YOGA AND BIOMECHANICS: A New View of Stretching Part 2

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Last time, Part 1 of this article, published in the July issue, looked at the interplay between flexibility, stretching and yoga and what type of stretches to do depending on the desired outcome. Part 2 here further discusses some commonly used terms, such as tight/loose, strong/weak and short/long, and elaborates on the biomechanics of what happens to muscles when they are loaded under tension, as well as muscle length and eccentric contraction. This article has been extracted from chapter 2 of the author's book *Yoga and Biomechanics: Stretching Redefined.* Read this article online https://spxj.nl/2ZcRCEZ

In Part 1, we found that although stretching is an example of an activity for improving flexibility, there are several different types of stretching and which is of most benefit depends on the desired outcome. In this article, Part 2, we discuss muscle structure, what tissue adaptations occur when muscles are loaded in tension, and how eccentric contractions can be used to increase muscle length.

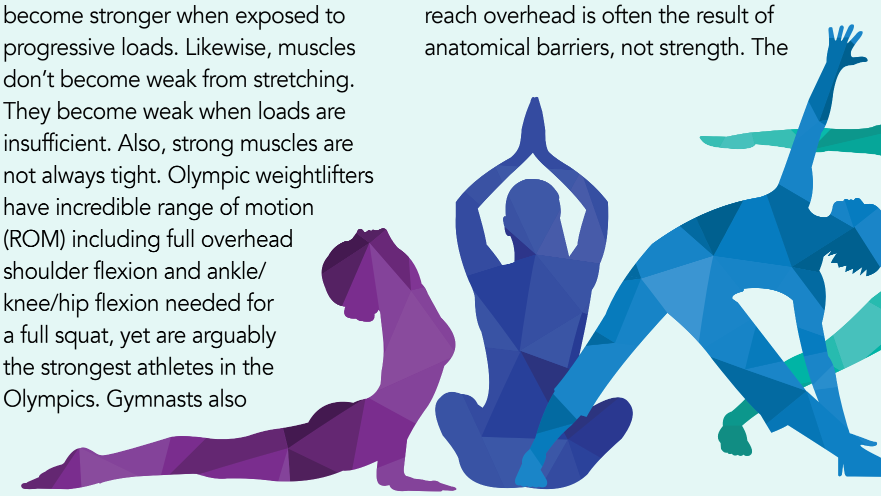
Muscle Length

As muscles are being stretched, they naturally resist deformation. Some muscles seem to resist deformation stubbornly well, while others tend to yield quite easily. We are apt to call those unyielding muscles tight. Colloquially, the definition for tight implies something pulled taut. If tight muscles are wound up like guitar strings, then an additional tension would not result in any further deformation. Alas, muscles are not wound up like guitar strings, and the term tight is derived more from imagery than mechanical behaviour.

We might conclude the resistance to deformation occurs because of a structurally shortened muscle, one that has developed a diminished length of tissue between the two attachment points (ie. origin and insertion). If this were true, then the opposing muscles would have inadvertently been fixed in a lengthened position, and one would expect them to have developed an excessive length of tissue between attachment points. I often hear this regarding the hip flexors. Clients, students, even yoga teachers have all told me they have 'tight' hip flexors and 'weak' glutes, considering this to be the reason why they stand in Mountain Pose with an anteriorly tilted pelvis. Less often, but still frequently, I hear the opposite. They explain to me their hamstrings are 'short' and their hip flexors are 'overstretched'. I'd like to deconstruct these scenarios in terms of muscle length.

The first problematic issue with the binary short/long line of thinking is that it further assumes that shorter muscles must be strong/tight and longer muscles must be weak/loose. Muscles, however, don't get strong by being held in shortened position. They become stronger when exposed to progressive loads. Likewise, muscles don't become weak from stretching. They become weak when loads are insufficient. Also, strong muscles are not always tight. Olympic weightlifters have incredible range of motion (ROM) including full overhead shoulder flexion and ankle/knee/hip flexion needed for a full squat, yet are arguably the strongest athletes in the Olympics. Gymnasts also demonstrate extreme flexibility, but are not lacking in strength and power, defying the presumed long/weak relationship.

Looking at the inverse relationship, the false assumption is that strong muscles adaptively shorten, and weak muscles adaptively lengthen. I tend to blame bodybuilders for the former impression. The primary training goal of a bodybuilder is aesthetic, to put on muscle mass, and bulk up, not to get stronger. The training methods for building muscle to those extremes are very specific, and while they do get stronger, strength training methods are somewhat different. Therefore, the classic image of a bodybuilder who can't straighten her arms or reach overhead is often the result of anatomical barriers, not strength. The



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●● THERE IS A FALSE ASSUMPTION THAT STRONG MUSCLES ADAPTIVELY SHORTEN, AND WEAK MUSCLES ADAPTIVELY LENGTHEN ●●

latter impression, that weaker muscles adaptively lengthen cannot possibly be true or a sedentary desk job would result in the longest muscles in the office!

