# Maximizing Hypertrophy: Possible Contribution of Stretching in the Interset Rest Period

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## SUMMARY

THE TIME THAT A MUSCLE IS UNDER TENSION DURING RESISTANCE STRENGTH TRAINING IS THOUGHT IMPORTANT IN MAXIMIZING THE HYPERTROPHIC RESPONSE OF SKELETAL MUSCLE. IMPLEMENTING STRETCHING IN BETWEEN SETS MAY INCREASE THE HYPERTROPHIC EFFECT BY ADDING TO TOTAL SESSION TIME UNDER TENSION AND AS SUCH INCREASE THE EFFECT OF VARIOUS NEUROMECHANICAL AND METABOLIC STIMULI THAT ARE THOUGHT IMPORTANT TO HYPERTROPHIC ADAPTATION. THIS REVIEW WILL EXPLORE THIS CONTENTION BY BRIEFLY DISCUSSING THEMES AROUND STRETCH AND RESTRICTED BLOOD FLOW, HORMONE RELEASE, SIGNALING PATHWAYS, STRETCH ACTIVATION CHANNELS, STRETCH-INDUCED HYPERTROPHY, AND STRENGTH AND POWER PERFORMANCE.

## INTRODUCTION

A strength training session consists of work and rest periods in its simplest form. To improve strength and power, an understanding of how to maximize the effects of the work and rest periods to produce the desired neuromuscular adaptation associated with various loading schemes is needed. With the advent of linear position transducer and force plate technology, there is an increased awareness of the kinematics and kinetics associated with a single repetition, set, and workout (13).

Improving our mechanical (kinematic and kinetic) understanding of the training stresses that will be imposed on muscle is important as it is thought that strength and power adaptation is mediated by mechanical stimuli and their interaction with other hormonal and metabolic factors. However, our knowledge in this area is in its infancy, the kinematics and kinetics associated with different resistance strength training programs, and associated adaptations will remain a topic of research for many years to come if researchers are genuinely interested in and committed to understanding the adaptational effects of resistance exercise.

Apart from the research that has investigated the effect of rest durations between sets on kinematics and kinetics (15,38,43,44), there has been a lack of research that has investigated how athletes might optimize the rest period to enhance session kinematics and kinetics. That is, there may be activities that can be engaged in during the rest period that may enhance the ensuing set and total workout kinematics and kinetics. The net result could be a session with increased mechanical, hormonal, and metabolic responses and hence the opportunity for improved strength and power adaptation.

This article will explore the concept of engaging in activities in the rest period in terms of increasing hypertrophic adaptation. That is, activities that can be performed during the rest period that promote recovery and/or maximize the mechanical stimuli associated with a training session to maximize the outcomes of training. Specifically, the role of stretching in the recovery period, as a means of increasing hypertrophic adaptation and subsequent strength and power performance, will be described.

Throughout the article, the terms passive and active stretching will be used. Passive stretching implies holding an extended range of motion with little or no neural activation. Active stretching on the other hand can mean (a) the muscle being active while being extended or lengthened (eccentric

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contraction) or (b) the muscle passively extended to end range of motion and then activated (isometric contraction) or (c) a combination of both.

## HYPERTROPHY LOADING PARAMETERS AND REST

Studies have shown that strength gains from resistance training are not only the result of neural adaptation but also because of increases in the cross-sectional area of muscle (10,23,25,54). Researchers and organization's recommendations (5,6,15,34,40,42,52) for increasing muscle hypertrophy include the use of moderate loading (70–85% of 1 repetition maximum [RM]) for 8–12 repetitions per set for 1–3 sets per exercise, for novice and intermediate individuals. Furthermore, slow to moderate repetition velocities are recommended (31), with slow tempos of 1–5 seconds concentric and 1–5 seconds eccentric (29). In terms of time under tension, these slower tempos and contraction velocities result in significantly greater time under tension (29). For experienced athletes, it has been recommended that a loading range of 70–100% of 1RM be used for 1–12 repetitions per set for 3–6 sets per exercise in a periodized manner (31).

