connective tissues remains unclear. Muscle length adaptations result from changes in the number of sarcomeres in series, which depend on the imposed length of muscles, not on the level of muscle activation and tension. This mechanism of muscle length adaptations, termed ‘myogenic’, has not been demonstrated in human muscles, but it has been intimated by therapeutic lengthening studies showing that both healthy and neurologically impaired human muscles can undergo increased length adaptations in the presence of muscle activations. Studies have suggested that optimal muscle function is probably achieved by increasing muscle length, length extensibility, passive elastic stiffness, mass and strength, but additional studies are needed to investigate these relationships, particularly for aged muscles and for muscles affected by clinical disorders, disease and injury. Such studies could contribute to the development of new intervention strategies designed to promote the passive muscle extensibility that enhances total muscle function, and ultimately improves the ability to complete functional activities and excel in athletic performances. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Skeletal muscle; Passive extensibility; Passive elastic stiffness

1. Introduction

The passive extensibility of skeletal muscles can be defined as the ability of skeletal muscles to lengthen without muscle activation. Passive extensibility is an important component of total muscle function because it allows for the maximal length of both non-activated and activated muscles. Maximal muscle length contributes to the maximal joint range of motion that is generally believed to influence functional activities and athletic performances. Accordingly, therapeutic interventions designed to increase the passive extensibility of muscles in order to achieve maximal joint range of motion are employed as an important component of physical rehabilitation and sports. Because efforts to improve the passive extensibility of human muscles are an integral part of therapeutic interventions, knowledge

of the basic anatomic and physiologic properties of passive extensibility is important to consider. Moreover, understanding the known mechanisms of passive extensibility adaptations and how they apply to interventions with human muscles could help to direct future studies that lead to new intervention strategies designed to promote favorable passive extensibility adaptations, functional activities and athletic performances.

The purposes of this article are to: (1) review the basic passive characteristics of skeletal muscles and the terms used to describe these characteristics; (2) review the anatomic and physiologic passive properties of skeletal muscles; (3) survey the evidence from non-human animal muscle studies of the proposed mechanisms of passive extensibility adaptations; and (4) discuss the results, limitations and clinical implications of studies with healthy, normal human muscles, neurologically impaired human muscles, and aged human muscles. The author hopes that the information will contribute to a better understanding of skeletal muscle passive extensi-

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bility and that the review will influence the direction of future studies.

2. Basic passive characteristics of skeletal muscles and descriptive terms

The terms used to describe the passive extensibility of skeletal muscles are often confusing because clinicians and researchers have used different terms to describe similar phenomena. The following section provides a brief overview of the basic passive characteristics of skeletal muscle and the terms used to describe these characteristics.

Studies conducted prior to and during the past century showed that the *total force produced* by skeletal muscles results from the summation of the *passive forces* and the *active forces*, both of which are influenced by the length of the muscle. The passive forces increase exponentially (curvilinear increase) as the muscle is stretched to its maximal length [1–8]. The active forces, produced by the interaction of actin and myosin contractile proteins, are greatest near the resting length of the muscle, and the active forces decrease as the muscle is either lengthened or shortened in relation to this mid-range muscle length [3,4,7–10]. As a result, the active forces show a parabolic force–length relationship (Fig. 1) [11]. Because the active forces cannot be measured directly, they are calculated by subtracting the passive forces from the total forces throughout the full length of the muscle. The results of these numerous studies have established the basic framework for how the change in the passive forces contributes to total muscle function when a muscle is stretched through its available length ex-

tensibility. The influence of the passive forces on the total force produced is depicted in the classic active and passive length–tension curves of skeletal muscles (see Fig. 1) [11].

The *muscle–tendon unit* is the gross anatomic and physiologic unit responsible for voluntary movements. Tendons, which consist of dense regular connective tissues and considered a part of the series elastic component of the muscle–tendon unit, exhibit minimal length extensibility characteristics [5,12,13]. Although there is some slight straightening of the connective tissues within tendons, for practical purposes the length of tendons can be considered constant, so the muscle belly is the primary part of the muscle–tendon unit that contributes to the overall passive length–tension relationships of the stretched muscle–tendon unit [5,12,13]. Accordingly, the term ‘muscle’ will be used in place of the terms ‘muscle–tendon unit’ throughout this article.

As a muscle is passively lengthened from a very short position that is without measurable passive resistance, it reaches a point where the first passive resistance to the stretch can be measured. This point of resistance is considered the *initial passive resistance*, and it defines an *initial length*, which is not identical to the *resting length* of the muscle (see Fig. 1). As the muscle is lengthened beyond this initial length, greater passive resistance is recorded until a *maximal passive resistance* is reached, corresponding to the point of *maximal length*. Stretch beyond this point results in rupture at the ends of the muscle fibers associated with the musculotendinous junction, which is documented in animals studies [14,15], and avoided in human studies because of obvious ethical reasons. In humans, the maximal length of muscles that is unrestricted by bony or other non-muscular tissue limitations, would correspond to the angular measurement of the *maximal passive joint range of motion*. Although maximal passive joint range of motion may be described clinically by the terms *flexibility*, or *passive stiffness* [16,17], the maximal passive joint range of motion that is measured clinically is one point that represents the maximal length of muscles. It should not be considered a measurement of the absolute length, the flexibility or the passive stiffness of muscles. Although some controversy exists about this terminology [18], the author suggests that the maximal joint range of motion should be called the ‘maximal joint range of motion’, a point that represents the maximal muscle length. The terms flexibility and passive stiffness more accurately depict a physiologic relationship of the passive resistive forces and the passive lengths of the muscle as it is stretched [16,19].