The second concern with this assertion is the widespread acceptance that stretching and/or exercise corrects postural deviations. It is an attractive concept to embrace, partly because of its simplicity and partly because of what we have all witnessed, but biology is rarely simple, and assessing a student in Mountain Pose, identifying muscles as either short or long, and assigning stretching or strengthening exercises to correct them is the epitome of simple. Perhaps, several decades ago, when we knew far less, the theory served us well, but today, the evidence forces us to reconsider (1*,2*,3). One of my friends and colleagues who also leads teacher trainings attended a course of mine where we elaborated on this idea. She shared with us that on day one of her trainings, everyone takes a photo of themselves in Mountain Pose. Then, months later, at the end of the training, they take a second photo for comparison, and share with the group the change they see in themselves. As would be expected, everyone is standing 'taller,' yet they *always* attribute it to psychological factors such as confidence, joy, and fulfilment. It is far easier to assume that the yoga stretched out a short tissue when you are looking at a static photo of a body without the context of human experience. There is no denying we have all witnessed such personal case studies where yoga has improved posture, but we must ask ourselves how much is attributable to actual muscle shortages or surpluses.

I can continue to deconstruct these postulations about muscle length, but I find it difficult to do so without falling into a logical fallacy trap. In part, this is because I don't really understand the logic behind these assumptions, which have been only explained to me through unsupported conclusions, based in weak reasoning and anecdotal evidence. These conjectures are difficult to refute with evidence when the theories have not, thus far, been validated. Recall with whom the burden of proof lies; she who is making the claim should provide the evidence. It is difficult to refute a claim that Shoulderstand is the 'panacea to most common ailments' (4) when no credible evidence is provided to support the claim. Additionally, many are invested in the long/short line of thinking and have been using language in their teaching to support it. It's never easy to change beliefs, even when evidence to the contrary is compelling. In the spirit of critical thinking, as you continue to read, I ask you to consider any number of other possible mechanisms that might contribute to the experience of muscle or joint tightness, 'faulty' posture, or other states of being we seem to want stretching to 'correct'.

Muscle Structure

To further explore the topic of muscle length, it will be useful to define a few more terms. It is often argued

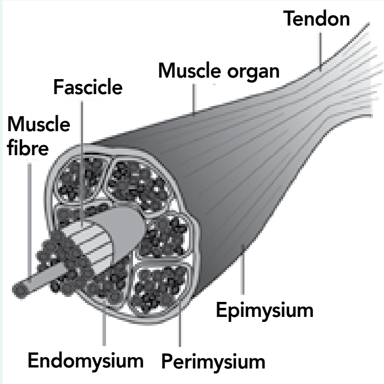
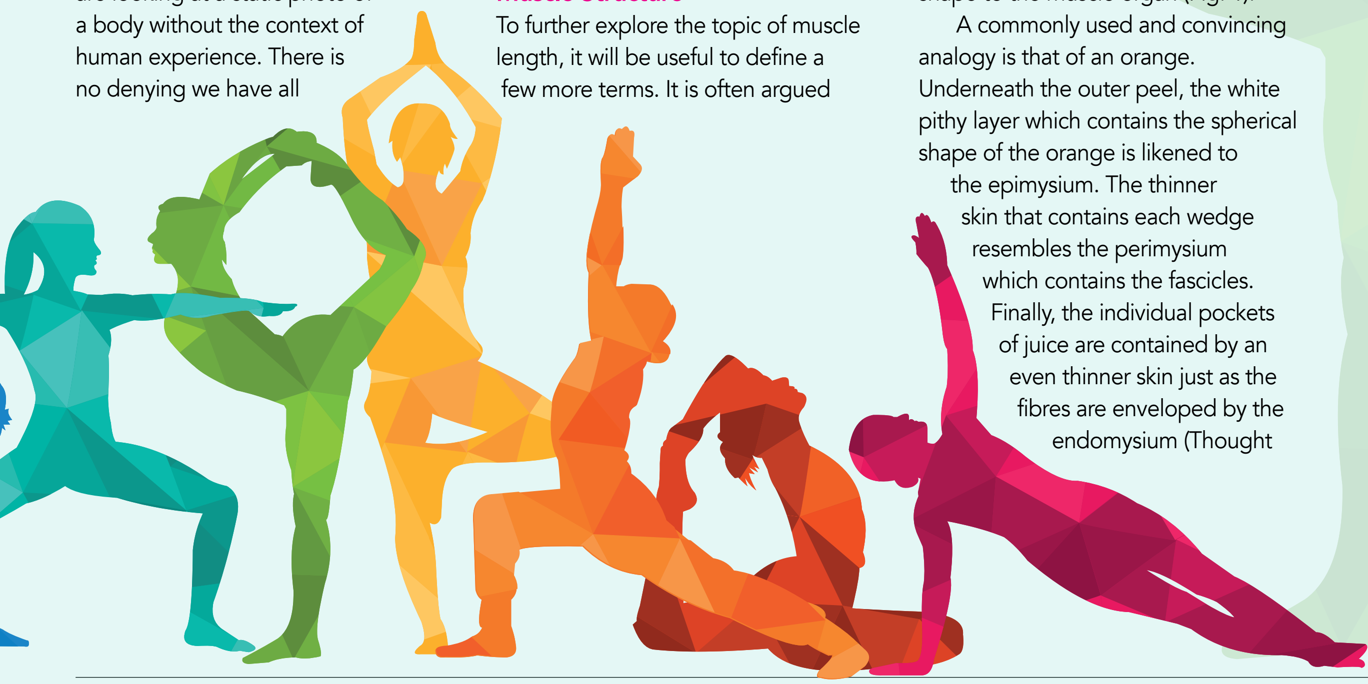


