Stretching scientifically part I: Myths, Facts, the Science

Research · August 2015
DOI: 10.13140/RG.2.1.4558.0005

1 author:

Valentin Uzunov
Portland State University
15 PUBLICATIONS **5** CITATIONS

SEE PROFILE

The Gym Press

Gym Coach
www.thegympress.net/journal

Gym Coach, Vol.1 (2007) 28-32 *Commentary Article*

Stretching scientifically part I: myths, facts, the science

Valentin Uzunov
Hataitai Gymnastics, Wellington, New Zealand.

ABSTRACT

Flexibility is an important part of the physical preparation of most sports, particularly in sports like gymnastics, diving, and dance where esthetic appeal of execution is also critical. Most coaches will recognize the necessity and benefits of a flexibility program and many will even have a fair understanding of the methods they may use, but very few will actually understand how their program works, or its short comings. This two part article will examine the physiological and neurological adaptation that occur with flexibility training, and explore the pros and cons to the common stretching methods used in sports today. Short term gains from stretching are predominantly the result of mechanical adaptations of the muscle and connective tissue. Long term changes are still not clearly understood, but 2 possible alternatives have been proposed: myofibrillogenesis which is the addition of sacromeres to the muscle fibers, and neural inhibition which is the suppression or refined control of the stretch reflex. A better understanding of the physiology behind flexibility training can greatly improve the coaches understanding of what works and what doesn't. This will allow coaches to design more effective flexibility programs and a reduced risk of injury from engaging in regular intensive flexibility training.

Key Words: stretch reflex, myofibrillogenesis, flexibility, physical preparation, sport science.

INTRODUCTION

In gymnastics the development of overall flexibility is critical and it is in every coach's and gymnasts best interest to make sure that their flexibility program is effective. The flexible gymnast has clear advantages over the not so flexible gymnast, by having an improved capability to learn skills faster, lower risk of injuries, ability to perform a greater variety of skills, and an improved aesthetic appeal, and usually being scored higher (20). Gymnastics programs, particularly in the developmental years, place a greater emphasis on passive flexibility, with little attention being given to the improvement of active and dynamic. As the gymnast gets older active and dynamic flexibility are more often than not still neglected even after the gymnast has developed the necessary passive flexibility. It is hypnotized that this trend in gymnastics training could be due to a lack of understanding of flexibility training; however time restraints and the gymnast's level requirements could also be responsible.

This article will examine the physiological adaptations that take place with flexibility training, including the limiting factors associated with flexibility. The information in this article will provide the necessary background knowledge for understanding the rationale of part II of this article which will discuss the advantages and disadvantages of the common stretching methods used in gymnastics and other sports. A better scientific understanding of flexibility training can greatly help the coach to identify errors in the flexibility program implemented, and thus make the necessary adjustments to correct them and prevent injuries, save time, improve functionality of program, and improve performance.

DISCUSSION and REVIEW

The definition of flexibility is "the ability to move a joint or series of joints through a full, non restricted pain-free range of motion" (21). Through training, the flexibility of any joint can be improved quite substantially; however there are also natural limitations and potential limiting factors as well, which will restrict the benefits from training. There are a number of factors that need to be considered with any training program (21):

- An injury or orthopedic conditions
- Excessive fat, or muscle bulk
- Skin - Especially scar tissue over a joint
- Connective tissue - Ligaments and tendons. Physiological changes can take place at these tissue sites, through flexibility training but its normally not directly intended (22) or the desired outcome

- Temperature of muscle - Optimal temperature for muscle elongation is between 39C (102F) and 43.4C (110F))

These are to an extent controllable. However there are also a number of uncontrollable factors as well that a coach should be aware of, and consider when planning and implanting a training program, or talent scouting:

- Joint Structure / Type - Each joint has a relative degree of freedom which it is design to go through(22).
- Collagen crosslink's / Age - Collage crosslink's present is the collagenous connective tissue, such as the ligaments, and tendons, increases with age, and the ability to stretch the collage gets harder (22).
- Genetics – People from south and northern Asia like Mongolia and China, tend to be more flexible (22).
- Fiber Type distribution - The more Type IIb muscle fibers it tends to be more elastic than Type Ib (22).
- Gender - Females tend to have more compliant connective tissue then males due to less muscle mass (22,11).