With regard to rest period durations, researchers recommended durations of 1–3 minutes depending on athlete training status (42). There is no doubt that longer recoveries allow more complete recovery of the energy and neuromuscular systems; however, short rest periods (1–2 minutes), coupled with moderate- to high-intensity loading and volume, have elicited the greatest acute anabolic hormone and metabolic responses to resistance exercise in comparison with programs using very heavy loads with long rest periods (32,33). Therefore, it is of interest how to magnify the mechanical, hormonal, and/or metabolic responses during the rest period to enhance hypertrophic adaptation. To achieve this, some understanding of the stimuli that determine hypertrophic adaptation is needed.

## STIMULI FOR HYPERTROPHY (RADIAL GROWTH)

There is no doubt that there are many interacting factors responsible for hypertrophic adaptation. To ensure that protein synthesis exceeds protein degradation, appropriate loading needs to optimize the muscles mechanical, metabolic, and hormonal responses; signaling pathways; nutrition and recovery (3,7,9,59,60,66). In terms of mechanical loading, Toigo and Boutellier (61) identified the distinct role of active tension in generating muscle hypertrophy, reporting that time under tension is one of the important stimuli in promoting cross-sectional or radial growth of skeletal muscle. They summarized that elongated muscles placed under tension triggered protein synthesis, which is important for muscle growth and is the reverse process to what can be observed during muscle atrophy associated with immobilization (61). It is also widely accepted that the eccentric or lengthening phase in resistance exercise training is an important contributor to muscle growth (16,20,25,28).

It seems that moderate to high forces and time under tension, particularly during lengthening contractions, and subsequent hormonal and metabolic responses are important stimuli for the radial growth of the muscle. Therefore, if hypertrophy is the goal of training, it would seem important to maximize these stimuli during a training session. There is no doubt that this occurs throughout the work periods during a resistance strength training session; however, it may be that what practitioners do during the rest period may provide an additional hypertrophic response, this contention to provide the focus of subsequent discussion.

Topics such as stretch-induced hypertrophy, restricted blood flow, hormonal and metabolic responses, and signalling pathways are briefly discussed. The discussion on these topics is far from definitive statements as there are substantive bodies of research associated with each of the topics listed. The aim of the discussion is to present a rationale why stretching during the rest period may add to the hypertrophic effect and thereafter stimulate dialogue and research among interested practitioners and scientists.

## STRETCH-INDUCED HYPERTROPHY

In terms of animal research, there is a body of literature that has documented stretch-induced hypertrophy. Goldspink (18) compared 2 conditions where rat soleus and extensor digitorum longus muscles were immobilized in shortened and lengthened positions and reported that the lengthened muscles increased in size, whereas shortened position muscles atrophied. This suggests that passive stretch at long muscle length stimulates protein synthesis and induces the growth of muscles.

Holly et al. (27) reported increases in length and cross-sectional area of 4 chicken wing muscles stretched to different extents over a 5-week duration. Essentially, length changes were complete after 1 week of stretching; however, increases in cross-sectional area of 73–206% were recorded over the 5-week duration of the study depending on the type of the muscle. The researchers concluded that muscle grew and adapted enzymatically (oxidative enzymes) to stretch, but these responses are dissimilar in twitch and tonic muscles.

Goldspink et al. (19) investigated the effect of passive stretch, electrical stimulation at 10 Hz, and a combination of both stretch and electrical stimulation, on the expression of insulin-like growth factor I (IGF-I) and the rates of protein turnover and growth of the rabbit extensor digitorum longus muscle. It was found that static stretch caused significant adaptive growth and increases in IGF-I either with or without electrical stimulation, whereas continuous electrical stimulation alone failed to induce muscle growth. Yang et al. (67) studying rabbit's lower limb muscles found that 6 days of passive stretch while immobilized in a plaster cast not only induced an increase in expression of IGF-I messenger RNA (mRNA) but also increased the percentage of fibers expressing slow myosin. This change in muscle phenotype was accompanied by a rapid and marked increase in muscle mass, total RNA content, and IGF-I gene expression.

