ABSTRACT

THE SCIENCE OF STRETCHING: A REVIEW

By

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Yoga, as a tradition, is practiced for overall benefits in physical and mental health. The current posture-centric yoga practiced in the United States, however, claims flexibility as a primary outcome. The science of stretching is not well understood by yoga teachers and practitioners, which suggests the methods of improving flexibility in a yoga class may not be entirely beneficial. This comprehensive literature review of the biomechanical and neurological mechanisms involved in stretching provides a current discourse for fitness professionals and stretching enthusiasts. Both acute and chronic adaptations are reviewed, as well the effects of stretching on healthy versus injured tissues. Common myths are dispelled, current data is presented, and what remains unknown is highlighted. Future biomechanical research using ultrasonic imaging technology on flexibility exercises as they are practiced in a yoga setting is needed to determine the safety and efficacy of the postures.

# THE SCIENCE OF STRETCHING: A REVIEW

A PROJECT REPORT

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# CHAPTER 1

# INTRODUCTION

Yoga is associated with movement, exercise, and an overall healthy and fit lifestyle (Clay, Lloyd, Walker, Short, & Pankey, 2005). Hatha Yoga, the popular physical form typically practiced in the West, is based in movement where the general design of a yoga class is a sequence of increasingly difficult postures (poses) that move the limbs through a variety of joint angles. The marketing of yoga by popular consumer media supports the posture-centric trend, successfully influencing mass opinion to associate the achievement of the poses with achievement of superior health. A national survey revealed yoga practitioners believe their health status improves as the frequency of practice increases (Ross, Friedmann, Bevans, & Thomas, 2013). A simple internet search on yoga produces almost 1.5 million web pages claiming a variety of benefits including stress reduction, elevated moods, a boost to the immune system, cardiovascular fitness, smarter dietary preferences, pain reduction, improved memory, enlightenment, a fit and toned body, and most of all, Cirque du Soleil standards of flexibility.

A 2012 study conducted by the popular consumer magazine, *Yoga Journal*, revealed that 20.4 million Americans (8.7% of the U.S. adult population) practice yoga for the top five following reasons (from least to most popular): physical fitness, improvement in general health, stress reduction, general conditioning, and flexibility ("Yoga In America study 2012," 2012). Flexibility is the number one motivating factor for Americans who practice yoga.

Through the process of bending, folding, and twisting the body into poses, stretching and flexibility have undeniably become a primary component of the average yoga class. Yet, the available research on yoga and flexibility only determines that a yoga intervention may improve range of motion and does not evaluate the possible biomechanical and neural adaptations due to stretching (Amin & Goodman, 2013; Cowen, 2010; Gonçalves, Vale, Barata, Varejão, & Dantas, 2011; Hewett, Ransdell, Gao, Petlichkoff, & Lucas, 2011; Mccarthy, Brazil, Greene, Rendell, & Rohr, 2013; Pullen et al., 2010; Tracy & Hart, 2013). In addition, most people who enjoy yoga and stretching, including those who teach yoga and stretching, cannot answer basic questions about what is occurring underneath the skin when the tissues are stretched. Some students are instructed only to stretch the muscle, while other teachers target stretching connective tissue; seemingly, the same yoga pose or stretching activity affects muscle in one class and connective tissue in another. Contrary to reason, when certain postures are deemed challenging, past life behaviors and character flaws transgress into present day to be worked through on the mat. Whether the nature of the practice is biomechanical or karmic, flexibility must be improved through frequent and aggressive stretching to achieve the most difficult postures and attain the associated rewards. Regrettably, anecdotal evidence about the benefits of stretching prevails.

Confusion and ambiguity arise when the stretch is met with a barrier or resistance. Reincarnation aside, conflicting accounts of the mechanism(s) limiting range of motion (ROM) and obstructing one's ability to achieve the pose are commonplace. Muscles are impetuously described as tight, tense, stiff, frozen, facilitated, contracted, short, locked, locked short, or knotted. Sometimes the opposing muscles are blamed for being weak,

loose, locked long, or inhibited. When connective tissue is at fault, it is labeled as tight, stiff, brittle, shortened, shrunken, dried up, or crystallized. In any case, the solution to the resistance is most often "just do more yoga, it will get better." Yet, this statement is never accompanied by a discussion of "what" will get better and how much and which kind of yoga will make "it" better. Further, it alludes to the idea that something needs to be fixed or repaired. Not one of the above descriptions provides the student with a clear explanation of how stretching is beneficial, only that you should stretch.

When teachers are not provided with a solid foundation in how the body works, the flow of accurate instruction to the student diminishes. Perhaps this is why teachers fall back on the golden rule of yoga, "Listen to your body." Divulging to students the mystical assertion that they are their own teachers, perhaps intended to empower the student, only results in a relationship where neither the student nor the teacher are empowered. In order to listen to and receive instruction from your own body, you must be educated in what to listen for. The dubious language around "tight" muscles and "stiff" joints does not lend to a clear, two-way conversation. Fortunately, an educated teacher has the power to restore order to the learning process.

Prior to researching this project, the author was a "neighborhood yoga teacher," teaching popularized yoga, and regularly attending similar types of classes. Stretch-induced injuries were alarmingly common among students and teachers alike, the author included. The author began to question the real effects of stretching, why it was important, and how the tissues mechanically responded. In her first biomechanics class, she was literally shocked to discover that scientists have conducted decades of clinical

and laboratory research on how well (or how poorly) different tissues can withstand stretching forces! Not only were there equations to calculate and graphs to plot, but there was an entire vocabulary to learn to keep the conversations clear and in a common language. Oftentimes, the facts were counterintuitive to common instructions in yoga class, the words were defined as something other than the colloquial uses, and "listening to your body" was not going to give you enough information to make the best choice if wanting to improve your health and flexibility, much less prevent injury. It became increasingly obvious that the complexity of muscle and connective tissue architecture, the fact that they are not discrete structures, and how the musculoskeletal and nervous systems work interdependently, is scantly understood by the general yoga population. The author quickly realized that an earnest conversation about stretching in the yoga community was long overdue.

The following work contained in the appendix is a comprehensive literature review of stretching, encompassing biomechanical and neurological adaptations, both acute and chronic. The investigation into the role of the neuromotor system during flexibility exercises helps dispel common myths about how stretching works. The known benefits of stretching are discussed in the context of a yoga practice, which provides for creative inquiry into how the poses may be taught differently from classical instruction. Finally, much is still unknown about the effects of stretching, and the author is careful to point to frequent assumptions which contribute to the overall misunderstanding of stretching. This review compiles the most recent soft tissue data into a manual for any fitness professional or stretching enthusiast.

# CHAPTER 2

## METHODS

The author's journey through the research and how this project came to be was long but expansive. The process of collecting and understanding data, then converting it into useful information when teaching yoga, required many stages. Some of the first articles pulled supported conclusions which were foreign to the author and quickly discarded. Only after several years of research, seminar attendance, and collaborations, were those original articles retrieved and included as critical content to the project at hand.

When the author began researching for this project, the topic was still unknown. Of course, it would be biomechanical in nature, but the direction was beyond the horizon. At the time, the author was focused on injuries caused by yoga and began a first draft with that in mind. While studying connective tissue injuries, the author became fascinated with the resilience, capabilities, and intelligent design of the human body, and began to wonder if all the tugging and pulling associated with a daily yoga practice interfered with this design. The author turned to the stretching science, and the original piece on yoga injuries was left behind.

The author scoured the databases (PubMed, Science Direct, SPORT Discus, and EBSCO) searching a variety of terms including stretching, flexibility, yoga, hamstring flexibility, biomechanics of stretching, tendon stiffness, creep, ligament injuries, tendon injuries, fibroblasts, collagen turnover, fascia, tensegrity, sarcomeregenesis,

mechanoreceptors, proprioceptive neuromuscular facilitation, and stretching tolerance. The author attended seminars by sports medicine professionals and fascia scientists, participated in a 6 day cadaver dissection, and searched the reference list of a dozen textbooks on biomechanics and neuromechanisms. Social media and online communities for researchers provided a platform to request articles directly from the scientists and engage in conversations about the outcomes. A thorough understanding of the entire body of literature regarding stretching was achieved during this time.

The author received a preliminary education on stretching through yoga teacher trainings and workshops, as most yoga teachers do, and was surprised to find the science would lead to complete re-education. While attempting to collect research articles validating what the author knew, looking for more evidence to confidently write about stretching, an impasse was reached. The effort was ephemeral; *that* supply of literature was scarce. Instead, the author uncovered articles that challenged what was taught in yoga, disputing the anecdotal concepts taught by respected yoga teachers (yogi-lore). The author had to be willing to unlearn and learn again before piecing all the science together into a complete thesis project.

While writing Chapters 1-6 of the appendix, the author still did not understand exactly where the project was going. Still working from the "stretch it and make it longer" framework, the author simply had not made sense of it all. It was not until Chapters 7-9 of the appendix that the author began to see flexibility as a motor skill that required training at end ranges of motion. Of course, biomechanics plays a role, but the nervous system is the actuator for movement--including stretching. The author went back to rework the early chapters of the appendix.

This process is explained here because it may help in navigating through the material. There are essentially two parts to this project contained in the appendix, Part 1: Biomechanics (Chapters 1-6) and Part 2: Neuromechanisms (Chapters 7-10). When the author presents the material in a lecture format, yoga teachers are eager and nodding with agreement during the biomechanics segment. The absolute nature of physics is reliable and predictable (once learned), and therefore, comforting. But during the neurosensory and neuromuscular chapters, the teachers are skeptical and confused. The variable nature of the nervous system, alternative methods of dissections, and departures from sacred cows of yoga instruction are unsettling. In time, it all comes together, the big picture becomes clear, and the science can be easily applied in practice. The author recommends approaching the material as it was assembled: after completion, go back and read the biomechanics chapters with the neuromotor system in mind.

# CHAPTER 3

# CONCLUSION

The science of stretching is not to be contained within the scientific community. The author is positioned to be a vessel through which the information would flow to the yoga community, including students, teachers, and other fitness professionals who have an interest in stretching. Fueled by personal momentum and an overwhelming response by yoga teachers worldwide who were reading the author's blog and wanting to learn more, this project was born. The demand was palpable. Three and a half years of study later, the author presents a literature-based research on stretching, complete with added information gleaned from the author's teaching experience and dozens of informal interviews and collaborations.

The information provided within this project is not a system, program, or protocol--rather a model for expected outcomes of stretching based on the presently available body of evidence. As a result, it is limited in "how to" instruction. On occasion, the author offers insights into how she has adapted her teaching methods and personal practice as a result of her interpretation of the literature--which are not to be received as a critical rating of different yoga schools and styles. These highlights are intended to spark inquiry, curiosity, and to elevate the standards by which yoga instruction is delivered. With a critical mind, the education provided by the contents of this book can be applied to any school of yoga to which you belong or any fitness program you follow. There is no list of "good" poses and "bad" poses, nor are there

instructions on how to sequence a class. There is no script for how to teach the following information. Instead, the following chapters in the appendix educate the reader in stretching science to better determine adequate stretching protocols.

# APPENDIX

## THE SCIENCE OF STRETCHING: A REVIEW

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# Introduction

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impetuously described as tight, tense, stiff, frozen, facilitated, contracted, short, locked, locked short, or knotted. Sometimes the opposing muscles are blamed for being weak, loose, locked long, or inhibited. When connective tissue is at fault, it is labeled as tight, stiff, brittle, shortened, shrunken, dried up, or crystallized. In any case, the solution to the resistance is most often "just do more yoga, it will get better." Yet, this statement is never accompanied by a discussion of "what" will get better and how much and which kind of yoga will make "it" better. Further, it alludes to the idea that something needs to be fixed or repaired. Not one of the above descriptions provides the student with a clear explanation of how stretching is beneficial, only that you should stretch.

When teachers are not provided with a solid foundation in how the body works, the flow of accurate instruction to the student diminishes. Perhaps this is why teachers fall back on the golden rule of yoga, "Listen to your body". Divulging to students the mystical assertion that they are their own teachers, perhaps intended to empower the student, only results in a relationship where neither the student nor the teacher are empowered. In order to listen to and receive instruction from your own body, you must be educated in what to listen for. The dubious language around "tight" muscles and "stiff" joints does not lend to a clear, two-way conversation. Fortunately, an educated teacher has the power to restore order to the learning process.

Prior to researching this project, I was your "neighborhood yoga teacher," teaching popularized yoga, and regularly attending similar types of classes. Stretch induced injuries were alarmingly common among students and teachers alike, myself

included. I started to question the real effects of stretching, why it was important, and how the tissues mechanically responded. Not afraid of a little (okay, a lot of) academic work, I set out to find some answers. In my first biomechanics class, I was literally shocked to discover that scientists have conducted decades of clinical and laboratory research on how well (or how poorly) different tissues can withstand stretching forces! Not only were there equations to calculate and graphs to plot, but there was an entire vocabulary to learn to keep the conversations clear and in a common language. Oftentimes, the facts were counterintuitive to common instructions in yoga class, the words were defined as something other than the colloquial uses, and "listening to your body" was not going to give you enough information to make the best choice if wanting to improve your health and flexibility, much less prevent injury. It became increasingly obvious that the complexity of muscle and connective tissue architecture, the fact that they are not discrete structures, and how the musculoskeletal and nervous systems work interdependently, is scantly understood by the general yoga population. I quickly realized that an earnest conversation about stretching in the yoga community was long overdue.

The science of stretching is not to be contained within the scientific community. I knew I could be a vessel through which the information would flow to the yoga community, including students, teachers, and other fitness professionals who have an interest in stretching. So began this project, fueled by personal momentum and an overwhelming response by yoga teachers worldwide who were reading my blog and

wanting to learn more. The demand was palpable. Three and a half years of study later, I present to you my literature-based research on stretching, complete with added information gleaned from my teaching experience and dozens of informal interviews and collaborations.

Finally, one important caveat is that you will find this project is limited in "how to" instruction. The information within is not a system, program, or protocol - rather a model for expected outcomes of stretching based on the presently available body of evidence. On occasion, I will offer insights into how I have adapted my teaching methods and personal practice as a result of my interpretation of the literature - which are not to be received as a critical rating of different yoga schools and styles. These highlights are intended to spark inquiry, curiosity, and to elevate the standards by which yoga instruction is delivered. With a critical mind, the education provided by the contents of this project can be applied to any school of yoga to which you belong or any fitness program you follow. There will be no list of "good" poses and "bad" poses, nor will there be instructions on how to sequence a class. There will be no script for how to teach the following information. Instead, the following chapters will educate you in stretching science so that you may better determine adequate stretching protocols for yourself and/or for your students. Good luck and Namaste.

# Chapter 1. Bodies In Motion

*"Movement is what we are, not something we do."*
*Emilie Conrad*

A glimpse out the window into nature reveals constant movement and change. Waves come and go with the tide, trees and plants grow toward the sun, and animals chase, lounge and stretch. Similarly, the human body is a structure designed to move and change. An examination of our parts and development demonstrates this: muscles that generate movement and grow bigger when exercised, fluid filled joint capsules for smooth gliding of surfaces that wear as we age, bones that withstand tremendous impact, smooth muscle that dilates and constricts to digest and move nutrients through the body, blood that flows, lungs that inflate and deflate, skin that regenerates, and so on. The source of movement is always the same: a force acting on the object changing position. Without force, there would be no movement, no change. Likewise, without movement, there would be no force.

In the human body, each unique movement and resulting joint position is caused by a force. The bones, soft tissues, fluids and cells are collectively subject to the forces associated with movement. Sensory nerves, called mechanoreceptors, collect information about force and deliver it to the nervous system, where the input is translated into mechanical, chemical, and electrical outputs. While different types of receptors respond to different stimuli, the general purpose is the same: to send information to the central nervous system (CNS) eliciting some sort of movement

response, whether it be cellular activity or full body movement. The human body is designed to engage in a constant interplay between forces; creating, preventing, and modifying movement.

Culturally, we are in an epidemic of insufficient movement. Moderate movement throughout the day, for which we have genetically adapted was required by the hunter-gatherer from whom we evolved, is our natural form of fitness (O'Keefe & Vogel, 2010). Slowly, beginning with the agricultural revolution, continuing with the industrial revolution, and now in our present state of technological advancement, our modern lives have become increasingly sedentary. We have strayed far from our ancestral behaviors of movement, and are now suffering the consequences. In an attempt to avoid the negative health consequences associated with the stationary lifestyle, we have created a short isolated period of movement we call exercise. In the timeline of human evolution, only recently have we contained movement as a scheduled workout essential to staying fit and healthy. Continuous movement throughout the day was essential to the survival of the average hunter-gatherer and adequately introduced forces to the tissues of the body.

Today's movement, however, is often an isolated exercise bout approached with a specific goal in mind: lose weight, strengthen the cardiovascular system, get stronger, be more flexible, reduce the risk factors for disease. Whatever the individual goal may be, the commonality lies in our dynamic nature. We are creatures that need to move, not static objects. Movement enhances the capabilities of our tissues, making

them stronger and more efficient. Movement and force adapt tissues both macroscopically and microscopically; some adaptations we see with our eyes, others we measure through health, fitness, lack of injury and disease. The widespread acceptance of the detriment to our health accompanied by inactivity resulted in an increase in the popularity of fitness, the subsequent rise in the number fitness and health professionals, and eventually the 1954 formation of the American College of Sports Medicine (ACSM) (Berryman, 2004). Today, exercise science a prevalent field of study, research and employment.

The ACSM defines exercise as a "physical activity that is planned, structured, and repetitive and [that] has as a final or intermediate objective of the improvement or maintenance of physical fitness" (Garber et al., 2011). The components of fitness, (Table 1) are physical activities classified by training goal. Interestingly, the ACSM classifies yoga as a "multifaceted" activity, including components of neuromotor fitness, strength training and flexibility (Garber et al., 2011). Perhaps the average yoga practitioner agrees, but the archetypal images and marketing of yoga still focus primarily on flexibility rather than controlled movement. The research presented in this project suggests the strength training and neuromotor training aspects of yoga deserve greater attention.

Perhaps the classical interpretation of strength training requires no explanation, where muscle power and hypertrophy are the typical associations. Connective tissue resilience in the presence of a stretch, however, may be a new consideration for you

and will be discussed at length in subsequent sections, as will the role of the various sensory receptors on movement, joint mobility, and stretch, because it too plays a significant role in flexibility. "Neuromotor exercise training," as ACSM states, is "sometimes called functional fitness training, incorporates motor skills such as balance, coordination, gait, and agility, and proprioceptive training" (Garber et al., 2011). Regardless of how yoga is interpreted, the popular consumer media presents it predominantly as a stretching activity which warrants a deeper investigation into the effects of the various types of flexibility exercises and the impact of yoga on joint mobility and tissue health.

**Table 1. ACSM Components of Fitness**

| **ACSM Components of Fitness** | **Examples** |
|---|---|
| 1. Cardio respiratory fitness | Long distance running |
| 2. Muscular strength and endurance | Weight lifting |
| 3. Body composition | Measure of body fat % |
| 4. Flexibility | Stretching |
| 5. Neuromotor fitness | Tai chi, qi gong, yoga |

A prerequisite to an intelligent discourse on the mobility and flexibility requirements for movement is understanding how muscle and connective tissues respond to the forces associated with movement. In order to satisfy this, an introduction to biomechanics will be presented. Biomechanics is often misunderstood to mean how the body moves (applied anatomy) and is ordinarily misused by yoga teachers. When people have different definitions for the same word, communication is greatly obstructed; they may as well be speaking a different language. Biomechanics is

the study of how forces affect biological systems. Human biomechanics, in the context of yoga, is therefore, the study of the effect of mechanical forces on structure, function, and movement. The following section imparts on you proficiency subject of force and the language of biomechanics.

## Recommended Reading

(Garber et al., 2011; Neumann, 2010)

# Chapter 2. Biomechanics

*"My ally is the Force, and a powerful ally it is."*
*Yoda*

In the science of physics, mechanics narrows the focus to the action of forces on objects. In the science of engineering, materials engineering narrows the focus to the action of forces on the matter out of which objects are designed. In the science of kinesiology, biomechanics is the study of structure and function of living objects and narrows the focus of movement to the action of mechanics on the human body. In biomechanics, this matter, or material, is the biological tissue out of which we are made and this finer point of focus is called tissue mechanics. Therefore biomechanics is not about how to *perform* a forward fold, but rather how the *forces act* on the whole body and its parts during any given combination of joint positions. The extensive assortment of postures offered in a yoga class are clearly great fodder for anyone with a biomechanical interest.

Biomechanics is more than just physics, however, and a modest list of related fields of study includes biology, chemistry, anatomy, physiology, physics, engineering, kinetics, kinematics, histology, and hydraulics. Just as inanimate objects occupy space and are subject to physical laws, humans are animate objects subject to the same. Furthermore, human movement combines the complexities of many systems acting within the living matter. Simply understanding a single muscle contraction requires some knowledge of mechanics, chemistry, electricity, cellular biology, and fluid

dynamics. In the context of health and longevity, osteopathic medicine incorporates concepts of pressure and force transmission into treatment protocols for holistic wellness (Findley & Shalwala, 2013). Thought-provokingly, the scope of classical yoga is more than just its' poses and merges a vast array of sciences and theories as well. Biomechanics encompasses an awe-inspiring sum of disciplines, from the intelligence of life to the physicality of material existence. Biomechanics completes a network of otherwise independent sciences.

Unlike an apple falling from a tree, the human nervous system responds to forces in an unpredictable manner. In Newtonian mechanics, another name for classical mechanics, if you dropped the same apple from the same tree while controlling for every possible variable, you would be able to reproduce the same results. Such variables may include releasing the apple from the same height, at the same acceleration, and during identical wind conditions. If a human jumped out a tree, you would *not* be able to reproduce the exact results on subsequent trials, even if controlling for every possible variable. Humans are impossible to completely predict and control. This means that the forward fold you do today is not the forward fold you will do tomorrow. Your body and mind have a history of yesterday's forward fold to include into today's forward fold, even if all other variables are controlled for. Humans simply don't behave like apples.

Generalized instructions, often called "cues," on how to position the body in a pose can be ineffective in reaching specific goals. For example, you often hear the instructions to bend the knees during a forward fold (FF) to protect the spine. To evaluate the biomechanics during a FF would be to establish how the forces are acting on each segment of the spine and how bending the knees would alter the forces. Perhaps redirecting force by deliberate placement of yoga props would have a greater impact on the intended outcome of the pose than simply bending the knees. You might discover that in order to satisfy the desired effect, you may have to give up on the standing forward fold and suggest an entirely *different* pose. This approach makes teaching yoga and stretching in any sort of group situation a challenge; no two people respond to forces identically.

## Where Forces Come From

Studying force builds upon an understanding of where forces come from and how the body receives and responds to them, just as studying nutrition lends to an understanding of where nutrients come from and how the body metabolizes them. Forces acting on the body can be divided in 3 categories: gravity, externally applied forces, and internally generated forces. These forces can be smartly utilized to achieve beneficial outcomes and poorly utilized to result in harmful outcomes.

The omnipresent, unavoidable force that deserves the first look is gravity. Gravity is an unrelenting and constant force we must invariably either push against or surrender to, continually fluctuating between the two. Everything in nature interacts with gravity if you pause and take a look; from the direction and turbulence of a flowing river to the phenomenon of plants growing upward through the earth's surface. It is the interplay of the human body with gravity that serves as the primary catalyst for our body's cellular signaling that regulates tissue synthesis or degradation. For example, astronauts that spend just 90 days in zero gravity environments lose tendon strength, even when performing preventative mechanical exercises (Reeves, 2006). We truly are mechanical beings and our tissues adapt to the forces to which we are exposed.

The second force comes from an externally applied object or the touch from another being on the body. Some loads are beneficial, others are not. An impact collision during sports could potentially cause a ligament to tear or a bone to break. A massage treatment intends to therapeutically direct force, just as an adjustment from a

yoga teacher does, although many have also been injured as a result of either. Yoga props, such as the weight of a sandbag across the sacrum in a standing forward bend, utilize gravity to produce a modified external force. These external loads further provide feedback via one's sensory receptors which are translated by the nervous system to monitor and control the degree of movement and stretch. The nervous system is then responsible for the third and final force acting on the body: the internal force of human movement.

Muscle tissue, excitable via an electrical impulse from the brain, generates force internally, providing for an infinite number of body positions. Skeletal muscle (as opposed to cardiac muscle and the smooth muscle of the organs and vessels) grants us the ability to express conscious movement, essentially enabling us to adapt our tissues at will. Muscles, embedded within a web of collagen based connective tissues, leverage the limbs to control joint articulations and influence the characteristics of forces, which further designate exactly how these forces are mechanically applied to body.