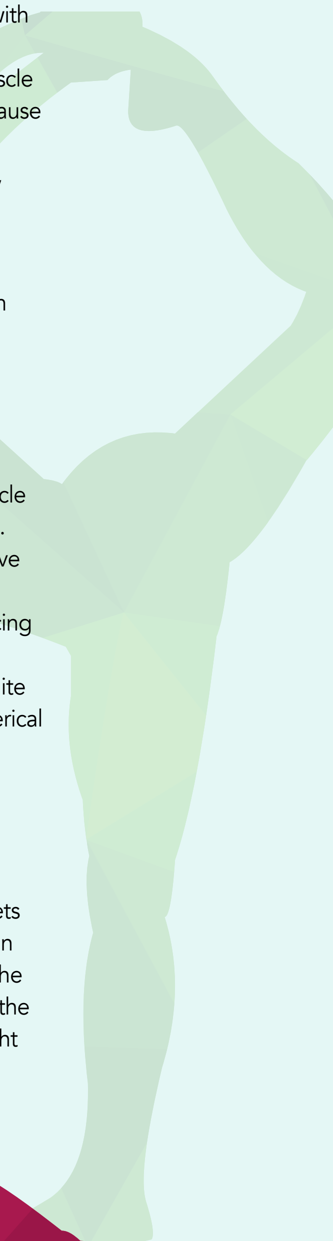
Figure 1: Muscle structure depicting the deep to superficial endomysium, perimysium, and epimysium

that stretching muscle will make it longer. We have already explained that stretching a muscle will decrease resistance torque and will improve tolerance for the stretch, but what are the components of muscle that might adaptively lengthen when loaded under tension? Let us next begin with the study of muscle structure.

Skeletal muscle is made of muscle cells, also called *muscle fibres* because of their long threadlike structure, and surrounding connective tissue, called the myofascia. Three layers of connective tissue help define the shape of the muscle body. At the deepest layer, the endomysium envelops each individual fibre. Bundled together, these fibres form *fascicles*, encased by the next connective tissue layer, the perimysium. Finally, these fascicles bundled together to form the muscle organ, enclosed by the epimysium. These fascial layers contour and give shape to the muscle organ (Fig. 1).

A commonly used and convincing analogy is that of an orange. Underneath the outer peel, the white pithy layer which contains the spherical shape of the orange is likened to the epimysium. The thinner skin that contains each wedge resembles the perimysium which contains the fascicles. Finally, the individual pockets of juice are contained by an even thinner skin just as the fibres are enveloped by the endomysium (Thought





Thought Provoker 1: Stretching Resistance

If you were to try to stretch an orange wedge, would the resistance you have to overcome come from the juice inside or from the surrounding connective tissue, or both?

If applied to muscle, when you stretch a muscle, is the resistance coming from the bags of connective tissue, or the proteins within, or both?

Table 1: Muscle contraction types

Contraction type	Change in length
Concentric	Sarcomeres shorten
Isometric	Sarcomeres do not change length
Eccentric	Sarcomeres lengthen

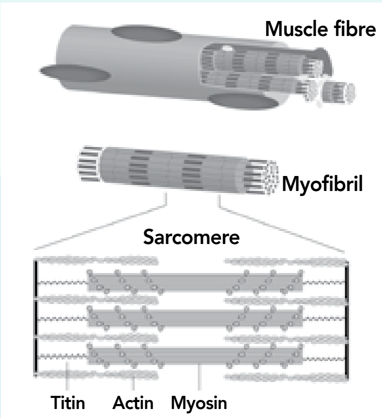


Figure 2: Muscle structure depicting the macro to micro fibre, myofibril, and myofilaments (actin, myosin, titin)

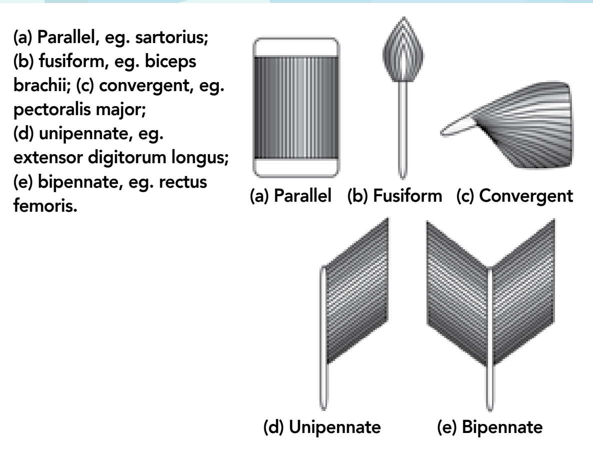


Figure 3: Some examples of different types of muscle morphology

Provoker 1).