According to many researcher, the optimal stretching technique or method remains ambiguous (3,5), partly because of the relatively little scientific research done on flexibility training. What research does exist is very scattered and inconsistent in methodology, and protocol to be comparable (1). However by understanding some of the scientific principles behind stretching and flexibility training a coach can design more effective flexibility programs.

The goal of stretching is to increase range of motion (ROM) of a joint by, essentially increasing the compliance of the *musculotendinous unit* (MTU is made up of the muscle and tendon collectively) while also decreasing the stiffness of the MTU (3). As a result of stretching the MTU undergoes either 'elastic' or 'plastic' soft tissue elongation. *Elastic elongation* is a temporary increase in the soft tissue length that may eventually leads to plastic elongation adaptations. *Plastic elongation* is a long term/permanent lengthening of the soft tissue. Research suggests that acute and long term flexibility limitations are most likely a combination of, neural, biomechanical and molecular process (3,4).

Biomechanical limitations can be characterized as either 'active' (contractile) or 'passive' (non-contractile) restraints (23). Contractile restrain come in the form of muscle fiber resistance to stretching, whereas the passive restrains to flexibility come from the connective tissue within and around the muscle fibers, predominantly the epimysium, perimysium, and endomysium, and possibly the tendons as well (figure 1) (9,11,23). It has been proposed that because of the relatively large amount of perimysium, and the orientation of the collagen network that make it up, it is the major contributor to passive resistance, and in preventing muscle overstretching (11).

Muscle fiber resistance to stretch is predominantly the result of muscle spindles within the muscle belly. Muscle spindles are "neuroreceptors imbedded in parallel to the muscle fibers, which provide the central nervous system with information about the momentary length and rate of change in length of the host muscle" (15). Depending on the rate of change in length of the muscle, the spindles respond either with a static or dynamic reflex (15). In a dynamic situation were a muscle is stretched rapidly (defined as anything above 5deg/s and past 80% of max ROM of the joint (12)*), spindle activity significantly increases causing the muscle to contract, this is knows as the stretch reflex (6,26). As a result of the contraction, tension builds up within the MTU and the surrounding connective tissue as end range of motion is approached (23). If this tension is maintained for several seconds, another neuroreceptor know as 'Golgi tendon organ" (located within muscle-tendon or muscle-aponeurosis junctions) senses the tension in the tendon and inhibits the response of the muscle spindles (26). In doing so it induces a force relaxation response (knows as autogenic inhibition), causing the muscle to relax allowing the muscle to stretch a little further before the he spindle is activated again (15).

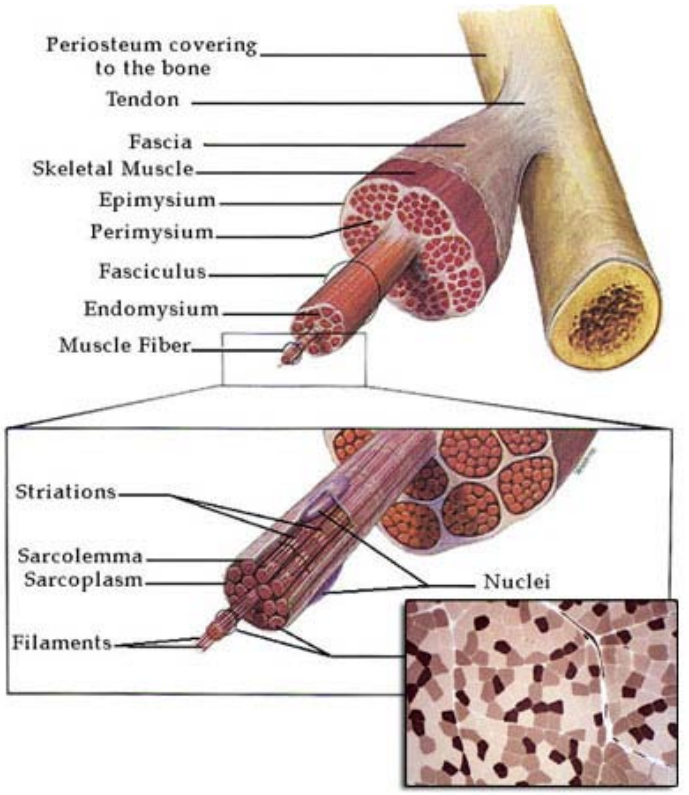

Figure 1 - *Parts of a muscle. A- Muscle is composed of fasciculi, which can be seen by the unaided eye as striations in the muscle. The fasciculi are composed of bundles of individual muscle fibers (muscle cells). B Each muscle fiber contains myofibrils in which the banding patterns of the sarcomeres are seen. C- The myofibrils are composed of actin myofilaments and myosin myofilaments, which are formed from thousands of individual actin and myosin molecules*