Unlike the previous studies that used chronic stretching protocols, a study by Coutinho et al. (12), which was conducted on eighteen 16-week-old Wistar rats for 3 weeks, found that stretching the immobilized soleus muscles for 40 minutes every 3 days did prevent the muscle shortening and reduced the magnitude of muscle atrophy as compared with a immobilized-only group (22 ± 40 versus 37 ± 31%, respectively). Furthermore, muscles that were submitted to a stretching-only group significantly increased the length (5 ± 2%), serial sarcomere number (4 ± 4%), and fiber area (16 ± 44%) compared with the contralateral muscles. This study showed that although the stretching was passive, hypertrophy still resulted. Tension is also an important regulator of skeletal muscle hypertrophy in vivo because when increased constant tension is applied to embryonic skeletal muscle fibers differentiated in a tissue culture environment, many of the same biochemical processes associated with muscle hypertrophy in vivo are also stimulated in vitro, for example, protein synthesis, total protein, and myosin heavy-chain accumulation (62).

Although much of the research in this area is from immobilization studies, it can be observed that active and passive stretch can induce changes in cross-sectional area of the muscle (14). Naturally, a muscle is stretched under load during strength training, and the slow tempos recommended for hypertrophic adaptation magnify the time under tension or stretch load. During the rest periods, if the muscles are actively or passively stretched, the additional mechanical stimulus may enhance the hypertrophic effect. This contention obviously needs to be researched in a systematic manner, ensuring that the stretching protocol does not significantly influence the ensuing repetition and set kinematics and kinetics.

## STRETCHING AND RESTRICTED BLOOD FLOW

It is fairly well accepted that passive and in particular active stretching is likely to disrupt regional blood flow and muscle oxygen consumption, which has been attributed to the local pinching of blood vessels (2,22,48,65). A number of other researchers have noted that the restricted blood flow differs regionally within a muscle, for example, blood flow reduced to a greater extent in the central zones of a muscle (26,30). These regional differences have been attributed to differences in regional tissue pressure, as well as the heterogeneous pattern of muscle fiber direction (architecture), which may induce shear forces and subsequent partial occlusion of blood vessels during stretch and contraction.

In this case, Poole et al. (41) found that not only did stretching reduce blood flow and therefore oxygen delivery and metabolite removal but also it altered red blood cell flow dynamics, which may further impair blood tissue oxygenation. These reductions were attributed to stretching causing (a) passive reductions in vessel diameter by compression and axial stretch and (b) pinching of blood vessels because of the different architectural arrangements between muscle fiber and fascicles.

Given the information on restricted blood flow above, it is likely that the ischemia associated with stretching if used in the rest period, in particular active stretching, would likely contribute to both fatigue and metabolite accumulation. The relative occlusion would reduce the clearance capacity of the circulatory system, resulting in accumulation of lactate, hydrogen ions, sodium ions, and phosphate in the working muscle, which has been implicated as important in the growth hormone (GH) response to exercise (21,63). In this case, Rodney et al. (45) and Schott et al. (53) found that fatigue and metabolite accumulation were important in increasing the strength and accretion of muscle mass. Furthermore, it has been argued that the enhanced hypertrophic response, noted with exercise that uses blood flow restriction, may be attributed to the recruitment of larger type 2 motor units and the resulting mechanical load on these muscle fibers (36). The exact mechanism by which acute changes in high-energy phosphates or other metabolites may trigger hypertrophic signaling is unknown. However, Meyer (36) suggested that there is ample evidence that metabolic sensors such as adenosine monophosphate–dependent protein kinase can play an important role in regulating skeletal muscle growth. For a comprehensive discussion of the effects of restricted blood flow on skeletal muscle, the reader is directed to a recent review by Manini and Clark (35).