The muscle fibre itself is made up of smaller myofibrils, which are made up of myofilaments (Fig. 2). These filaments are made up of proteins organised into *sarcomeres*. Sarcomeres are known to be the smallest contractile functional unit of a muscle, although some muscle physiologists have recently proposed that it might be the half-sarcomere. Sarcomeres are arranged in series, end to end, to form the long fibrils. Sarcomeres are only a few microns (one millionth of a metre) in length – for perspective, the width of a strand of human hair is about 25 times wider than a sarcomere is long!

The proteins within the contractile sarcomere (*actin*, *myosin*, and *titin*) interact together to generate muscle force. According to the Sliding Filament Theory, actin and myosin link together to form cross-bridges. Through consecutive linking and unlinking, cross-bridge formations pull the actin toward the centrally located myosin to create a concentric contraction. During an isometric contraction, the cross-bridges still generate a force, but the sarcomere lengths remain the same. Finally, during an eccentric contraction, an opposing force greater than the force generated by muscle pulls the actin away from the centre, lengthening the sarcomeres (Table 1). When the sarcomere is actively stretched (possibly even beyond cross-bridge formation), titin interacts with calcium and maybe even actin to contribute to force production (5*).

In recent years, the role of titin has shed light onto the previously uncertain and unexplainable mechanisms of eccentric contractions. At the risk of veering off topic, the significance for investigating eccentric contractions here is to grasp that muscles do generate force in the absence of cross-bridge formations. Although, since the introduction of the Sliding Filament Theory in the 1950s, concentric contractions have been explained thoroughly, lengthening contractions have not been so well understood. Perhaps it is this uncertainty which led to the speculation that lengthening a muscle is a factor of relaxing a muscle and that fewer cross-bridges would make for less resistance to the stretch. The re-evaluation of titin's role (it was originally considered a passive element, holding the sarcomere together when stretched beyond the actin–myosin overlap) has advanced our understanding of muscle force production from a two filament model (actin and myosin) to a three filament model (5*).

Admittedly, these last few paragraphs provide only a gross oversimplification of muscle physiology. Additionally, the Three Filament Model, at the time of this writing, is just a proposed possible mechanism for eccentric contractions by a select group of researchers – the theory is still new, somewhat controversial, and has yet to make its way into kinesiology textbooks. As this is not a muscle physiology textbook, I believe it is okay to offer new perspectives, as long as I am transparent about their overall position in the scientific community, and I offer this perspective here in an effort to define terms, which, as I've argued, is essential to clear communication. Simplified, sarcomeres are contractile functional units that produce force via actin–myosin binding at shorter lengths and actin–titin binding at longer lengths (5*).

Muscle Morphology and Force Generation

Now that we understand the structural components of muscle we can study how they are arranged. Muscle morphology explains how muscle fascicles are positioned in relation to the tendon (Fig. 3). Some run parallel to the tendon (at a 0° angle) and some run at a pennation angle. In parallel muscles, 100% of the longitudinally transmitted force transfers to the tendon. In pennate muscles (Fig. 4), if the pennation angle is given, a trigonometric equation can determine the percentage of longitudinal force transferred to the tendon. An example of this force transfer is attempting to move an object horizontally by pushing on it directly from the side versus pushing it at an angle from the top of the object. Both methods can displace the object horizontally, but the amount of force transfer is determined by the angle of application.

Practically speaking, pennate muscles accommodate more muscle fibres into their oblique architecture than parallel muscles, thereby increasing their capacity to produce greater overall force. The amount of force not transferred to the tendon is minimal compared to the amount of total force gained by the pennate morphology. Most muscles are morphologically pennate, the significance regarding muscle length being that most muscle fibres are obliquely oriented. An increase in muscle fibre length, therefore, is not always a purely longitudinal growth with respect to attachments points. If the pennation angle also increases, the additional length is even further away from the longitudinal axis relative to the tendon (and attachment points). Ultimately, the amount of additional muscle tissue length between the two tendon–bone attachments is geometrically determined.

In this regard, muscle length is defined by the length of the muscle fascicles. If we expanded our definition of 'muscle' to include the entirety of the muscle organ, which includes the connective tissue layers that merge to become a tendon where the ends of muscle fibres do not reach, we end up with a slightly different interpretation of muscle length. Using the orange analogy, the former definition only accounts for the juice inside the bulbs, whereas the latter accounts for the surrounding materials as well.

Tissue Adaptation

Interestingly, during development, if a limb is immobilised, it will present with a greater proportion of tendon length than mobilised limbs (6). In other words, when muscle fibres are not growing longer, connective tissue replaces it. Again, using the orange analogy, if a bulb were to lose some juice at one end, the space would be filled with extra enveloping material. The envelope itself would not become architecturally smaller, only the proportion of outer material to inner material. Is it possible then that when muscle lengthens, the fibres fill more length of the connective tissue envelope rather than expanding the entirety of the envelope?