The terms *passive extensibility* and *passive length extensibility* can be considered synonyms describing the distance the muscle can be stretched while offering passive resistance to the stretch. Passive extensibility is the distance between an initial muscle length and the

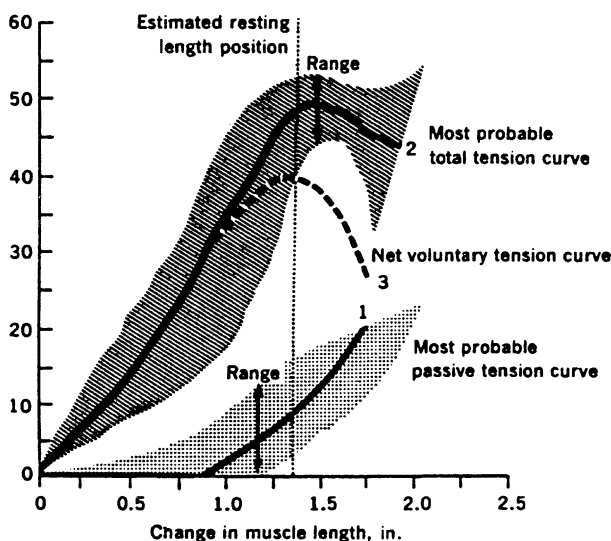


Fig. 1. Classic length–tension curves for skeletal muscle. Net voluntary active tension is predicted by subtracting passive tension from total tension. (With permission from Astrand and Rodahl [11].)

maximal length, both of which are dependent on the passive resistance to the stretch. Passive extensibility influences the maximal length because the maximal length is the end point of a muscle's length extensibility. The passive curve is usually constructed by plotting the passive length extensibility between an initial length and the maximal length in relation to the corresponding number of passive resistance points (Fig. 1).

Skeletal muscles demonstrate *viscoelastic properties*. That is, they exhibit *viscous behaviors* that depend on the rate of the applied stretch, and *elastic behaviors* that depend on the load of the applied stretch [20]. It may be difficult to separate and measure the viscous and elastic behaviors as a muscle is stretched from an initial length to the maximal length, the so called *dynamic phase* [18,21–24] or the *stretch phase* [25] of muscle lengthening. Accordingly, *passive viscoelastic stiffness*, *passive elastic stiffness* and *passive stiffness* are terms that are frequently used interchangeably to describe a muscle's physiologic response during this dynamic phase of the stretch. Passive elastic stiffness is defined as the ratio of the change in the passive resistance or passive force (ΔF) to the change in the length displacement (ΔL), or $\Delta F/\Delta L$. This physiologic response is usually measured at a slow constant rate of applied dynamic stretch in order to avoid stretch-reflex activations. As the velocity of stretch is increased the viscous behaviors of muscles contribute to increased passive resistance and increased passive elastic stiffness. This rate-dependent response has been demonstrated in animal muscles [26] and in human muscles in the absence of stretch induced muscle activations [27]. *Passive compliance* is defined as the reciprocal of passive stiffness ($\Delta L/\Delta F$), so the two terms represent the same physiologic response to stretch viewed from reciprocal perspectives. A muscle with a steep rise in the passive curve is stiffer, or less compliant than a muscle with a shallow rise in the passive curve. In contrast, a muscle with a passive curve that has a shallow curve is less stiff, or more compliant than a muscle with a passive curve that has a steep rise. Because some viscoelastic energy is lost immediately after muscles are stretched, they demonstrate decreased passive resistance when returned to their original shortened position at the same rate. This effect is manifested in a *hysteresis loop*, and the loss of stored viscoelastic energy can be calculated as the difference between the stretch phase (dynamic phase) and the return phase of the hysteresis loop [28,29].

Numerous studies have examined the influence of a constant, sustained stretch at the end of the dynamic phase of the stretch in efforts to study human muscle passive viscoelastic properties [21–25,29–33]. This constant stretch is referred to as a *static phase* [18,21–24] or a *holding phase* [25] of the muscle stretch. Again, because stored viscoelastic energy is lost immediately after muscles are stretched, they demonstrate *viscoelastic*

stress relaxation, expressed by the slope, or the percent decline in the passive resistance over time [21–25,30–33]. In addition to this stress relaxation (load relaxation), skeletal muscles also show *creep*, or strain relaxation (lengthening relaxation) when a constant load is applied [20]. Creep can help to explain the immediate increases in passive joint range of motion (muscle length) that have been measured in response to therapeutic stretching procedures.

3. Structures and mechanisms contributing to passive properties of muscle

When resting muscles are passively stretched, the resistance produced by the passive properties is thought to be influenced by several structures and mechanisms. These include: (1) stretching stable cross-links between the actin and myosin filaments, called the 'resting filamentary tension,' and perhaps resistance from the actin and myosin filaments directly (series elastic components); (2) stretching non-contractile proteins of the endosarcomeric and exosarcomeric cytoskeletons (series elastic components); and (3) deformation of the connective tissues located within and surrounding the muscle belly (parallel elastic component). As stated earlier, for practical purposes the length of tendons can be considered relatively constant and non-contributory to the measurable passive length–tension relationships of a stretched muscle [5,12,13].

3.1. Filamentary resting tension

The passive resistance that may result from stretching stable interactions or cross-links between the actin and myosin filaments was first proposed by Hill [34–36] and expanded by others (see [37], for a review). The stable bonds have been explained by a very low level of actively generated resting tension believed to impart passive resistance because the actin–myosin cross-bridges resist the stretch a short distance from the stable position before the contacts slip and reattach at other binding sites. This proposal was expanded to suggest that actin and myosin filaments are linked by a small number of slowly cycling cross-bridges, the so called 'Cross-bridge Population Displacement Mechanism' [38]. If this very low level of activity exists in completely relaxed human muscles, it is probably not measurable using surface electromyography (EMG). Instead, the passive state in human muscles is operationally defined by the presence of minimal, or negligible EMG activity [17,18,39–42].

In addition to the possibility that some passive resistance may reside in actin–myosin cross bridges, recent X-ray diffraction studies have provided evidence that actin and myosin filaments show extensibility properties that contribute to the stiffness of active muscle [43–46].

Whether the extensibility of actin and myosin filaments contributes to the resistance of a passively stretched non-activated muscle is unclear and worthy of future studies.

3.2. *Sarcomere cytoskeletons*

Recent studies have indicated that much of the passive resistance of a stretched relaxed muscle comes from non-contractile filamentous connections within two sarcomeric cytoskeletons, termed the endosarcomeric and exosarcomeric cytoskeletons. Filamentous connections between the thick myosin filaments and the Z-discs of the sarcomere have been shown to contribute to this passive resistance [47], particularly when the sarcomere is stretched beyond the actin and myosin overlap [48]. The filamentous connections of the endosarcomeric cytoskeleton are comprised of large, thin filaments of a giant protein that has been named ‘titin’ (also called connectin; molecular weight = 2600–3000 kDa) [49–53]. The titin protein attaches into the ‘M’ line region, or central area of the myosin filament, courses longitudinally and attaches into the Z-discs at the ends of the sarcomere. The titin protein is believed to be the major sub-cellular component of the endosarcomeric cytoskeleton that resists passive lengthening of a relaxed muscle [49–53]. Slow twitch muscle fibers (type I) have greater passive stiffness than fast twitch muscle fibers (type II), and the differences may reflect different isoforms of titin within each fiber type [54].