## Forces and Movement

Forces acting on the body, also called loads, are applied in a variety of ways (Figure 1). Normal[1] forces (as gravity always is) can place a material under tension or compression. Shearing[2] forces (as many manual therapy techniques employ) may also be combined with compression and tension to result in bending or twisting (toque). In

[1] normal = perpendicular to surface
[2] shearing = parallel to surface

the human body, bones and cartilage are generally considered compressive tissues because they withstand compression *and* develop in the presence of compression, just as tendons and ligaments are considered tensile tissues because of their resilience *and* adaptation to tension. The architecture of these biological tissues determines the type of load it prefers while the type of load it receives influences its architecture. It is your classic "chicken or the egg" conundrum: which came first is indeterminable.

Friction is an internally created force resulting from the resistance between two surfaces involved in movement. Friction can be external to the body, such as the resistance experienced when swimming through water, or internal, such as when the articulating cartilaginous surfaces of your joints are degenerated (osteoarthritis, e.g.). Furthermore, mechanical resistance within the body is a complex *fluid friction* for which the effect of on movement is not included here. The review of the stretching data assumes normal levels of fluid friction within joint capsules, thereby no interference with joint mobility, and is a considerable limitation of this project.

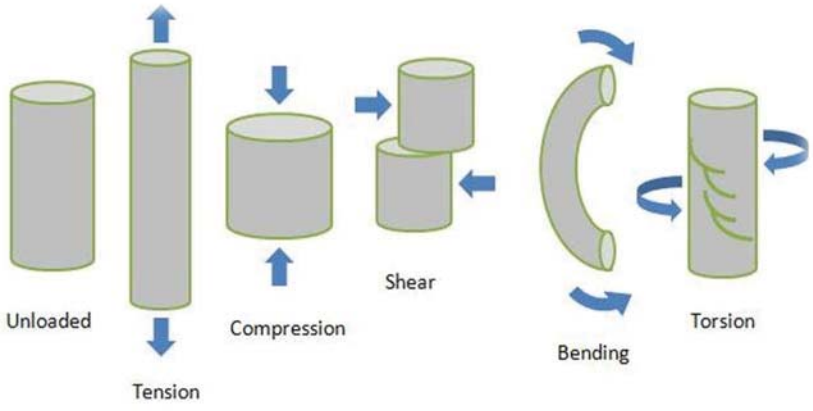



**Figure 1. Types of Forces**

Mobility can be measured both passively and actively. Passive joint mobility relies on an external force (gravity or another individual) to move the limbs. Active mobility is generated by the internal muscle force and often results in a lesser range of motion than passive mobility. Mobility is generally limited by compressive tissues: the shape of the bones at the articulating surfaces, the cartilaginous lining, and the fluid within synovial joint capsules. Joints, however, can also be hypermobile, in which case the supporting ligaments surrounding a particular joint are not able to constrain movement to normal ranges of motion where the joint is protected from excessive loads (Hauser et al., 2013). Hypermobility, often appearing as flexibility, is quite the opposite as ligaments receive the stretch rather than the tissues involved in flexibility.

Flexibility, the primary topic here, is measured by the behavior of the neighboring tensile tissues, mainly the muscle-tendon unit and myofascia[3]. The benefit of flexible tissues is improved efficiency of movement by reducing the amount of energy exerted to move the limbs, and conversely, the drawback of flexible tissues is a delayed rebound effect, in turn increasing energy expenditure during repetitive movements (Shrier, 2004). Fluid friction between the layers of soft, tensile tissues may be a limiting factor in flexibility assessment. Future chapters present the role of hydration in the behavior of connective tissues, yet friction is not adequately accounted for. Once again, quantifying fluid friction is beyond the scope here (as it is also in much of the literature) and is a limitation of this project. Flexibility, here, is assumed to be a mechanical

[3] Myo is a prefix meaning "muscle"

response under an applied tensile load and no interference of internal fluid friction is assumed.

Commonly, mobility and flexibility are taught as discrete factors in movement. If joint mobility is limited, then flexibility is *not* the limiting factor. Conversely, if joint mobility is free and clear of limitations, but movement is impeded, then flexibility *is* the limiting factor. Such a model assumes that an increase in flexibility automatically transfers to better utilization of mobility, which is not necessarily the case (Moreside & McGill, 2013). As humans do not behave as apples, neither do they behave as robots. Movement is an expression of the interaction between compressive and tensile tissues but also includes the contribution of the nervous system. The compartmentalization of mobility and flexibility will seem obscure and futile, therefore, as the interconnectedness of the body is revealed in subsequent chapters. The exploration of the continuity of musculoskeletal tissues suggests a harmonious continuum of mobility and flexibility (as opposed to discrete and distinct definitions). A more precise model of how external forces are managed by the body, how internal forces are expressed throughout range of motion, and how the nervous system regulates movement will be presented.

Tree pose can help you evaluate the difference between your passive and active range of motion at the hip and knee joint. To test your passive range of motion, reach down and grab hold of your right ankle and place your foot as high as you can against the inner left thigh. To test your active range of motion, position the foot without using your hands at all. Try both sides. This will help you determine your passive and active range of hip abduction, flexion, rotation and knee flexion.

## Force Characteristics

Much like in the famous film Star Wars, not all forces are equal. The threat of a journey to the dark side looms during any movement. Forces can vary in magnitude, duration, direction, location, rate, and frequency (Table 2). If any one of these qualities is too great, it can injury in the human body. For example, the anterior cruciate ligament (ACL) of the knee withstands tensile loads during gait as it curbs tibiofemoral translation, however, a swift change of direction, a powerful collision, or excessive repetition could cause damage. Likewise, not enough loading could cause tissue weakening. The characteristics of forces regularly applied to the tissues determines the resiliency against those same forces. Biological tissues adapt based on the demands imposed upon them.

**Table 2. Force Characteristics**

| Characteristic | Description |
|---|---|
| Magnitude | How hard? |
| Duration | How long? |
| Direction | At what angle or vector? |
| Location | Where? |
| Rate | How fast? |
| Frequency | How often? |

A concept worth considering which goes against the classical rules of "alignment" in yoga is that of progressively increasing "misalignment" variables. Regular loads of varying characteristics will optimize tissue strength and reduce the likelihood of tissue damage during unexpected movements. Returning to the ACL example, if you

only load the ACL in one direction, the sagittal plane, for example, it will never adapt to withstand loads in the transverse plane, and thus be unprepared for an accidental twisting of the knee. While it is impossible to know without a Jedi-mind which unexpected forces the body will encounter, systematically increasing or altering the magnitude, duration, direction, location, rate, and frequency of loads will provide mechanical stimulus to improve tissue strength and minimize degradation.

## Responses to Load

The architecture and structural characteristics of muscles, bones and the connecting tissues are constantly changing. Wolff's Law states that bone will remodel its architecture to adapt to an applied load. Herein lies the basis for the often recited "weight bearing exercises increase bone density." When a force is applied to a bone (it bears weight), it gets stronger (increases density) and becomes better equipped to resist fractures and breaks. The corollary to Wolf's Law is Davis' Law, which refers to the adaptable nature of the soft musculoskeletal tissues. Although not widely cited, perhaps because it is not as well developed or clearly defined; Davis' Law reinforces the adaptable nature and constant structural remodeling of human living tissue induced by the application of loads.

If you only load the shoulder joint during Plank Pose in perfect alignment, you will be adapted for Plank Pose in perfect alignment. If you were to trip and fall, however, the likelihood that you will land in a perfectly aligned Plank Pose is slim. Consider practicing plank off centered to the left, off centered to the right, and with the hand placement staggered and at varying widths. Perhaps elevate the feet by placing them on blocks or on a workout bench. Be certain to progressively increase the direction, duration, and frequency of the loads to prevent injury and allow time for tissue remodeling.

Since muscle, bone, and connective tissue favorably respond to mechanical loading, the Progressive Overload principle - loading must progressively increase above normal loading patterns to stimulate growth - applies (Figure 2). An obvious example is the effect of weight lifting, where a gradual increase in volume (number of repetitions) and intensity (percentage of maximum capacity) are the defining aspects of progressive overload. After just a few weeks of loading muscles through lifting weights, muscles get stronger and the demand can safely increased by gradually adding more weight (increasing the load). Try to lift more than the capacity of the muscle at that time and the lift will be unsuccessful, possibly injurious. Take a few weeks off and the muscles may not retain the capacity to lift even the original load. Less obvious is the adaptation of a ligament. Increasing the demand on a ligament also initiates strengthening, although at much slower rate than muscle tissue. Apply a tensile load far greater than the capacity of a ligament and that tissue weakens or fails. Oppositely, an insufficient tensile loads fail to strengthen the ligament which will then be unable to accept increasing loads. Thus, the *Goldilocks Principle* is in effect: too much and too little are unfavorable, but when you get it just right, the adaptations are favorable.

Applied stress (another word for load) requires a fine balance between stressing the tissue just enough to improve the load bearing capacity and stressing the tissue conservatively enough to prevent substantial injury. Determining how much load and which degree of force characteristics and appropriate for a classroom full of yoga students is not a simple task. Unless you have magnetic resonance imaging (MRI) vision,

you cannot be sure the poses you are teaching to the group are appropriate for everyone.

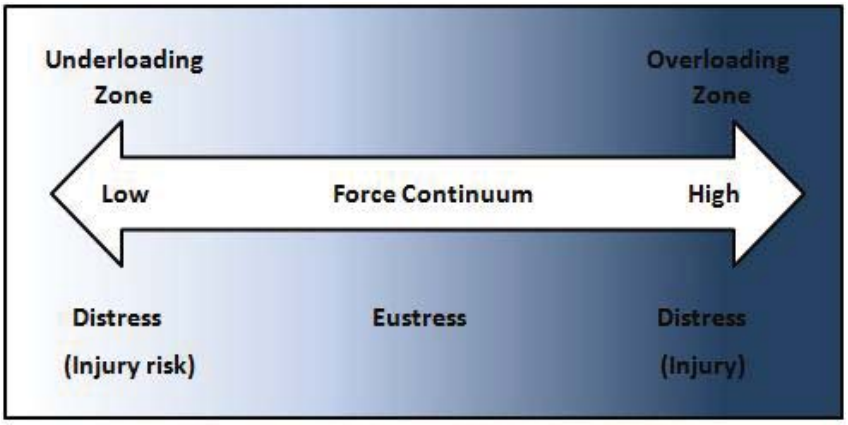



**Figure 2. Progressive Overload**

One thing is certain, however, and it is that movement is an essential component to tissue remodeling. Movement changes joint angles, in turn stretching the soft tissues opposite to the direction of movement. Theoretically, hip flexion stretches the hip extensors and hip extension stretches the hip flexors - although modern interpretations of anatomy challenge the simplicity of this lever based model (Franklyn-Miller et al., 2009; Meyers, 2009; Scarr, 2012; van der Wal, 2009). Regardless of the preferred anatomical model, if one's physical activities are varied enough, joint ranges of motion are likely to be preserved and achieving adequate flexibility can then be incidental.

If you have ever worn a cast, you have firsthand experience with the importance of regular movement; the muscles responsible for moving the immobilized joint degenerated during the time when demand was limited or absent. After the cast is

removed, movement leads to muscle regeneration. Similarly, failure to adequately load bones is a risk factor for osteoporosis, too little (or too much) friction within a joint capsule may lead to osteoarthritis, and insufficient loading of the muscle-tendon unit may reduce the swiftness by which tendons transfer the force generate by muscle to bone. With gradual and progressive overloading, however, the effects of immobilization and insufficient loading can, to some degree, be reversed. That is not to say you can move your way past aging and eventually, death. Some elements of degeneration are certain and cannot be avoided.

A critical observation regarding progressive overload is the nature of adaptation to overloading and underloading. Bones, when exposed to consistent compressive stresses grow in strength and, particularly in developing children, length. Bones do not develop into shorter, more compressed segments in response to gradual overloading. Bones may *break* in response to overloading beyond their present capacity, unable to withstand the stress, but they do not *lose* longitudinal length. By that rationale, tensile connective tissue would not grow longer in response to stretching. Ligaments would get stronger, perhaps even shorter, in order to develop a greater capacity to withstand tensional stresses. Ligaments may *tear* in response to overloading beyond their capacity, unable to withstand the stress, but they do not *gain* longitudinal length. The notion that stretching and flexibility training is not a matter of increasing tissue length is central to the following investigation.

In yoga, handstands are becoming increasingly popular. Students want to learn them and teachers want to teach them. However, if a student has insufficient loading history on her dorsiflexed wrists, the tendons and ligaments may not be able to bear the load of the body while stretched at the required joint position. The average office employee who types on a computer with her wrists continually in palmar flexion, are unlikely adapted for the volume of weight bearing poses on the arms (downward included) as are found in a typical vinyasa yoga class. It is quite common for these students to complain about wrist pain. Progressively and gradually increasing the amount of load on the wrists - either by offering alternative poses (spinal balance > plank > one leg/arm variations, etc.), props to decrease extreme joint angles (e.g. a wedge), or even changing the sequence of the class - would be a prerequisite to attempting handstands.

## Specificity

The SAID Principle (Specific Adaptations to Imposed Demands) states the body will adapt to the forces most frequently applied (i.e. the movements most frequently performed). For example, playing soccer will not improve your tennis swing. Biceps curls, however, *may* improve a tennis swing by a little: some movements have greater carryover than others. Biceps adaptively become stronger due to weight training, potentially improving the strength and power of your tennis swing. Of course, the skill required for precise ball control cannot be achieved by weight training. The only way to improve a tennis swing is to practice the tennis swing.

The yoga pose Warrior 2 will, therefore, only improve your Warrior 2. The assumption that yoga (and stretching) will improve sports performance is somewhat farfetched. Yoga will improve yoga, not tennis. Additionally, yoga poses are static postures which are held usually between 30 and 90 seconds. Most physical activities and sports require a motor skill (i.e. movement) which will not improve by holding a static posture. Practicing yoga improves flexibility and increases available range of motion (Amin & Goodman, 2013; Cowen, 2010; Gonçalves et al., 2011; Hewett et al., 2011; Mccarthy et al., 2013; Pullen et al., 2010; Tracy & Hart, 2013), but without training and skill acquisition at these new ranges of motion, performance will not improve. If you are practicing yoga to improve upon some aspect of your movement curriculum, increased flexibility is all you should expect to achieve.

In the context of adaptations to load, the SAID Principle states that the body will adapt to the loads to which it is most frequently exposed. If an adaptation to tensile

loads such as stretching is increased flexibility, it is important to understand the mechanisms of flexibility. As previously mentioned, flexibility training does not necessarily increase tissue length but rather its *capacity* to lengthen. The distinction may be unclear without a clear definition of terms around stretching and flexibility, a lesson in the mechanical behavior of the biological tissues being stretched, and the cellular processes that regulate adaptations. The following chapters provide just that.

## Recommended Reading

(Hall, 2012; Nordin & Frankel, 2001)

# Chapter 3. Stretching and Flexibility

*"Our own physical body possesses a wisdom which we who inhabit the body lack. We give it orders which make no sense."*
*Henry Miller*

Stretching and flexibility are practically synonymous in the world of yoga. While many people practice yoga to increase their flexibility, others refuse to practice yoga because they aren't flexible enough. Some people are flexible *because* they practice yoga, others practice yoga because they are *already* flexible. The connection between stretching and flexibility motivates yoga practitioners to stretch more often, stretch harder, stretch longer, even stretch in heated rooms, to gain greater flexibility despite the lack of understanding of the science behind it.

## What is Stretching?

The word "stretch" requires some distinction. For our purposes here, the word stretch is defined as a tensile load. Stretching does not automatically assume any tissue elongation, the load may or may not be great enough to distend the tissue receiving the stretch. Stretching activities apply a tensile load to the target muscle and surrounding connective tissues. The word "tight" describes nothing other than a sensation; there is no mechanism that causes something to be "tight." The significance lies in the falsely assigned colloquial meaning of "tight" - which assumes the tissue is shortened and must be released/lengthened by a stretch. Unfortunately, it is quite common for the "tight"

sensation to be already overstretched, in which cases the remedy of stretching will not alleviate the discomfort.

## What is Flexibility?

The American College of Sports Medicine (ACSM) writes a brief single page about flexibility in the 26 page position statement titled **Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory, Musculoskeletal, and Neuromotor Fitness in Apparently Healthy Adults: Guidance for Prescribing Exercise** (Garber et al., 2011). The stand is that "flexibility exercises" increase range of motion and may improve postural stability and balance while evidence of injury, pain, and soreness prevention or remedy is lacking. ACSM cautiously declares that the sole "goal of a flexibility program is to develop range of motion in the major muscle-tendon groups in accordance with individualized goals." It says *develop*, not maximize nor create. Develop means to bring out the capabilities of, to boost progression. Flexibility is, therefore, a matter of range of motion.

Returning temporarily to the seemingly obscure and futile distinction between mobility and flexibility, the capacity for the soft tissue to distend when stretched is how flexibility is typically evaluated. Think of an elastic material, like a rubber band. If stretching a rubber band is met with an internal material resistance (the visual explanation for the sensation "tight"), then the rubber band is inflexible. Conversely, applying a tensile load to a flexible rubber band will distend the material, but it can only

stretch so far before the material starts to tear. Keep stretching it and it may eventually tear completely. The rubber band's capacity to resist damage when stretched, like human muscle and connective tissue, is called tensile strength (we are supposed to feel "tight").

While this compartmentalized analysis of flexibility, how much elongation and tensile strength for which the tissue has a capacity, is an adequate analogy for the behavior of the soft tissues of the musculoskeletal system, it has very little to with range of motion. Humans are animated creatures whose movement is initiated, modified, restrained, and regulated by the nervous system. More accurate measures of flexibility, must therefore, eventually include sensory input and motor output; but not before a thorough study of tissue properties such as tensile strength is completed.

The tissues involved in mechanical flexibility have both active and passive elements. Muscle tissue is active, due its excitability, where the relatively inert connective tissue is passive. The research of the past decade on fascia and connective tissue greatly challenges the inactive framework into which connective tissue has been relegated (Schleip, Findley, Chaitow, & Huijing, 2012; Schleip, 2003a, 2003b; C. Stecco et al., 2007; C. Stecco, Macchi, Porzionato, Duparc, & De Caro, 2011; Willard, Vleeming, Schuenke, Danneels, & Schleip, 2012), but for the present purposes the differentiation is well served.

Just as passive and active mobility is measured separately, so is passive and active flexibility. During passive flexibility tests or exercises, a partner or device holds a

limb at the joint position which stretches the target tissue (antagonist). Active flexibility requires the individual to move and hold the limb into the specific joint position and the stretch is often limited by the fleeting endurance of the agonist. These delineations, however are only theoretical.

In practice, the distinction between active and passive flexibility is not as discrete. As long as the individual has any amount of neuromuscular activation (i.e. is conscious), the range of motion may technically be considered active as it is possible the nervous system is preventing the joint position. True and entire passive range of motion propounds joint positions accomplished without any neuromuscular interference, as when anesthetized, for example, and neural responses are suppressed (Bennett, Hanratty, Thompson, & Beverland, 2009; Krabak, Laskowski, Smith, Stuart, & Wong, 2001). Because the musculoskeletal system is under direct control of the nervous system, when conscious, the individual is aware and allowing the movement (even when relaxed under a guiding hand of a physical therapist). There will always be some level of neural "noise" resulting in muscle activity.

Unfortunately, the literature does not adhere to any particular nomenclature or procedure and confounding data containing active and passive results is a laborious task (Gajdosik, 2001). The data on flexibility and mobility are collapsed into range of motion evaluations and rarely account for neural contributions. Regardless, flexibility has earned its own category in the ACSM Components of Fitness, and stretching exercises are promoted as effective methods of developing range of motion.

A supine hamstring stretch can help you evaluate the difference between your passive and active hamstring and calf flexibility. To test your passive flexibility, use a yoga belt under your shoulder blades looped around your foot as the device which holds your leg (photo top). If you do not have 90° of flexibility, this variation will not work for you. Instead, go to a door way and rest the leg on the wall as the other leg stretches through the doorway. To test your active range of motion, from a supine position lift one leg and hold it there (photo bottom). Try both sides. Individuals with limited flexibility will find the agonist will have to work much harder during the active test and will fatigue much sooner.

## Types of Flexibility Exercises

As evident during the passive versus active discussion, it is clear not all stretching is the same. Stretching can vary by frequency (how many times per day and how many days per week) and by repetition and duration (which can vary as greatly as changing joint positions with every breath, a single stretch for 15-60 seconds, multiple stretches for 15-60 seconds each, or one time for 3-5 minutes). The ACSM, which provides a single page dedicated to flexibility, separates and defines the various types of stretching exercises by 4 distinct categories (Table 3) (Garber et al., 2011). Following the descriptions of stretching exercises, however, minimal suggestions for frequency, repetitions, durations, of the stretch are provided.

Further, the relevance of the stretching techniques and the associated outcomes are inconclusively discussed in only four sentences. The brevity of the ACSM Position Stand leaves the reader with only the most basic guidelines and with no applicable understanding of stretching. Probably most valuable are the comments on the effects on sports performance (inversely affects strength and power when stretching precedes activity) and injury prevention (insufficient evidence to make a correlation), dispelling two widespread, albeit unsupported beliefs, about stretching, especially in yoga. It is highly plausible that the relatively meager content on flexibility is because stretching and flexibility are natural byproducts of movement, physical activity, and sport specific training. Allover flexibility may be considered incidental if one's physical activities are varied enough to preserve the joint ranges of motion with which one is born.

**Table 3. ACSM Stretching Techniques**

| Stretching Technique | Description |
|---|---|
| 1. Ballistic stretching | Bouncing stretches heavily affected by momentum |
| 2. Dynamic stretching or slow movement stretching | Slow repeated movements that progressively increase in range |
| 3. Static stretching<br>a. Active<br>b. Passive | Holding a position for a specified length of time<br>Active: Recruits the agonist muscle to hold the position<br>Passive: Position is held or supported by an external force |
| 4. Proprioceptive Neuromuscular Facilitation (PNF) | An isometric contraction against resistance (usually provided by a partner) prior to a static stretch of targeted muscle to override stretch receptors |

Assuming momentarily that flexibility is a factor solely of the mechanical behaviors of soft tissue, then the thorough study of how stretching affects these properties is warranted. Collagen based soft connective tissues, presented collectively, will begin the investigation. *In vivo*, tendons, ligaments, and fascia behave somewhat differently due to architecture, function, location in the body, and individual genetics. When critical to the conversation, differences will be highlighted, but overall, the *similarities* will be presented. Rather than getting lost in the minutia and overwhelming complexity of similarities and differences, an overview of tissue behavior should immediately equip you to make informed choices about your flexibility routines and your yoga practice, independent of the yoga school of which you are a follower.

## Recommended Reading

(Alter, 2004; Garber et al., 2011)

# Chapter 4. Tissue Mechanics

*"Don't fight forces, use them."*
*R. Buckminster Fuller*

When any material is stretched, it naturally resists elongation. This internal resistance is called tension. The greater the internal resistance, the greater tensile strength a material possesses. When muscles are stretched, they develop tension; not the kind of tension that we complain about and get massaged to eliminate, but mechanical tension. During a forward fold, the hamstrings develop tension as they resist elongation equaling the applied tensile load, ultimately determining their tensile strength. Muscle tension is a totally normal, naturally occurring phenomenon.

Musculoskeletal tissues are not able to self-initiate elongation. Stretching occurs *only* through one of the three force sources previously discussed: gravity, externally applied load, and muscle contraction. Muscle contractions initiate muscle shortening, which in turn may elongate other tissues in the body, including other muscles. A more comprehensive review of muscle contractions will be presented in chapter on muscle. For now, the lever based model where a stretch initiated by muscle contraction occurs on the *opposite* side of the muscle contracting. For example, during a biceps curl, the contraction (shortening) of the biceps brachii applies a tensile load to the triceps brachii and all the surrounding connective tissue, which, in turn, elongate.

## The Stress Strain Curve

Connective tissue is all over the body and plays an integral role in human movement by absorbing the tensile loads associated with changing joint positions. These tissues, made of collagen fibrils, the most abundant protein in the body, can be stretched under a tensile load, or stress (signified by the Greek letter Sigma, σ). The resulting elongation, or deformation, is called strain (signified by the Greek letter Epsilon, ε) (Figure 3). The maximum amount of deformation a material can withstand is called a mechanical limit. If you stretch collagen *within* that limit, it behaves elastically, meaning it readily returns to its original length. If you stretch it *beyond* that mechanical limit, it behaves plastically, meaning it will be permanently deformed. To use an analogy, think of a rubber band stretched beyond its mechanical limit where the structural integrity of the rubber is compromised. In bundles of collagen, permanently deformed presents as damaged tissue, or frayed fibers, which weaken the mechanical capabilities. Continue to increase the stretch and eventually you will get total failure. Your rubber band will snap apart.