I've been insinuating that muscle proliferates to meet rising demand. Hypertrophy, the enlargement of muscle fibres, is the muscle growth that occurs when we load muscles (ie. lifting weights). *Sarcomerogenesis*, the addition of sarcomeres in series, occurs when we load muscles eccentrically. *Sarcomerolysis*, the removal of sarcomeres in series, occurs when we load muscles concentrically (7*). Unsurprisingly, muscle architecture adaptations are load dependent.

Sarcomerogenesis is regulated by chronic stretch. As bones grow longer during development (provided immobilisation is not a condition, as noted above), the growth spurt is met with sarcomerogenesis, meeting the movement demands of the child for optimum function (8). Other examples of chronic stretch include surgical limb lengthening and casting to specific joint angles (9*). Conversely, during deconditioning or immobilisation, sarcomeres are lost, and excess connective tissue accumulates (10*). In spite of what you may read online, passive stretching alone may not be enough to preserve, or increase, sarcomeres.

Biomechanics tells us load matters. Muscle contractions, of any type, are needed to regulate sarcomere quantity, presumably because of their energetic expense. Contractile tissue is expensive to operate – it requires an energy source to function. Muscles burn calories. In the evolution of human existence, having enough calories to survive is a modern luxury enjoyed by the privileged. The human body has evolved to conserve energy and would likely not preserve unused tissues with a high energy cost.

A passive stretch is a tensile load indeed, but when muscle contractions are paired with stretching, the load parameters are more effective in regulating sarcomere production and loss (11). A recent meta-analysis concluded that stretch training alone produced 'trivial' changes in fascicle length and angle, reinforcing previous statements I've made (12). We know stretching does influence muscle architecture, but not as much as it may seem.

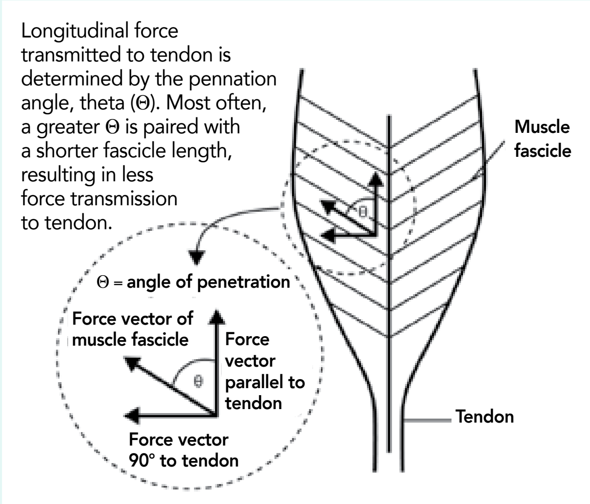


Figure 4: Muscle architecture of a bipennate muscle

Muscle Architecture and Mobility Versus Flexibility

Arguably, muscle architecture plays a greater role in mobility than flexibility since muscles produce the internal force to create movement. If flexibility is measured passively, mobility is measured by how someone performs through a ROM. It has been shown that increased passive ROM does not transfer to twist and reach activities or improvements on an elliptical machine (ie. functional tasks) (13*). What, then, is the benefit of improved flexibility without training in these new ranges? At surface level, it is easy to accept that ROM is limited by an insufficient length of tissue (ie. short muscles). It is also easy to explain without a detailed conversation about muscle architecture and physiology or differences between flexibility and mobility. When these factors are taken into consideration, however, it challenges the idea that short muscles are the sole cause of limited flexibility.

Whereas a number of studies do show improvements in ROM simultaneous to increases in fascicle

IT MAY BE HELPFUL TO THINK OF CONCENTRIC CONTRACTIONS AS ACCELERATORS AND ECCENTRIC CONTRACTIONS AS DECELERATORS

SARCOMEROGENESIS, THE ADDITION OF SARCOMERES IN SERIES, OCCURS WHEN WE LOAD MUSCLES ECCENTRICALLYS

length, many do not. Muscle extensibility (think 'stretchability' of the tissue) has been shown to increase 13% after a stretch training intervention with no changes in fascicle length or amount of tendon deformation (14*). In other words, the muscle portion of the entire organ (inclusive of the enveloping connective tissue layers) resisted the stretch less, while the tendon portion did not become more pliable. Unless the definition of muscle length as an architectural adaptation is clarified, increased pliability could easily be falsely interpreted as a sarcomeric muscle lengthening.

Moreover, increased ROM has been shown to be altered by many activities other than stretching. In addition to the effects of anaesthesia and tolerance on ROM discussed in Part 1, core endurance activities (15*), breathing techniques (16*), somatic practices (17*), and foam rolling (18*) have all improved ROM. Improvements have also been observed after interventions involving joints other than the target joint. Foam rolling one limb has increased ROM in the contralateral limb (19*). Static stretching of the upper limb has produced significant changes in lower limb ROM and vice versa (20). None of these activities are associated with muscle lengthening by any mechanical definitions. If adaptive tissue lengthening is not the driving factor, we are therefore left to consider other mechanisms of action.