Intermediate sized protein filaments, with diameters of about 10 nm, midway between actin (6 nm) and myosin (16 nm), contribute to the exosarcomeric cytoskeleton of muscle fibers [49,50,55,56]. One protein, called ‘desmin’ (also known as skeletin; molecular weight = 55 kDa) is the major subunit of the intermediate protein filaments forming the Z-discs [56]. It serves to interconnect Z-discs transversely, and to connect Z-discs with organelles, but not with the T-tubule system [55]. Desmin also extends longitudinally from Z-disc to Z-disc outside of the sarcomere [50,56], and because of this longitudinal arrangement between Z-discs outside of the sarcomere, the protein contributes to the exosarcomeric cytoskeleton. Desmin lengthens as the sarcomere is stretched, so its elasticity is thought to contribute to the passive resistance of a stretched muscle.

The potential contribution of the ‘resting filamentary tension’, coupled with the possible resistance from the actin and myosin filaments directly, and the resistance from the titin and desmin non-contractile proteins, indicate that multiple sub-cellular components within muscle fibers contribute to the passive resistance one feels when stretching a relaxed, non-activated muscle. Because these components reside within the substance of muscle tissue directly, the passive resistance to stretch and the passive elastic stiffness are influenced by the

amount, or the mass of muscle tissue. This proposal has been supported by studies showing that the passive compliance at the elbow is negatively related to the volume of the arm [57], and that passive elastic stiffness increases with increased strength of muscles [17,24,58].

3.3. *Connective tissues*

As a muscle is stretched, the passive resistance is also influenced by a lengthening deformation of the connective tissues of the endomysium, perimysium, and epimysium of the muscle belly. The endomysium consists of a dense weave network of collagen fibers about 100–120 nm in diameter that surround the surface of individual muscle fibers and attach into the basement membrane of the sarcolemma [59]. The endomysium surrounding muscle fibers also attaches perpendicularly to adjacent muscle fibers [59] and interconnects with the perimysium [59,60]. The perimysium consists of tightly woven bundles of collagen fibers, 600–1800 nm in diameter [59] which interconnects groups of muscle fibers known as fascicles [59,60]. The perimysium interconnects with the epimysium which surrounds the entire muscle belly [59,60].

Although all three components of the connective tissues that package the muscle belly contribute to the resistance when a muscle is passively stretched, the relatively large amount of perimysium [62] with its well-ordered crisscross array of crimped collagen fibers surrounding muscle fasciculi [60–62], is considered the tissue that is the major contributor to extracellular passive resistance to stretch [59,62]. Examination of the perimysium with light microscopy [61,62] and scanning electron microscopy [63] revealed that the orientation of the crimped collagen changes as the length of the muscle changes. The crimped arrangement of the perimysium, a system of sheets with a three-dimensional weave surrounding muscle fasciculi, becomes uncrimped as the muscle is lengthened. The perimysium undergoes a mechanical deformation and realignment that should contribute to the exponential, or curvilinear increased resistance when a muscle is stretched. Some of the increasing resistance that a clinician feels as a relaxed muscle is stretched maximally probably stems from lengthening of the extracellular connective tissues of the muscle, primarily the perimysium.

4. **Animal muscle studies: passive extensibility characteristics and adaptations**

Numerous experimental non-human animal muscle models have shown that anatomic and physiologic length extensibility and passive elastic stiffness adaptations of skeletal muscles can be induced by different experimental methods, including immobilization,

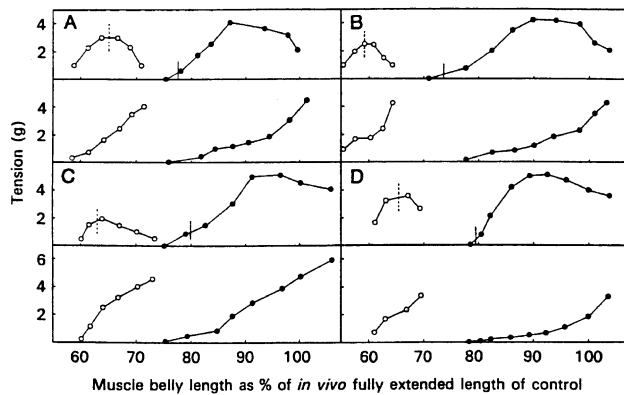


Fig. 2. Length–tension curves for young muscles (A–D) immobilized in shortened positions (○) and their controls (●). After immobilization the active and passive curves were shifted toward the left, indicating shorter muscles lengths. The passive curves also had less passive extensibility between the initial and maximal lengths, and a steeper rise indicating greater passive elastic stiffness ([8, p. 464], reprinted with the permission of Cambridge University Press).

denervation, local contraction by artificial stimulation, or a combination of these methods. The results of some of these animal muscle studies have suggested that muscle length adaptations result from a ‘myogenic’ mechanism, not a ‘neurogenic’ mechanism. In other words, the mechanism for length adaptations appear to reside within the muscle tissue directly, independent of neurological activity.

Researchers have used passive length–tension curves to provide information about changes in the passive forces, lengths, length extensibility and passive elastic stiffness of muscles in light of histologic and histochemical changes in the muscle. Following experimental interventions, the position and steepness of the curves may change, indicating changes in the muscles’ passive properties (Fig. 2). A shift of the curve to the left indicates a shorter muscle, and a shift of the curve to the right indicates a longer muscle. Increased displacement between the initial point of the curve (left) and the end point of the curve (right) would indicate greater passive extensibility. Decreased displacement between these two points would indicate less passive extensibility. A steeper passive curve indicates that the muscle has increased passive elastic stiffness (decreased compliance), whereas a shallower, less steep passive curve indicates that the muscle has decreased passive elastic stiffness (increased compliance).

