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The Effect of Varying Eccentric Velocity on Muscle Hypertrophy

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ABSTRACT

Resistance training is essential to muscle hypertrophy as it fatigues fibres through time-under-tension (TUT). As myocyte energy depletes, metabolites accrete, leading to inflammation to increase cell size so it is adapted for future stimuli. TUT can be measured by varying eccentric velocities: i.e., the rate at which a muscle lengthens under load. A longer period of lengthening will lead to greater metabolite accretion and inflammation. However, it is unknown whether TUT has a threshold or if it can gradually increase and lead to more muscle growth. Through a literature review and an experiment, this project investigates the effect of varying eccentric velocity on muscle hypertrophy. Previous research in the field of muscle physiology and metabolism were explored, with an emphasis on eccentric training. The supplementary experiment measured shoulder growth in response to the medial deltoid exercise called lateral raises, where different eccentric velocities were assigned to groups. Individualistic daily calorie and protein intake were controlled to ensure that sufficient nutrients were available for recovery and performance. Post-experimental research suggested that high-velocity eccentric training was best for hypertrophy due to greater levels of force production. This was consistent with the experiment, which found that those with a fast-velocity eccentric, a lower TUT, experienced greater growth. They also exhibited greater strength gain due to a neuromuscular junction adaptation. These findings are significant for designing exercise regimens that are optimal for the prevention and rehabilitation of musculoskeletal injuries and disorders. The review's findings suggest that fast-velocity eccentric contractions are ideal for increasing muscle size and strength.

3.0 Keywords: Muscle hypertrophy, eccentric training, contraction velocity, medial deltoid

1. INTRODUCTION

Time-under-tension (TUT) is the duration a muscle actively contracts during resistance training. Time-under-tension as a contributor to muscle hypertrophy is rarely discussed in scholarly circles but is worth more consideration. This paper explores the effect of manipulating TUT for working muscles during resistance training, and if increased TUT equates to increased hypertrophic effects. First, a foundation of relevant muscle functionality will be established through a literature review of muscle hypertrophy. The concepts of metabolic stress and mechanical tension as mediators for muscle hypertrophy will be the central focus of the review. Next, the different types of muscle contractions will be outlined to determine which is most conducive to muscle hypertrophy. Altering muscular contraction velocity as a method of manipulating TUT will also be explored. Finally, a supplemental experiment measuring the effect of varying TUT on muscle hypertrophy will be outlined. This experiment measured shoulder growth over a 6-week period in response to different contraction velocities. The results of this experiment will be analysed and discussed. Specifically, we will explore what caused the experimental results and how the literature supports them. Conclusively, this paper will discuss the resistance training regimen most conducive to muscle hypertrophy, incorporating findings from the literature review and experiment.

2. LITERATURE REVIEW

2.1 Causes of Muscle Hypertrophy

2.1.1 Metabolic Stress

Metabolic stress following resistance training leads to numerous hypertrophic effects that impact the subcellular structure of myocytes, most notably the accretion of metabolites within the cells. Sufficient training intensity has been shown to elicit fast glycolysis for quick energy generation, in the form of ATP.[1] Lactic acid is released as a by-product of this process, which

dissociates in the blood plasma to form lactate. As resistance training progresses, plasma lactate saturates in the arteries and veins surrounding working muscles, increasing occlusal pressure and leading to an inhibition of blood flow, furthering metabolite accumulation.[2,3] Blood pools more in these areas due to a process known as reactive hyperaemia; a transient increase in blood flow following arterial and venous occlusion.[4] This mode of concentrating metabolites will lead to numerous hypertrophic effects, either by activating certain metabolic pathways within the myocytes or through extrinsic cellular interactions. The effectiveness of every metabolic stressor discussed in this section is dependent on the intensity and duration of resistance training. Essentially, as intensity and duration increase, metabolic stress will also increase.