Biomechanics, how the human body responds to and adapts to force, is the main topic here. If you recall the different types of stretching discussed, those that involve force production of the target muscles are those that include isometric and eccentric contractions. Although conventionally not discussed in terms of stretching, eccentric contractions including resisted stretches, body-weight exercises, and loaded training are all worthy of reviewing in our expanded view of stretching as a tissue under tension.

Thought Provoker 2: Muscle Relationships

In Standing Forward Bend Pose, an instruction we often hear is 'contract your quadriceps to relax your hamstrings'. In actuality, on the way into the forward bend from Mountain Pose, as in the beginning of a Sun Salutation, the hamstrings are eccentrically lengthening to control the descent. While the pose is held, the hamstrings are isometrically contracting to counterbalance the load of the trunk and prevent falling forward. What does the reciprocal relationship in the instruction inaccurately imply about how muscles work? Is there a cue you could use instead that would emphasise control of the movement rather than individual muscles?

Eccentric Contractions

Eccentric contractions are lengthening contractions against a greater opposing load. Muscles cannot willingly lengthen. When a muscle contracts, an electrical signal from the nervous system (an impulse) travels along a neuron to the muscle. At the neuromuscular junction, where the neuron meets the muscle cell membrane, the impulse prompts an electrochemical and then a mechanical sequence of events leading to cross-bridge formations. Skeletal muscle can only voluntarily concentrically contract. In order for an isometric contraction to occur, opposing forces must be equal to the force produced by the contracting muscle. In eccentric contractions, opposing forces are greater. The opposing force could come from an external load, like a free weight, or from the internal load produced by surrounding muscles.

When first learning about muscle actions, the process is often described in a binary relationship, contracting, and relaxing. For example, you may have learned that to flex the elbow, the biceps brachii concentrically contract and the triceps brachii relax. It is quite the contrary, however, because if the triceps were relaxed, the elbow would flex rapidly and clumsily without a decelerating mechanism. The triceps eccentrically contract and contribute to the ideal combination of forces necessary to achieve the given movement. It may be helpful to think of concentric contractions as accelerators and eccentric contractions as decelerators. This model of opposing actions is still a reductionist view of elbow flexion. Such a local agonist and antagonist relationship around a single joint ignores the global contractile contributions of other muscles, both proximally and distally, needed to achieve any given movement. Muscles work individually and collectively as motors, brakes, springs, and struts – they work in far more complex patterns than our experimental models have accounted for in controlled laboratory settings (21). Putting aside a detailed exploration of muscle mechanics, for it is somewhat off topic, we will keep our focus on the effects of eccentric training on muscle architecture.

The length–tension relationship of muscle describes the amount of force a muscle can produce at different lengths (Fig. 5). Muscles are generally weaker at very short and extreme long lengths and strongest in mid-range. Additionally, we are somewhat stronger eccentrically than concentrically. Graphically depicted, the length–tension curve represents the ideal length for optimum force production. In response to eccentric training, that curve shifts to the right (22). Eccentric training improves force production capabilities at longer muscle lengths, which may be useful in activities that utilise longer end ranges (ie. require force production at long muscle lengths) such as gymnastics, martial arts, and potentially, yoga (Thought Provoker 2).

Sarcomerogenesis is one of the proposed mechanisms by which the length–tension relationship shifts. It has recently been shown that fascicle length increases after a 10-week eccentric intervention using the high-magnitude body-weight exercise, the Nordic Hamstring Curl (Fig. 6) (23). Body-weight eccentric exercises resembling many common yoga pose transitions, however, have been shown to not provide a great enough load magnitude to shift the curve (24*). These body-weight exercises, some of which resemble Mountain Pose to Warrior III Pose (Fig. 7), or gliding into and out of Hanuman's Pose (Forward Splits) (Fig. 8), were effective as overall strengthening exercises and, in another study, these same exercises did reduce the time to return to sport when compared with conventional exercise (25*). The outcomes of eccentric exercises including sarcomerogenesis appear to be load dependent as well.

That is not to suggest that eccentric exercise is the only way to improve end range force production or alter muscle architecture. Training at long muscle lengths (26*) and isometric training of a muscle in a lengthened position has been shown to increase fascicle length (27*), replicating the effects of eccentric training. In any case, the over-arching theme is that load parameters matter. It appears that contraction type (concentric, isometric, eccentric) is less important than specificity and intensity on causing changes in muscle architecture – a concept that should not be surprising at this point. Of the types of stretching previously discussed, proprioceptive neuromuscular facilitation and resistance stretching utilise contractions. Based on the above evidence, whether the intensity is high enough to promote any substantial muscle remodelling is still doubtful.

Regarding yoga, we know training at long range improves long-range performance, providing load parameters are sufficient. In the absence of any research examining muscle architecture and yoga asana, we are left to draw parallels using the available sport science research. If eccentric loading associated with common yoga transitions potentially improves strength but falls short of shifting the length–tension curve to the right, isometric training at end range may satisfy that specific adaptation. It certainly builds a case for holding postures while cueing to co-contract the ostensibly binary/opposing muscle groups to incite a high-magnitude isometric contraction ('hug muscles to the bone', for example).