4.1. Muscles immobilized in shortened positions

When muscles were acutely immobilized in shortened positions they showed a decrease in the total force produced, resulting from decreases in both the active forces and the passive resistive forces [8,64]. Muscle atrophy from immobilization in shortened positions has been associated with decreased tensile properties and a

reduced capacity to resist stretching to the point of rupture [65]. Muscle length also decreased [64,66], a change brought about by a reduction in the number of sarcomeres [6,8,67]. The soleus muscles of young mice immobilized in the shortened position showed a decreased growth in length because of decreased postnatal addition of sarcomeres [68]. Muscles immobilized in shortened positions showed significant loss of tissue protein because of decreased synthesis and increased degradation, primarily at the ends of the muscle fibers [69]. Muscles immobilized in the shortened position also presented decreased initial lengths [67] and maximal lengths, decreased extensibility, and apparent increased passive elastic stiffness (Fig. 2) [6,8,13]. When the immobilization was removed, the muscles readapted to gain their original sarcomere numbers and lengths.

Although decreased lengths have been attributed to a loss of sarcomeres, increased passive elastic stiffness, demonstrated by increased steepness of the passive curves, has been attributed to changes in the connective tissues of the muscles [6,8]. Muscles immobilized in shortened positions showed an apparent greater abundance [6,63] and remodeling [63] of connective tissues in the early stages of immobilization. Greater abundance of connective tissue was shown in the mouse soleus muscle by an increase in the relative concentration of hydroxyproline in relation to muscle fiber tissue [63]. Concurrent histological analysis showed that the early increased concentration (after 2 days of immobilization) occurred in the perimysium, followed by increased concentration in the endomysium after one week [63]. Acute immobilization of muscles in shortened positions causes decreased muscle weight [70] and muscle fiber atrophy [71]. Accordingly, the increased passive elastic stiffness observed in muscles acutely immobilized in shortened positions probably resulted from relative increases in the amount of connective tissues.

Relative amounts of connective tissue accumulation in muscles may be influenced by passive stretch and muscle activation. Studies with mouse soleus muscles [72] and rabbit soleus muscles [73] have provided evidence that the apparent connective tissue accumulation in inactive, immobilized muscles can be prevented by passive stretch [72,73] or by active stimulation [73]. The lack of connective tissue changes was demonstrated in mouse soleus muscles that were immobilized in a shortened position but passively stretched for 15 min every two days for a period of 10 days, even though there was a loss of muscle fiber length [72]. Connective tissues also did not change in rabbit soleus muscles that were periodically activated over a reduced range of motion, even though there was a reduction in the number of sarcomeres similar to when animal muscles were immobilized in shortened positions [73].

Evidence for connective tissue remodeling was provided by scanning electron microscopy of the soleus

muscles of mice immobilized in the shortened position for 2 weeks [63]. After 2 weeks the collagen fibers of the perimysium were oriented at more acute angles to the muscle fiber axis than were the collagen fibers of non-immobilized muscles fixed in the same position. This collagen fiber arrangement at the immobilized shortened length resembled the arrangement found in non-immobilized muscles held in lengthened positions. As a result of the remodeling, greater tension per unit of passive elongation would produce increased resistance to passive stretch. The passive curves were shifted to the left and appeared steeper, indicating that the muscles were shorter and stiffer after immobilization in the shortened position. The passive curves for the muscles of young animals and for the muscles of adult animals were similar [63].

The decreased lengths and passive extensibility of muscles acutely immobilized in shortened positions were brought about by a loss of sarcomeres in series. Decreased maximal passive force was probably influenced by a decrease in muscle mass because of associated muscle atrophy. Decreased muscle mass would result in the loss of the subcellular proteins described earlier (myosin, actin, titin and desmin), and this change would decrease both the maximal active force and the passive resistance to stretch. The relative amounts of connective tissues may increase and remodel, and contribute to a relative increased passive elastic stiffness. The relationship of acute changes in the intramuscular proteins and changes in the extracellular connective tissues, and how these changes influence the form and position of passive curves is worthy of further study.

4.2. Muscles immobilized in lengthened positions

Acute immobilization of muscles in lengthened positions has brought about an increase in muscle lengths because of increases in the number of sarcomeres [7,8,68]. As with muscles immobilized in shortened positions, these sarcomere adaptations occurred at the ends of the muscle fibers. The addition of sarcomeres was accompanied by increased protein synthesis [74] and weight gain [70,74] after immobilization. The increased number of sarcomeres, however, was not as great (19% increase) as the loss of sarcomeres in muscles immobilized in shortened positions (40% loss) [7]. Accordingly, these studies suggest that lengthening adaptations of muscles that start with normal lengths may not be as obvious as the lengthening adaptations of muscles that start with an abnormally shortened length. The active and passive curves for adult muscles immobilized in lengthened positions were shifted to the right, indicating that they were longer compared with those of adult controls. As with muscles immobilized in shortened positions, muscles immobilized in lengthened positions

readapted to their original lengths when the lengthening immobilization was removed.

In young muscles immobilized in lengthened positions the muscle belly length was decreased, so the curves of the experimental muscles were shifted to the left [8]. This evidence suggested that tendons of young, growing animals elongate more readily than in adult animals. In young mice with muscles immobilized in either shortened or lengthened positions, the overall muscle belly lengths decreased, with concomitant increases in tendon lengths [8]. Thus, in young animals shorter muscle bellies may result in strength deficits that are independent of the imposed lengths of the muscles during immobilization.

4.3. Evidence for a myogenic mechanism of length adaptations

Studies of peripheral denervation of skeletal muscles have revealed obvious loss of the ability of the animal to generate voluntary active tension. After denervation, the passive curves showed gradual changes over a period of weeks, with longer initial lengths, decreased extensibility between their initial lengths and their maximal lengths, and steeper passive curves compared with those of controls [75,76]. Denervation studies have also revealed that the length adaptations from immobilizing muscles may result from a myogenic mechanism, not a neurogenic mechanism. In adult rats, denervated muscles immobilized in shortened positions showed muscle belly shortening after 8 weeks [66], and a similar change was observed in adult cats after 4 weeks, with loss of up to 35% of the sarcomeres [67]. The muscle belly shortening and increased passive elastic stiffness were essentially the same as those observed for innervated muscles immobilized in shortened positions.