Increased muscle fibre recruitment is one of the effects of metabolic stress following resistance training that can have hypertrophic effects. As resistance training progresses, more muscle fibres are incrementally recruited to sustain the contractions for the working muscle group holistically, eventually leading to full muscle group fatigue.[5] Before understanding muscle fatigue, it is important to note that total number of adenine nucleotide molecules within a myocyte remain constant.[6] Essentially, the ratio of ATP, ADP, and AMP must always be the same. For example, if one molecule of ATP is used by a muscle fibre for contraction, then two molecules of ADP will be converted to one molecule of ATP and AMP. The muscle now has another ATP molecule for energy, and the extra AMP will be degraded by AMP-deaminase to IMP and ammonia. Thus, the ratio is maintained. When muscles have an insufficient ATP supply, they will fail to generate sufficient force for the movement, leading to fatigue. Additionally, the continuous amount of ammonia molecules generated from maintaining the adequate ratio will be accumulated in blood plasma. These will be converted to urea nitrogen, which is harmful in high concentrations.[7] Muscles will inhibit further metabolism of ATP molecules to mitigate the downstream deleterious effects by urea nitrogen. The muscle fibres that are receiving blood from vessels with high concentration of this harmful substance will cease to function. But, if the resistance training is maintained, then other fibres not affected will be recruited. The generation and accumulation of the metabolites will cause muscle fibres not being employed for resistance training to become active, because the ones previously used are fatigued while the movement is maintained. Hence, metabolic stress is necessary to exhibit full activation of muscles.

Introducing the significant role of myokines must first be met with an overview of hypertrophic myocyte anatomy. Muscle cells are one of the few exceptions to the cell theory, as they are multinucleated. Additionally, the process for muscle growth would first involve increasing the myonuclei, and then increasing the size. This is because the extra genetic material in the new myonuclei is necessary for growth and development of the cell. The myonuclei are created through the differentiation of satellite cells: multipotent, muscle stem cells.[8] These satellite cells infiltrate and proliferate within myocytes to increase the myonuclear saturation, giving rise to greater size and strength capacity for the muscle.[9] Therefore, satellite cell accretion is a necessary precursor to muscle hypertrophy.

Resistance training also leads to the synthesis of myokines that contribute to hypertrophic adaptations over time. Myokines are cytokines synthesized and released within skeletal muscle cells during muscle contractions.[10] Some myokines that have been abundantly explored in the scientific literature are Interleukin-6 and Interleukin-15 (IL-6, IL-15). IL-6 is an essential regulator of satellite cell-mediated hypertrophic muscle growth.[11] IL-6 signaling has been associated with myogenesis through the regulation of the proliferative capacity of satellite cells. Satellite cells are activated and undergo asymmetric division to both maintain the satellite cell pool, and generate daughter committed myoblasts.[12] Myoblasts are mononucleated precursors that differentiate further to form multinucleated muscle fibres.[13] Hence, IL-6 mediating the infiltration and proliferation of satellite cells in myocytes leads to hypertrophic muscle effects. IL-15 is a different myokine that is used to increase protein accretion to induce muscle hypertrophy.[14] Specifically, proteins are accumulated in sarcomeric myosin-actin chains in myotubes, the microstructures of skeletal cells. Increasing these miniscule functional units enables a muscle to increase in volume and strength capacity. Thus, IL-15 is a significant myokine that allows for increases in muscle size. Both IL-6 and IL-15 are important myokines for inducing muscle hypertrophy.

Muscle fatigue because of resistance training can lead to heightened production of reactive oxygen species (ROS). ROS, both radical and non-radical oxidizing agents, in low concentrations can lead to muscle hypertrophy. The two most potent ROS regarding positive benefits are superoxide and hydrogen peroxide (Farooqui, 2008). ROS are primarily generated as the by-product of the mitochondrial electron transport chain (ETC). It has been shown that 0.2-2% of electrons in the ETC do not follow the normal transfer order and leak out to interact with free oxygen to yield radical or non-radical ROS.[15,16] These ROS are in the mitochondrial matrix, specifically the FMN site and the CoQ binding site. They are created during the transfer of electrons from NADH to CoQ. The accumulation of ROS can lead to acute hypoxia in the vessels surrounding working muscles, producing a superoxide burst in arterial endothelial cells.[17] Although chronically elevated levels of ROS have been implicated in negative effects on health, acute accumulation enhances cellular signaling pertaining to hypertrophy.[18–