The relationship between stress and strain is depicted graphically (Figure 3). Sometimes named a Load Deformation Curve, stress (load) and strain (deformation) are represented by the y-axis (vertical) and x-axis (horizontal), respectively. The various slopes, curves (linear and non-linear portions), areas under the curves, and points literally draw us a behavioral picture of our connective tissues.

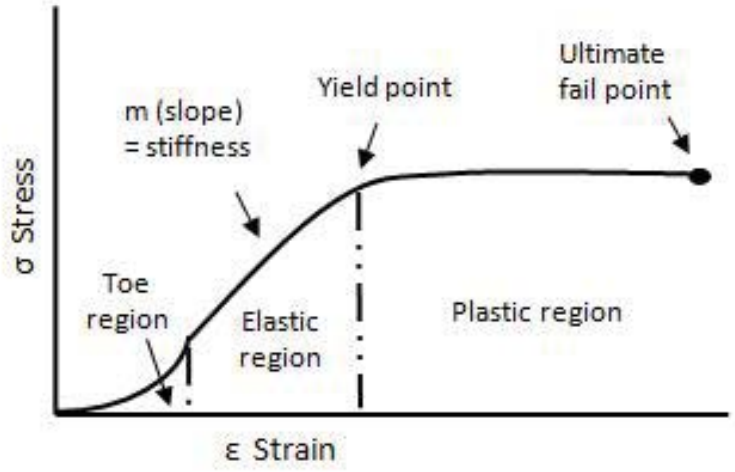



**Figure 3. The Stress Strain Curve**

The graph is divided into three main regions. The first region of deformation is called the toe region. Collagen fibers have a little crimp to them which straightens out upon initial loading returns when the load is removed. Keep stretching and the material yields to the stress a bit more. As long as you stretch below the yield point, the deformation is reversed upon unloading. Moving to the right of the curve, as you move past the elastic region and yield point, any additional stress will cause you to enter the plastic region, where deformation is not reversed. Plasticity in ligaments, for example, interferes with joint stability by compromising the ability to return to its resting length after the movement has ended. Continue to stretch beyond the plastic region, and you will reach the ultimate fail point, full tissue tear. During normal movements, tendons and ligaments extend between about 2-5% (Knudson, 2006) (Table 4).

**Table 4. Connective Tissue Strain**

| Elongation | Region of the Curve | Condition |
|---|---|---|
| 1-2 % | Toe region | Crimped fibers straighten |
| 1-4 % | Elastic region | May return to resting length |
| 4-8 % | Plastic region | Micro-failure, may not return to resting length |
| 8-10 % | Ultimate fail point | Macro-failure, complete tissue damage |

The differing shapes of the curves exhibit the inconsistent response to stress. In the elastic region, deformation is linear, meaning that the amount of strain is proportional to the amount of stress. The crimped toe region is somewhat non-linear (C. Stecco et al., 2009) where lower stresses result in greater strain. In ligaments, the non-linear plastic region, reveals that it only takes a small increase in stress to cause more and more strain. Progressively loaded movement, not excessive elongation, signals collagen production, thereby increasing the capacity to withstand loads. The exact appearance of the stress strain curve varies among individuals partially due to loading history.

The non-homogenous capacity for strain in human connective tissue strain is also due to genetic factors. Women generally have greater capacity for strain, but less capacity for stress (Kubo, Kanehisa, & Fukunaga, 2003; Kubo, Kanehisa, Miyatani, Tachi, & Fukunaga, 2003; Magnusson et al., 2007). Age-related changes in elasticity and collagen may be due partly to changes in activity levels associated with aging and more research is needed to understand the true effects of aging (Kragstrup, Kjaer, & Mackey, 2011). We do know however, that as we age, if we do not load the tissues through stretching and movement, they will become more unyielding (Hall, 2012). The human

body synthesizes and degrades collagen, adjusting the mechanical properties based on demand.

## Mechanical Range

In mechanical terms, range is how much strain (deformation) a material can withstand while stiffness is how much stress (load) something can withstand. A significant misunderstanding around flexibility is the failure to distinguish between range and stiffness. Most people talk about stiffness as something that is inhibiting range - wanting to stretch out or massage away their stiffness. Just as "tension" won't be used to describe muscle pain and discomfort, neither will stiffness. Colloquial uses of these mechanical terms interfere with the understanding of actual mechanics. Our tendons, ligaments, muscles; they are all stiff and should be stiff, to some degree. Joint materials that lack sufficient stiffness are known to be lax. Joint laxity is more a matter of instability and excessive compliance, not the same as increasing the range of the tissue. While both values are derived from the same stress strain curve, they are actually quite different.

Range is how *far* something can stretch, how much it yields, and is expressed by the x-axis of the curve. A curve continuing farther to the right represents a material with a greater range than one with a very short curve (when measured from left to right, assuming the units are the same). As previously noted, the elastic range for

human connective tissues is somewhere around 4% - a range not exceeding 6% is generally advised to avoid substantial injury (Levangie & Norkin, 2011).

The value of strain (ε) on the graph further demonstrates how a material will behave when the range is tested. A low value of strain at the ultimate fail point along the x-axis is called brittleness. Glass, an extremely brittle material, has almost no range and will not deform plastically. Glass will not yield under load and then will suddenly fail and shatter. The opposite of brittle is ductile. Ductile materials have very long horizontal distances on the stress strain curve in the plastic regions. Copper wire, and most metals are excellent examples of ductility: you can deform the material plastically with a high tolerance for failure.[4] Luckily, human connective tissues are not as brittle as glass or trying to touch your toes could prove to have devastating consequences, and not as ductile as copper wire or coming out of a forward bend would be an arduous task.

Connective tissues, neither too brittle nor too ductile, although limited in extensibility or range, should have enough range to perform most functional movements (recall the anesthetized subjects). Adapted mechanical ligament shortness is unlikely the culprit limiting an individual's flexibility. The mechanical term "range" refers the limits of extensibility and is not the same "range" in the ACSM definition of flexibility as having adequate *range of motion (ROM)* (Garber et al., 2011). If your collagen fibers maintain structural integrity at a range of about 4-6%, and normal

[4] Incidentally, malleability, the ability to deform under compressive loads is the corollary to ductility, the ability to deform under tensile loads. Technically speaking, ligaments are the slightest bit ductile and bones are the slightest bit malleable.

movements distend connective tissue between 2-5%, then your stretching activities should probably not need to emphasize pushing the limits of this range. One should, therefore, be keenly aware of a variety of potential factors limiting ROM including, but not limited to articular fibrosis (scarring), osteoarthritis in the joint, neuromuscular interference.

## Stiffness and Elasticity

Stiffness, also called the modulus of elasticity, or Young's modulus, is represented on the linear component of stress strain curve (between the end of the toe region and the yield point) as the ratio of stress to strain, and is represented by this equation: $E = \sigma / \varepsilon$, or the slope of the line ($m=\Delta y/\Delta x$) (Figure 3). The steeper the slope of the elastic region, the greater the stiffness, meaning you can apply more load and get less deformation. Inversely, a gradual slope will indicate less stiffness (greater compliancy), where a smaller load will result in more deformation. In mechanical terms, stiffness is a measure of how much load a *material* (steel, for example) can take before it deforms. In biomechanical terms, stiffness is a measure of how much load a *tissue* (tendon, for example) can take before it deforms. Essentially, stiffness represents how strong and resilient a material is under stress.

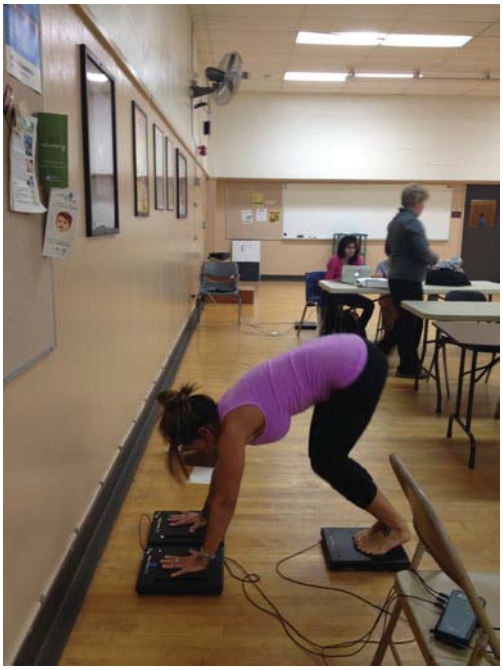
Elasticity is the tendency for a material return to resting length after the tensile load is removed - like when releasing a slingshot. In rhythmic movements such as walking, the connective tissues store and release potential energy to help propel the

limbs, economizing the energy production needs imposed on muscle tissue for movement (Fukunaga, Kawakami, Kubo, & Kanehisa, 2002; Kubo, Kawakami, Kanehisa, & Fukunaga, 2002). The profoundly elastic behavior of human tendons using ultrasound photography has been measured *in vivo*, shedding new light on muscle versus tendon behavior during the stretch shortening cycle (SSC)[5] . During a single movement from dorsiflexion to plantar flexion, the muscle shortened and the tendon-aponeurosis[6] length remained mostly unchanged. But when performed with a prior counter movement (beginning in plantar flexion, moving to dorsiflexion, and returning to plantar flexion), the muscle length remained mostly unchanged and the tendon-aponeurosis lengthened and shortened (Kawakami, Muraoka, Ito, Kanehisa, & Fukunaga, 2002). The potential for tendons to store energy reduces the force output and energy expense required by muscle.

[5] Stretch Shortening Cycle is an active stretch followed by active shortening of muscle tissue, making use of stored elastic energy. Lowering down (flexing the hips, knees, and ankles) before jumping is an example of the SSC.

[6] aponeurosis - a flat, broad tendon-like structure

You apply the Stretch Shortening Cycle when you jump forward from downward dog to standing forward bend. You contract your muscles before you jump, flexing your hips, knees, and ankles before you jump, stretching and elongating your tendons, storing potential energy. The energy is released as your tendons return to resting length and you spring or "float" forward.



Tendons are particularly stiff and elastic due to their positioning and role in longitudinally transmitting muscle force to bone. Progressively overloading muscle will simultaneously load tendons, which will adapt accordingly (Kubo et al., 2009). The threshold for tendon strain which elicits adaptations occurs at higher loads (70% 1 RM)[7] where light loads (50% MVC)[8] and static stretching are insufficient (Arampatzis, Karamanidis, & Albracht, 2007; Konrad, Gad, & Tilp, 2014; Kubo, Kanehisa, & Fukunaga, 2002a, 2002b; Kubo, Kanehisa, Miyatani, et al., 2003; Kubo et al., 2009; Kubo, Kanehisa, Kawakami, & Fukunaga, 2001). Tendon underloading due to a sedentary lifestyle slows collagen turnover, leads to net collagen degradation, reducing both stiffness and elastic; chronic unloading may even result in atrophy (Kjaer, 2004; Reeves, 2006).

Tendon stiffness is a desirable quality. Imagine that a tendon complex is *not* stiff, or compliant, meaning it deforms very easily. The tendon would lengthen, absorbing the distance left behind by the shortened muscle, and the bone would not move. You would have to generate *more* force by contracting the muscle *even more* until finally, the tendon would eventually stop deforming and the bone would move; analogous to feverishly pumping on the gas pedal in your car for minimal displacement. Precious calories and ATP, or fuel, would be wasted on movement. In addition, the greater the stiffness at end ranges of motion, the better equipped you are to avoid injury under unexpected high loads at unexpected angles (Kovaleski et al., 2014a,

[7] 1RM = 1 repetition maximum
[8] MVC = maximum voluntary contraction

2014b). It is important to understand reducing stiffness does improve flexibility; reducing stiffness only results in a lower ratio of load to deformation.

Stiffness, being a mechanical property represented by the stress strain curve, is also affected by genetic factors. Although the literature is amass with contradictory results, age and gender are known to play a significant role (Knudson, 2006; Magnusson, Simonsen, Aagaard, & Kjaer, 1996; Reid & Mcnair, 2004). One study determined that tendon stiffness decreases 10% in the elderly adults, although the full impact of age versus reduced loading habits associated with aging is unknown (Reeves, 2006). High intensity resistance training restored the mechanical stiffness in the elderly subjects, but did not influence the physical dimensions of the tissue (Reeves, 2006). Some evidence suggests women remodel connective tissue at a slower rate and do not respond as efficiently to mechanical loading (Magnusson et al., 2007), and women are more prone to ACL injuries, possibly resulting from less myotendinous hamstring stiffness and thereby less stability (Blackburn, Bell, Norcross, Hudson, & Kimsey, 2009).

Consider your ratio of stretching to strength training. Then consider your personal goals in relation to a sport you play or an activity/hobby you may have. Additionally, consider your goals for aging. Most yoga practitioners need not be convinced of the merits of good flexibility (adequate range of motion). Improvements in tendon stiffness, are however, not often discussed in the yoga class room. The addition of a full body exercise (with load - at least 70% 1 RM), such as a squat, to your movement repertoire may initiate adaptations to those connective tissues and keep you performing well. It is likely that the your bodyweight Chair Pose is not loading your tissues at 70% 1RM. Please consult a qualified trainer if weight training is new to you.

When reviewing the literature on the topics of stiffness and elasticity it is particularly advisable to review the methods sections. Technological advancements in laboratory equipment have improved the measures by which properties such as stiffness can be quantified. Until recently, stiffness could not be measured by the ratio of stress to strain of a tendon *in vivo*, and approximations using range of motion and flexibility tests were standard. Stiffness was frequently measured in the units of torque required to passively move a limb to the end range of motion, telling us nothing about the actual deformation of a specific tissue (Magnusson, 1998). The end range of motion could have been due to hip joint friction but blamed on a "stiff" hamstring which may have easily yielded across a healthy joint. Improvements in ultrasound imaging allow for detailed digital images of tendon displacement during an isometric contraction, resulting in more definitive data. Stiffness, more recently, includes measurements of length in relation to force, which gives a much more accurate value and promotes a clear and mechanically sound discourse. Unfortunately, these methods of data collection fail in a side-by-side comparison as the values are measurements of entirely different mechanisms.

A careful dissection of the definitions of mechanical terms is also advisable as the literature will often use the terms stiffness and elasticity interchangeably, although not formally identical in meaning. Additionally, Young's modulus is not *exactly* the same as stiffness in all materials, but when it is calculated, it may be to normalize[9] for more

[9] For math enthusiasts: to normalize is to mathematically adjust values

accurate comparisons across samples (Reeves, 2006). Young's modulus refers to the stiffness of an elastic material, expressly a linearly elastic material like an inanimate rubber band, which human connective tissue is not. The elastic modulus is derived from Hooke's law which states that the deformation is proportional to the applied load: for each additional unit of load, a proportional unit of deformation will occur.[10] Human connective tissue, is in fact viscoelastic, where the effect of *time* on mechanical properties is pronounced as viscous behaviors blend with elastic behavior.

## Recommended Reading

(Kawakami et al., 2002; Kubo, Kanehisa, et al., 2002a; Kubo et al., 2001; Kubo, Kanehisa, Miyatani, et al., 2003; Levangie & Norkin, 2011)

[10] For math enthusiasts: F=kx; F = force, k=stiffness, x=displacement

# Chapter 5. Viscoelasticity

*"The only reason for time is so that everything doesn't happen at once."*
*Albert Einstein*

## Connective Tissue Properties

Viscoelasticity is a combination of viscosity and elasticity: viscosity is the measure of a fluid material's strain resistance to shear and tensile stresses, elasticity describes a solid material's ability to return to resting length stress induced strain. When a stress is applied, viscous fluids resist deformation and elastic materials readily deform, but when the stress is removed viscous material remain deformed and elastic materials return to original shape. Honey, when compared with water, is an example of a viscous fluid. A teaspoon of honey resists deformation when poured off the spoon whereas a teaspoon of water readily deforms into a long stream. Neither honey nor water are elastic; if they were, upon up-righting the spoon, the fluids would recoil back to the original dollop cradled in the spoon's basin. A rubber band is an example of an elastic material which returns to resting length when the load is removed. To envision a viscoelastic material, combine the qualities of a rubber band with the qualities of honey where the honey slows the elongation of the rubber band and the rubber band aids the honey in returning to resting length. Since human connective tissues are neither exclusively fluid nor solid, they display viscoelastic behaviors.

Viscoelasticity is, additionally, a function of time resulting in connective tissue strain patterns conditional to the duration of the load. Honey gradually stretches off the

spoon into a cup of tea but at an increasing rate over time. Viscous fluids, therefore, inherently resist strain as a non-linear function of time. Upon unloading, viscosity dampens the elastic rebound bringing forth smooth and controlled movements rather than the jerky and abrupt movements that would be the case if our connective tissues were solely elastic; like the damper preventing a screen door from slamming by slowing the rate of elastic recoil. Human connective tissues, as well as the comprising individual collagen fibrils (Svensson, Hassenkam, Hansen, & Magnusson, 2010), exhibit this combination of non-linear viscous behavior over time and linear elastic behavior.

Viscoelasticity is further influenced by both temperature and rate of applied load. A warmer viscous fluid will resist strain less than a colder vicious fluid; warm honey pours off the spoon more rapidly than cold honey. Generally, but not always, a slowly applied load will resist strain less than a rapidly applied load. Try pushing honey through a syringe at different rates; the slowest effort will be met with the least resistance. The science of fluid behavior is overwhelmingly complex, far beyond the scope here, and it should simply be understood that viscoelastic materials are less able to resist strain from warmer and slower than cooler and abrupt loads. Whether human connective tissues are affected by external temperatures (i.e. heated yoga class rooms) has not yet been researched albeit unlikely considering the internal body temperature is both naturally warm and remarkably stable. Very thoroughly researched, however, are the following time and rate dependent phenomena unique to viscoelasticity: creep, stress-relaxation, and hysteresis.

## Creep and Recovery

Creep is the viscoelastic property defined by an initially rapid increase in strain followed by a slower increase in strain at a *constant stress* (σ) over time (Figure 4). Gummy worms, made of hydrolyzed collagen, also display creep when stretched, observed by stretching the two opposite ends of a gummy worm. Following the immediate change in length, the candy will continue to elongate even if you don't increase the force of the stretch. Upon unloading, the worm recovers. In human tissues this response is not limited to collagen fibers and connective tissue but is also present in the muscle-tendon unit (Ryan et al., 2010).

Creep is the phenomenon responsible for slow prolonged tissue strain associated with a sedentariness; sitting in a slouched positions for hours without changing positions. The greatest strain (i.e. steepest slope of the curve) has been measured to occur within the first 15-20 seconds (Ryan et al., 2010), after which the strain slows considerably. When human connective tissues creep, they become temporarily, but not permanently elongated. Recovery time is not instantaneous, the damper effect upon load removal is temporally represented by the graph (Figure 4). Creep is the phenomenon which provides a student with a temporary experience of increased flexibility after a yoga class - until the tissues have recovered.

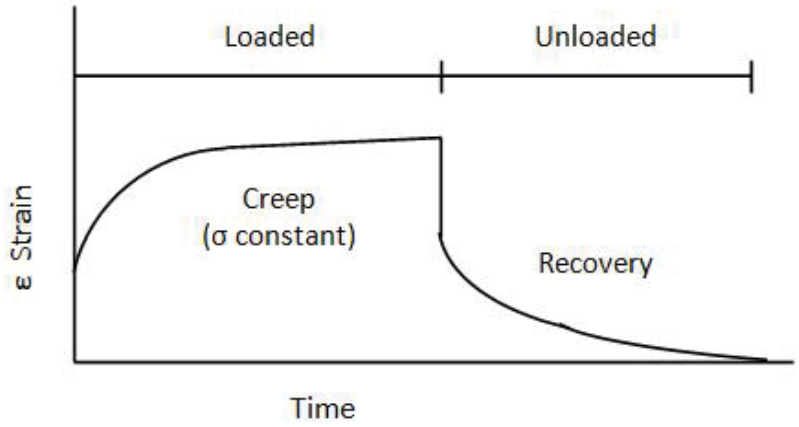
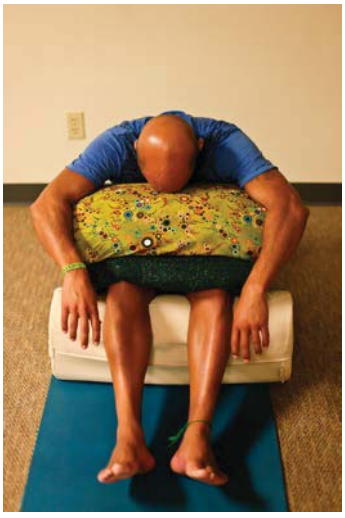



**Figure 4. Creep and Recovery**

A popular form of passive stretching in yoga presumably utilizes creep to improve upon flexibility by promoting passive tensile loading of connective tissue for long periods, usually 3-5 minutes. Students are instructed to minimize muscle activity which will in turn transfer the load to the connective tissue. The plausibility of a truly passive stretch aside (this method assumes passive stretching with little to no EMG activity is achievable), the efficacy of this style of stretching is questionable. The yin yoga teachers assert stretching connective tissue for 3-5 minutes will improve flexibility by increasing the mechanical range. Evidence supports serial deposition of contractile proteins in the lengthening of muscle tissue, thereby increasing muscle tissue range (Zöllner, Abilez, Böl, & Kuhl, 2012), but should not be carelessly extrapolated to connective tissue. The research regarding time dependent non-injurious plastic deformation of collagen fibers is well over one hour at very low strain rates of 1-1.5% (Currier & Nelson, 1992; Schleip, 2003b; Threlkeld, 1992), which are not parameters maintained by the joint angles of the postures and the durations of the stretch. The

alternative scenario is a tissue damaging plastic deformation at high forces (60kg) and 3-8% strain rate, causing tearing, inflammation, and eventually scar tissue formation (Schleip, 2003b; Threlkeld, 1992), none of which suggest increased flexibility by mechanical lengthening due to passive stretches.

During prolonged passive stretches (longer than 60 seconds), minimize creep by using props to control the total strain. The body will get a continuous stretch (tensile load) without continuous elongation. After several minutes in the pose, slowly come up and recover. YES!



## Stress-Relaxation

Stress-relaxation explains the reduction in stress needed over time for a viscoelastic tissue to be held at fixed length (Figure 5). After the initial strain, a quick decline in load needed to hold the length is followed by a slower decline in load over time. The phenomenon can also be experienced when stretching a gummy worm; hold it at a fixed length and your effort will slowly decline. *In vivo*, stress relaxation during dorsiflexion occurs within the first 15-20 seconds by up to 78% (Gajdosik, Lentz, McFarley, Meyer, & Riggin, 2006). In the laboratory setting, human cadaver crural fascia[11], stress decreased by 40% in 120 seconds during constant strain (C. Stecco, Pavan, Pachera, Caro, & Natali, 2013). Environmental conditions, architecture and composition of the tissues, individual variances, and exceptions (increases in stiffness rather than relaxation, called *strain-hardening*, have also been observed), reduce stress-relaxation to little more than an illustrative tool (Currier & Nelson, 1992; Yahia, Pigeon, & DesRosiers, 1993). Connective tissue research in recent decades attempts to explain viscoelastic phenomena and their exceptions by the inclusion of neural control and cellular activity in mechanical behaviors providing insight into how human connective tissues do not behave as inanimate materials and are unceasingly influenced by the nervous system (Findley, Chaudhry, Stecco, & Roman, 2012; Schleip, Jäger, & Klingler, 2012; Schleip, 2003a, 2003b). That is not to say the mechanical properties should be

[11] crural fascia = deep fascia of the lower leg

disregarded: even the sensory nerve endings and cellular behaviors are force dependent and, therefore, requires an overview of force terminology.