The reports that muscle length and associated physiologic changes may be independent of the level of muscle activation were supported further by studies of muscles stimulated with tetanus toxin [77,78] or electrical stimulation [79]. Local injection of tetanus toxin into the soleus muscles of guinea pigs produced a shift in the passive curve toward the left, indicating decreased length, and a 45% decrease in sarcomere number [78]. The shortening adaptations were similar to those found after the muscles of cats were immobilized in shortened positions [6]. Analysis of the changes in sarcomere numbers in the soleus muscles of guinea pigs after length and tension were varied independently indicated that the length of muscles, not the tension, appeared to be the determining factor in sarcomere number regulation [77]. Contraction of abnormally shortened muscles, however, may hasten sarcomere loss. Electrical stimulation of the sciatic nerve induced a 25% decrease in sarcomere numbers and increased passive stiffness within 12 h [79], whereas 5 days of shortening by immobilization in

plaster casts alone was required to produce similar changes [78]. Spastic gastrocnemius muscles in very young mice have been shown to grow in length at only 55% of the rate of growing bone, whereas the rate of growth of normal gastrocnemius muscles was 100% of the rate of growing bone [80]. Although the regulation of sarcomere numbers may be independent of the level of muscle activation, increased or decreased muscle activation appears to influence the rate of the regulation.

5. Human muscle studies: passive extensibility characteristics and adaptations

5.1. Methodological considerations

Measuring the passive extensibility of human muscles presents a formidable challenge because of difficulty applying research technologies and methodologies to objectively isolate and study specific muscles. Although the use of computer technologies that permit simultaneous integration of the velocity of stretch, angular displacement, passive resistance, and EMG activity have improved objective testing, operational definitions not used in animal studies are needed to describe some key measurement phenomena in humans. Two measurements that require operational definitions are: (1) defining what is meant by a passive muscle stretch, and (2) defining the end point of muscle stretch.

A passive muscle stretch can be operationally defined when there is minimal, or negligible EMG activity recorded through surface electrodes [17,18,39–42,58]. Based on ethical considerations, human subjects are usually asked to relax and maintain EMG silence in the targeted muscles in order to achieve a passive muscle stretch. Although this method may be clinically relevant, ensuring complete muscle silence is not possible using surface EMG because low level activity may go undetected. Even so, recent studies have indicated that low level, minimal EMG activity in targeted human muscles may be unrelated to immediate measurements of their maximal length [23,28,29,32] and to their viscoelastic properties [25,28,30–33]. This makes sense in light of the evidence from animal studies showing that muscle length adaptations depend more on the imposed length positions than on the amount of activation within the muscles [77]. Moreover, optimal normal muscle function requires maximal length extensibility in the presence of muscle activations, so it seems plausible that passive length adaptations in humans can occur in the presence of low level EMG activity.

Accurately identifying the end point of muscle stretch is another potential problem because to do so requires operational definitions that are based on psychophysiological phenomena [17,81]. The end point of the

muscle stretch can be established by observing increased EMG activity because relaxed muscles may demonstrate increased involuntary stretch-induced muscle activations near their terminal lengths [17,27,30,39–42]. The subject's perception of the end point of stretch that is based on discomfort or pain (their stretch tolerance) is also used, both alone [22,28,30,32,82] and in combination with increased EMG activity [17,27,39,40,42,58]. Accurately identifying the end point of the stretch is important because measuring passive extensibility and viscoelastic properties, and the changes in these properties that result from interventions, depend on accurately defining the end point of maximal muscle length.

As stated earlier, passive elastic stiffness can be represented by the ratio of change in passive resistance to change in passive length ($\Delta F/\Delta L$), and passive compliance can be represented by its reciprocal ($\Delta L/\Delta F$). To arrive at these direct measurements requires invasive research methods that are not usually possible with humans. Instead, passive elastic stiffness in human muscles can be represented by the ratio of the change in passive torque (ΔT [N m]) to a change in size of the joint angle (ΔA [°]), ($\Delta T/\Delta A$) [17,27], or by using other comparable units of resistance (i.e., stress in N m/cm²) and angular change (i.e., radians) [18]. Passive compliance in humans is measured by the reciprocal ratio of the change in the size of the joint angle to the change in the amount of passive torque ($\Delta A/\Delta T$) [39,40,83,84].

5.2. Immediate effects of stretching human muscles

The preponderance of studies with human muscles have used the maximal joint range of motion to represent a measure of passive muscle length and passive extensibility. Many of these studies have focused on two-joint muscles because they can be stretched to their maximal psychophysiological length without bony limitations. The hamstring muscle group is a two-joint muscle group that crosses the hip and the knee, and the literature abounds with clinical studies targeting this muscle group. The results of these studies provide good insight into the immediate and longer-term length responses of these human muscles to clinical muscle lengthening interventions. The investigations with the hamstring muscles have primarily employed two different tests that used maximal joint range of motion as the dependent variable. These tests include: (1) the passive unilateral straight-leg-raising test [85–97], and (2) variations of active and passive knee extension with the hip held in flexion [18,39,41,97–101].

The passive straight-leg-raising test represents hamstring muscle length by the angle of hip flexion with the knee held in extension. It is considered an indirect test for hamstring muscle length because the pelvis has been shown to move during the test [90,92,102,103], and

because maximal hip flexion range of motion may be limited by structures other than the hamstring muscles, such as the deep fascia of the lower limb and neurological tissue [41,98]. Within these potential limitations, however, comparisons of the straight-leg-raising test with more selective tests for hamstring length have indicated that the test probably provides a clinically valid indication of hamstring muscle length [39–41], particularly if the angle of the thigh in relation to the pelvis is isolated [90,92,95,102,103].

Because of the indirect nature of the passive straight-leg-raising test, hamstring muscle length has also been represented by the angle of knee flexion after active knee extension [97,98,104] or passive knee extension [18,39–41,97,101] with the thigh flexed. Several studies, however, have emphasized the importance of ensuring that the pelvis is stabilized in order to achieve valid test results, both in a clinical setting [104] and in a controlled research setting [39–41]. If the pelvis is not stabilized, the proximal attachment of the hamstring muscles may move distally and the gluteus maximus muscle and low back extensor muscles also may contribute to the knee flexion angle [104].