## STRETCHING AND HORMONE RELEASE

With regard to the restricted blood flow research cited previously, Takarada et al. (57,58) have reported very large acute increases in plasma GH levels, resulting from low-load occlusion training. It is likely that during ischemic conditions, such as stretching during rest periods, metabolites and ions accumulate rather than dissipate, which in turn lead to GH secretion and increased levels of IGF-1. According to Perrone et al. (39), stretch-induced skeletal muscle growth may involve increased autocrine secretion of IGF-1, because IGF-1 is a potent growth factor for skeletal muscle hypertrophy, and stretch elevates IGF-1 mRNA levels in vivo. They believed that extracellular matrix protein type I collagen and stretch stimulate the autocrine secretion of IGF-1 but with different time kinetics. This endogenously produced growth factor may be important for the growth response of skeletal myofibers to both types of external stimuli (39).

## STRETCHING AND SIGNALING PATHWAYS

Several factors intrinsic to the muscle are involved in the hypertrophic response of muscle, such as intracellular signaling (transduction of mechanical tension into intracellular signals) (11). Many potential key molecules (e.g., IGF-I, myogenic growth factor, protein kinase B (PKB)/serine/threonine-specific protein kinase (Akt) mammalian target of rapamycin (mTOR) and

P70$^{S6K}$) have been identified as having a role in the hypertrophy associated with skeletal muscle (4,11,37). It is beyond the scope of this article, however, to extensively review the possible mechanisms by which tension is transduced into intracellular signals. Nonetheless, a brief discussion of 1 enzyme (Akt, also referred to as PKB) will give insight into the regulatory effects of contractile activity on adaptation, in particular, the effect of stretch. Akt is thought important in the activation of the hypertrophic response in skeletal muscle (4,50). Although muscle contraction is known to activate Akt in skeletal muscle composed of various fiber types (49), the extent to which different mechanical stimuli activate Akt for the most part remains unclear.

A study found that passive stretch for 10 minutes significantly increased Akt activity (2-fold) in the fast-twitch extensor digitorum longus muscle, in which the researcher of the study concluded that mechanical tension might be a part of the mechanism by which contraction activates Akt in fast-twitch muscles (49). However, a study by Russ (47) found that stimulation frequency in muscle contraction did not contribute significantly to the signaling pathway that is thought important for hypertrophic adaptation, but passive and particularly active muscle tension were the critical signaling factors. Russ (47) also documented that the degree of Akt phosphorylation in isometric contractions was substantially less than forced lengthening (eccentric) contractions and concluded that if tension was indeed the main stimulus for Akt activation, then it was not surprising that using forced lengthening type contractions induced greater levels of Akt. This is consistent with findings that eccentric contractions for the most part induce greater hypertrophic adaptation than other contraction modes (16).

The implications of these findings although speculative are that holding a passive or active stretch at a long length may increase time under tension and/or the magnitude of the tension, which in turn will increase Akt activation and subsequent hypertrophic signaling. For further information on this topic, the readers are referred to the article of Sandri (51), which explains the signaling pathways in hypertrophy and atrophy of the muscles more clearly.

## STRETCHING AND STRETCH-ACTIVATED CHANNELS

Stretch-activated channels (SACs) are calcium- and sodium-permeable channels that increase their permeability in response to mechanical loading such as stretch. There is evidence that these channels may play a vital role in mechanotransduction, linking mechanical deformation and cell signaling (8). There is a great deal of research in this area. A snapshot of the importance of stretch on SACs is provided in a study of Spangenburg and McBride (56). They (56) showed that eccentric contractions increased the degree of Akt or p70$^{S6K}$ phosphorylation, whereas 2 different SAC inhibitors reduced activation of the Akt or p70$^{S6K}$ signaling pathway, the signaling pathway thought to be a major regulator in the initiation of protein synthesis in skeletal muscle. They concluded that in skeletal muscle, SAC activity appears to only occur during lengthening or stretch-induced contractions; therefore, SACs are only contributing to hypertrophic signaling mechanisms after eccentric or lengthening contractions.