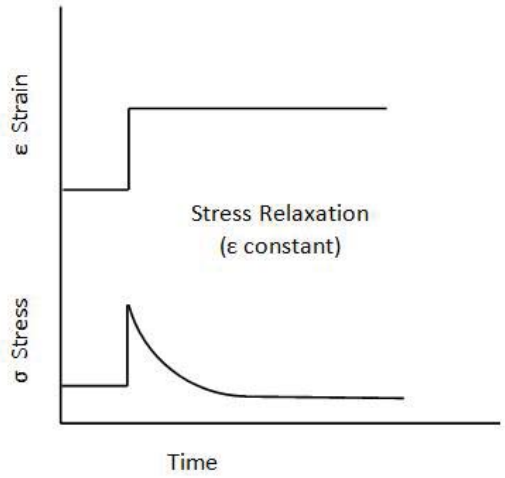



**Figure 5. Stress-Relaxation**

## Hysteresis

The hysteresis graph charts the energy needed to strain the tissue, both upon loading and unloading (Figure 6). During these two stages, the load deformation curve differs; it takes more force to achieve certain length upon loading than unloading, creating a loop with the two unique curves. Less energy is required to unload and the excess (area inside the loop) dissipates as heat. If collagen were a perfectly linear elastic material abiding by Hook's Law, no energy would be lost between the loading and unloading phase and the curves would be represented by an identical, single sloped line both on loading and repeated, albeit in the opposite direction, upon unloading. Collagen is not perfectly linearly elastic (no material *actually* is) and, therefore, not all of the mechanical energy can be conserved for the recoil (unloading) phase and must be

produced by muscle force. For example, a tendon hysteresis value of 22% translates into a remaining 78% of potential energy available for returning the tendon to its resting length; a highly efficient system for cyclical activities such as walking rather than generating muscle force for both positions (Kubo, Kawakami, et al., 2002). The less energy lost (and lower the hysteresis value), the more efficiently your body will move.

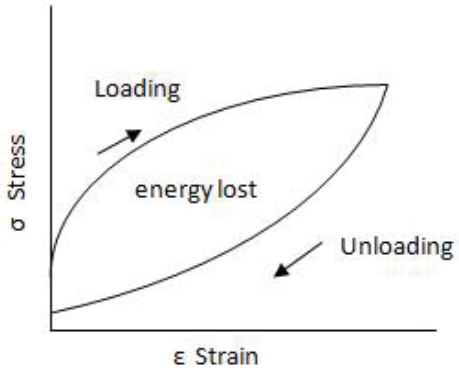



**Figure 6. Hysteresis**

## Cyclic Effect

Hysteresis and stress-relaxation are further defined by a cyclic effect. During hysteresis, as the stretch repeats, the energy lost (area inside the loops) decreases and the load required to reproduce deformation decreases as the number of cycles increases (Figure 7). During repetitive jumping (i.e. Achilles tendon is stretched and released), the first few jumps will be powered more by muscle force than by tendon elasticity. As the number of jumps continue, the Achilles tendon increases its energy storage capacity. Correspondingly, during stress-relaxation, the load required for deformation decreases with subsequent stretches (Figure 8); the Achilles tendon elongates with less muscle force. The combination of these two graphs suggests that

repeated loading and unloading may be an efficient means by which to use stored energy, thereby conserving energy used by muscle activity (Kubo et al., 2001). However, the level at which temporary internal molecular and structural alterations in response to repeated loading contribute to viscoelastic changes in modifying mechanical behavior is not fully understood (Einhorn, O'Keefe, & Buckwalter, 2007).

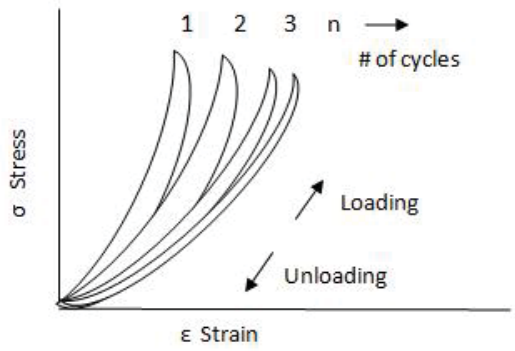



**Figure 7. Cyclic Effect - Hysteresis**

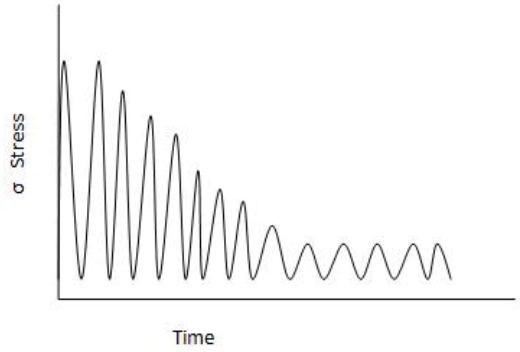



**Figure 8. Cyclic Effect - Stress Relaxation**

Cyclic exercises, such as plyometrics, have shown to be effective training methods in significantly increasing tendon stiffness with no significant change in muscle architecture accompanied by a decrease in the intramuscular connective tissue stiffness. (Fouré, Nordez, McNair, & Cornu, 2011). The investigators suggest that the increased elastic storage capability is a factor of intramuscular decline in stiffness, and the efficiency of load transfer is a factor rise in tendon stiffness (Fouré et al., 2011). Additionally, load transfer efficiency via elasticity is diminished when movements lack grace and ease, and transitions are abrupt (Figure 9). Playful bouncing movements and stealth leaps that absorb energy between transitions produce the sinusoidal shaped graph (Figure 10) (Schleip & Müller, 2013). As with acute changes, chronic adaptations

are also thought to be matter of internal structural alterations (i.e. chemical bonds within collagen fibers), rather than changes in material viscoelasticity (Fouré et al., 2011).

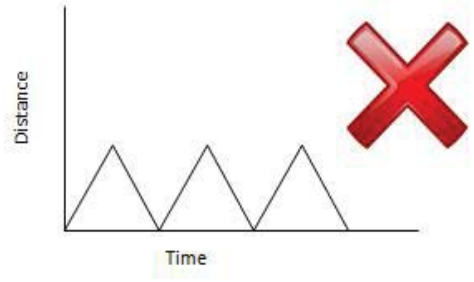



**Figure 9. Abrupt Change in Direction**

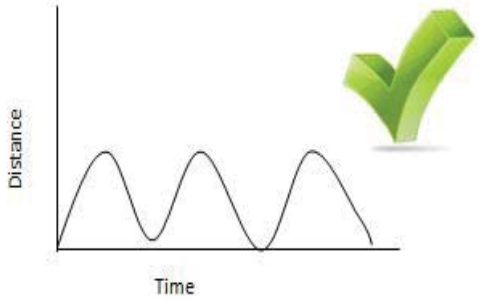



**Figure 10. Smooth Change in Direction**

As with previously discussed tissue behaviors affecting performance, age, gender and training affect viscoelastic properties. Where tendon stiffness significantly decreases with age, hysteresis significantly increases among middle aged and elderly women (Kubo, Kanehisa, Miyatani, et al., 2003). Establishing any steadfast conclusions about averages, however, is nearly impossible due to extreme inter-subject variability (Fukashiro, Hay, & Nagano, 2006; Kubo et al., 2001; Kubo, Kawakami, et al., 2002; Muramatsu, Muraoka, Kawakami, & Fukunaga, 2002). Training improves viscoelastic variables; high intensity resistance training and the cyclic activities such as running have shown to decreases hysteresis, an adaptation which reduces inertia[12] (Reeves, 2006; Schleip & Müller, 2013).

[12] Inertia = resistance to change in speed and direction of motion

Thus far, mechanical behavior of human connective tissues and the effects of stretch have been the focus. However, changes to the internal architecture, including degradation due to age and insufficient loading, and synthesis due to training and progressive overload, are regulated by complex cellular processes. Tensile loads signal these cells, which command the constant connective tissue remodeling process occurring beneath the skin, well beyond our everyday awareness. Connective tissue is far more than the passive component of movement to which it has been relegated in the past.

## Recommended Reading

(Currier & Nelson, 1992; Einhorn et al., 2007)

# Chapter 6. Connective Tissue

*"I believe that more rich golden thoughts will appear to the mind's eye as the study of the fascia is pursued than any other division of the body."*
*Dr. AT Still*

Connective tissue is responsible for the continuity of the body, continuously transitioning one structure *into* another structure. Connective tissues, although given distinguishing names, cannot physically be distinguished. The individual names assigned to structures like tendons, ligaments, and fascia that you see in anatomy books are delineations created by an anatomist to help compartmentalize the multiple roles of connective tissue. Connective tissue, although often referred to as a passive elastic structure, is very much alive with cellular activity and does much more than simply connect things. It is a binding material, holding parts together, while simultaneously a separating material, allowing for movement between structures.

Broadly defined, a connective tissue is composed of living cells surrounded by a nonliving matrix, appropriately named the extracellular matrix (ECM). Connective tissue textures span from solid and firm to soft and pliable to fluid and can be differentiated into three types:

- Fluid: blood and lymph
- Proper: tendons, aponeuroses, ligaments, and fascia
- Supportive: bone and cartilage

The soft connective tissues referenced in this project fall under the "proper" category (and will continue to imply "proper"), but are not as distinct from the "supportive"

category as the above list suggests. Both proper and supportive connective tissues contain collagen as the primary ECM material and the cells which manufacture that collagen are only specialized versions of the same cell. Regardless of similarities and differences, the three types of connective tissue run throughout the body so that no body part or system stands alone and all act as a conduit for communication between the various parts and systems.

Connective tissue proper is a web of fibrous tissue that covers the entire body, surrounds each muscle and muscle fiber, all bones and joint capsules, every body cavity and every organ, even nerve fibers. Connective tissue proper is literally everywhere. Connective tissue proper is entirely continuous with no demarcated beginning and end, making the identification and naming of individual structures more a matter of tradition than an obvious visual distinction. In recent years, the surge of fascia research has made ambiguity around nomenclature particularly apparent; by using the term fascia loosely it cannot be ascertained which structure is under investigation. In 2009, fascia scientists proposed a recommended language and naming of connective tissue proper that included 12 categories of fascia, including aponeuroses, interosseus membranes[13], and the periosteum[14] but excluding tendons, ligaments, and joint capsules (Table 5) (Langevin & Huijing, 2009; Schleip, Jäger, et al., 2012). While such detailed distinctions are essential for scientific study and evaluation, it would prove to be a tedious and futile

[13] Interosseus membrane = a connective tissue membrane between two bones (e.g. radius and ulna of forearm)
[14] Periosteum = connective tissue membrane surrounding bone

task for our purpose here and the term fascia will be used comprehensively, unless specification is warranted.

**Table 5. Fascia**

| Fascial Tissue | Location |
|---|---|
| 1. Dense connective tissue | Tendons and ligaments |
| 2. Non-dense or areolar tissues | Adipose & reticular (deep dermal layer) fascia |
| 3. Superficial fascia | Subcutaneous fascia (under the skin) |
| 4. Deep fascia | Fascia around the muscular system |
| 5. Intermuscular fascia | Fascia connecting muscle organs |
| 6. Interosseal membrane | Fascia between bones |
| 7. Periosteum | Fascia surrounding bones |
| 8. Neurovascular tract | Neural and vascular fascia |
| 9. Epimysium | Fascia around the muscle organ |
| 10. Intramuscular & extramuscular | Fascia within muscle organ |
| 11. Perimysium | Fascia surrounding muscle fascicles |
| 12. Endomysium | Fascia surrounding muscle fibers |

Adapted from (Langevin & Huijing, 2009)

## Fiber Arrangement

More important than the specifics of fascial nomenclature to the overall topic of stretching is the understanding of arrangement. Connective tissue collagen fibers can be arranged loosely or densely and regularly or irregularly. Regular tissues, e.g. tendons, demonstrate directionally organized collagen fibers, whereas the fibers of irregular tissues are multidirectional and may appear disorganized. Proper fascia (deep fascia or fascia profundus), intermuscular fascia (the epimysium), and the intramuscular fascia (the endomysium and perimysium) are more densely arranged than the superficial fascia (which lies just beneath the skin and contains pockets of adipose

tissue). Variation across tissues in inevitable due to structural adaptations to imposed demands, however, the visceral fascia varies the most in density and regularity (Schleip, Findley, et al., 2012; Schleip & Müller, 2013).

The specific fiber arrangement is a response to the mechanical demands placed on the fibers, a conditioning to support future demands of the same nature. Tendons, which longitudinally receive and transmit force from muscle to bone, are more linearly arranged than ligaments, which surround multi-directional mobile joints, and thereby receive and resist forces across multiple vectors. The form occurs *because of* the function and the function further reinforces the form.

Additionally, the orientation of collagen fibers have preferred directionality of tensile loading, as human connective tissues behave differently depending on the direction of load, termed *anisotropy* (Findley et al., 2012). Isotropic materials behave similarly regardless of direction, where anisotropic materials do not. Stretch an isotropic theraband, for example, and the deformation will be identical if the material is stretched horizontally, vertically, or diagonally. Human connective tissue deformation depends on the vector at which the load is applied. In bone, anisotropic behavior is well understood; bones favor compressive forces on a longitudinal axis versus compressive forces on a transverse axis. The research, however, on exactly *how* the anisotropic connective tissue proper behaves is limited due to complex behavior of multi-layered, 3-dimensional, interconnected and architecturally varying nature of connective tissue (Chaudhry, Max, Antonio, & Findley, 2012; Findley et al., 2012; C. Stecco et al., 2009).

The collagen fibers of tendons run longitudinally, receiving load from muscle contractions, and are, therefore, adapted to accept and withstand longitudinal loads. Tendons are anisotropic, meaning the direction of the load determines the behavior of the tendon. During the Splits Pose, students tend to allow gravity to pull them into the pose, relaxing the muscles which would normally load the tendons longitudinally. When the hip flexion and hip extension ranges of motion are not there, the student may turn the pelvis to the side or lean onto on hip (photo top). The external load (gravity) is now obliquely applied to the tendon, a direction that has less capacity to withstand stress. By supporting the pelvis with a bolster, or the thighs with some blocks (photo bottom), the student can be supported while working to keep the stretch active by contracting the muscles of the hip.

## The Effect of Stretching on Fiber Arrangement

When most people think of stretching, they think of muscle tissue and of relieving "stiff" muscles, however, muscles are made up of contractile proteins embedded in layers and layers of connective tissue (inter and intramuscular fascia, or collectively, myofasica) responsive to tensile loading. Myofascia, although looser and more irregular then tendons or ligaments, is also composed of crimped collagen fibers with deliberate directionality. Myofascia, which receives multi-directional tensile loads associated with movement, is arranged in a lattice pattern. The dynamic geometry of myofascia adjusts the lattice framework angles between 20° and 80° to effectively receive and transmit force associated with muscle contractions (Schleip et al., 2006).

Research on the myofascia of rats shows us that daily running results in an organized lattice architecture and the presence of crimp in the collagen fibers (Wood, Cooke, & Goodship, 1988). In just 3 weeks of immobilization in rats, the lattice configuration becomes disorganized and crimp flattens (Järvinen, Józsa, Kannus, Järvinen, & Järvinen, 2002). Crimp allows for changes in length at low load and organization is associated with greater ability to transmit force. In humans, children who jump and bounce playfully with little effort posses organized and crimped myofascia. Untrained adults, who move less elastically, tend to have disorganized and un-crimped myofascia (Schleip & Müller, 2013).

Not all training is the same; the type of stretch reaches different structures based on architecture and location. When muscles are in a relaxed position, none of the connective tissue structures receive a load. During usual muscle work (i.e. weight lifting), the contracting muscle transfers a load longitudinally to the parallel tendon and transversely to the circumferential fascia as the muscle swells. Classic stretching refers to the passive, static stretching characteristic of gentle yoga. Here the load is transferred to the outermost layer of the myofascia (epimysium) and the extramuscular fascia (profundus). The transverse intramuscular fascia don't receive a load, neither do the tendons (Fukunaga et al., 2002; Schleip & Müller, 2013). Passive stretching is, in fact, unfavorable for tendons and reduces stiffness because it is unloaded (Konrad et al., 2014). Active stretching, however, which includes muscle contraction resisting tensile loading, transfers the load the all connective tissue structures with the exception of the transverse intramuscular fascia. Although collagen fibers are a non-living material, it is apparent by the adaptations to loading that connective tissues are more sophisticated than the inanimate materials tested in an engineering lab and living cellular activity is responsible for fiber production, eternally adjusting and altering the arrangement based upon the magnitude, duration, direction, location, rate, and frequency of loading.

Active stretching utilizes muscle contraction to transmit force to the tendon for adaptive capabilities. (Schleip & Müller, 2013). A more thorough discussion of resistance stretching will come later, but for now, it can be thought of as a pandiculation. Pandiculation occurs involuntarily when fatigued or drowsy as in yawning, but can be voluntarily triggered when seeking the satisfying "morning stretch" that mimics how cats stretch - a natural phenomenon where the body tenses and stretches at the same time (Bertolucci, 2011). Animals in nature pandiculate, as in when cats stretch, reaching and tensing simultaneously. Sometimes they claw into the carpet to assist them, other times they leverage the force internally. In Downward Facing Dog Pose, you would "tense" the muscles you are stretching (hamstrings, calves, lats, etc.)

## Components of Connective Tissue

Connective tissue proper, in addition to living cells and a fibrous extracellular matrix is also composed of water, ground substance, and other compounds. The fibers of the non-living ECM are made up primarily of the proteins collagen, elastin and reticulin. The interfibrillar glue-like ground substance contains polysacchrides called proteoglycans (PGs) and glycosaminoglycans (GAGs) (which become PGs when bound to a protein) (Alter, 2004). Other compounds within the extracellular matrix environment are fibronectin, integrins, and laminins which transmit force through physical connectivity between the cells and the ECM, regulating cellular function and interacting with the abundance of water. The combination of these protein and carbohydrate compounds create a mechanically and chemically responsive framework that inhabits cell life, redefining CT as an actively living tissue with adaptability that can be trained and deliberately altered through applied loads such as movement and stretching (Alter, 2004; Halper & Kjaer, 2014; Kjaer, 2004; Meyers, 2009; Schleip, Findley, et al., 2012).

Just as fibrillar arrangement varies, the quantities of the ECM components and the type of cells in each category of fascia and connective tissue proper also varies. In the past, the role of identifying differences among structures was in the hands of the anatomist. Since one tissue literally becomes the next, the scientist would create a dividing line based on macroscopically visual changes and location, identifying one side as unique from the other side. The recently proposed fascial nomenclatures take these

traditional divisions into account but has expanded to include the behavior and disparity of the microscopic components.

## The Extracellular Matrix

Collagen is the most abundant protein in the body, making up connective tissue proper as well as skin, bones, intervertebral discs, blood vessels, and more. (Kadler, Baldock, Bella, & Boot-Handford, 2007). Type-I collagen, in addition to types II, III, IV and VI represent approximately 95% of the 28 types identified to date, all differing in molecular structure depending on function (Kadler et al., 2007; Kragstrup et al., 2011; Schleip, Findley, et al., 2012). Collagen type-I is the most common, making up 80% of this 95%. Types I, III and V appear in long strands. In general, type-I and type-III collagen are found in ligaments and tendons. The crimp in the relaxed state of the fibers of collagen types I and III are straighten when pulled tense - making up the toe region. Other non-fiber forming types of collagen contribute to the structure and function of the three dimensional ECM (Hauser et al., 2013; Schleip, Findley, et al., 2012). The connective tissue properties discussed in prior chapters are the result of the high proportion of type-I collagen in connective tissue proper.

Elastin is the second fibrous protein that works in conjunction with collagen in the extracellular matrix. The recoil capabilities of elastin make it the perfect tissue for skin, blood vessels, the lungs, and of course, tendons. Elastin compliments collagen by providing extensibility and recoil while the stiffer collagen can withstand high loads

(Halper & Kjaer, 2014). Elastin has been shown to extend as much as 100% to 150% its resting length (Schleip, Findley, et al., 2012). Most connective tissue proper contains only 2-5% elastin, although the ligamentum flavum of the spine is made up of considerably more elastic fibers - the purpose for which is unknown as the reminder of the spine is thoroughly braced and enveloped by much stiffer, collagen dominant ligaments (Schleip, Findley, et al., 2012; Zorn, 2007). Recent ideas about elastin question its role since the stiffer collagen is a preferable quality for our musculoskeletal connective tissues. Some suggest it plays a major role in healing, holding the ECM together during tissue tears until the clotting and early stages of collagen production can repair the wound (Zorn, 2007).

The fibers of the ECM are produced by the cells that live within the matrix; the cells literally produce their own environment, like a spider and its web. When collagen molecules are said to arrange in response to mechanical stresses, technically cellular activity is responsible. The collagen does not arrange itself nor are the cells "smart" enough to know how build the ideal environment. Only through the sensation of directional force do the cells "understand" how to build the collagen network around them. The cells, in conjunction with other compounds, continually synthesize and degrade collagen, collectively called connective tissue remodeling.

## Cellular Activity

Fibroblasts are the undifferentiated cells that produce collagen. When a force is felt by a fibroblast, it appropriately differentiates into a specified cell that builds the necessary ECM framework. Fibroblasts differentiate into osteoblasts, chondroblasts, desmocytes and tenocytes which produce bone, cartilage, ligaments or joint capsules, and tendons respectively. (Levangie & Norkin, 2011); all connective tissue has the same origin and is only differentiated over time in response to force (Guimberteau, 2004). When the ECM is stretched, the fibroblasts, housed within, sense the load and signal the remodeling of collagen, elastin, and ground substance (Balestrini & Billiar, 2009; Langevin, Bouffard, Badger, Iatridis, & Howe, 2005; J. Wang, Yang, Li, & Shen, 2004; Weidenhamer & Tranquillo, 2013). The conversion of mechanical forces into the biochemical reactions such as cellular productivity, gene expression, and tissue remodeling is called *mechanotransduction* (Ingber, Wang, & Stamenović, 2014; Kjaer, 2004).

Communication of forces between fibroblasts and the extracellular matrix and its fluid environment, is modulated with the assistance of additional ECM components including collagen, fibronectin, integrins, and laminins. Fibronectin is an extracellular protein which has the ability to bind to collagen fibers and cell membrane proteins called integrins. Integrins are receptors which attach to the cytoskeleton within the cell and fibronectin outside the cell, sensing applied loads and transmitting the force through the cell membrane and into the fibroblast (Halper & Kjaer, 2014; Ingber, 2003,

2008; Kjaer, 2004). Laminins, extracellular proteins which also bind to integrins, modulate the cell normal functions including influencing differentiation and resisting aptosis[15] (Halper & Kjaer, 2014; Ingber, 2003, 2008; Mackey, Heinemeier, Koskinen, & Kjaer, 2008). Called cell signaling, the same collagen fibers can be connected to multiple neighboring cells, allowing for communication among cells across distal locations of the stress sensitive structural environment (Khan & Scott, 2009). This intra and interconnectivity between the cells and the ECM maintains a balance of forces that stretches and shape the cell, maintaining the tensional environment essential for tissue renewal and growth (Chicurel, Chen, & Ingber, 1998; Ingber, 2003).

Collagen synthesis accelerates with loading and exercise, building stronger and more resilient connective tissue (Heinemeier et al., 2007). *Mechanotherapy* is the use of exercise to initiate tissue repair via the mechanism of mechanotransduction (Khan & Scott, 2009). When joint positions change, the resulting tissues stretch. The cells within the tissues also sense the stretch and are signaled to behave according to the characteristics of the load. In healthy connective tissue, a deliberate orientation of the cell helps direct the alignment of the collagen fibers (Boccafoschi, Bosetti, Gatti, & Cannas, 2007; J. Wang et al., 2004). In injured tissues, collagen synthesis is stimulated as the balance of internal cellular tension and external mechanical stretching is disrupted, resulting in a disorganized collagen alignment (Weidenhamer & Tranquillo, 2013). The disorganized fiber arrangement, called fibrosis, is the essential initial

[15] aptosis = programmed sudden cell death

scarring of the injuring that closes the wound. To reduce the likelihood of permanent scarring, which impedes force transduction, fibers must reorganize as cell secrete collagen under a deliberately directed load (Balestrini & Billiar, 2009). Stretching through movement, manual therapies, and eventually loaded exercise therefore aid in tissue recovery and much as in tissue development (Balestrini & Billiar, 2009; Heinemeier et al., 2007; Langevin et al., 2005).

Collagen not only synthesizes, but also degrades with loading. The combined effects are called collagen turnover. Collagen synthesis peaks about 24 hours after heavy loading and continues for another 2 or 3 days and, in response to exercise, collagen degradation peaks and subsides sooner than synthesis (Kjaer, 2004; Kjaer et al., 2009). The result is overall degradation during the first 24 hours, synthesis around 48 hours, and an approximate equilibrium at 72 hours after exercise (Schleip & Müller, 2013). Additionally, collagen half life is approximately 1 year. Translated, this means that in 6 months 1/3 of your collagen fibers have been renewed and remodeled, in 1 year, 50% and in 2 years 75% (Kjaer et al., 2009; Magnusson, Langberg, & Kjaer, 2010; Neuberger & Slack, 1953; Schleip & Müller, 2013). Results with collagen turnover are slow and the reconstruction rate is substantially slower than the decay rate (Einhorn et al., 2007). The impact of loading on connective tissue is not as immediate and obvious as muscle tissue and will take anywhere from 6 months to 2 years to perceive personal increases in mechanical strength resulting from habitual loading.