Numerous stretching studies employing these tests have documented increases in the maximal joint range of motion, and presumably hamstring muscle length, immediately after stretching exercises [29,90,91,96,99,100]. Both static (constant) stretching procedures [29,90,91,96,99,100] and proprioceptive neuromuscular facilitation (PNF) techniques [96,99,100] increased maximal joint range of motion. It is interesting to note that PNF techniques have been shown to be more effective even though they caused increased EMG activation of the hamstring muscles compared to static stretching [87,99,100]. Further studies are indicated to examine these differences. The immediate increases in joint range of motion from stretching may have resulted from a lengthening ‘creep’ response that is well known to occur in most biological tissues [20], and that this lengthening creep is probably independent of low level EMG activity in the hamstring muscles.

In addition to measuring the maximal joint range of motion to indicate hamstring muscle length, several studies have attempted to measure their length extensibility and viscoelastic properties. With the subjects side lying, controlled testing procedures of passive knee extension with the pelvis stabilized showed that the absolute hamstring passive compliance ($\Delta A/\Delta T$) during dynamic stretching for men and women with similar straight-leg-raising angles was less for men (stiffer) ($n = 15$) than for women ($n = 15$) [39]. No difference was found, however, when the passive compliance ratios were controlled for body mass, which indicated that passive compliance and passive stiffness was related to the size and mass of the muscles [39]. Length extensi-

bility, measured by the percent change beyond the initial length and controlled for femur length, also did not differ between genders.

Using the same controlled testing procedures, men with clinically short hamstring muscles (straight-leg-raising $\leq 65^\circ$) ($n = 12$) were shown to have passive curves that were significantly shifted to the left (decreased initial and maximal lengths) with decreased length extensibility compared to men without short hamstring muscles ($65^\circ < \text{straight-leg-raising} < 80^\circ$) ($n = 12$) [40]. The maximal passive torque, however, did not differ significantly between the two groups. A more recent study using passive knee extension in the seated position, indicated that men with clinically short hamstring muscles (identified by the toe-touch test) reached a lower maximal knee extension angle, lower maximal passive torque, and decreased stiffness compared to men without clinically short hamstrings [23]. This study, however, tested subjects sitting and did not ensure pelvic stabilization, so the testing methods did not account for the potential contribution of pelvic movement. Also, lumbar flexion and thoracic flexion range of motion have been shown to influence the toe-touch test [105]. Different subject selection criteria and different testing methods [23,40], could possibly account for the differences reported in these studies. Future studies would need to standardize objective testing methods to allow for more accurate comparisons.

Studies have also reported the immediate effects of stretching on the dynamic and static viscoelastic properties of the hamstring muscles. Within the limitations of using the testing protocol of passive knee extension in the seated position, the hamstring muscles have been reported to show decreasing dynamic resistance to stretch over repeated stretching trials and decreasing static resistance over time (90 s) when the muscles were held in a lengthened position [21,22]. Decline in the passive resistance during a static stretch while the hamstrings were held in a lengthened position was considered a measure of the muscle’s viscoelastic stress relaxation because the decline was not influenced by the presence of low level EMG activity [22,33].

Immediate increases in maximal hamstring length and maximal passive torque have been associated with viscoelastic stress relaxation without changes in EMG activity [29,33]. As a result, concomitant increases in hamstring length and maximal passive torque after short-term stretching have been attributed to immediate increases in subjects’ tolerance to the stretch, without a change in the muscles’ passive viscoelastic properties. Increased muscle length and increased maximal passive torque, however, may result from muscle lengthening creep (strain) in relation to increased lengthening tension (stress). Again, this appears to happen independent of low level EMG activity.

5.3. Long-term effects of stretching human muscles

Studies have also shown that both static stretching [41,95,101] and PNF techniques [89,93] for the hamstring muscles increase range of motion over time (ranging from 3 to 10 weeks of stretching). Thus, the cumulative effects of a stretching regimen appear to lead to more permanent adaptations in hamstring muscle length and extensibility. Similar to the results of immediate stretching exercises, PNF techniques were more effective than static stretching [89] or ballistic stretching [89,93], but an explanation remains wanting and worthy of future study.

As with the results of immediate stretching, some researchers have reported gains in hamstring muscle length and maximal passive resistance from stretching over time without a change in their viscoelastic stress relaxation [25,82]. Accordingly, increased subject tolerance to stretch has been suggested as the proposed mechanism of this adaptation, not changes in the mechanical properties of the muscles [25,82]. These reports, however, did not acknowledge the possibility that anatomic and physiologic length adaptations within the muscles could have increased their functional lengths in the presence of EMG activity, and thus allow for the appearance of increased subjects' tolerance as the primary explanation. Increased sarcomere addition in lengthened animal muscles can occur independent of the level of muscle activation [77]. The increased hamstring muscle length could have resulted from similar changes, and this would permit the muscles to be stretched farther and have greater resistance to the stretch prior to a stretch induced muscle activation [41] or before the stretch was stopped by the subjects [25,82]. The passive length and extensibility adaptations could have occurred in the absence of changes in the viscoelastic properties measured within the muscles' original length, but additional studies would need to be conducted to verify this possibility. Even so, these studies have indicated that long-term stretching exercises increase the hamstring muscles' functional length extensibility, without changing their viscoelastic stress relaxation properties. It should be noted that these studies were over relatively short time periods (<10 weeks). Longitudinal studies are needed to examine the effects of longer-term stretching regimens (>10 weeks) on the length extensibility and viscoelastic properties of normal human muscles.

5.4. Effects of lengthening interventions on neurologically impaired muscle

Length adaptations in muscles affected by altered neurological activity have supported the notion that passive length extensibility adaptations in humans may be independent of the level of neurological activation.

Studies of children with cerebral palsy and hypoextensible calf muscles showed that they have muscle shortening and increased passive elastic stiffness compared with findings in children with typical development [83]. In a different study, nine children with hypoextensible calf muscles were casted for 3 weeks with these muscles placed in the lengthened position [106]. Four children showed passive curves that were shifted to the right with decreased slopes, indicating longer muscles with decreased passive stiffness, whereas five children had passive curves that were shifted to the right without a change in the slopes of their passive curves. In other words, similar changes in the muscle lengths were observed in both groups, without similar changes in passive elastic stiffness. In the same study, the ankles of five children with hyperextensible calf muscles were casted in shortened positions. The passive curves shifted to the left with increased slopes, indicating decreased length and increased passive stiffness, in four of the five children.