## STRETCHING AND STRENGTH PERFORMANCE

If stretching is to be used within a single workout and repeated workouts as with hypertrophy training, then some understanding of its influence on muscular performance is fundamental. A more full appraisal of the effects of stretching on muscular performance can be found in 2 recent reviews scrutinizing the effects of acute and regular stretching (46,55). The author of a systematic review of 23 articles reported that an acute bout of stretching did not improve isometric and isokinetic force or jump height, and the results for running speed were contradictory (55). Shrier (55) stated that the contradiction for running speed could be attributed to running economy, which was found to be improved by stretching, whereas force and velocity of contraction were decreased after stretching.

Rubini et al. (46) concluded that although most studies found acute decreases (−4.5 to −28%) in strength (isometric, isotonic, and isokinetic) and jump performance (−3.2 to −7.3%) after stretching and that such decreases seemed to correlate to the length of the stretching protocol, the number of exercises and sets, and the duration of each set, in most studies, exceeded the ranges normally recommended in the literature. For example, 30 minutes of passive stretching of the plantar flexors decreased voluntary strength by 28% (17), 20 minutes of passive stretching resulted in a 10% decrease in the plantar flexors voluntary peak torque (24), 7% decrease of maximal isometric voluntary contraction after 10 minutes of acute passive stretching (64), and nonsignificant decreases in explosive force production (0.3–3.6%) after a 5-minute warm-up of either treadmill run or bicycling and 1–4 minutes of static stretching (1,68).

It seems that as the stretching duration decreases, the negative effect of static stretching on performance reduces to nonsignificance. Given these findings, it may be speculated that a 30- to 60-second stretch between sets may have a limited effect on subsequent set kinematics and kinetics, especially as the duration between the stretch and onset of performance increases. However, such a contention needs to be investigated with regard to both active and passive stretching. Furthermore, the influence of active and passive stretch on agonist-antagonist or upper- to lower-body work to rest protocols would benefit from research exploration.

## CONCLUSIONS

From the literature reviewed, both passive and active stretching will influence the time under tension and the associated neuromuscular, metabolic,

and/or hormonal responses. Active stretching by the nature of the added contractile activity should provide greater time under tension and hence adaptational effect. With regard to hypertrophy, it is unlikely that hypertrophic adaptation is caused by one single mechanism, and more likely, it depends on the integration of multiple local and systemic factors, which are influenced by the mechanical stimuli presented to the muscle and the resultant hormonal and metabolic responses. The tension developed within a session (i.e., the magnitude and time under tension) appears to be the critical mechanical stimuli in this process.

Our understanding of how to develop tension throughout the work period during resisted strength training has been well researched, that is, using slow contraction velocities (slow tempos), maximizing both the eccentric and the concentric contraction duration. However, it has been argued that there seems compelling reasons to stretch (active and/or passive) during the interset rest periods while hypertrophic strength training, as it is likely to increase the total time under tension of the muscle, which may have a number of mechanical, neural, metabolic, and hormonal advantages compared with not stretching.

At this stage, it is yet to be determined if the stretching protocol will adversely affect the kinematics and kinetics of the ensuing sets (i.e., magnitude of the tension) given variables such as force are also thought critical to adaptation. The status of the stretching literature does not assist our understanding in any great depth as to the negative influence of stretching on a traditional strength session because the literature for the most part has focused on the influence of stretch on expressions of force and power over excessively long stretch durations. The proposed negative influence of stretch on set and session kinematics and kinetics may be minimized by stretching the antagonist while training the agonist or stretching the lower body while training the upper body and vice versa. The net effect of such work to rest paradigms warrants investigation.

If stretch does influence the set and session kinetics negatively, we need to quantify the magnitude of this decrement and determine whether the increase in time under tension and associated neuromuscular adaptations compensates for the decrement in mechanical stimuli such as force and work. Furthermore, the differential influences of active and passive tension/stretching also need investigation, and how one can maximize tension during the work period also is worthy of research (using weights to stretch muscles at extended positions, pause training at long muscle lengths). Once a greater understanding of these questions is gained, the application of the findings into long-term training protocols assessing hypertrophic adaptation is needed.

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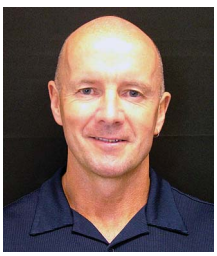

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