[22] This is hormesis, a phenomenon in which a harmful substance gives beneficial effects at a low concentration.[23] At a relatively low concentration, ROS will stimulate extra Calcium (Ca^{2+}) release from the sarcoplasmic reticulum of myocytes.[24] Ca^{2+} binds to the C-component of the actin-filament in sarcomeres, exposing the binding site for the myosin head which generates a cross-bridge and stimulates muscle contraction.[25] Thus, increased Ca^{2+} release will enable the muscle to generate more force for contraction, which will lead to faster fatigue, and hypertrophic effects. ROS can also influence hypertrophy by mediating transcription of highly conserved stress proteins called heat shock proteins (HSPs).[26] These abundant proteins are synthesized in response to harmful agents such as environmental stresses, infection, gene transfer, or in this case, ROS. All functions of HSP can be attributed to molecular stress sensing and a subsequent protein folding synthesis response.[27] Exercise is the main stimuli associated with a robust increase in different HSPs in skeletal muscle tissue. Specifically, HSPs facilitate the cellular remodeling process during muscle growth.[28] Following resistance training, major oxidative damage to muscle proteins triggers HSP expression, to ensure that muscle growth is effective, efficient, and systematized. Clearly, the generation of ROS, a metabolic stressor, is an effective vehicle for inducing muscle hypertrophy following resistance training.

Cell swelling is a by-product of metabolic stress following resistance training that proves to exhibit hypertrophic effects. Cell swelling may physiologically regulate certain cell functions that enhance muscle growth.[29] During resistance training, veins surrounding the muscle tissue are compressed, restricting the removal of blood. However, arteries continue to supply blood and oxygen to the working muscles, thereby creating an increased concentration of intramuscular blood plasma. Higher intra-arterial pressure leads to plasma leakage from capillaries into the interstitial space, building a plasma extracellular pressure gradient.[30] This dense gradient, through passive diffusion, causes excess plasma to diffuse back into the muscle. The cascading signaling response is facilitated by integrin-associated volume osmo-sensors within the muscle fibres.[31] As the membrane of the myocytes undergo hydration-induced stretching, the aforementioned sensors activate anabolic protein-kinase transduction pathways. This hyperhydration has a direct effect on amino acid transport systems such as phosphatidylinositide 3-kinase modulating glutamine and methyl aminoisobutyric acid transport in muscle.[31] Improving the rapidity of amino acid transport, and subsequent protein accretion will enable hypertrophy. Fast-twitch fibres in particular are sensitive to osmotic changes, possibly related to a high concentration of water transport channels called aquaporin-4 (AQP4).[32] Given that fast-twitch fibres are the most susceptible to hypertrophy, it is possible that cellular hydration influences the hypertrophic response following resistance training, which includes protein accretion.[33] Thus, cell swelling proves to possess some hypertrophic effects.

2.1.2 Mechanical Tension

Mechanical tension on muscle contractions during resistance training plays an important role in the hypertrophic process. Mechanical tension applied to muscles has the ability to trigger a cascade of biochemical reactions from physical stimuli such as kinase activity, sarcomere stiffness, and rearrangement of myocyte architecture.[34] This section of the literature review will focus on the physical and biochemical cellular and subcellular manifestations of mechanical tension.

Mitogen-activated protein kinase (MAPK) is a regulator of gene expression that has been shown to contribute to the adaptive response in muscles that generates growth.[35] Specifically, MAPKs are involved in relaying extracellular stimulations to intracellular responses. This includes satellite cell proliferation and differentiation. One group of MAPKs, c-Jun amino-terminal kinases (JNKs) has been shown to be a mediator of hypertrophic responses in muscle cells.[36] Indeed, Aronson et al. (1998) found c-Jun mRNA levels elevated in Northern blot analysis of muscle samples taken from subjects who underwent an exercise regimen.[37] This is because the JNK pathway mediates cellular responses to environmental stressors, which includes rigorous activity.[36,38,39] More specifically, the metabolic stressors previously covered in this literature review can facilitate the JNK pathway, including high plasma pressure and myokine synthesis.[39] Relaying mechanical extracellular stimuli to intracellular signaling pathways proves to be the primary function of MAPKs, which recognizes them as mediators of muscle hypertrophy following resistance training.