Prolonged passive stretching of muscles in a state of severe contracture from long-term hypertonicity and shortening also may promote lengthening adaptations and increased range of motion in children with cerebral palsy [107]. Therapeutic stretching of hypoextensible calf muscles of adult hemiparetic stroke patients has been shown to increase dorsiflexion range of motion [108].

As stated earlier, the observed lengthening changes in neurologically impaired muscle may result from changes in the muscle directly, and not from changes in neurological excitability. The enhanced strength and function of the antagonist muscle group after surgical lengthening of the agonist muscle groups [109,110] supports the hypothesis that functional changes result from direct changes in muscle length, not from changes in motoneuron excitability. This proposal was also supported by the finding that splinting spastic muscles of patients with brain damage changed range of motion without altering the integrated EMG activity of the muscles when compared with the activity in muscles that were not splinted [111]. A more recent study examined the effects of three weeks of dorsiflexion casting on the reflex characteristics of spastic calf muscles of children with cerebral palsy [112]. They reported that the casting brought about increased dorsiflexion range of motion and that the angle of reflex excitability elicited by a rapid dorsiflexion stretch also was shifted toward increased dorsiflexion. The soleus and tibialis anterior coactivation EMG tracings, however, did not change as a result of the casting.

The notion that the length of muscles can be influenced by clinical interventions without influencing the underlying neurological excitability of human muscles, is supported by the results of these studies. As stated earlier, increased functional length and maximal resis-

tance to stretch have been documented after regimens of passive stretching of unimpaired, normal muscles [25,41,82] without significant changes in the amount of EMG activity [25,82]. It seems reasonable that neurologically impaired muscles would also show passive length extensibility adaptations to imposed positional changes in muscle length, although the adaptations will be more difficult to achieve than for non-neurologically impaired muscles.

5.5. Effects of aging on passive extensibility characteristics

Aging studies have indicated that a normal muscle's mass, strength, length and passive elastic stiffness are positively related. For example, the strength of the calf muscles in humans is known to decline with aging [42,113–116], probably brought about by a loss of functional motor units [117–120], and a decrease in the number [120–122] and size [120–124] of both slow twitch (type I) and fast twitch (type II) muscle fibers. Aging is also well known to be associated with decreased dorsiflexion range of motion [17,27,42,125,126], generally believed to be caused by decreased calf muscle length.

The results of a recent study in our laboratory showed that active older women (60–84 yr; $n = 33$) had significantly weaker concentric strength of the calf muscles than younger women (20–39 yr; $n = 24$) and middle aged women (40–59 yr; $n = 24$) [58]. The older women also had significantly decreased active and passive dorsiflexion range of motion, decreased passive length extensibility, decreased maximal passive resistive torque, and decreased passive elastic stiffness within the last half of their available, tolerated stretch range of motion [17]. The shape of the passive curves within the stretch range of motion that was common among the three groups appeared similar (Fig. 3) and the passive elastic stiffness did not differ among the three groups within this common range, a finding reported previously [127]. The complete curves for the middle-aged and older women, however, appeared truncated in relation to the curves for the younger women because the middle-aged and older women had less dorsiflexion range of motion and less passive resistive torque at their maximal tolerated functional limit (Fig. 4) [17]. In other words, the older women appeared to have less maximal muscle length and less maximal passive resistive torque, and this resulted in less passive elastic stiffness within their tolerated, yet decreased available stretch range of motion.

The notion that aged muscles loose passive length extensibility and passive elastic stiffness may conflict with popular clinical opinions that aged muscles are passively stiffer, thought to be cause by increased amounts of connective tissue. The muscles of older people have been shown to have increased fat and

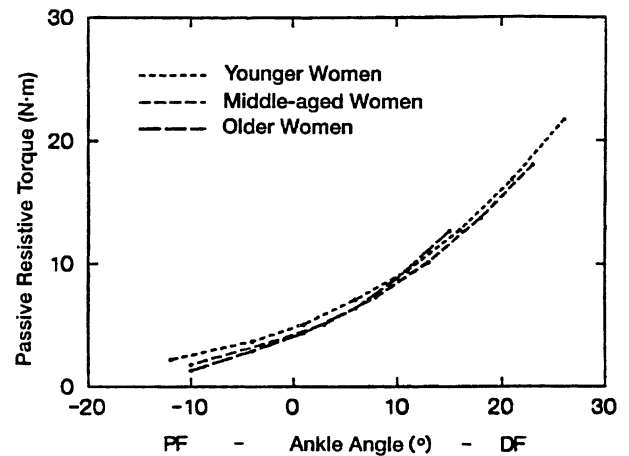


Fig. 3. Passive curves for calf muscles of three age groups of active women superimposed to illustrate their similarities within their common ranges of passive extensibility. PF = plantar flexion, DF = dorsiflexion ([17, p. 835], reprinted with permission from The American Physical Therapy Association).

connective tissue [120,128–130], but the relative contributions of increased fat compared to increased connective tissue have not been related to changes in passive extensibility, maximal passive resistive torque or passive elastic stiffness. Increased passive elastic stiffness was not observed in our study [17], which suggested that if lost muscle mass was replaced by fat and connective tissue, the amount of fat and connective tissue was probably insufficient to counteract the lost muscle mass; this would be necessary to increase the passive elastic stiffness. Furthermore, animal studies have indicated that the apparent connective tissue accumulation that may occur in inactive muscles can be prevented by active stimulation [73], and that exercise has been shown to prevent connective tissue accumulation of aging muscles [131]. A minimal level of physical activity may prevent the accumulation of connective tissue in the aged muscles of active people. The passive elastic stiffness of very inactive, sedentary people may be different from the active women we tested. Moreover, other passive muscle properties that were not measured in our study [17] may change with aging. Another recent study in our laboratory showed that the calf muscles of older men did not demonstrate a robust velocity dependent increased passive resistance to rapid stretches like the calf muscles of younger men [27]. The application of this method could offer additional insight into the influence of aging on the viscoelastic properties of muscles. Additional research is needed to examine this possibility, as well as how the muscles of older people respond to long-term muscle stretching regimens. The literature appears deficient on this topic.