Where a mechanical stimulus begins the process of remodeling for tissue resilience, unloading does not result in an equal stimulus for degradation (Kjaer, 2004; Kjaer et al., 2009). After two weeks of tendon immobilization, collagen synthesis initiation is not hindered when resuming activity (Moerch, Pingel, Boesen, Kjaer, & Langberg, 2013). It is also understood that loading is not the *only* factor involved in collagen synthesis. In fact, gender plays a definitive role in connective tissue mechanical strength; men possessing greater mechanical properties, due primarily to hormonal influences (Kjaer, 2004; Mackey et al., 2008; Magnusson et al., 2007). The chemistry of enzymes, proteins, hormones, and cells additional fibroblasts gets very complicated very fast and will and be deferred to the more relevant fluid nature of connective tissue (Heinemeier et al., 2007; Kragstrup et al., 2011; Mackey et al., 2008).

## Ground Substance and Water

The sugary interfibrillar ground substance functions primarily as a stabilizer by binding to the fibers of the ECM, cells, and water. It is the relationship of these proteoglycans (PGs) and glycosaminoglycans (GAGs) to water which allows for the adhesive stabilizing properties, absorbing excessive forces on the ECM. PGs and GAGs also aid with the frictionless sliding of collagen fibers, and add density to the interstitial space, protecting cells from penetrating bacteria. The stored water holds nutrients and waste, transporting essential nutrients in and nonessential waste out as interstitial fluid flows. This fluid flow is regulated by the balance of tensional and compressive forces

that occur with movement and exercise. Chronic inactivity retards interstitial fluid flow and the polysaccharides risk dehydration, increasing the glue-like properties, and decreasing the sliding and transporting abilities. Rather than shuttling undesirable waste out, waste may remain trapped within the ECM. Water is required for nearly all biochemical reactions and the interstitial water is particularly dynamic (Meyers, 2009; Schleip, Findley, et al., 2012).

Of the great proportion of water in the human body, 70% is intracellular and 30% is extracellular. Of that extracellular fluid, 2/3 is in the interstitial space of the ECM. As the fibroblasts and integrins stretch and the PGs compress, the water molecules break and form bonds. The ECM and its components act as a sponge, endlessly swelling and squeezing water seeking a state of equilibrium. The fibroblasts transcribe the rate of flow, sensed via the cilia, into chemical and metabolic (Donnelly, Williams, & Farnum, 2008; Ng, Hinz, & Swartz, 2005). At faster rates of flow associated with inflammation, fibroblasts differentiate into myofibroblasts (specialized cells that increase collagen production and possess smooth muscle contractile proteins to help constrict a wound) (Hinz, 2010). Moreover, collagen production aligns in response to the direction of the fluid flow, suggesting that early stages of inflammation provides useful mechanical information for healing tissues (Ng et al., 2005). Ideally, the matrix building myofibroblasts are eliminated by aptosis when the inflammation subsides (Micallef et al., 2012). An overproduction of collagen would result in a pathological and fibrotic scar therefore chronic inflammation should be mitigated. In healthy tissue even tiny

amounts and slow rates of interstitial fluid flow have been shown to regulate expression of the enzymes[16] produced by the fibroblast which degrade and remodel the ECM (Zheng et al., 2012). Interstitial fluid flow is not only integral to injury recover, but also to the everyday process of collagen turnover that maintains the structural characteristics required by imposed mechanical demand.

Water is thought to be the main contributor to the mechanical phenomenon referred to as strain hardening. Typically, mechanical strain is associated with a decrease in stiffness. However, in certain conditions of repetitive loading, the opposite behavior is observed; a temporary increase of stiffness accompanies a stretch (Yahia et al., 1993). During the initial stretch, it has been determined, water is squeezed out of fascia, causing a decrease in stiffness, but upon subsequent loading water absorption rates increase and stiffness increases (Schleip, Duerselen, et al., 2012). Stretching protocols that may enhance strain hardening and the implications of ECM hydration in sports performance have yet to be researched. However, it is clear that the binding capabilities of water to the ECM plays an important mechanical role, elevating connective tissue above simply a static and passive viscoelastic material.

[16] matrix metalloproteinase (MMP)-1 and MMP-2 and tissue inhibitor metalloproteinase (TIMP)-1 and TIMP-2

## Injury and Tissue Repair

Soft tissue injuries occur by way of one or two mechanisms: a single abrupt, forceful impact or repeated lower magnitude loads that weaken the tissue over time. These repetitive stress injuries account for roughly half the injuries associated with exercise and sport (Schechtman & Bader, 1997). In yoga, it is much more likely that your hamstring tendon will fatigue to the repetitive low magnitude load of a forward fold over a number of years than for a single stretch to cause tissue trauma - although not impossible. Sports related repetitive stress injuries can largely be avoided through conservative increase in the magnitude, duration, direction, location, rate, and frequency of the load while considering the 2-3 day turnover rate of collagen. Nonetheless, injuries occur.

Tissue traumas are referred to as sprains and strains, referring to ligamentous and myotendinous structures respectively. One would say "he sprained his ankle" (referring to the ligaments of the ankle), but "he strained his hamstring" (referring to the hamstring tendon); a strain is not a sprain to lesser degree. In reference to tendon strains, a new term "tendinopathy" has been adopted in recent years intended to replace tedonitis or tendinosis, both of which refer to a specific pathology within the tissue that cannot be diagnosed without a histological examination (Abate et al., 2009; Mehta, Gimbel, & Soslowsky, 2003; Sharma & Maffulli, 2006). Consistent terminology is essential to accurate communication among fitness and health professionals to ensure successful protocols for optimal tissue healing (Mueller-Wohlfahrt et al., 2013).

In spite of descriptive anatomical and trauma terminology, not all connective tissues have the same load bearing capabilities. As a whole, tendons are stronger than ligaments, due to both a lower quantity of collagen type-3 (which is weaker than the predominant type-1) and exposure to higher mechanical demands because of location in the body. Some tendons, however, are weaker than ligaments and not all tendons (or ligaments) share the tensile strength of all the other similarly named structures (Thornton & Hart, 2011). Exceeding load bearing capacity and failure to adapt tissue in a timely manner (due to cellular function and ECM turnover) during repetitive loads within capacity are the primary considerations in the origins of tissue injury (Selvanetti, Cipolla, & Puddu, 1997).

Connective tissue injuries are classified by three grades (Table 6) (Levangie & Norkin, 2011). Grades I and II are tissue traumas distinguished by micro-damage ranging from a few torn fibers to a partial tear of the tissue. The tissue is still functional and it may or may not be associated with pain. Nerve endings do not often penetrate the surface of tendons or ligaments, although are plentiful in fascia (more in Chapter 9). As a result, pain is not a good indicator for anatomical damage of grades I or II and can vary greatly among individuals (Abate et al., 2009). A grade III injury is a full tear at the macrosopic level that interrupts the function of the tissue. In some cases, as in an torn anterior cruciate ligament (ACL), the neighboring ligaments are able to keep the joint functional to some degree. In others, as in an Achilles tendon rupture, the joint becomes dysfunctional. An avulsion is a grade III injury characterized by a tearing away

from the insertion point, or bone. A rupture is a grade III injury that occurs in the span between insertion points. Low load repetitive stress injuries progressively leads to any grade. Due to detection of symptoms such as redness, swelling, sensitivity, or restricted ROM during earlier grades, grade III injuries are more often caused by a single over-load (Abate et al., 2009; Selvanetti et al., 1997).

**Table 6. Grades of Connective Tissue Sprains**

| Injury Classification | Characteristics | Location on the Stress Strain Curve |
|---|---|---|
| Grade I | A few damaged fibers, micro-damage | Plastic region |
| Grade II | Partial tear, micro-damage | Plastic region |
| Grade III | Full tear (rupture), macro-damage | Ultimate fail point |

Healing occurs in 3 elaborate and well defined, yet overlapping phases with vaguely distinct transitions (Table 7) (Abate et al., 2009; Bedi et al., 2012; Diegelmann & Evans, 2004; Gurtner, 2007; Kannus, Parkkari, Järvinen, Järvinen, & Järvinen, 2003; Oliva, Via, & Maffulli, 2011; Sharma & Maffulli, 2006). Tendons and ligaments are minimally vascularized, relying on blood flow from neighboring muscle tissue or bone (Kjaer, 2004). The blood from muscle only reaches approximately 1/3 of the way into a tendon and blood sourced from bone does not extend much beyond the insertion point (Sharma & Maffulli, 2006). Thus it can be deduced that a partial tear near the distal or proximal end of the tissue has greater opportunity for healing than a mid-span trauma. It is important to note that healing, or tissue regeneration, should be distinguished from scar tissue formation. The immediate fibrotic scar which closes and seals the wound is only the first phase, but the subsequent phases are essential for restoring tissue

function, although a remolded tissue post-injury rarely returns to pre-injured capacities (Gurtner, 2007; Hauser et al., 2013). The following healing process refers mainly to tendons and ligaments, although damaged fascia would undergo a similar process of collagen repair.

**Table 7. Connective Tissue Repair Phases**

| Repair Phase | Duration | Processes |
|---|---|---|
| Phase I: Inflammatory Response | Begins immediately post injury and lasts 3-7 days | Pain, redness, swelling, temp increase<br>Vasodilation and hematoma<br>Inflammatory mediators regulate<br>Mast cells and macrophages arrive<br>Coagulum initiates repair process<br>Mesenchymal stem cells (undifferentiated cells)<br>Fibroelastic scar builds groundwork for collagen<br>Type-III collagen peaks (signals next phase) |
| Phase II: Repair and Proliferation | Begins day 3-7 and lasts 4-6 weeks | Cytokines released<br>Increase in GAGs<br>High water content<br>Mast cells and macrophages leave<br>Collagen fibrils bundle into fibers |
| Phase III: Remodeling and Maturation | Begins around 4-6 weeks and lasts 1-3 years! | Type-I collagen replaces weaker type-3 collagen<br>Decrease vascularity<br>Cell death ceases excess collagen production<br>Collagen organizes, aligns with axis of loading |

The inflammatory response (phase I) begins immediately after injury and continues over the following 3 to 7 days. Following an initial and immediate vasconstriction, vasodilation floods the area with red and white blood cells (RBCs and WBCs) from outlying sources. The blood coagulates as a temporary band-aid until the tenocytes proliferate and initiate a fibroelastic scar, binding the torn fibers together

again. Mast cells release granules such as histamines and heparin (anticoagulants that regulate the blood clot) and protect against pathogens (infectious microorganisms). Histamines increase permeability to injury site, a benefit of inflammation, allowing for transportation of inflammatory mediators, cells, proteins, hormones, other materials needed for healing. Mesenchymal stem cells (MSCs) assist in tissue reconstruction. Macrophages clean up and digest material waste caused by the injury. Type-III collagen, the thinner and weaker collagen begins forming; the peak of which marks the end of phase I (Abate et al., 2009; Gurtner, 2007; Sharma & Maffulli, 2006).

The repair and proliferation phase (phase II) continues with an elevated collagen type-III synthesis and lasts roughly 4-8 weeks. The macrophages deposit transforming growth factor ß1 (TGF-ß1) which promotes collagen production (Bedi et al., 2012). As collagen content increases, mechanical strength improves, allowing for low loads to mechanically signal directional fiber alignment. The increase in fibroblast activity causes an increase in the release of cytokines, which are a collection of proteins that signal cells to differentiate, produce, and eventually terminate through aptosis. Cytokines include a broad category of mediators such as interleukins (ILs), proteins such as the MMPs and their inhibitors TIMPs, and several additional growth factors (GFs) (Bedi et al., 2012; Oliva et al., 2011). Cellular biology is well beyond the scope of study here, yet the incredibly complex chemical and molecular processes should be momentarily appreciated. GAGs and water are in abundance, simultaneously binding and unbinding, regulating molecular and cellular movement within the wound site (Kosir, Quinn, Wang,

& Tromp, 2000). Collagen fibers bundle together, increasing thickness and diameter, and arrange longitudinally, increasing length. The associated increase in material strength marks the transition into the next phase (Abate et al., 2009).

During the final and longest phase of healing, remodeling and maturation of collagen fibers are the dominant function. This 2-3 year long process is first marked by an increase in fibroblast volume as the resilient collagen type-1 replaces the weaker type-3. After fibrotic production reaches a peak, fibroblast volume reduces by aptosis and the collagen matrix forms cross links and aligns along the axis of loading. The increase in the tissue's capacity for tensile loading and a decline in vascularity marks the later stage of phase III (Abate et al., 2009; Gurtner, 2007; Sharma & Maffulli, 2006). Mechanical demands to signal collagen organization during phase III is essential.

Traditionally, the protocol for connective tissue injury rehabilitation involved rest and immobilization during phase I and II. Recently, these methods have been questioned, particularly in regards to immobilization. Immobilization is associated with disorganized collagen arrangement which has been shown to lack mechanical strength and reduce mechanical stiffness (Hauser et al., 2013; Järvinen et al., 2002; Killian, Cavinatto, Galatz, & Thomopoulos, 2012). Research shows that early controlled mobilization after injury, even as early as the final part of phase I, result in better healing outcomes, and that classic muscle contractions and resistance stretching can stimulate collagen production and fiber alignment; the foundation of mechanotherapy (Boyer, Goldfarb, & Gelberman, 2005; Hauser et al., 2013; Järvinen et al., 2007; Järvinen,

Järvinen, & Kalimo, 2013; Kannus et al., 2003; Khan & Scott, 2009; Killian et al., 2012; Kjaer & Heinemeier, 2014; Prado et al., 2014). While movement immediately post injury should be restricted to allow for fibroelastic scar formation and avoid re-tearing, unloaded movement should gradually be introduced as soon as the scar can withstand associated loads; early mobilization, when compared with immobilization, lends to a more effective healing response, significant improvements in wound site stiffness and strength, and fewer adhesions (Boyer et al., 2005; Gurtner, 2007; Järvinen et al., 2007, 2013; Kannus et al., 2003; Killian et al., 2012; Kjaer & Heinemeier, 2014; Prado et al., 2014; Sharma & Maffulli, 2006). Excessive stresses or strain during phase II and even phase III, however, may re-injure the tissue, reigniting the inflammatory response of phase I. This cycle of recurring inflammation and scarring is referred to as *smoldering fibrogenesis* and results in a disorganized, weakened collagen matrix (Thornton & Hart, 2011).

Cryotherapy, or the topical application cooling agents (such as ice), is another common treatment for musculoskeletal injuries which has recently come under fire due its anecdotal origins (van den Bekerom et al., 2012). In laboratory rats, ice has been shown to reduce inflammation, a crucial phase of healing that should not automatically be curbed (Deal, Tipton, Rosencrance, Curl, & Smith, 2002). Reviews of human experiments, however, are less clear in the effect of icing, which may not penetrate the dermis at a level deep enough to influence inflammation and seems to provide mostly an analgesic effect (van den Bekerom et al., 2012). In healthy human subjects, an ice

bath immersion reduced response times and neuromuscular control in the immediate 30 minutes post-cryotherapy, suggesting that activities requiring sharp movement skills be suspended until neuromuscular function resumes (Macedo, Alonso, Liporaci, Vieira, & Guirro, 2014). Applying heat to an injury is another form of thermal treatment that seems to have an analgesic effect and temporarily increases flexibility (Bleakley & Costello, 2013). Injured subjects who apply ice may resume normal activities more quickly than those who apply heat, an average of 13.2 days versus 33.3 days, respectively (van den Bekerom et al., 2012). When considering temporal factors for tissue regeneration after an injury, however, normal loading during earlier phases may not be better. While a definitive conclusion about thermal treatment and mobilization after injury cannot be made, protocols can be selected based on the available research on tissue healing and compared with individual needs to choose the best available options to promote the most favorable conditions (van den Bekerom et al., 2012).

When stretching and sheer forces are adequately applied, the results can influence inflammation, fluid flow, and fibrosis. Inflammation not only delivers cytokines to the wound site, but is also associated with increase rate of interstitial fluid flow. Sheer forces in the form of vigorous foam rolling and massage techniques would only cause faster flow rates, signaling myofibroblast differentiation, potentially increasing fibrotic scarring; not an effective treatment protocol if attempting to reduce scar tissue, effective if wanting to stimulate collagen production. Ultra-slow and light massage would decrease fluid flow rate, signal aptosis and give directionality for fiber

arrangement (Schleip, Findley, et al., 2012). Slow passive stretching in as little as 10 minutes twice per day, has been shown to reduce inflammation and fibrosis in laboratory rats (Corey, Vizzard, Bouffard, Badger, & Langevin, 2012). Of course, research on humans and passive stretches associated with yoga is needed to make any meaningful conclusions about the effects of yoga on scar tissue formation or elimination.

Suggested foam rolling techniques:

- To treat scar tissue, strokes should be slow - 1 millimeter per breath cycle (inhalation *and* exhalation).
- To increase tonicity of connective tissue, strokes should be fast - the length of tissue in one direction per breath cycle (inhalation *and* exhalation).

When most people think of stretching, they think of gaining flexibility (improving ROM) and the redistribution of tensile loads, which are the product of movement, is overlooked. The present investigation has begun to shed some light on alternative target goals of stretching including increasing tissue resilience with adequate loading, *not* increasing mechanical range, influencing fiber arrangement and injury repair via mechanotransduction. A more global, less dissected view of movement and stretch demands more global, less dissected view of anatomy. Only then can the human body's relationship to tension and stretch be fully understood.

## Recommended Reading

(Langevin & Huijing, 2009; Schleip, Findley, et al., 2012; Schleip, Jäger, et al., 2012; Schleip & Müller, 2013)

# Chapter 7. Tensegrity: Tensional Integrity

*"You never change things by fighting the existing reality. To change something, build a new model that makes the existing model obsolete."*
*Buckminster Fuller*

Art and architecture tangibly express what we observe in nature and in our own form. Our engineering developments are only limited by our understanding of how to manipulate and utilize compressive and tensile forces. A structure designed with too much compression implodes, just as too much tension tears it apart. An equilibrium of forces driven by an internal communication of load distribution, a regulation of tension and compression, stabilizes a structure independent of external environments. Such an equilibrium is the basis for a tensegrity system.

Historically, humans designed column structures that stabilized through the continues compression of its parts, perhaps because the available construction materials could best withstand compressive forces and gravity, particularly when stoutly assembled. The massive Gothic churches and Egyptian pyramids are a testament to the solidity and permanence of compression structures. Likewise, anatomists have historically explained our bodies to be continuous compressive structures, where the stacking bones of the skeleton provide us with our stability. Our spines have been described as a columns, the discs behaving as mortar, connecting adjacent structural components. Recent technological advances in materials exhibiting extreme tensile durability, however, has made tension structures like suspension bridges possible (Pugh,

1976). The availability of new materials has spurred a growth of tensional architecture, changing how we understand mechanical design, both manmade and in nature.

R. Buckminster Fuller[17], the architect, designed equilibrium systems that balanced the robustness of compression and the durability of tension. He garnered fame with the geodesic dome, a structure that brilliantly utilized the inherent stability of geometric shapes. Struts under compression combined with tendons[18] under tension arranged in geometrical configurations withstand considerable forces. Independent from gravity and a capacity to accept loads in any direction, a tensegrity system can maintain its structure even when tilted on any axis (Guimberteau, 2005; Pugh, 1976). The interaction and transmission of forces through interrupted compressive components and uninterrupted tensile components establish and maintain a stable balance of forces where an increase in compression causes an increase in tension (Pugh, 1976). This continuity of tendons and discontinuity of struts is the defining aspect of a tensegrity structure, appropriately named by combing the words *tensional* and *integrity*.

The human form similarly relies on geometry and a shared distribution of loads. In the tensegrity model of the human body, bones are the discontinuous "struts" pushing out and getting compressed, where connectives tissues are the continuous tensile "tendons" pulling in, and getting stretched (Figure 11). The suspension of the compressive parts within the tensile surroundings is seen in every aspect of the human body from the cytoskeleton and membrane of the cell to the vertebra, discs, and

[17] Buckminster Fuller Institute http://bfi.org/
[18] In architecture, the tensile components of a tensegrity system are also called tendons.

ligaments of the spinal "column" (Chicurel et al., 1998; Ingber et al., 2014; Ingber, 2008; Vora, Doerr, & Wolfer, 2010; N. Wang et al., 2001). Globally, the human form demonstrates independence from gravity by maintaining shape when standing on our heads, standing on our hands, or lying flat on our backs; the stability of a tensegrity structure depends on all of its parts, not just those at the foundational level. Quite a departure from the continuous compression model described by anatomists of the past!

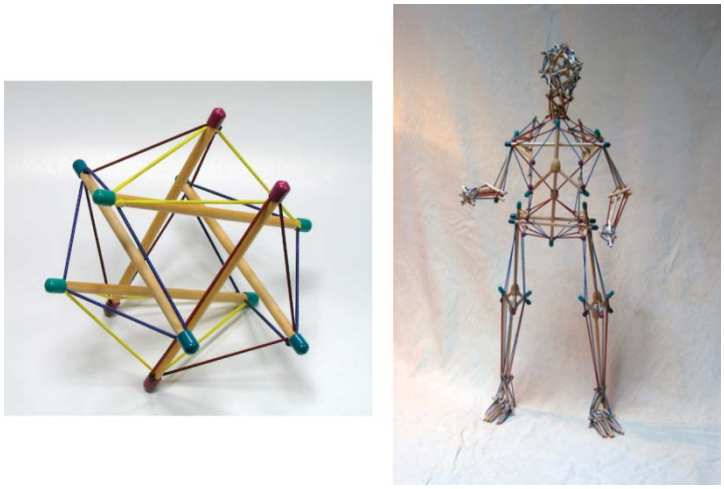




**Figure 11. Tensegrity Models**

The basis for mechanotransduction lies in the pre-stressed tensional and compressive construction of the cell. Cell signaling via integrins and fibronectin expands the tensegrity system outward to another degree. Continued connections, or adhesions, to yet more distal structures continue to create tiers, somewhat of a hierarchy of force communication (N. Wang et al., 2001). The physical and chemical stability of a cell depends each level interaction, from those closest to those most

distally. A theoretically "unloaded" cell is suspended in a pre-stressed homeostasis based on the current environment. Moving and stretching our limbs shifts the equilibrium of the cell until loading ceases, whereupon the cell returns to its natural state. Characteristics of load (magnitude, duration, direction, location, rate, and frequency) alter cellular activity. The significance of the tensegrity model, called biotensegrity when referencing living structures, is that explains the mechanism by which movement is essential to overall health and wellness of the human body, beginning at the cellular level (Ingber, 2008).

Additionally, the human body is filled with fluid in every otherwise empty space; cells, tissues, and organ are all partially fluid. The human body is approximately 60-70% water, 70% of which is contained within cellular walls. (Schleip, Findley, et al., 2012). The tensegrity model does not account for the mechanical laws of fluid dynamics and rheology[19], particularly in regards to the fluid rich cells, which are no longer viewed simply as fluid sac floating within an extracellular space, but rather as a combined rheological and mechanical entity whose shape influences and is influenced completely by its surroundings.

Just as the cell exists in a pre-stressed tensional state, so does the entirety of our form. The "global inter-tissue tension" is present in every element, from collagen fibers to cell membrane, including hydrogen bonds and even muscle tone (Guimberteau, 2005). Viscoelastic stiffness of the collagen fibers is just one contributor to the pre-

[19] rheology = the study of how non-Newtonian fluids (liquid matter and soft solids) flow

stressed state, cellular stiffness and strain hardening are others (Ingber et al., 2014; Schleip, Findley, et al., 2012). Contracting muscle proteins regulate the state of pre-stress by increasing tension on the neighboring collagen tissues, as is apparent by the muscular insertions directly into the fascia lata (Huijing, Maas, & Baan, 2003). Minimal muscular force generation has been shown to improve shoulder joint stability (measured by stiffness and viscosity increases), acting as a damper, controlling and polishing movement (Zhang et al., 2000). Through muscle excitation, we adjust the multi-directional tensional load on *all* of the connective tissues, beyond those in the immediate vicinity.

## Continuity and Connectivity

One of the first mantras you learn when studying anatomy is "tendons connect muscle to bone, ligaments connect bone to bone." This model classifies tendons as in-series, and ligaments as in-parallel structures in regards to how muscle moves bones. When observed in parallel, as dissection is traditionally performed, the ligaments would be slack on the inactive, flexed side of the joint angle. A new model has been presented by van der Wal (2009) who approached a dissection from a new perspective, with function in mind. Ligaments, when observed in series to the muscle tendon unit, are under constant compressive or tensile loads, thus able to act in all joint positions (van der Wal, 2009).