Applied mechanical tension can be generated by the force of the weight as well as the stretch endured by the muscles being worked. The addition of the stretch in conjunction with the weighted force leads to an additive effect that pronounces muscle fatigue, leading to a hypertrophic response.[40–42] One enzyme that is often observed when studying the stretch of a muscle is p70S6k, a protein kinase that targets the substrate S6 ribosomal protein.[43,44] Phosphorylation of S6 results in protein synthesis at the ribosome. Hornberger and Chien (2006) checked the effect of mechanical stretch on the prevalence of p70S6k and witnessed a sizable increase.[42] This suggests that phosphorylation of S6 increased drastically following mechanical stretch, which would allow the ribosome to synthesize more usable proteins that could be accreted to the damaged muscle fibres. Hence, applied mechanical tension enables the muscles to fatigue and grow more.

Mechanical tension leads to localized muscle tissue damage that can accelerate growth and recovery. Lengthening muscle contractions in particular have been shown to produce ultrastructural damage such as microscopic tears in contractile proteins within the muscle cells.[45,46] This promotes muscle protein turnover, and increased protein accretion in the damaged area, leading to an increase in overall size within the sarcomeres. Resistance training initially causes myotrauma: damage to the underlying muscle tissue.[47] Indeed, Staron et al. (1990) found the cross-sectional area of specific muscles to be sizably lower.[47] However, cross-sectional areas of the same muscle in different participants, ones with adequate rest and nutrition, experienced sizable amounts of type-II muscle fibre increases. This suggests that the microscopic shearing on the muscle proteins was a result of the mechanical tension from the resistance training but was also a precursor to a larger muscle post-recovery. Another noticeable change was a substantial increase in mitochondrial density in the targeted muscle, which is to be expected given that a now larger cross-sectional area of the sarcomere would require more energy to sustain sufficient cross-bridge formations.

Mechanical tension applied through resistance training has been shown to alter ionic concentrations within the working muscles. The muscle damage that ensues resistance training most drastically affects the Ca^{2+} abundancy.[48] Mitochondrial Ca^{2+} increases have been observed, due to shear tears along the length of the sarcoplasmic reticulum, which harbors reservoirs of Ca^{2+} ions.[49] Additionally, T-tubules, which are located at the A-I junction in muscle fibres can be severely distorted or damaged, which would lead to rapid equilibration of the intracellular and extracellular spaces, causing an influx of Ca^{2+}.[50,51] The change in intracellular calcium concentration then leads to a cascade of chemical activations such as the previously mentioned MAPK, resulting in muscle hypertrophy.[52] Hence, the distortion of muscle architecture caused by mechanical tension can alter the growth of the muscle.

The most potent effect of mechanical tension as physical stimuli relates to a change in muscle architecture that induces damage, fatigue, and biochemical changes that translate to muscle hypertrophy. Prado et al. (2005) assert that the contractile performance of skeletal fibres largely depends on the myosin heavy chain (MHC) isoform and the stiffness of the titin spring.[53] The MHC is the actin-based motor protein that generates mechanical force from ATP.[54] The titin-spring is a spring in sarcomeres that is activated by active mechanical tension. This provides the muscle with extra force during the stretch.[55] Using heavy resistance training during this stretch will disrupt the stiffness of the spring, and essentially degrade the elasticity of the I-band region which consists of the titin isoform. Now with regard to a different ultrastructure, the Z-band showcases a broadening and disruption effect during a mechanically active stretch, as demonstrated by Friden et al. (1981).[50] Indeed, the lattice pattern in the Z-bands became disorganized, suggesting that high tension leads to mechanical disruption of the interdigitating arrays of actinin-tropomyosin microfilaments.[56] As suggested by Sasuki et al. (1982), the Z-band's tropomyosin and actinin microfilaments have been shown to be susceptible to reconstitution by Ca^{2+}-activated factor (CAF).[56] This is a growth factor that is typically released by damaged muscle fibres after accumulation of Ca^{2+} due to sarcoplasmic reticulum disruption. Hence, the altercations in muscle architecture that result from resistance training are diverse and prove to be highly effective in triggering muscle damage, which will act as a precursor to muscle hypertrophy.