Studying the passive extensibility characteristics of the calf muscles of subjects from across the life span has offered additional insight into the passive extensibility of

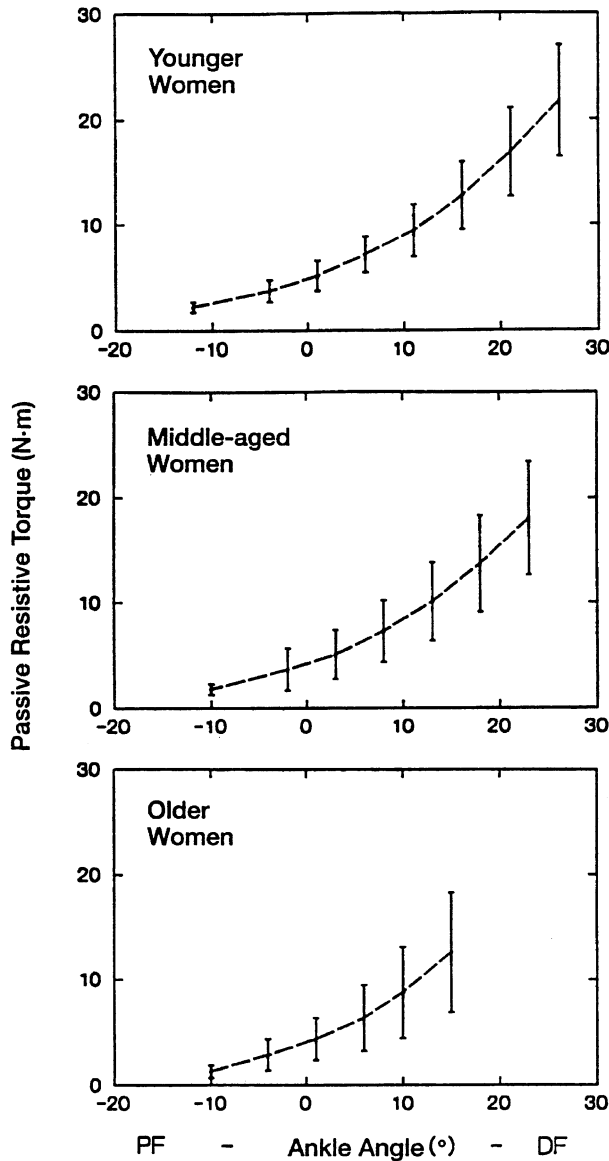


Fig. 4. Passive curves (\pm S.D.) for the calf muscles through the full, defined stretch range of motion for the younger women ($n = 24$), middle aged women ($n = 24$) and older women ($n = 33$). The curves for the older and middle-aged women were truncated because of decreased maximal muscle lengths and decreased maximal passive torques. PF = plantar flexion, DF = dorsiflexion ([17, p. 833], reprinted with permission from The American Physical Therapy Association).

human muscles. Based on the limited evidence available, it seems reasonable to suggest that stronger and longer muscles are associated with increased passive elastic stiffness within the subjects' available and tolerated stretch range of motion. Isometric strengthening exercises have been shown to increase the maximal passive torque and passive elastic stiffness of the hamstring muscles without affecting their viscoelastic stress relaxation [24]. It makes sense, therefore, that clinical interventions should be designed to achieve stronger muscles within a maximally tolerated stretch range of motion in

order to achieve optimal muscle function. Because the mass of skeletal muscles is positively related to strength and increased passive elastic stiffness, a larger and stronger muscle is also a passively stiffer muscle. It should be emphasized that this positive relationship between strength and passive extensibility properties was based on an aging model with active women [17]. The relationships may be different in patients with clinical disorders, disease or injury.

Therapeutic stretching, and other regimens of muscle lengthening, appear to increase the muscle's functional length, its ability to withstand a passive load, and its passive elastic stiffness. This could partially explain why stretching exercises are believed to help prevent muscle strains in athletic performances. Clearly, more research is needed to examine the relationship of the effects of muscle lengthening and muscle strengthening interventions on a wide range of clinical conditions for people from throughout the life span. It should be noted that the majority of the studies in the literature have focused on the response of normal, healthy muscles of non-disabled people. Human muscles from people of all ages that are affected by clinical disorders, disease or injury, either directly or indirectly, may respond differently. Future studies are needed to study the passive extensibility characteristics of a wide variety of people in order to identify the most appropriate applications of current interventions, and to develop new strategies that promote the most favorable functional outcomes and athletic performances.

6. Conclusions

Basic studies with animal muscles have shown that the passive extensibility of muscles is influenced by the size and length of muscle fibers (series elastic components) and by the amount and arrangement of connective tissues (parallel elastic components) of the muscle belly. Resistance to passive muscle lengthening is influenced by the amount of contractile muscle proteins, non-contractile muscle proteins, and extracellular connective tissues that readily adapt to imposed load and length demands. The interrelationship of these structures and how they contribute to passive extensibility characteristics and adaptations remains unclear. Muscle length adaptations in animal muscles result from changes in the numbers of sarcomeres in series, which has not been confirmed in human muscles. Animal studies have indicated that passive length adaptations may be independent of, but influenced by the muscles' level of activation, suggesting a myogenic mechanism for length adaptations. Although this mechanism has not been confirmed in human muscles, it has been intimated by non-invasive studies with healthy human muscles and with neurologically impaired human

muscles showing that human muscles can undergo passive extensibility adaptations that also may be independent of their level of activation. Studies have suggested that optimal muscle function is probably enhanced by increasing muscle length, length extensibility, passive elastic stiffness and strength, which are positively related using an aging model. These relationships, however, may be different for patients with different clinical disorders, diseases or injuries. Additional research is needed to examine the relationship of these muscle characteristics and the influence of therapeutic interventions on passive extensibility, particularly for the muscles of older people and for muscles affected by clinical disorders, disease and injury. Studies are needed to develop new, evidence-based intervention strategies that promote optimal passive extensibility that enhances total muscle function that ultimately improves the ability to complete functional activities and excel in athletic performances.

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