*"Most deep and superficial RDCT [regular dense (collagenous) connective tissue] layers (as muscle compartment walls) are organized in series with muscle fascicles. Collagenous fibers running from bone to bone—thought to be stressed passively by displacement of the articulating bones—hardly occur. Instead, there occur broad aponeurotic layers of RDCT to which relatively short muscle fascicles insert, which, on the opposite side, are directly attached to skeletal elements. Such configurations of muscle fascicles attached to the periosteum of one articulating bone and via a layer of RDCT indirectly attached to another articulating bone, could be considered "dynamic ligaments." Such "dynaments" are not necessarily situated directly beside the joint cavity or in the deep part of the joint region. By describing the dynament as an architectural unit of the musculoskeletal system, we mean a unit of RDCT connected to the periosteum of a skeletal element with muscle fascicles in series attached to it."* (van der Wal, 2009)

Alas, a departure from the classical lever system of movement is long overdue. Within the framework of the biotensegrity model, muscle activity does not occur in the body in the same way appears in the anatomy books where individual muscles are described by the joint articulation they command. For example, the single joint action of flexing the elbow has classically been explained as an action solely of the elbow flexor

muscles. Muscles, however, don't act individually. Try engaging the quadriceps of one leg now. You will feel non-localized tensional change in your lower back, buttocks, lower leg, feet and more. The muscle force you generate is communicated through connective tissue in all 3 dimensions, conserving energy by modulating appropriate stiffness, mobility, and stability when moving a joint (Scarr, 2012). Through the deepest layers of myofascial connective tissue, force is transmitted radially to larger structures, both proximally and distally. Anywhere in the human body, local changes in compression (the flexed biceps brachii) elicit global responses in tension; loads distribute to all components, even those most distal (Guimberteau, 2005) as the forces travel along the fascial network to any and all structures (Vora et al., 2010).

The deep fascia, in particular, a previously discarded tissue in dissection labs and histology research, is now intricately studied for its role sensory input and motor output (Findley & Shalwala, 2013). The inclusion of intramuscular fascia into movement analysis tells us that the muscle acts on far more than simply the origin and the insertion (Day, Copetti, & Rucli, 2012). Roughly one third of the force produced by a muscle is transmitted laterally through the epimysium and perimysium to other structures and muscles, including antagonist and synergists (muscles with "opposite" and "supporting" actions) substantially reducing the amount of force transmitted longitudinally to the tendon unit (Huijing & Baan, 2008; Huijing et al., 2003; Huijing, van de Langenberg, Meesters, & Baan, 2007; Huijing, 2009; Passerieux, Rossignol, Letellier, & Delage, 2007; Patel & Lieber, 1997). The perimysium has been described as both a tubular lattice and

honeycomb elastic structure with four distinct organizational strata that adjusts tension based on attached muscle fiber insertion (Passerieux et al., 2007). Previously, movement was thought to be well timed and executed interaction of muscle contractions in relation to other muscle contractions. Today, the known viscoelastic properties of intramuscular fascia is understood to capture, store, and release (elasticity) muscle force. Movement is now thought to be a well timed and executed interaction of muscle contractions in relation to the mechanical behavior of fascia and connective tissue (Smith, 2006). Fascia has even been revolutionarily declared the antagonist to muscle (Smith, 2006).

Moreover, muscles fibers are not definitively contained within the boundaries of the muscle organs depicted in anatomy textbooks. Muscle fibers insert into non-tendinous connective tissues as seen with the gluteus maximus (GM) muscle which inserts primarily (entirely in some individuals) into the iliotibial band (ITB) of the fascia lata as opposed to the femur (Huijing et al., 2003; Langevin & Huijing, 2009). GM fibers, as well as but not limited to latisimus dorsi, erector spinae, multifidis and transversus adbominus act to tense the layers of thoracolumbar fascia (Vleeming, Pool-Goudzwaard, Stoeckart, van Wingerden, & Snijders, 1995; Willard et al., 2012). Layers of myofascia merge longitudinally to become tendon, but also laterally to become deep fascia and the periosteum, to become ligaments and joint capsules. Fascia does not start and end at muscle fibers; it begins at skin, our most superficial organ and webs through the body

literally merging with and becoming bone. The division between structures in your anatomy books is a construct of the anatomist - they are not real.

The muscles of the lower limb interact with the muscle of the upper limb and spine by transferring load through the thoracolumbar fascia (TLF) and into other structures, expanding the classical categorization of "back muscles" well beyond the immediate spinal region (Vleeming et al., 1995; Willard et al., 2012). The mechanical tension within the thoracolumbar fascia is regulated by muscle contractions and relaxations (Vleeming et al., 1995). Muscles with fibers inserting directly into the middle layer of the TLF include the transversus abdominus, biceps femoris, gluteus maximus, erector spinae and multifidis. Additionally, the TLF becomes increasingly stiff upon repeated loading caused by strain hardening due to hydration changes in the extracellular matrix (Willard et al., 2012). A flexed spine narrows and lengthens the TFL, in turn storing energy to maximize efficiency of the spinal erectors when extending the spine (Willard et al., 2012). Spinal muscles are not large force generators because they are very generally short and very close to the fulcrum. Mathematically, the accepted biomechanical model of the spine could not explain the loads able to be carried by the human spine (Gracovetsky, 2008).

The inclusion of the lumbodorsal fascia (another name for the thoracolumbar fascia, depending on the scientist) into the new spinal model supported the calculations. The phenomenon is called "muscle relaxation" or "flexion relaxation" (Gracovetsky, 2008). When the spine flexes between 45-90°, the lumbodorsal fascia is pulled taut

enough take on the majority of load allow the spinal muscles to relax (measured by electromyography silence). The load is transferred between the lower and upper extremity via the lumbodorsal fascia. Upon return from flexion, the lumbodorsal fascia slackens enough at about 45° for the spinal muscles to pick up the remaining, more lightly loaded completion of the lift.

Interestingly, chronic low back pain (CLBP) patients have been shown not load share in the same way as healthy patients during the flexion relaxation response. Subjects with CLBP tend to show continuous spinal muscle activation through all degrees of flexion, unable to distribute the load through the passive connective tissue and thereby placing greater demand on the muscle tissue (Colloca & Hinrichs, 2005). Further, viscoelasticity must be considered. Because of creep, prolonged spinal flexion (just 5-10 minutes) will continue to deform the lumbodorsal fascia, reducing the ability to transfer load. Upon returning from prolonged flexion, the spinal muscles will need to compensate for the excessively stretched lumbodorsal fascia (Shin, D'Souza, & Liu, 2009; Shin & Mirka, 2007). Fatigued spinal muscles with a reduced capacity to bear the load may be a contributor to CLBP (Shin et al., 2009). The lumbar flexion induced by prolonged sitting (for just one hour) is enough to interfere with normal flexion relaxation patterns (Howarth, Glisic, Lee, & Beach, 2013). Regular periods of recovery may mitigate creep and reestablish the flexion relaxation response (Shin & Mirka, 2007).

A yoga intervention prescribing mostly forward folds and twists was effective in reducing the symptoms of chronic low back pain (K. Williams et al., 2009). Classically, forward bends in yoga are taught with a "neutral" spine and flexion is advised against. Only the low load Cat/Cow Pose moves the spine through full range of flexion and extension. Research shows, however, that healthy backs are able to transfer load to the lumbodorsal fascia between 45-90° of flexion. Sequentially flexing the spine through every segment of the spine is a motor skill that may be developed. Consider gradually increasing the load in a flexed spine positions by adding controlled roll-ups and roll-downs. Please consult an experienced professional if spinal flexion is new to you.

In yoga, flexing the lumbar spine is typically avoided due to the belief that a flexed spine places undue pressure on the vertebral discs. Considering the tensegrity model, it is unlikely that load would be transferred solely to the discs. The fibrocartilage discs are collagen based structures, continuous with the collagen ligaments of the spine and the collagen lumbodorsal fascia (Colloca & Hinrichs, 2005). Where the viscoelastic responses caution against prolonged flexion, complete avoidance of flexion could potentially have its own interference in the combined functionality of muscle and connective tissue. Knowing that collagen adapts to the demands placed upon it, if we never flex our spines (even with increasing load) the lumbodorsal fascia may degrade in the absence of sufficient loading support adequate load transfer. Furthermore, despite urban legend, squat lifting and stoop lifting do not place significantly different compressive and shearing loads on the spine (Nordin & Frankel, 2001; van Dieën, Hoozemans, & Toussaint, 1999). The recent fascia and tensegrity research explains the mechanical logic behind these findings (Gracovetsky, 2008) Position, posture, or yoga pose should be selected by the function it is intended to achieve. Mechanotherapy may require more spinal flexion than has been classically taught in yoga classes.

The involvement of all structures in force transmission across any given joint position is widely accepted, while simultaneously under intense scrutiny (Huijing & Baan, 2008; Ingber et al., 2014; Maas & Sandercock, 2010; Meyers, 2009; Passerieux et al., 2007; Schleip, Findley, et al., 2012; A. Stecco et al., 2009; van der Wal, 2009). If the old model, the free body diagram (Figure 12), is now reduced to a simplistic

homogeneous model, a new model for heterogeneous problem solving where quantifying muscle activity is dependent upon the complexities of 3-dimensional myofascial force transmission should be closely and cautiously analyzed. Yet, the tensegrity model is not without limitation as human tissues are constantly in a dynamic state of turnover, influenced not solely by mechanical, but also by chemical, thermal and rheological processes (Ingber et al., 2014). During these early stages of discovery, in particular, we should avoid generalizing localized research findings to the entire musculoskeletal system (Maas & Sandercock, 2010). For example, the evidence that force may be transmitted across locations where connective tissue is firmly anchored into bone via the periosteum is lacking (Meyers, 2009). Such theories provide a foundation for forward and radical thinking, but are prone to become "buzzwords" which dilute scientific understanding among the mainstream. The question of how, and if, a tensegrity model will translate into a practical application for the movement professional is still unclear. Perhaps new insights into injury recovery, allowing the structure to operate, albeit at a lower level of performance, during the healing process may be revealed (Maas & Sandercock, 2010). Regardless, the presence of debate of the biotensegrity model within the scientific communicate tells us that have entered a new era in the understanding of human movement and the questions open to investigation based on a tensegrity model of the human body are extensive.

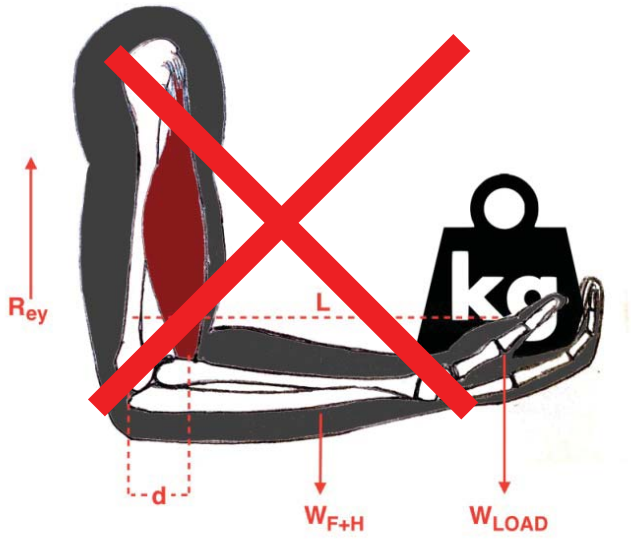



**Figure 12. Free Body Diagram**

## Recommended Reading

(Meyers, 2009; Pugh, 1976; Scarr, 2012; van der Wal, 2009; Vora et al., 2010)

# Chapter 8. Muscle Tissue

*"An effective human being is a whole that is greater than the sum of its parts."*
*Ida P. Rolf*

Skeletal muscle tissue, although fully embedded within the network of connective tissue, is unique for its ability to respond to a stimulus and generate force, called excitability (or irritability). A stimulus in the form of an electrical impulse from the Central Nervous System (CNS) signals a chemical reaction in the muscle tissue. An energy system then provides the fuel for the muscle to generate force which it relays to surrounding tissues via the CT. Muscle tissue is referred to as the *active component* of the musculoskeletal system because of its excitability. Connective tissue is referred to as the *passive component* because it is governed by the mechanical forces to which it is exposed. Based on the tensegrity model, active and passive components are indiscrete as there is constant mechanical interaction. Muscle plays a major role in collagen viscoelastic behavior, including stiffness, as muscle force is transmitted to the connective tissues (Kawakami et al., 2002). If connective tissue is the marionette, muscle tissue is the hand to provides the leverage on the strings to create movement.

## The Motor Unit

A motor unit is comprised of a motor neuron and all of the muscle fibers ( muscle cells) it innervates. Each neuron (nerve cell) emerges from the spinal cord and divides into smaller branches that terminate at the muscle cell membrane. This point of

contact is called the neuromuscular junction (Figure 13). A single neuron reaches 10s to 100s to 1000s of fibers. The number of muscle fibers per motor unit determines the degree of control and precision. An eye muscle may be activated by as a little as one muscle fiber per neuron, whereas a quadriceps neuron may stimulate several hundred fibers.

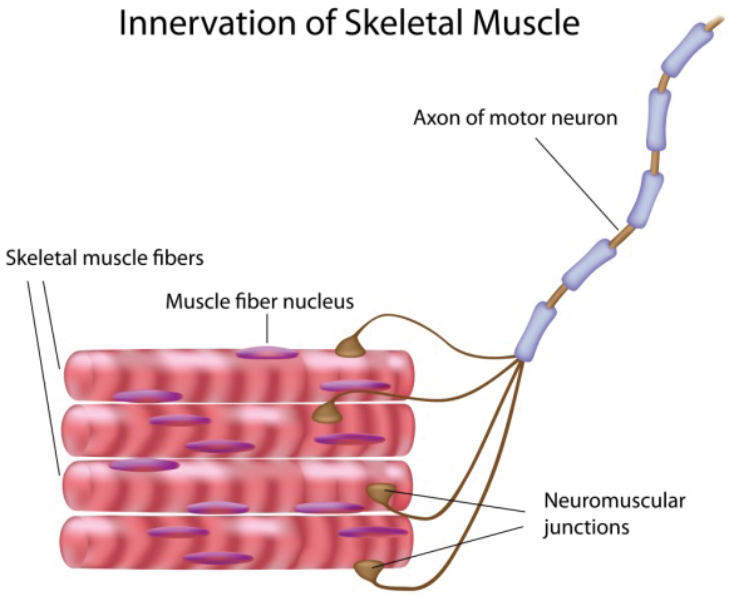



**Figure 13. Muscle Innervation**

All the muscle fibers in a motor unit contract together when a motor neuron is stimulated, referred to as the *All-or-None Principle*. A stronger signal from the neuron will not result in a stronger muscle contraction. Only additional neuromuscular signals, thereby firing an increased volume of muscle fibers, will result in a stronger contraction. Only the number of motor units needed to generate the force to complete the job at hand will fire. While the same motor units prefer to fire, when the fibers fatigue, the

motor units involuntarily take a break and others pick up the slack (Bawa & Murnaghan, 2009). Motor unit activation is governed by movement and position rather than by distinct muscle organs.

To explain, the neuromotor system does not understand what a hamstring is, it only understands the movement. The motor units recruited for a specific movement are not dictated by the name of the muscle organ, but by the desired movement. When moving from standing into a forward bend, the brain fires the motor units necessary to achieve the movement, *not* simply the "hip flexors" as if we were designed with single plane muscle actions in mind. Other muscle groups are all working in balance to mobilize and stabilize in 3 dimensions in order to achieve the desired movement. Likewise, one cannot stretch an isolated muscle organ (e.g. only the hamstrings).

A large portion of the research uses a straight leg raise test (SLR), where the subject lies supine and one leg is passively raised until the end range of hip flexion, as the preferred method to measure hamstring flexibility. Research on strain patterns in cadavers showed substantial strain when compared to the hamstring strain in the lower leg, foot, the IT band (240% strain), the achilles tendon (100%), the plantar fascia (26%), and the lumbar fascia (145%) - even on the other side of the body (45%) (Franklyn-Miller et al., 2009). A similar study in both cadavers and *in vivo* showed significantly less IT band strain in the live subjects where neuromuscular activity was a factor (Clark et al., 2009). To assume the SLR is providing information about the behavior of the hamstrings is to assuming a single muscle can be stretched and overlooks the global aspect of

movement. The true function and behavior of muscles, linked with antagonists and synergists, never be understood if studied in isolation (Huijing & Baan, 2008; Huijing et al., 2003, 2007; Maas & Sandercock, 2010). When joint positions change in 2 dimensions, structures in 3 dimensions are involved. Muscle names, are therefore, the result of an arbitrary compartmentalized, albeit standardized, method of dissection useful in when studying of anatomy, but not when studying movement.

During a hamstring stretch, all the posterior structures are getting a stretch. The degree of the stress and strain on the structures depends on mechanical variables within the structures, neuromuscular activity, body position and joint angle. Even the slightest ankle eversion during a supine hamstring stretch changes the direction of the load and the sensation of the stretch. Since the tissues adapt according to previous loading history and no two people share an identical loading history, the difficulty in achieving consistent results in human studies is apparent. Try Reclining Big Toe Pose with a strap. First try pressing the inner edge of the foot toward the ceiling, pulling the outer edge of the foot toward the floor. Notice how the stretch changes. Then reverse it - outer edge toward the ceiling, inner edge toward the floor. Did you notice a change in hip joint position? The hamstrings are not the only limiter/contributor to hip flexion.

## Muscle Structure

A skeletal muscle cell is also called a muscle fiber because of the long threadlike nature of its structure. Muscle fibers (encased by the endomysium) are bundled into fascicles (encased by the perimysium). Fascicles are bundled into muscle organs (encased by the epimysium) (Figure 14). Each muscle fiber has a sarcoplasm (the muscle equivalent to the cytoplasm in cell biology) and similarly contains cellular organelles required for cell metabolism like nuclei, ribosomes , and mitochondria. The cell membrane, the sarcolemma, plays a crucial role in muscle excitation by regulating the chemical environment within the cell and lies deep to the endomysium. Additionally, the sarcoplasm contains myofibrils, the contractile structures within the cell.

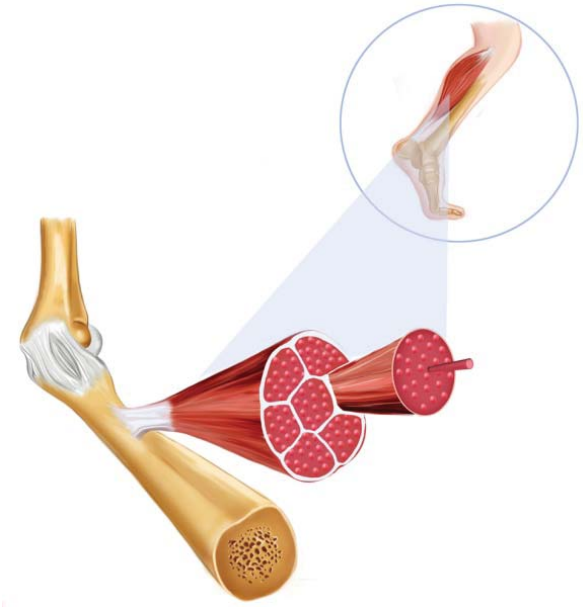


**Figure 14. Muscle Fascicles**

Myofibrils appear as repeating units in-series, called sarcomeres. The sarcomere is generally considered to be the smallest contractile functional unit of muscle. However, many muscle physiologists have recently adopted the half-sarcomere as the smallest contractile unit (Edman, 2012; Leonard, DuVall, & Herzog, 2010; Panchangam & Herzog, 2012; Rassier, 2012; Seiberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013). The sarcomere is made up of smaller structures called myofilaments such as actin and myosin. Proteins located on the actin filaments (troponin and tropomyosin) interact with calcium ($Ca^{2+}$) to bind to myosin projections (myosin heads), forming cross-bridges that enable the two myofilaments to slide past each other, causing the muscle to contract.

A full sarcomere is the segment of the myofibril between two Z discs and is divided by bands or zones. The anisotropic A band is composed of all the myosin filament plus any overlapping actin filament at a given position. The isotropic I band is composed of the actin filament. Further dividing the sarcomere, the H zone is the central part of the A band where no actin overlaps. The M band is the thick filament central part of the H zone. A half sarcomere is defined by the region between the M band and a Z disc.

The most widely accepted mechanism of muscle contraction is the Sliding Filament Theory where the full sarcomere is the contractile functional unit and the actin myosin cross-bridge is the force generating mechanism (Figure 15). A myosin head cocks and binds to an activated molecule on the actin filament to form a cross-bridge.

The actin is then pulled toward the myosin in what is called the power stroke, followed by the release. This repeating reformation of the cross-bridges draws the Z discs toward center, shortening the length of the fiber generating tension (force). During shortening, the actin is pulled toward the center of the sarcomere and the A band holds both myosin and actin. At maximum shortening, the H band and I band disappear. When a muscle fibril is stretched to maximum length, the A band is only occupied by myosin, as the actin is pulled away from center. According to the Sliding Filament Theory, the greatest force is produced at resting length due to the maximum number of possible cross-bridge formations (Figure 15).

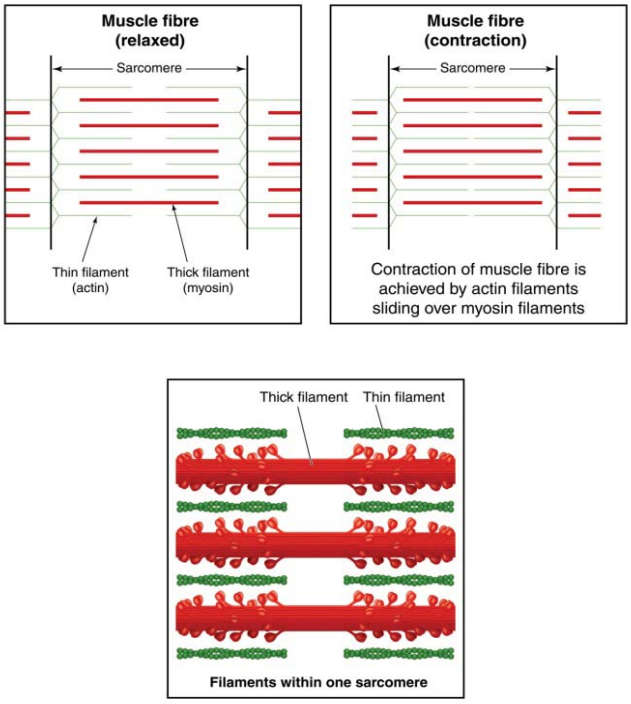



**Figure 15. Sliding Filament Theory**

The Sliding Filament Theory, however, has not been able to explain a measurable increase in force production after a contraction when a muscle is activated *while*

stretched and held (when compared to the level of force production during muscle activation *after* the stretch) (Edman, 2012; Minozzo & Lira, 2013; Rassier, 2012; Shim & Garner, 2012). This Residual Force Enhancement (RFE) has even been measured at stretched lengths where sarcomeres are stretched beyond the region for cross-bridge formation (Leonard & Herzog, 2010). Several proposed theories attempt to explain this behavior, all of them subject to a fair share of both support and criticism. None of the theories are able to fully satisfy the explanation nor can they be fully dismissed (Minozzo & Lira, 2013). Moreover, the role and significance of RFE in everyday activities, such as walking, is scantly understood (Seiberl et al., 2013).

Future research of additional myofilaments and structural proteins making up the muscle fibril (beyond actin and myosin) may modify the classic Sliding Filament Theory (Alter, 2004; Levangie & Norkin, 2011; Nordin & Frankel, 2001). Titin is perhaps the most widely discussed protein as current research on its role in muscle contraction and force and Residual Force Enhancement is greatly debated (Edman, 2012; Leonard et al., 2010; Leonard & Herzog, 2010; Minozzo & Lira, 2013; Powers et al., 2014; Rassier, 2012). Titin is an elastic protein surrounding the myosin filaments and continuing to the Z lines, connecting the half-sarcomeres. Originally, titin was thought to passively control myosin position during contraction by providing passive tension during elongation (Alter, 2004; Herbert, 2005), receiving the tensile load and holding the sarcomeres together during a "passive" stretch when the sarcomeres are not producing tension via cross-bridging. Titin, also subject to viscoelastic phenomena, creeps, and has limited

mechanical range. More recently however, titin is thought to play an *active* role in muscle force production when the sarcomere is stretched beyond the active myosin-actin overlap. Titin has not only been shown to increases stiffness by binding with $Ca^{2+}$, but is also concluded to bind and interact with actin forming a force producing interaction in the absence of cross-bridges (Leonard et al., 2010; Leonard & Herzog, 2010; Minozzo & Lira, 2013; Powers et al., 2014). Some researchers are even proposing a Three Filament Model (actin, myosin, and titin) as a more complete theory of muscle contraction (Herzog, Leonard, Joumaa, DuVall, & Panchangam, 2012). This research challenges the popular belief that a relaxed muscle is better equipped to stretch and elongate, and suggests that contractions during a stretched state are much more complicated than the current paradigm depicts.