In theory this tension-relaxation effect could continue indefinitely, but unfortunately the relaxation response is non-linear and adheres to the law of diminishing returns (12). This means that gains over time are constantly decreasing, with the greatest changes observed in the first 20sec (12). This tension-relaxation response has never been tested for extensive periods of time according to the authors knowledge (beyond 180sec) (12), and the effects of very long term (over several hours or days) passive hold stretches in not entirely known.

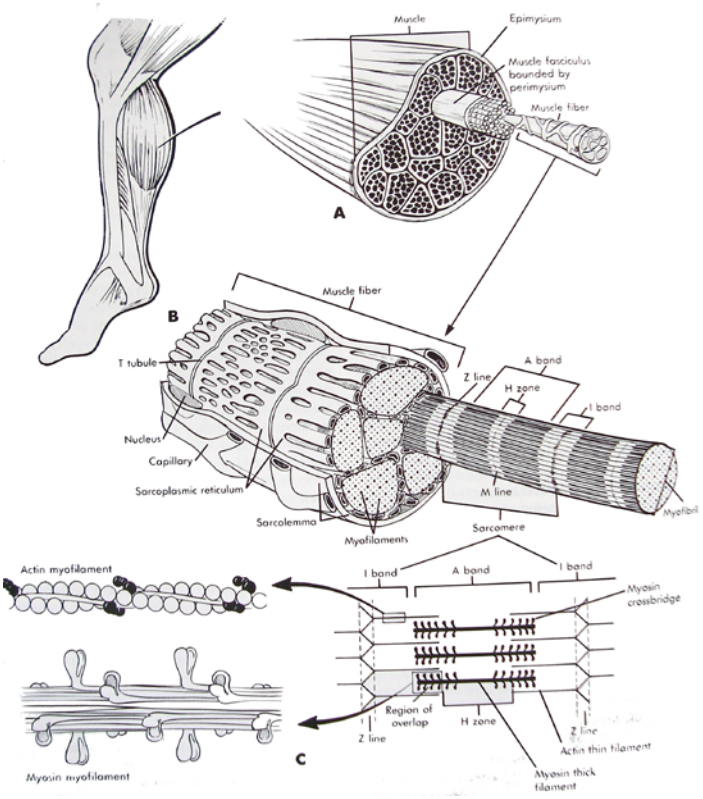

Figure 2 - *The relationship between muscle fibers and the connective tissues of the tendon, epimysium, perimysium and endomysium. Close-up shows and expanded view of a single muscle fiber. S.I. Human Physiology, 4th Ed.Wm.C. Brown, publ.*

When a muscle is stretched, the initial elongation takes place in the sarcomeres, which are smallest functional units the muscle fiber (21) (figure 2). As the muscle is stretched the initial overlap of actin and myosin decreases resulting in elongation of the muscle. When the muscle reaches its maximum length, additional stretching tension starts to be absorbed by the surrounding connective tissue. As the tension increases, the collagen fibers in the connective tissue align themselves along the same line of force as the tension (11,25). Essentially during a stretch the muscle fiber is pulled out to its full length sarcomere by sarcomere, and then the connective tissue takes up the remaining slack. When this occurs, it helps to realign any disorganized fibers in the direction of the tension. This realignment is also what helps to rehabilitate scarred tissue back to health (25) following an injury. The relatively large amount of perimysium has been suggested to be the major to extracellular source for passive resistance to stretch (12).

Even though the endomysium, perimysium, epimysium merge together to form a major part of the muscle tendon, the adaptation of the tendon to stretching is still unclear (5). In fact evidence seems to suggest that the tendon structure is not significantly affected with stretching (5), rather the myotendinous junction might be the site of structural changes in response to the altered loading (4).