Co-contraction can also be used to develop an internal resistance during transitions in asana. If eccentric contractions are the decelerators, these lengthening contractions can be emphasised by 'putting on the brakes.' Imagine you were lowering from Plank Pose all the way to the floor. The path of least resistance would be to go quickly, letting gravity take you down. But if you were to lower slowly, you would have to create some internal resistance to modulate the pull of gravity. You could further develop this internal resistance if you were to try to push your way back up into Plank Pose while simultaneously trying to pull your way to the floor. Naturally, this approach would ensure slow descent, as well. The point here, however, is not to move slowly, but to explore control through a ROM at a higher demand than that which you might be accustomed to during your yoga practice. Stretching (or lengthening) against external resistance can be used with equipment (eg. resistance band or weight), explored with a partner (eg. resistance stretching), and in body-weight activities, like yoga, using co-contraction.

My intent here is not to establish a right way of teaching asana, but rather to recognise how various approaches have different outcomes. Cueing students to relax their hamstrings under the assumption that a passive stretch is the only way to improve flexibility is incomplete. Eccentric training has been shown not only to increase flexibility, but also to increase fascicle length (28). Furthermore, always cueing to relax the hamstrings reinforces outdated concepts about stretching and fails to highlight beneficial principles of progressive loading, specificity, and adaptation.

●● DURING AN ECCENTRIC CONTRACTION, AN OPPOSING FORCE GREATER THAN THE FORCE GENERATED BY MUSCLE PULLS THE ACTIN AWAY FROM THE CENTRE, LENGTHENING THE SARCOMERES ●●

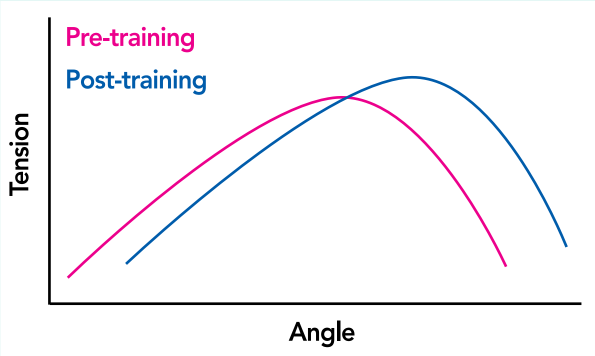


Figure 5: Length–tension curve before and after eccentric training. Force production increases at longer muscle lengths (ie. greater joint angles). [Illustration modified after Brughelli and Cronin (22)]

Figure 6: Nordic hamstring curl

Figure 7: The Diver (resembling Warrior III Pose)

Figure 8: The Glider (resembling Hanuman's Pose)

●● ECCENTRIC TRAINING HAS BEEN SHOWN NOT ONLY TO INCREASE FLEXIBILITY, BUT ALSO TO INCREASE FASCICLE LENGTH ●●

Key Points

- When people discuss their muscles, they often equate short/tight with strong and long/loose with weak, leading to the false assumption that strong muscles become short and weak muscles become long.
- Muscles don't get strong by being held in a shortened position; they become stronger when exposed to progressive loads.
- Muscles don't become weak from stretching; they become weak when loads are insufficient.
- Strong muscles are not always tight – gymnasts have both great flexibility and strength.
- The Sliding Filament Theory of muscle contraction explains concentric contractions well, but lengthening contractions have not been so well understood.
- Simplified, sarcomeres are contractile functional units that produce force via actin–myosin binding at shorter lengths and actin–titin binding at longer lengths.
- Eccentric contractions, including resisted stretches, body-weight exercises, and loaded training, are important in our expanded view of stretching as a tissue under tension.
- Eccentric contractions are lengthening contractions against a greater opposing load.
- Regarding yoga, we know training at long range improves long-range performance, providing load parameters are sufficient. Parallels from sports science research build a case for holding postures while cueing to co-contract the ostensibly binary/opposing muscle groups to incite a high-magnitude isometric contraction.

Equipped with this understanding, you are prepared to make educated choices in how you teach that are outcome specific and population dependent. Instead of debating with other teachers what the right way is, we can now debate about which options we think are best given a desired result.

Stretching Redefined

Some time ago, when I was developing the narrative for this book, I took an informal survey of everyone on my mailing list and asked them 'what is stretching?' I intentionally made the question open-ended, hoping to get answers that weren't led by my own words. The responses varied, of course, but three distinct themes came up: ROM, tissue lengthening, and sensation. When we look at conventional stretching as it is described and studied in the research, these themes are absolutely central to the conversations of flexibility, muscle architecture, and tolerance. It seems we can all agree on some basic concepts, but where we get lost is in the details.

In my informal survey, some answers described stretching as a function of muscle relaxation with an insinuation that a stretch is the opposite of a contraction. Others described stretching as an activity designed to bring 'strength,' 'suppleness,' and 'elongation' to the muscle. I'm hoping you are, at this point, asking what type of stretching would develop strength and how it is measured, while also wondering what exactly 'suppleness' might mean. A clear definition for 'elongation' is also needed (is it deformation, tolerance, or sarcomerogenesis?) before determining the accuracy of that perspective. Some responders separated muscle from connective tissue, implying they can be stretched separately and alluding to the notion that different approaches to yoga target different tissues. In our review of conventional stretching thus far, we have discussed very little about connective tissue outside of the fascial layers providing the structure for the muscle organ. In order to establish how stretching might affect tendons and ligaments, we would first need to define the properties of the tissue, and then the type of stretching, the load parameters, etc. In other words, the details.