2.2 Types of Muscle Contractions

There are different types of muscle contractions employed by the human body during resistance training, each of which has unique characteristics. There are two kinds of dynamic contractions, eccentric and concentric.[57] Eccentric contractions occur when the muscle length is increased, usually for decelerating or controlling motions. This is also known as the "negative" of a resistance-based exercise. Differently, concentric contraction occurs when the muscle length is shortened. The third kind of muscle contraction, which is not dynamic, is isometric contraction.[57] Isometric, also known as a static hold, is when muscles contract without motion or length changes. This is typically done to actively stabilize a joint. Given this information, we will explore which type is most beneficial to centralize in a hypertrophy-focused training regimen.

2.3 Advantages of Increasing Time-Under-Tension During Muscle Contractions

This literature review has explored the hypertrophic effects of metabolic stress and mechanical tension, and the research has led to the belief that increasing the TUT for a muscle will exhibit hypertrophy. Regarding metabolic stress, a longer TUT for the working muscle will lead to more metabolite accumulation. Muscle contractions for longer periods of time will lead to restricted blood flow, allowing for more muscle fibre recruitment. Myokine activation will also increase, leading to a greater number of satellite cells infiltrating and proliferating, undergoing myogenesis. Lastly, reactive oxygen species (ROS) and cell swelling will

rise, leading to more damage and alterations to muscle architecture. With alternative regard to mechanical tension, MAPK and other myokines will be synthesized and released to a greater degree, resulting in more gene expression and stimulation of myogenesis. A longer duration will also lead to more muscular stretching that can disrupt microstructures such as the Z-band and titin-spring. Finally, activation of p70S6k will become more abundant, leading to a greater expression of S6 ribosomal protein, which will lead to elevated protein synthesis and accretion within the muscle tissue. When varying the TUT to test this hypothesis, it is best to focus on the eccentric movement of a muscle because a slower negative during resistance training is generally regarded as safer to maintain than a concentric or isometric movement, and has a greater plethora of data surrounding its safety.[51] Hence, it is predicted that increasing TUT in eccentric muscle contractions will lead to more hypertrophy.

3. EXPERIMENT

3.1 Methods

The experiment supplementing this literature varies the velocity of eccentric contractions during the medial deltoid exercise called dumbbell lateral raises to observe the change in shoulder width as a result of muscle hypertrophy. Four volunteering participants were split into two even groups. Each group was assigned a different eccentric velocity for the exercise being performed. Each participant in a group had another participant in the other group that had a similar starting shoulder width, in order for post-experimental comparative analysis to be consistent. For instance, participant E1-1 had a comparatively similar starting shoulder width to participant E4-1. Group E1 had an assigned velocity of 1-second, meaning that the lowering of the dumbbell after reaching shoulder height, or the peak, had to be completed over the course of one second. The eccentric velocity for this group was $90^{\circ}/s$, as the range of motion for the exercise is generally 90 degrees because the arm starts in a vertical position near the hip and rises to a horizontal position in line with the shoulder joint. Group E4 had to do the same motion, but lower the weight over the course of four seconds. This group had an eccentric velocity of $22.5^{\circ}/s$, precisely a quarter of the speed of the previous group. Having two different velocities that were magnitudes apart allowed for distinct comparisons after the experiment, to determine how pronounced the effect of increasing TUT would be. Participants were asked to start with a dumbbell weight that was comfortably performed for one complete set of twelve repetitions. This ensured that the exercise technique of the participants was consistently accurate, so as to avoid injury, and employ the correct muscle fibres. The experiment was conducted over the course of 6-weeks, with 3 sets of 12 repetitions every day for 6 days a week as the training regimen. This regimen provided the participants with a sufficiently rigorous routine to promote muscle fatigue, while giving them enough time for adequate recovery. The participants were offered the opportunity to increase the weight of the dumbbells during every session of training if the stimulus was not challenging enough. Shoulder width measurements were taken after the final weekly training session. Measuring tape was wrapped around the shoulder width of the participants, specifically over the peak of the medial deltoid muscle. Consistently measuring this point measured the maximum change in the muscle's size. The participants were provided with a diet set at maintenance level with elevated protein content. Essentially, the caloric intake was calculated to be at a daily maintenance, but the protein content was set to be 0.5 g/lb of body weight. This ensured that the participants had enough nutrients and protein to facilitate muscle growth. Additionally, the participants were suggested to sleep 8 hrs/night so as to promote adequate rest and recovery.