Passive tissue stiffness as a limiting factor might seem like a logical limitation to joint flexibility, but there is contradictory evidence to suggest otherwise. One study concluded that passive muscle is 4 orders of magnitude more compliant compared to the tendon, which means that these connective sheaths and muscle fibers are likely to receive most of the mechanical stress that is generated by passive stretching (4). In one other study examining passive stiffness, it was found that long term adaptations to static stretching showed no change in resistance within the muscle, but maximal ROM improved regardless (5). The same study reported that subjects with low levels of flexibility demonstrate a lower stretch tolerance and an increased resistance to stretch when compared to subjects with good flexibility (5). From the limited information available from studies it is still unclear as to the exact influence passive stiffness has on a joints range of motion.

From the available evidence it has been concluded that immediate and ***short term*** gains in flexibility following a stretching session (particularly using passive stretching methods as done in most studies) are the result of increased stretch tolerance and the viscoelastic properties of muscle and connective tissue, rather than actual physiological changes (3,4). The gains observed immediately following a stretching session last about 1hr (5). As a result of regular flexibility training it has been suggested that neural physiological adaptations do eventually occur, which effect the activation of the muscle spindles and perhaps the Golgi tendon organ (4). However, these mechanisms have not been determined yet, and more research is needed in this area (4). Thus given all the available information and the adaptable nature of living tissue, the biomechanical and neurophysiologic rationale to flexibility seem to explain short-term, reversible change, but seems to be inadequate to explain long-term permanent changes (4).

One proposed mechanism for long term-permanent flexibility adaptations is *myofibrillogenesis**. The addition of sarcomeres in series near the MTU junction (4). It is suggested that the force generated during stretching is likely transmitted via a series of protein-protein interactions which may lead to a chain of biological signals that ultimately lead to myofibrillogenesis (4). Up to date, the only evidence to support myofibrillogenesis as an explanation for permanent adaptations comes from animal experiments and orthopedic procedures where muscle is

indirectly lengthened, such as distraction osteogenesis* (4). One study showed that if the extensor digitorum lateralis muscle in rabbits is lengthened by 3mm by distraction, there was an increase in sarcomere length from 3.1 to 3.5µm. When that stretch was maintained for several days the sarcomere length returned back to a value of 3.1µm which suggests the addition of sarcomeres (4). However there could be an alternative explanation. Several past experiments have shown that the 'mean single sarcomere' does not consistently correspond to the response of the entire cell (24). This means that it's quite likely that not all sarcomeres are stretched to a 3.5µm, and some may actually have contracted prevent tearing. Thus over the course of the held stretch over a long period of time, it may have resulted in a uniformed sarcomere response which could have resulted the return of the average sarcomere length of 3.1µm. Alas there is no systematic way to analyze individual sarcomere length changes in the fibers that occur during end-held active contractions with or without stretch, and thus all evidence is hypothetical (24). Even though stretch induced myofibrillogenesis may seem like a reasonable explanation to permanent flexibility training adaptations, there are is no recorded evidence in humans that myofibrillogenesis does occur through conventional stretching methods.

Anecdotal evidence in support of myofibrillogenesis following long term flexibility training using conventional methods comes from observations of retired gymnasts, dancers, divers, acrobats and other athletes who have engaged in regular flexibility training over years practice. These retired athletes who may do little exercise if any (particularly stretching) still manage to retain a much higher than normal level of flexibility, many still able to achieve splits or bridges years after they retiring. This suggests that permanent and semi-reversible changes have occurred. Some sources suggest that with extensive training, the stretch reflex of certain muscles can be controlled so that there is little or no reflex contraction in response to a sudden stretch. While this type of control provides the opportunity greatest gains in flexibility, it also provides the greatest risk of injury (25). Only consummate professional athletes and dancers at the top of their sport are believed to actually possess this level of muscular control (25).