## Muscle Force

Not all muscle contractions are the same. A typical muscle contraction, called a concentric contraction, shortens the muscle by pulling the Z lines together. Muscle contractions can also occur during resting or elongated sarcomeric positions. An isometric contraction occurs when muscle contracts but does not change length, an eccentric contraction when a muscle contracts but gets longer (Table 8). Muscle fibers can only shorten (concentrically contract) on their own, they cannot independently lengthen. When a muscle fiber is lengthened, it must be lengthened just how connective tissue is lengthened - against an applied load. In the case of isometric and

eccentric contractions, opposing muscle fibers, gravity, or an external force that is equal to or greater than the force generated by the contraction will override the shortening mechanism. When the opposing applied force is equal to the contraction force, the fiber length remains the same as cross-bridges remain static. When the opposing force is greater than the contraction force, the fiber lengthens as cross-bridges are broken and reformed. As previously mentioned, the force generated from an eccentric contraction (concentric and isometric, as well) is likely the result of a more complex mechanism than simply cross-bridge formation.

**Table 8. Muscle Contractions**

| **Muscle contractions** | **Change in muscle length** |
|---|---|
| Concentric | Muscle fibers shorten |
| Eccentric | Muscle fibers lengthen |
| Isometric | Muscle fibers length remains constant |

The Sliding Filament Theory only accounts for muscle force production along the a single spring longitudinal axis formed by actin and myosin. Recently, it has been discovered that myofilaments expand radially and new models have been developed with multi-spring configurations which depend on the radial distance between filaments (C. D. Williams, Regnier, & Daniel, 2010, 2012; C. D. Williams, Salcedo, Irving, Regnier, & Daniel, 2013). Such directionalities in force production are further supported by the architecture of the perimysium which contributes to the muscular determination of 3 dimensional functionality (Maas & Sandercock, 2010; Passerieux et al., 2007). Additionally, muscle fibers are not continuous throughout the entire longitudinal axis of

muscle between origin and insertion, in spite of how they are depicted in anatomy books (Fukunaga, Kawakami, Kuno, Funato, & Fukashiro, 1997). Instead, muscle fibers are so closely connected to the fascial network, the connective tissue web moves in response to the force output of the excited myofibrils (C. Stecco et al., 2011) and muscles are able to draw force from neighboring and opposing muscles (Huijing & Baan, 2008; Huijing et al., 2007; Maas & Sandercock, 2010).

Prior to such advancements in research, the theory regarding muscle force production which assumed the greatest force producing capabilities is directly related to the number of cross-bridges available, prevailed. A "relaxed" muscle, or an isometric contraction, would therefore be in position to develop more tension than a shortened muscle, as in during the concentric phase (Figure 15), or an elongated muscle, as in during the eccentric phase. Concentric specific or eccentric specific training has been shown to alter the length tension relationship of muscle (Brughelli & Cronin, 2007; Kilgallon, Donnelly, & Shafat, 2007), reminding us that necessary levels of flexibility is best determined by sport specific requirements and that force production at those end ranges of motion, whether concentric or eccentric contractions, should be trained. While eccentric training has been shown to increase levels of flexibility, the important observation is the improvement in performance (measured by force production) at these newly available end rages of motion as a result of resistance training on the eccentric arc of the movement (Brughelli & Cronin, 2007; O'Sullivan, McAuliffe, & Deburca, 2012).

In yoga, concentrically contracting your hip flexors would take you from standing to a forward fold. The eccentrically contracting hip extensors control the movement on the way down by resisting the forward fold. Gravity and the concentric contraction are stronger than the force output of the eccentric contraction and the movement continues. Without any eccentric contraction, you would flop into your forward bend at an acceleration of 9.8 $m/s^2$ (the acceleration of gravity). However, most yoga instruction suggests you relax your hamstrings once you are in the forward fold. How much force production do you have at this end range of motion? Try maximally contracting your hamstrings instead of trying to relax them.

## Muscle Length

Muscle fibers grow longitudinally, in-series, adjusting the growth rate in the presence of a passive stretch, called sarcomeregenesis (Zöllner et al., 2012). Similarly, an increase in the number of sarcomeres *in-parallel* resulting from an active stress is called, myofibrillogenesis (Zöllner et al., 2012). Sarcomeregenesis occurs as a response to chronic lengthening of the sarcomeres, upon which the number increases, restoring the sarcomeres to the original length, and establishes the inherent ability to develop and degrade muscle tissue to satisfy efficiency and function (Zöllner et al., 2012). During periods of bone growth associated with development, muscles respond to a growth spurt with sarcomeregenesis, balancing a child's musculoskeletal system enabling optimum function (Herbert, 2005). When immobilized during periods of bone growth during development, the aponeurosis or tendon length increases, rather than muscle length (Heslinga & Huijing, 1990). Immobilized muscles held at short length accumulate excess connective tissue, possibly interfering with yield during a stretch. But when immobilized muscles are held at resting length and isometric contractions can occur within the muscle fibers, the connective tissues accumulation is prevented (P. Williams & Catanese, 1988). Contractile muscle tissue, therefore, requires active stimulation and adapts best when allowed to contract regularly. Stretching in combination with muscle contractions (i.e. eccentric exercise) provides favorable adaptive conditions for muscle tissue and connective tissue (Brughelli & Cronin, 2007; Butterfield, Leonard, & Herzog, 2005; Kjaer & Heinemeier, 2014; Lynn, Talbot, & Morgan, 1998; O'Sullivan et al., 2012).

Oppositely, immobilized muscle adapts to the length at which it is held by *reducing* the total number of in-series sarcomeres (P. Williams & Goldspink, 1978). This is often falsely interpreted to mean that muscles will profoundly shorten, thereby limiting range of motion, in the absence of a regular stretching routine. Just 30 minutes per day of "stretch" is, in fact, enough to prevent sarcomere loss (P. Williams, 1990). For example, the office worker who sits at a desk most of the day with bent knees and the hamstrings held in a shortened state, eliminates his/her risk of losing sarcomeres simply by straightening his/her legs for at least 30 minutes per day (e.g. standing, walking, sleeping with legs straight). Lost sarcomeres is more likely the result of immobilization rather than a sedentary lifestyle. When end ranges of motion are sparsely explored, however, excess connective tissue accumulates where sarcomeres may be lost, limiting potential joint positions (P. Williams & Catanese, 1988). This reduction in range of motion is less like a factor of muscle length and more likely contributed to limited capacity for performance based on muscle interaction with connective tissue structure. Isometric muscle contractions at end ranges of motion (i.e. contractile activity while in an elongated state) may be suitable pre-training to eccentric exercise, providing favorable adaptive conditions for developing and preserving range of motion.

An example of active stretching is stretching with isometric contractions at end ranges of motion. Sit in Wide Leg Forward bend and tie a long yoga belt around your big toes (photo top). Then "hit out" on the belt, isometrically contracting at this end range of motion. Hold for 30 seconds, relax, repeat. Then try to pull away from the belt (toward midline), cocontracting to create internal resistance. Another method is to move toward the end range of motion against imaginary resistance, eccentrically contracting through internal resistance, holding the end range for 30 seconds. When moving to Standing Twisted Big Toe Pose, resist twisting as if you were pushing through a wall (photo bottom). Use a rope wall to help leverage the resistance, and combine isometric contractions at end range of motion by pushing the foot down onto the chair, contracting the hip extensors in an elongated position.

Sarcomeregenesis is more important for control at end ranges of motion than it is for adding length to the tissues. Attachment points do not change, therefore the myotendinous unit does not change in length. What does change is the capacity to perform (increased force production), the capacity to withstand loads, and the capacity for the nervous system to perceive and control certain joint positions. Sarcomeregenesis is an adaptive response to eccentric exercise, such as downhill running, as a reduction in sarcomere is an adaptive response to concentric exercise, such as uphill running (Butterfield et al., 2005; Lynn et al., 1998). An individual who has lost sarcomeres based on a sport requiring high force production in the concentric range has adapted to perform optimally in these conditions. Simply improving flexibility in the absence of eccentric conditioning may improve range of motion but my not translate into moving well at the end range.

Finally, injured muscle fibers in the form of damaged or separated sarcomeres, should not impair flexibility if healing is complete. The repair process is similar to that of collagen injuries. An initial hematoma is followed by an elaborate cellular response. Myoblasts secrete muscle proteins which develop into myofibrils. In healing muscle tissue, however, these myofibrils do not reconnect end to end, but rather insert into the surrounding connective tissue (Järvinen et al., 2007). Essentially, the muscle fiber is never fully repaired, only built into the surround scaffolding. Recovery time for damaged muscle proteins is substantially less than for damaged collagen and are generally functional again after anywhere from 3-7 days (Järvinen et al., 2013). Any

remaining scar tissue may, however, interfere with the mechanical capabilities around the wound and if range of motion does not return it may be indicative larger scale injury. Any small amounts of disorganized collagen based on typical muscle repair is unlikely to diminish range of motion for the long term.

In summary, mechanical muscle length, while adaptable to loads, is unlikely to be the primary contributor to inflexibility. Muscles are not likely to be short, and stretching "tight" muscles is not likely to make them longer. Muscle's propensity to generate force, even in passive scenarios, suggests that range of motion is regulated by muscle force outputs (Gajdosik, 2001). Muscle activity, acting on all surrounding connective tissues (including joint capsules), controls movement and prevents certain joint positions which are unfamiliar and perceived to be a potential for injury (recall the increased range of motion available to anesthetized subjects). Regulation and control are not mechanical limitations, they are neurological limitations. The nervous system, which receives spatial and sensory input and generates a motor output, is the primary factor of flexibility.

## Recommended Reading

(Leonard & Herzog, 2010; Passerieux et al., 2007; C. D. Williams et al., 2010, 2013; P. Williams & Catanese, 1988; P. Williams, 1990; Zatsiorsky, V. M., Prilutsky, 2012; Zöllner et al., 2012)

# Chapter 9. Nervous System

*" Moving and feeling are two sides of the same coin"*
*Thomas Hanna*

The nervous system is literally the brains behind the movement. Divided into several components, both by structure and function, the nervous system collects information (input) and transforms it into a response (output). Motor control, motor skill development, joint positions, and flexibility are learned behaviors utilizing sensory pathways triggered by the sensation of stretch and motor pathways which respond. The structural and functional divisions of the nervous system (Figure 16) are:

Central Nervous System (CNS)
- Brain and spinal cord
- Control centers

Peripheral Nervous System (PNS)
- Cranial and spinal nerves
- Communicates between CNS and rest of the body

Sensory Division (afferent)
- Somatic and visceral nerve fibers
- Conducts impulses from receptors to CNS

Motor Division (efferent)
- Motor nerve fibers
- Conducts impulses from CNS to muscles and glands

Somatic Nervous System
- Voluntary motor
- Conducts from CNS to skeletal muscle

Automatic Nervous System (ANS)
- Involuntary motor
- Conducts from CNS to cardiac muscle, smooth muscle, and glands

Sympathetic
- Mobilizes body during activity, “fight or flight”

Parasympathetic
- Conserves energy, promotes restoration and cell regeneration

Enteric
- Governs the gastrointestinal system

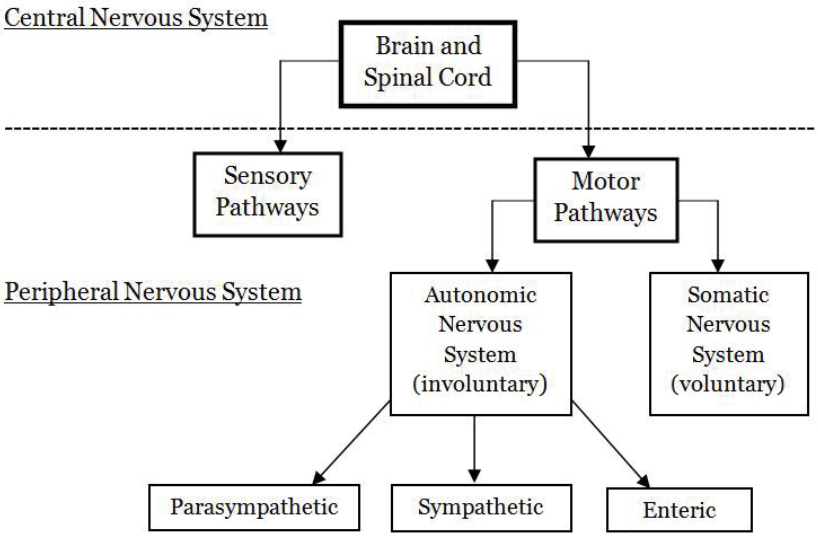



**Figure 16. Divisions of Nervous System**

## Sensory Receptors

Sensory receptors are nerve endings that sense a stimulus and deliver it to the nervous system where a response is initiated. The associated stimuli depends on the type of receptor, but the general purpose is the same; to send mechanical information to the central nervous system (CNS) to illicit a mechanical response. Proprioceptors are specialized receptors providing kinesthetic awareness, enabling determination of joint positions and where the limbs are in space. The primary receptors involved in stretching and range of motion are the muscle spindles and the golgi tendon organs (GTOs).

Muscle spindles are stretch receptors that sense information about muscle length. Spindles are microscopic bundles of specialized muscle fibers encapsulated in

connective tissue that are embedded into the contractile fibers of muscles, tendons and joints. These modified spindle fibers (called intrafusal fibers) lie in-parallel to the regular contractile muscle fibers (called extrafusal fibers) by attaching to the ends. Muscle spindles are, therefore, stretched alongside the muscle stretch, detecting a change and the rate of change in muscle fiber length.

When the CNS perceives this change in muscle length as potentially injurious, the afferent (sensory) muscle spindle initiates a reflex-like response resulting in the efferent (motor) response. The stretched muscle quickly contracts (excitation) to counteract the stretch. For example, if you catch your foot while walking, your quadriceps will stretch at a relatively rapid rate. The muscle spindle detects the unusual rate of stretch and the quadriceps excite to extend the knee and prevent a fall. Simultaneously, the muscle spindle initiates an opposing reflex-like response, inhibiting the antagonist muscle from excitation. In the example above, the hamstrings would relax as the quadriceps excite, aiding knee extension. Termed reciprocal inhibition, the reflex is tested at the pediatricians office during the knee-jerk test. A tap on the patellar tendon with a rubber mallet signals the stretched quadriceps to rapidly contract while the hamstrings briefly relax (Hall, 2012).

In yoga class, a popular stretching technique, fueled by anecdote, is the supposed voluntary activation of the reciprocal inhibition reflex to increase range of motion. The claim is that by contracting the quadriceps during a forward fold, the hamstring will "relax" and allow for a "deeper stretch", thereby improving range of

motion. Unfortunately, this popular instruction has no scientific support (Sharman, Cresswell, & Riek, 2006). In fact, the opposite has been shown to be the case and contracting the quadriceps prior to a stretch to utilize reciprocal inhibition was less effective than static stretching alone for increasing hamstring yield over a 4 week trial (Davis, Ashby, Mccale, Mcquain, & Wine, 2005). Reciprocal inhibition (also call reciprocal innervation), is instead, an instantaneous mechanism that allows for smooth coordinated movements between the stabilizing coactivation (also called co-contraction) of the muscle groups (Alter, 2004). The misconception of reciprocal inhibition as a tissue lengthening mechanism is common and readily accepted, despite the lack of evidence.

Golgi tendon organs (GTOs) are contraction receptors that sense information about tensile forces on the tendon from muscle contractions. GTOs are encapsulated nerve endings which lie in-series with extrafusal muscle fibers, most commonly where the muscle transitions to tendon at the myotendinous junction and aponeuroses. When a muscle contracts, the tensile load is transferred longitudinally to the tendon, stretching the tendon stimulating these in-series receptors. GTOs act as a protective mechanism; they regulate tension on the tendon by relaxing the muscle before tension on the tendon becomes too high. The afferent (sensory) signal stimulates an efferent (motor) muscle inhibition, reducing the stretch on the tendon. When the muscle passively lengthens, however, most of the tensile load is absorbed by the extrafusal muscle fibers and *not* transmitted to the tendon. Static stretching exercises, therefore,

lack sufficient force transmission to stimulate the GTOs; they are much more responsive to a strong muscle contraction (Purves et al., 2004). Golgi receptors located within ligaments, called Golgi end organs, and joint capsules are similarly arranged in-series with extrafusal muscle and respond to the similar stimuli.

The Golgi reflex loop, also fueled by anecdote, is claimed to be the primary mechanism in several Proprioceptive Neuromuscular Facilitation (PNF) stretching techniques. The thought is that by contracting the muscle strongly for 5-10 seconds prior to a stretch, the stimulated GTOs will signal muscle relaxation (called autogenic inhibition), whereupon the "relaxed" muscle will now get a "deeper stretch" (Alter, 2004). Once again, this notion unsupported by the literature, providing no evidence of increased muscle relaxation resulting from a *preceding* contraction (Konrad et al., 2014; Konrad & Tilp, 2014; Lim, Nam, & Jung, 2014; Magnusson, Simonsen, Aagaard, & Kjaer, 1996; Sharman et al., 2006). Any immediate changes in ROM (usually around a 10% increase) due to GTO activity is a temporary neurological allowance and ROM will return to baseline shortly after an acute PNF stretching session (Sharman et al., 2006).

Muscle spindles and GTOs are well known sensory receptors, often (misleadingly) discussed in context of stretching. Other receptors, however, collectively referred to as mechanoreceptors also receive proprioceptive input. These mechanoreceptors, appearing as encapsulated receptors and free nerve endings, innervate the deep fascia, myofascia, intermuscular septa, and in particularly high volume, the retinaculum (C. Stecco et al., 2007). When these mechanoreceptors are

stimulated, via muscle contractions transmitting force to local and non-local fascia through the tendon-fascia continuum, the CNS receives proprioceptive information and adjusts muscle activity accordingly, altering the tensional environment of surrounding structures (Schleip, 2003a).

## Mechanoreceptors

Mechanoreceptors, sensory nerve endings which respond to mechanical forces, are positioned most frequently in the literature in reference to the receptors residing in fascia. Fascial mechanoreceptors outnumber muscle receptors by 6:1, and within a motor nerve, sensory fibers outnumber motor fibers by almost 3:1 (Schleip, 2003b). Based on these ratios, during locomotion, muscle activity comes second to sensory input. The term "musculoskeletal system," which alludes muscles are the movers of bones, gives no indication of the profound role of proprioceptive *input* via connective tissue and the nervous system on movement.

Mechanoreceptors are distinguished by 4 main types: Golgi organs, Pacini corpuscles, Ruffini corpuscles, and Interstitial receptors (Abate et al., 2009; Day et al., 2012; Schleip, 2003a; C. Stecco et al., 2007, 2010, 2011; Willard et al., 2012). The arrangement of the mechanoreceptors, in addition to GTOs and muscle spindles, are organized according the connective tissue architecture and along the lines of force transmission (Scarr, 2012; van der Wal, 2009).

Golgi organs, as previously discussed, respond to slow and strong stretches. The GTOs located within the tendons (discussed previously) account for only 10% of the total Golgi organs in the body. The remaining 90% of Golgi organs lie in ligaments, joint capsules, aponeuroses, intermuscular septa and so on. Golgi organs trigger muscle inhibition, or relaxation (Schleip, 2003b). Perhaps this is why many people feel the instinct to stretch a muscle that is sore, cramped, or feels "tight" or "tense".

The Pacini corpuscles (and the smaller Paciniform corpuscles) sense vibration, rhythmic oscillating, and rapid changes in pressure (Schleip, 2003b). The Pacini group of mechanoreceptors, while found in dense connective tissue throughout the body, are heavily localized in the spinal ligaments and deep within joint capsules (Schleip, 2003b; Yahia, Rhalmi, Newman, & Isle, 1992). The mechanoreceptors contribute primarily to kinesthetic awareness.

Ruffini corpuscles respond to chronic sensations and quiet the sympathetic nervous system (Schleip, 2003b). Responding particularly well to lateral tensile forces and prolonged pressure, Ruffini receptors are dispersed among all the dense connective tissues, although they do appear in higher quantity in the superficial joint capsules, the peripheral ligaments, and in the dura mater (Schleip, 2003b; Yahia et al., 1992). It is possible that the long sustained stretches and placement of props in restorative yoga trigger the Ruffini receptors and contribute to the calming of the nervous system (Figure 17).

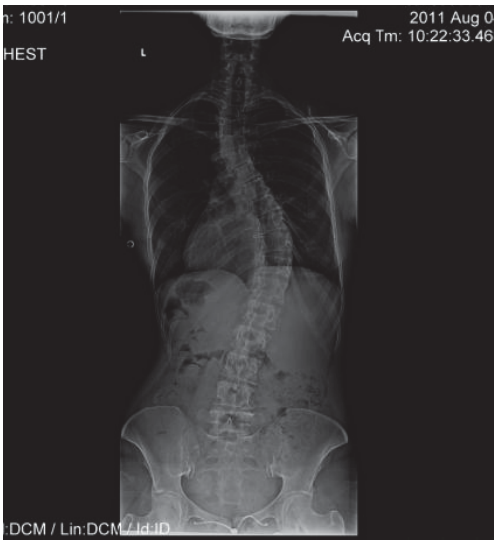

**Figure 17. Restorative Yoga**

Finally, the ubiquitous interstitial receptors comprise 80% of the mechanoreceptors and are located throughout the body, including the periosteum and bone tissue (Schleip, 2003b). They have influence on the automatic nervous system (ANS), affecting heart rate, digestion and respiration through changes in blood flow rate (via vasodilation) (Schleip, 2003a, 2003b). The interstitial fibers can be further divided into two equal groups that respond to either to light forces, called low-threshold pressure units (LTP units), or strong forces, called high-threshold pressure units (HTP units). Both units sense prolonged loads and rapidly applied loads, but only the HTP units may also function as pain receptors.

The nervous system clearly has a tremendous impact on range of motion, perhaps more so than mechanical limitation. Researchers tested this theory, measuring

passive knee joint ROM and the mechanical properties of the patellar tendon, in two groups of subjects. Both groups, the "flexible" and the "inflexible," showed no significant differences in tendon stiffness, stress, strain, elasticity, despite extensive differences in range of motion. The interference of joint friction aside, this research suggests that passive knee joint ROM is governed far more by neural input, than by mechanical properties (Bojsen-Møller et al., 2007). During passive flexibility tests, although the subject is not actively moving the limb, the conscious subject is still receiving sensory information and regulating, in some cases a limiting, movement with some sort of motor output. Muscle "tightness" is a sensory experience (e.g. pain or discomfort) and not a mechanical description of muscle activity, muscle shortness, nor muscle connective tissue stiffness.

Muscle "tightness" is a sensory experience and not a mechanical explanation. We often describe a muscle as "tight" when we think it will not relax, or is resisting a stretch. If that were accurate, then an overstretched (elongated) muscle could not be "tight." My scoliosis clients complain about the stretched side feeling "tight" and are always trying to stretch that side (right side, photo below). The compressed side (left side, photo below) is ignored. If "tight" means "short", then the self prescribed stretching treatment is exacerbating the situation. Training *both* sides to *contract* at various ranges is a more effective method than passively stretching the stretched "tight" side.



## A Few Words on Pain

Pain science is difficult to discuss in the context of mechanical science. Pain is truly an individual experience; mechanical loads that cause pain in one person may not cause pain in another. Additionally, just the *belief* that pain is caused by a mechanical input may lead someone to sense pain. Research has shown that medical diagnoses of structural "problems" leads to greater experiences of pain (Lin et al., 2013). Mechanical science is measurable and predictable, but pain science is not.

Nociceptors are the receptors which receive a warning signal as the sensory input to the brain. Pain is an output by the brain - not an input. That is not to say that pain is "all in your head," even though it is. Pain is determined by *how* the nervous system processes nociceptive input. Not all nociceptive input is processed as pain. The brain collects nociceptive "danger" signaling which is processed through an individual's unique synaptic connections to determine a response. While many of these synapses are associative, to a past experience or an MRI result, for example, reducing nociceptive input is a possible solution. Research on patients with chronic low back pain (CLBP) shows that proprioceptive input strongly inhibits spinal cord processing of nociception (Leinonen et al., 2003). Subjects with CLBP presented less lumbar proprioception than healthy patients and were not able to detect small perturbations in lumbar positioning (Leinonen et al., 2003; Taimela, Kankaanpää, & Luoto, 1999). Considering the pervasiveness of mechanoreceptors in fascia, stimulation of these receptors by reducing nociceptive processing, thereby reducing back pain, has been hypothesized. Static

stretching of the back reduced pain sensitivity in laboratory rats (Corey et al., 2012), suggesting that mechanical loading of the lumbodorsal fascia not only trains the flexion-relaxation response, but could also inhibit nociception.