3.2 Analysis

The weekly shoulder width measurements of the participants can be found in Table 1. After the 6-week experimental period, it was found that the percent difference between starting and final shoulder width was nearly two-folds greater for the 1-second eccentric group than the 4-second eccentric group, as showcased in Table 2. Figure 3 and Figure 4 illustrate the results of Table 1 and Table 2, respectively. The participants with a faster negative exhibited significantly higher muscle growth than their slower negative partners who had similar starting shoulder width. This is contradictory to the hypothesis originally proposed which suggested that increased TUT via slower eccentric velocity would lead to more muscle growth. The fast-velocity eccentric group also developed more strength over the course of the 6-week period. Indeed, participants E1-1 and E12 increased the weight of the dumbbell by 10 lbs and 12.5 lbs, respectively. This is greater than the slow-velocity eccentric group, which added 5 lbs and 7.5 lbs for participants E4-1 and E4-2, respectively. After witnessing these results, a thorough literature review regarding fast-velocity eccentric contractions was done to determine why faster negatives were more conducive to muscle hypertrophy. Note, this project is primarily concerned with muscle hypertrophy, so a discussion of potential strength gains will not be included. However, strength gains as a result of this regimen are worth exploring in the future.

4. DISCUSSION

4.1 Advantages of Eccentric-Focused Resistance Training

Resistance training should center on the eccentric phase of an exercise as muscles are stronger during this period. Muscles exhibit greater strength and are more prone to fatigue during the eccentric loading phase, leading to a stronger hypertrophic effect during recovery.[58]

As demonstrated in an experiment by Colliander and Tesch (1990), the torsional force, or torque, generated by eccentric muscle contractions is statistically more significant in comparison to isometric or concentric contractions of the same exercise and weight.[59] Generating torque is crucial to inducing mechanical tension on muscle fibres, which has been shown to be essential in eliciting muscle breakdown and subsequent regeneration, as asserted previously in this literature review.[59,60] Indeed, Higbie et al. (1985) found that eccentric contractions of a resistance exercise generated greater voltage on electromyogram data, implying greater influx of Ca^{2+}, which would be required by more rigorous exercise utilizing more musculature.[61] Note, although the force generated by eccentric contractions is shown to be substantially more than isometric and concentric contractions, the oxygen consumption and heat generation is considerably less.[62,63] The experiments conducted by Hill (1960), and Elmer and LaStayo (2014) found that participants generated equal power during eccentric contractions as other modes of muscle contraction, but with a fraction of the oxygen consumption, and with a miniscule change in mean muscle temperature.[62,63] This suggests that eccentric contractions generate higher force with a lower cost; thus, it is logical to emphasize eccentric loading in training regimens if the goal is muscle hypertrophy with adequate recovery.