CONCLUSIONS

Flexibility gains are mostly likely due to a combination of neural, mechanical, and molecular adaptations and process. At this point, all these process are not completely understood, which leads more researchers to agree that the optimal method for training is still not known. Flexibility is essential restricted by the stretch-reflex, which prevent the muscle being stretched to much as a preventative mechanism. Changes in the muscle fiber are believed to occur at the MTU, with any additional strain being placed on the connective tissues. Temporary flexibility gains from a short bout of flexibility training are most likely the result of viscoelastic properties of the muscle and connective tissues, plus the increased stretch tolerance. These gains last only about 1hr, before they revert to normal. The process for long term adaptations is not known. Two methods have been proposed. The first one is believed to be the result of a series of protein to protein interactions which lead to myofibrillogenesis. Myofibrillogenesis has been inconclusively been been used to explain plastic elongation in in-vitro studies on animal muscle, and through orthopedic procedures using distraction osteogenesis. The second method proposed suggests that long term adaptation leads to decreased response from the stretch-reflex. Neither of these methods are conclusive and thus it is still unclear what the long term adaptations from flexibility training are. Regardless it is clear that through such training substantial gains can be achieved and maintained.

Address for correspondence: Valentin Uzunov, H*ataitai Gymnastics, Wellington, New Zealand*.
valentin.uzunov@gmail.com.

REFERENCES and RECOMMENDED READINGS

1- Sharman, M.J, Cresswell, A.G & Riek S. (2005). Proprioceptive Neuromuscular Facilitation stretching. *Sports Medicine*. 36(11):929-939
3- Rees, S.S., Murphy, A.J., Watsfield, M.L., McLachlan, K.A., & Coutts A.J., (2007). Effects of proprioceptive neuromuscular facilitation stretching on stiffness and force production characteristics of the ankle in active women. *Journal of Strength and Conditioning Research*. 21(2):572-577
4- Deyne P.G. (2001). Application of passive stretch and its implications for muscle fibers. *Physical Therapy*. 81(2):819-827
5- Magnusson, S.P., Simosen, E.B., Aagaord P., et al. (1995)Viscoelastic response to repeated static stretching in human skeletal muscle. *Scandinavian Journal of Medicine and Science in Sports*. 5:342-349
6- Mahieu N.N., McNair P., Muynck, M.D., Stevens V.,Blanckaert, I., Smits N.,Witvrouw E. (2006). Effect of static and Balistic stretching on the Muscle-Tendon Tissue properties. *Medicine and Science in Sports Medicine*, 39(3):494-501
9- Kubo K., Kanehisa H., Fukunaga T. (2002). Effects of resistance and stretching training programs on the viscoelastic properties of human tendon structures in vivo. *Journal of Physiology*, 538(1):219-226

11- Kubo K., Kanehisa H., and Fukunaga T. (2001). Is passive stiffness in muscle related to elasticity of tendon structures. *European Journal of Applied Physiology*, 85:226-232
12- McNair P.J., Dombroski E.W., Hewson D.J., and Stanley S.N. (2000). Stretching at the ankle joint: Viscoelastic responses to hold and continuous passive motion. *Medicine and Science in Sports & Exercise*, 33(3) 354-358.
15- ADMOTION (2002). Vibration training: Mechanisms and possible mechanism relation to structural adaptations and acute effects. [url] http://nz.powerplate.com/EN/technology/scientific_support.aspx Accessed 13.11.08
16- Anderson D.M. (2005). Mosby's Medical Dictionary (Ed7). Elsevier Science Health Science division.
20- Sands. B.(1984). Coaching Women's Gymnastics. Champaign: Illinois, Human Kinetics Publishers, Inc.
21- Prentice W.E.(1994).Rehabilitation techniques in Sports Medicine. St Louis: Missouri. Mosby- Year Book, Inc.
22- Sands W.A (2006). From lecture notes on Flexibility and Strength at USA High Performance Clinic. National Training Center, Texas. USA
23- Chris M. (NA). Flexibility and Stretching: Two Sports physiotherapists show why flexibility is so important, and explain the science behind it. [URL]: http://www.pponline.co.uk/encyc/0833.htm. Accessed: 19.10.07
24- Telly I.A. (2005). The Role of Sarcomere Dynamics in Muscular Contraction. Zurich: Swiss Federal Institute of Technology Zurich. PhD of Natural Science.
25- Appleton B.D. (1994). Stretching and Flexibility. Retrieved November 11, 2007. Website: http://people.bath.ac.uk/masrjb/Stretch/stretching_1.html
26- Alter M.J. (1998). Sport Stretch (2nd Ed). Champaign: Illinois, Human Kinetics.