## Recommended Reading

(Konrad et al., 2014; Konrad & Tilp, 2014; Lim et al., 2014; Schleip, 2003a, 2003b; Sharman et al., 2006)

# Chapter 10. Stretching for Increasing ROM

*"Every cat in the world gets up in the morning and it stretches its body ."*
*Gil Hedley*

Stretching and flexibility exercises are supported by the body of evidence as efficacious methods of increasing flexibility and improving range of motion. Yet, the mechanism by which flexibility develops is inaccurately conveyed and perpetuated by oral tradition. Adjustments in mechanical range (i.e. permanent tissue lengthening) of connective tissue is as unlikely as stretching a rubber band would increase its resting length. Chronic collagen remodeling may be an attempted explanation for adaptive increases in mechanical range, but based on the rates of collagen turnover, those adaptations would take 2-3 years. Most research citing significant increases in range of motion are conducted in less than 8 weeks (Weppler & Magnusson, 2010).

Another hypothesis states adaptations to the neural reflex mechanism in response to flexibility training were responsible for improvements in range of motion (Guissard & Duchateau, 2004, 2006), although this has since been disproven (Hayes et al., 2012). This theory establishes relaxation of the contractile components of the target muscle as the mechanism by which greater lengthening occurs. If this were the case, EMG activity would *decrease* at these new end ranges of motion (ROMs), marking muscle relaxation. Research shows, however, EMG activity and force production may actually *increase* at these new increased ROMs; muscle relaxation is not a prerequisite for increased range of motion (Magnusson, Simonsen, Aagaard, Sørensen, & Kjaer,

1996; Reid & Mcnair, 2004). The remaining, most widely accepted, theory explaining increases in flexibility resulting from stretch training is an increase in tolerance for stretching.

## Stretch Tolerance

Stretch tolerance is the ability for an individual to withstand the discomfort associated with the tensile load at end range of motion. This "sensory theory" emerged because all human trials measuring end range of motion utilize subject tolerance as the limiting factor (Weppler & Magnusson, 2010). Unlike animal studies where tissues can be extracted and tested for inanimate properties, range of motion studies on humans are limited by the subject's willingness to stop the stretch in the presence of pain and discomfort. Stiffness may have a reputation for being the cause of inflexibility, but the argument does not hold up in the literature where tolerance is the well established limit to flexibility (Bojsen-Møller et al., 2007; Knudson, 2006; Konrad & Tilp, 2014; LaRoche & Connolly, 2006; Magnusson, Simonsen, Aagaard, & Kjaer, 1996; Magnusson, Simonsen, Aagaard, Sørensen, et al., 1996; Magnusson, 1998; Reid & Mcnair, 2004; Weppler & Magnusson, 2010; Ylinen et al., 2009).

Because we do not understand the exact mechanism behind tolerance, it is difficult to control for in a study. Researchers on opposing sides of the theory, attempt to prove and disprove the stiffness argument as an attempt to invalidate or validate (respectively) tolerance. In one study, stiffness was reduced after 5 immediately

consecutive stretches, the effect was temporary, and baseline ROM returned within 60 minutes. The same subjects, in the same trial, did not show reduced stiffness after 3 weeks, suggesting the changes were due to the temporary time function of viscoelasticity (Magnusson, 1998). In another study stiffness decreased after 4 weeks of a much higher stretching volume and the researchers hastily concluded that reduced stiffness was the source of increased ROM (Marshall, Cashman, & Cheema, 2011). Even *if* stretching adaptively reduced stiffness, and there is evidence that it may even *increase* stiffness improving force transmission (Knudson, 2006; Shrier, 2004), it would not mean that stiff subjects have less ROM. If stiffness were the cause of inflexibility, the increases in muscle-tendon stiffness due to strength training would interfere with movement. The illogical reasoning marrying strong with inflexible is inaccurate and deduction that range of motion is limited by stiffness is outdated (Magnusson, 1998).

The peripheral structure providing sensory input for stretch tolerance has never been identified, resulting in further challenges when researching the theory (Magnusson, 1998; Weppler & Magnusson, 2010). The language around the sensation of stretch between studies has not yet been standardized, including terms such as resistance, tightness, stiffness, pain, and discomfort to measure end ranges of motion (Weppler & Magnusson, 2010). The individual, perceptual nature of these barriers to flexibility are impossible to quantify will vary drastically among subjects, offering little insight to protocol specifics which would affect tolerance. As a result, most research

compares either time and frequency variables or stretching techniques (i.e. types of flexibility exercises) against a control.

## Stretching Techniques

Flexibility research focuses primarily on the efficacy of Proprioceptive Neuromuscular Facilitation (PNF) stretching and static stretching techniques. PNF stretching encompasses at least 9 different methods with slightly varying applications (repeated, held, and alternating; concentric, isometric, and eccentric contractions) and durations. Detailed distinctions of PNF methods is not necessary, the important commonality is the strong, often maximum, contraction of either, or both, the agonist and antagonist (target muscle). Static stretching is either active, an agonist contracts in order to stretch the target muscle, the antagonist (Fasen et al., 2009; Meroni et al., 2010; Winters & Blake, 2004), or passive, a external force stretches the target muscle. When lying supine, an active hamstring stretch on one leg would require ipsilateral contraction of the hip and knee extensors to hold the leg up (Figure 18 top). The name *active stretching* is misleading since the target muscle is theoretically passive; the active muscle is holding the joint position. Additionally, during active stretching the target muscle will not be entirely passive and the recruitment of active muscles spans well beyond the simply the agonist (recall tensegrity). Using a yoga belt (Figure 18 bottom) in the same supine position is an example of a passive stretch, where theoretically no muscle contraction is required to hold the joint position.

The ASCM recommends static stretching and PNF stretching; dynamic and ballistic stretching may be useful in sports specific training, yet these methods are considered less effective for improving range of motion (Garber et al., 2011; O'Sullivan, Murray, & Sainsbury, 2009). Warm-ups alone, such as 5 minutes of jogging or 10 minutes of stair stepping, increase flexibility and can be further increased (although minimally) by static stretching but not dynamic stretching, however, the improvements in ROM are significantly reduced after just 15 minutes of rest (de Weijer, Gorniak, & Shamus, 2003; O'Sullivan et al., 2009). The acute benefit of a warm-up may be the result of a change in viscoelasticity associated with increased blood flow to the muscle tissue, which returns to baseline when activity stops. Chronic adaptations to stretching, whether viscoelastic or tolerant, are a response to static or PNF stretching, which push flexibility limits, vary mostly by muscle contractions, and abide within the general timeframes recommended by the ACSM.

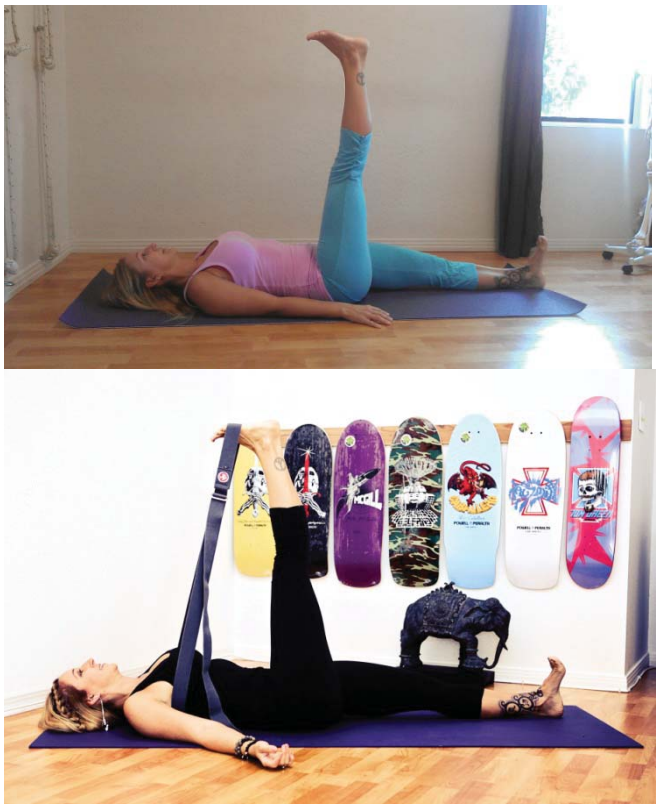


**Figure 18. Active vs. Passive Stretching**

The ACSM guidelines allow for a diverse range of repetitions, duration, and weekly total stretching bouts. The published recommended stretching protocol for a target muscle group is 10-30 second repetitions, totaling 60 seconds, 2-3 times per week. The stretch duration should be adapted to suit individual tolerance, but overall, holds longer than 30 seconds are not believed to produce greater results with the exception of elderly populations where 30-60 holds are suggested (Garber et al., 2011). Perhaps the most comprehensive study evaluating the ACSM guidelines studied 6 combinations of repetitions and hold times against a control in a 12 week study. All 6 groups stretched the hamstrings, just 3 minutes a day (in 15 second, 30 second, or 45 second intervals), 3 days per week, for a total of 9 minutes per week. While they

progressed at varying rates, in the end, all groups improved hip flexion against the control and the differences between groups was insignificant (Sainz de Baranda & Ayala, 2010). Prior research concluded that 60 second durations were no more effective than 30 second durations, nor was increasing the stretching doubt from 1 to 3 times per day (Bandy, Irion, & Briggler, 1997). Considering that most yoga classes are 90 minutes and students attend multiple times per week, the considerable amount of time is spent in hip flexion may be considered excessive when improvements are achieved in just 3 minutes per day.

Flexibility is transient as the adaptive nature of the body lends to a "use it or lose it" scenario where sparsely accessed joint positions may eventually become inaccessible. When comparing active and passive stretching over a 6 week trial, one study found that active stretching not only improved ROM more than passive stretching, but the active group *maintained* the new ROM after a subsequent 4 weeks devoid of stretching, while the passive group did not (Meroni et al., 2010). Another research team, who also found greater ROM improvements in an active group versus a passive group, considered a possible explanation to be an improvement in function of the active (agonist muscle) rather than the extensibility of the target muscle (Winters & Blake, 2004). In a 2 week trial, passive stretching resulted in greater ROM than active stretching, perhaps not enough time to improve the functionality of the agonist (Davis et al., 2005). Another group of investigators assert passive stretching increases tolerance but active stretching increase muscle fiber length (Riley & Dyke, 2012), still

supporting the notion that flexibility is largely a factor of muscle contractility and function, rather than relaxation and an absence of cross-bridge connections.

When reviewing the body of literature as a whole, neither active nor passive stretching stands out as the superior technique, in terms of pure range of motion assessments. As a result, researchers add variables in search of more definitive results. A passive stretching intervention, in combination with nerve gliding (plantar flexing and dorsiflexing during a straight leg raise to stretch and slacken the nervous tissue), was compared against active and passive stretching over an 8 week trial. After week 4, the active and passive with nerve gliding groups improved the most, although all groups improved somewhat (with the exception of the control group). After week 8, the purely passive group (one leg up a wall with the other on the floor through a door way) gained the most range of motion (Fasen et al., 2009). The nerve gliding variable was unable to stand out among the data as beneficial to stretching although it should not be ruled out and more research is needed.

Other methods of flexibility training, incorporating a more global approach, may include motor control exercises (see insert), stimulating awareness of joint position and proprioception. Subjects were divided into 3 groups: passive myofascial stretching (i.e. long kinetic chain stretches) alone, passive myofascial stretching with hip and trunk motor control exercises, and core stabilization exercises with no stretching. Both stretching groups greatly improved range of motion with no significant difference due to the inclusion of the motor control exercise. The core stabilization group produced small

but insignificant improvements in ROM suggesting that non-localized training may allow for greater freedom of movement. The effects of the motor control exercises, however, on functional tasks and performance is not known - the study only measured passive ROM (Moreside & McGill, 2012).

The significance of the Moreside & McGill (2012) study is the similarity of the 6 week training protocol to a yoga class. The motor control exercises included hip rotations and leg lifts plus poses similar to Spinal Balance and Side Plank. The "myofascial" stretching involved both upper and lower extremities resembling poses like king dancer or tree pose- stretching globally from hand to foot instead of localized single joint stretches. Myofascial stretching was not compared with single joint stretching, however.

PNF stretching, although utilized more by athletic trainers than yoga teachers, is accepted as the most effective, method of ROM training (Sharman et al., 2006). The disputable proposed mechanism (recall the argument against autogenic inhibition) for its effectiveness aside, a comparison of PNF against passive stretching is warranted. A very recent study positioned a PNF group against a passive stretch group for 8 weeks. Similar ROM improvements were reported, suggesting that neither is superior for individuals interested in developing flexibility. Passive flexibility training, however, comes at a cost. Subjects in the passive group showed greater electromechanical delay[20] (EMD), compromising swiftness and efficiency of movement, while the PNF group maintained EMD (Minshull, Eston, Bailey, Rees, & Gleeson, 2014). If PNF stretching preserves neuromuscular performance, particularly at end ranges of motion where delays in force transmission risk injury, then it is the superior stretching method. Finding a partner to assist in PNF stretching can be a barrier to consistency, but fortunately self-directed PNF stretching as effective (Schuback, Hooper, & Salisbury, 2004). Other variables, including tendon stiffness and rate of force production, may play a role in EMD values, particularly between sexes where women have been shown to have more compliant hamstrings (Blackburn, Bell, Norcross, Hudson, & Engstrom, 2009; Blackburn, Bell, Norcross, Hudson, & Kimsey, 2009). It is prudent to suggest passive stretching may be particularly ineffective in preventing injuries among the

[20] Electromechanical delay (EMD) = the time between muscle activity measured by electromyography (EMG) and change in joint position

women, who would benefit from focusing on the eccentric muscle contractions to improve neuromuscular performance.

Based on the data presented above, a few parameters for stretch related activities can be derived. In individuals with severely limited range of motion, passive and active stretching of the target muscle for 1-3 repetitions of 30 seconds a few times a week will suffice. The mechanism that improves range of motion is most likely tolerance. Contraction of the target muscle is also essential, and therefore PNF stretching or isometric contractions at the end range of motion are preferred, particularly if the individual does not participate in other activities that challenge force production at end ranges of motion. Holding a stretch for 30 seconds after an eccentric contraction has been shown to be more effective in improving flexibility than eccentric contractions alone (O'Sullivan et al., 2012). In individuals with adequate range of motion, stretches need not be focused on ROM improvements but rather strengthening throughout ROM (i.e. isometrics at end ranges of motion and eccentric contractions). Weakness during eccentric contraction means less neural control and more likelihood of injury (Fyfe, Opar, Williams, & Shield, 2013). Unfortunately, those with a high tolerance for stretching tend to seek passive stretch at the end range of motion, rather than strength and resistance.

While stretching effectively improves flexibility in individuals with severely compromised range of motion (a characteristic shared by subjects in most of the literature), rarely is stretching studied in populations who are already flexible. In a

ballet specific study, *immediate* effects of static versus PNF stretching were measured, determining which technique would best warm-up the dancer for a performance (there was no difference). Any chronic adaptation or even lingering effect to range of motion or lingering effect was not studied (Rubini et al., 2011). Additionally, some research teams regard participation in yoga as an exclusionary factor when screening subjects (Lim et al., 2014). One may pause and question how much flexibility is really necessary for the average person if scientists are not concerned with individuals presenting average flexibility. The unattainable yoga poses are possibly unattainable because the average person does not need them, and need not be chased by tugging and pulling on the limbs since stretching alone will not improve the neuromuscular control at these new ranges of motion. Enhanced tolerance for stretching is not the equivalent to useful range of motion and flexibility alone serves no purpose if neuromuscular control is absent.

Lotus Pose is highly esteemed as the ultimate seated posture for meditation. In most yoga classes, teachers and students alike, will use their hands to pull the limbs into place, each foot in the opposite hip crease. This passive range of motion, however, is void of neuromuscular control. Consider practicing yoga poses until you are able to actively move into them, without assistance from yourself or a teacher.

## Myths About Stretching

For generations, stretching was prescribed by coaches, trainers and therapists as a panacea for impaired performance. So trusted are these implications, improved range of motion has become associated with superior capabilities. Among the yoga community, icons are made of those who can put their feet behind their head and gurus

are made of those who teach them how. Of course, the motivation for these yoga poses may not be to enhance sports performance, but surely the cultural praise for stretching overlaps athletic genres and reinforces the dogma. The most widely accepted, albeit dubious, benefits of stretching include enhanced performance, injury prevention, and relieving sore muscles (Ingraham, 2003; Shrier, 2004).

The research on these topics is overwhelmingly ambiguous and inconclusive due to the variability of factors from mechanical properties to neural activity, the varying adaptations from acute to chronic, the varying stretching techniques, the unlimited scheduling possibilities, and the highly variable nature of conducting human trials. However, static stretching during the hour prior to performance is not recommended by the ACSM (Garber et al., 2011) as passive stretching is known to temporarily reduce strength, and interfering with agility and power type exercises, but baseline viscoelastic properties return approximately 60 minutes after the stretching bout (Fowles, Sale, & Macdougall, 2000; Garber et al., 2011; Gergley, 2013; Kokkonen, Nelson, Eldredge, & Winchester, 2007; Magnusson, Simonsen, Aagaard, & Kjaer, 1996; Magnusson, 1998; McHugh & Cosgrave, 2010; McMillian, Moore, Hatler, & Taylor, 2006; Rubini, Costa, & Gomes, 2007; Shrier, 2004). A comprehensive meta analysis concluded that static stretching interferes with acute force production capabilities of muscle (Simic, Sarabon, & Markovic, 2013), but stretching should not be disregarded as detrimental. Chronic stretching, both static (Kokkonen et al., 2007) and contraction based stretching (Rubini et al., 2007) may actually *improve* performance in the long term. The ACSM does

recommend dynamic stretching to warm-up prior to performance (Garber et al., 2011), which has been shown to improve acute power and agility (McMillian et al., 2006), while static stretching prior to performance diminishes strength (Gergley, 2013) and reaction time (Behm, Bambury, Cahill, & Power, 2004). Stretching sessions are therefore recommend by the ACSM to occur as individual training sessions rather than in preparation for performance (Garber et al., 2011).

Perhaps the hardest veil to lift is the one over injury prevention. Yoga practitioners have been convinced that their flexibility will keep them injury free well into their senior years. Unfortunately, stretching has no known effect on injury prevention (McHugh & Cosgrave, 2010; Thacker, Gilchrist, Stroup, & Kimsey, 2004). Eccentric weakness at end ranges of motion is actually thought to be a contributor to injury and re-injury (Fyfe et al., 2013). Flexibility without precise neuromuscular control not only serves no purpose, but is disadvantageous and a risk factor.

Finally, stretching does not prevent or reduce muscle soreness. Termed Delayed Onset Muscle Soreness (DOMS), due to the delay between soreness and the causing activity, it is thought to come from the mechanoreceptors in the epimysial layer of muscle fascia (Gibson, Arendt-Nielsen, Taguchi, Mizumura, & Graven-Nielsen, 2009). The old theory that associated DOMS with lactate build up has been disproven. Lactate *does* build up during high intensity activities, but is used by the body as fuel and is not a painful waste product. Lactate and muscle soreness are not related. Stretching before, after, or before and after exercise, has little to no effect on DOMS (Herbert, de

Noronha, & Kamper, 2011; Lund, Vestergaard-Poulsen, Kanstrup, & Sejrsen, 1998). If you do happen to feel better during or after your yoga class it is likely a temporary analgesic effect and the DOMS will return shortly (Ingraham, 2003). The meditative and breath-focused techniques in yoga classes, which have been used in pain management, may be an additional source of decreased perception of soreness (Boyle, Sayers, Jensen, Headley, & Manos, 2004). Currently, there is no known treatment or prevention for DOMS other than patiently waiting for it to subside beginning approximately 48 hours after onset.

The invalidation of the purported claims about stretching are well known in the Exercise Science community where research is shared among coaches, trainers, and therapists. Many also understand the effects of stretching, hence the popularity of PNF stretching over passive stretching. In contrast, the yoga community focuses its research efforts on other topics, mainly in the field of mental health. A single study on yoga and flexibility reports improvements in back and hamstring flexibility after just one 90 minute Iyengar class per week for 6 weeks (Amin & Goodman, 2013). While the outcome is interesting, it is hardly a contribution to the vast body of evidence around stretching and flexibility. A non-yoga study comparing seated hamstring stretches to standing hamstring stretches found that both groups improved in flexibility with no change in lumbar curvature (Borman, Trudelle-Jackson, & Smith, 2011), raising some very interesting questions for yoga teachers blame "short" hamstrings for flattened lumbar curves, tucked tailbones, and poor posture. Another non-yoga study successfully

challenged the notion that strengthen the rhomboids and stretching the pectorals will resolve a forwardly rounded shoulder posture (Hrysomallis, 2010). Simply strengthening muscle, or even improving muscle endurance, does not always equal a shorter resting length. Just as stretching hamstrings to improve flexibility does not equate to a longer resting length. Until yoga educators embrace an evidence based practice and participate in advanced flexibility research, myths about stretching will continue to circulate as fact. Yoga therapy will continue to be perceived only as practice for spiritual and mental health, and will struggle to emerge as a respected physical practice consisting of teachers educated in biomechanics and Exercise Science.

## Recommended Reading

(Bojsen-Møller et al., 2007; Garber et al., 2011; Knudson, 2006; Konrad & Tilp, 2014; Magnusson, Simonsen, Aagaard, Sørensen, et al., 1996; Magnusson et al., 2007; Sharman et al., 2006; Weppler & Magnusson, 2010)

# Conclusion

What now? Allow me to share with you my own personal journey through the research and how this project came to be, the process I went through to understand the data, and, most importantly, the *time* it took me make enough sense of it to be useful in my teaching.

When I began researching for this project, I had no idea what the topic would be. Of course, it would be biomechanical in nature, but the direction was beyond my horizon. At the time, I was focused on injuries caused by yoga and began a first draft with that in mind. While studying connective tissue injuries, I became fascinated with the resilience, capabilities, and intelligent design of the human body. I started to wonder if all the tugging and pulling associated with a daily yoga practice interfered with this design. I became fascinated with stretching science and that original piece on yoga injuries was quickly discarded.

I received my first education on stretching though yoga teacher trainings and workshops, as most yoga teachers do, and it had never occurred to me that the science would re-educate me. I began collecting research articles validating what I knew, looking for more evidence to help me confidently write about stretching. The effort was ephemeral; *that* supply of literature was scarce. Instead, I stumbled across articles that challenged what I thought I knew, and before long, found myself confronted by anecdotal concepts taught to me by respected yoga teachers (yogi-lore). I had to be

willing to unlearn and learn again before I would be able to piece all the science together into a complete thesis project.

While writing chapters 1-6, I still didn't understand exactly where I was going. I was still working from the "stretch it make it longer" framework - even though I had read all the literature and collaborated with several colleagues well versed on the topic. I simply had not made sense of it all. It was not until I wrote chapters 7-9 that I began to see flexibility as a motor skill that required training at end ranges of motion. Of course, biomechanics plays a role, but the nervous system is the actuator for movement - including stretching. I had to go back and rework the early chapters.

I explain this process to you here because it may help you in applying the material to your yoga practice and your yoga teachings. There are essentially two parts to this project, Part 1: Biomechanics (Chapters 1-6) and Part 2: Neuromechanisms (Chapters 7-10). When I present the material in a lecture format, yoga teachers are eager and nodding with agreement during the biomechanics segment. The absolute nature of physics is reliable and predictable (once learned), and we must find that comforting. But during the neurosensory and neuromotor chapters, the teachers are skeptical and confused. The variable nature of the nervous system, alternative methods of dissections, and departures from sacred cows of yoga instruction are unsettling. I recommend approaching the material as I did when assembling it: go back and read the biomechanics chapters with tensegrity, muscle contractions, and stretching tolerance in

mind. Eventually, it all comes together, and the big picture becomes clear. You will then realize, as I did, that flexibility is just one small component of moving well.

Despite its size, this project is extremely narrow in scope. The content provided here focuses only on the effects of stretching. Yoga is much more than stretching, I understand. Movement is also more than stretching. But if we are going to stretch in yoga class and associate the benefits with possibilities ranging from mechanical to spiritual, we had better be well versed in the science of stretching. We will be better equipped to design classes, select stretching techniques for our clients, and communicate with other professionals about our methods. I hope my work empowers you all in whichever style(s) of yoga you teach and practice.

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