Another reason as to why force output during the eccentric phase is greater than other types of muscle contractions involve the characteristics of sarcomeres. The elasticity and stored energy of cross-bridges become pronounced during the eccentric loading phase of an exercise.[64,65] Indeed, Huxley (1957), and Huxley and Simmons (1971) found that muscle fibres maintain an unknown amount of residual energy after the concentric movement, which is then added upon by the force generated during the eccentric phase. This cumulative force effect leads to an energy output that is greater than concentric alone. The aforementioned authors assert that the increased force during stretch is induced by rapid detachment and reattachment of cross-bridges during the eccentric phase. Essentially, cross-bridges in sarcomeres quickly release and reattach numerous times over as the weight is lowered. This is different from concentric contractions, where the entire muscle length shortens, leading to all cross-bridges moving in one direction and holding at the final shortened length. During an eccentric contraction, the cross-bridges release to increase the length, but incrementally, and very quickly, reattach to shorten the length, which momentarily slows down the lengthening process and generates additional additive force during the movement. Additionally, the quicker the movement, the lesser the number of cross-bridges formed during the contraction, and the higher rate of cross-bridges detachment.[66] There is a shorter window of time for myosin and actin to rebind, so although there is a temporary cross-bridge created to shorten the sarcomere, the period is not sufficiently long enough, and the muscle will continue to lengthen but with more force placed upon it per unit time, leading to more fatigue and damage. Additionally, the flexible fragment of the myosin tail close to the globular head, or S2 complex, will not be fully extended during a fast-lengthening period, which results in compression of the S2 complex, decreasing the pulling force applied to actin.[3,67] This allows the velocity of the filament's movement to essentially approach zero for a minuscule amount of time, at which cross-bridges can rapidly form before being detached. Given that the cross-bridges will form quickly, and the myosin is fully stretched, breaking the cross-bridges will require more force. This greater force requirement must be generated by the muscle, leading to more fatigue.[68] Given how quick this process is, and how it occurs frequently during the lengthening process, it is clear why the force generated during the eccentric loading phase is high, and why it has a low associated energy cost.

The presence of titin is another explanation as to why muscles are stronger during the eccentric phase of contraction. As previously mentioned in this literature review, titin is a protein that increases stiffness in the sarcomeres and leads to stiffness during the active stretch.[69,70] Indeed, Bagni et al. (2002) found that sarcoplasmic stiffness increased after cross-bridge formations due to titin.[71] Powers et al. (2014) found that titin-based force during active stretching was an inherent property of skeletal muscle, explaining up to 85% of the extra force generated during the eccentric phase.[72] The titin binds to the thin actin filament at one end of the Z-band in the sarcomeres, and tightens that section of the band to increase structural rigidity. Note, in these experiments, titin-binding affinity was unhindered by Ca^{2+} saturation, which is consistent with previous theory mentioned in this literature review as Ca^{2+} is expected to accumulate during muscle contractions. In fact, one study by Joumaa et al. (2008) found that force enhancement was partly caused by a calcium-induced increase in titin stiffness, and this added to the extra cross-bridge stiffness effect previously mentioned.[73] It is unclear as to what this mechanism is, but the results were consistent with the existence of some causal pattern. An overview of the

scientific literature surrounding the protein titin suggests that it enhances force production within skeletal muscle tissue during the eccentric loading phase by increasing structural rigidity.

4.2 Advantages of Fast-Velocity Muscle Contractions

Lengthening muscles during the eccentric phase of an exercise should be done at a relatively fast velocity as it is the most beneficial for muscle hypertrophy. Contractile force production proves to be higher at faster velocities due to the greater amounts of torque generated.[74,75] Moreover, these higher forces are more pronounced during the eccentric phases of an exercise. Indeed, Farthing and Chilibeck (2003) found that peak torque generated between fast- and slow-velocity contractions, for both eccentric and concentric phases, was significantly higher for the fast-eccentric group.[76] The set velocities were $180^{\circ}/s$ and $30^{\circ}/s$.[76] Faster eccentric velocity is also suggested for exercises that target muscles with a higher density of type-2, intermediate- or fast-twitch muscle fibres. This is because the volume and size that type-2 muscle fibres possess is substantially more than that of type-1.[77] Additionally, these fibres are capable of lifting with tremendous force, and are recruited most during heavy weightlifting. Utilizing a fast-velocity eccentric for muscle contraction will require more concentrated force, a specialty of the type-2 muscle fibres. Hence, for muscle hypertrophy, using fast-velocity eccentric phases should be the primary goal of training.