I highlight these varied and sometimes contradicting perspectives for you here to explain the importance of coming together to agree on terminology. As the reader, you don't have to agree with my definitions or interpretations of the literature; however, we must at least agree on the words we use so that we can form our opinions knowing we are talking about the same thing. If you are talking about how muscle tissue behaves during a stretch and I'm talking about how the collagen in connective tissue behaves, we will always be tuned to different channels. This reminds me of the John Godfrey Saxe poem of the six blind men and the elephant. Each blind man's position near the elephant influenced how they perceived the animal. One man likened the elephant to a tree stump (feeling the leg). Another argued that the elephant is like rope (being near the tail). While yet another likened the elephant to a spear (feeling the tusk). And so on. While all the men were partially right, they were all wrong. This poem is also referenced in a research paper about spinal stability (29*), which we return to in a later chapter in the book. Regarding stretching, the poem also serves to highlight the importance of continuing to define our terms.

In order for us to discuss stretching in terms of connective tissue, it is important that we re-establish the definition of stretching as a load; a tensile load. This will keep us within a framework of biomechanics while including forces that may not fit into any conventional type of stretching. For example, a concentric muscle contraction applies a tensile load to the tendon (because the force produced by a muscle pulls on the tendon, in turn pulling on the bone to create, or prevent, movement across a joint). Most would not consider a concentric contraction to be a tendon stretch, but in fact, it is. A passive stretch also applies a tensile load to a tendon, albeit a lesser load due to less muscle force. An isometric contraction at end range, which we have established is a type of conventional stretch, might apply a great tensile load to a tendon, depending on degree of muscle contraction. If we want to ascertain how a tendon responds to a stretch, we have to include all types of tensile loading, not just passive stretching. If you recall the conversation I had with my colleague about stretching tendons (See Part 1 of this article), these were some of the concepts we had to go over to move forward in our discussion. At the conclusion of the book, when in a conversation about stretching, it is my goal for you not to be bound by the limitations of the blind men discussing the elephant.

Incidentally, my favourite response to my stretching survey was from a medical writer who flatly declared 'I have no idea how to describe stretching'. It takes a vast amount of education to be willing to say, 'I don't know'. It also creates a perfect starting point to a discussion on the finer points of loading, stretching, and tissue adaptation (discussed in subsequent chapters in the book). Admittedly, my 'what is stretching?' probe was somewhat of a trick question.

References

Owing to space limitations in the print version, the references that accompany this article are available at the following link and are also appended to the end of the article in the web and mobile versions. Click here to access the references https://spxj.nl/31XFfcl

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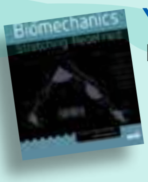

Yoga Biomechanics: Stretching Redefined

By Jules Mitchell, Handspring Publishing 2019;
ISBN 978-1-909141-61-2, Buy it from Handspring
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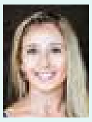
Yoga Biomechanics: Stretching Redefined provides a unique evidence-based exploration into the complexities of human movement and what a safe, effective yoga practice entails. The emphasis is taken off flexibility and centred around a narrative of body tissue adaptation. Conventional approaches to modern yoga are examined through a biomechanist's lens, highlighting emerging perspectives in both the rehabilitation and sport science literature. Artfully woven throughout the book is a sub-text that improves the reader's research literacy while making an impassioned plea for the role of research in the evolution of how teachers teach, and how practitioners practise. Yoga teachers and yoga practitioners alike will discern yoga asana for its role in one's musculoskeletal health. Yoga therapists and other allied healthcare providers can apply principles discussed to their respective professions. All readers will understand pose modifications in the context of load management, reducing fears of injury and discovering the robustness and resilience of the human body.

Contents



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Jules Mitchell MS, CMT, ERYT500 is a Las Vegas based yoga educator, yoga teacher, and massage therapist. Her unique approach blends the tradition of yoga with her extensive study of biomechanics to help yoga teachers develop their craft, and empower them through education. It is her passion to share the most useful and applicable findings from exercise science with the yoga community, and to build confidence in students and teachers by giving them a well-grounded understanding of related research. She leads her own advanced teacher training, teaches workshops and immersion courses worldwide, and offers an ongoing selection of online education and mentoring programmes. As an adjunct faculty member at Arizona State University, she serves as a yoga consultant on various research studies measuring the effects of yoga therapy on special populations including pregnant women, women with depressive symptoms associated with perinatal loss, and patients with cancer. Her future research goals include studying the effects of asana on tissue adaptation, and bridging the gap between research in exercise science and the practice of yoga.

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YOGA AND BIOMECHANICS:
A New View of Stretching Part 2

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