5. CONCLUSION

This paper discussed the concept of time-under-tension, and whether it is a significant contributor to muscle hypertrophy. The preliminary literature review suggested that increased time-under-tension would induce more metabolic stress and mechanical tension, leading to more muscle fatigue, and subsequent hypertrophy. However, the supplemental experiment suggested that the opposite was true. Indeed, fast-velocity eccentric contractions were superior to the slow-velocity variations.

Further exploration of the literature supported this experimental finding. Specifically, the centrality of this superior mode of training is the concept of torsional force. Indeed, torque increases significantly during fast-velocity contractions. Coupled with eccentric-type contractions that are more conducive to higher loading on the working muscles, this method of training proves to be ideal for fatiguing the fibres, and generating hypertrophy. Although this topic requires more inquiry to definitively determine its validity, the promises of improving muscle mass through fast-velocity eccentric contractions appear to centralise on a method of resistance training that is superior to all others. Including fast-velocity eccentric exercises in one's regimen should increase muscle mass, and perhaps strength. This will prove fruitful for the prevention, and perhaps rehabilitation of musculoskeletal injuries and disorders.

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9. APPENDIX

Table 1. **Table 1: Weekly shoulder width measurements for each participant during the six-week experiment.**

Participant shoulder width (cm)		E1-1	E1-2	E4-1	E4-2
Week	1	47.9	43.2	47.3	44.7
	2	48.7	46	47.6	44.8
	3	49.2	47.7	47.9	45.7
	4	49.2	48.4	47.9	47
	5	50.3	48.5	48.5	47.2
	6	50.8	49	48.5	47.8

Table 2. **Table 2: Percentage difference between initial and final shoulder width for all four participants**

Participant	E1-1	E4-1	E1-2	E4-2
Difference between initial and final shoulder width (%)	5.88	2.51	12.6	6.7

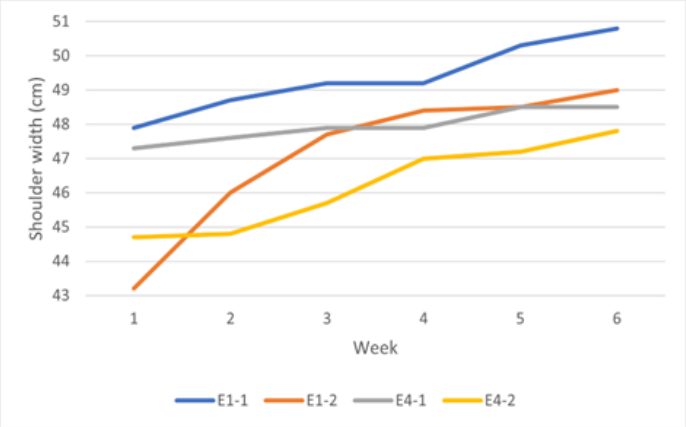

Figure 3. The changes in shoulder width (cm) in response to lateral raises (6x/week) over the course of 6 weeks. Participants of groups E1 and E4 were assigned 1-second and 4-second velocity eccentrics, respectively.

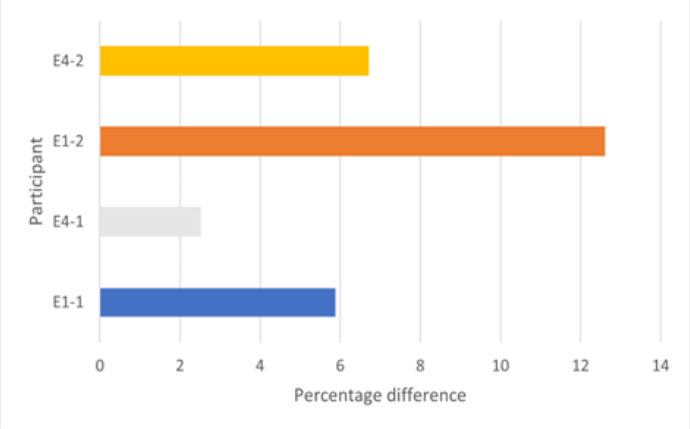

Figure 3. The percentage difference in shoulder growth between participants that had similar starting shoulder widths, but with different assigned velocity eccentrics.

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