Review:

Muscle Damage in Resistance Training —Is Muscle Damage Necessary for Strength Gain and Muscle Hypertrophy?—

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The objectives of this review are to delineate muscle damage induced in resistance training, discuss how muscle damage is associated with increases in strength and/or muscle cross-sectional area, and clarify whether "pain" is necessary for "gain" in muscle function and muscle volume in resistance training. Resistance training induces muscle damage, especially in the initial phase of training when unaccustomed eccentric actions are performed. Muscle damage is best indicated by loss of muscle strength and range of motion, and delayed onset muscle soreness does not necessarily reflect the magnitude of muscle damage. Resistance training employing eccentric biased exercises has potential for increasing not only eccentric strength but also isometric strength, and seems to be superior to exercise based solely on concentric actions. Since muscle damage and soreness are induced to a greater extent in eccentric muscle actions than with isometric or concentric actions, it is believed that muscle damage is necessary for size and strength gain. However, it appears that muscles become less susceptible to muscle damage as training progresses. We concluded that muscle damage and/or muscle soreness are not necessarily indispensable for exercise-induced muscle adaptation.

Keywords: eccentric exercise, muscle soreness, creatine kinase, adaptation, training

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1. Introduction

In order to produce training adaptation, a physiological system requires overload stimulus exceeding the level to which it is presently accustomed. Training frequency, volume, duration, and intensity are the variables that provide overload. "Pain" during or after training regularly accompanies overload situations, and it seems that in order for a training adaptation or "gain" to occur "pain" is often a pre- or corequisite. This may partially shed light on the phrase popular in sports culture, "No Pain, No Gain." Physical pain generally occurs when any tissue is being damaged, and is one of the symptoms of inflammation. It is anecdotally recognized that muscle damage is effective for gain in muscle strength and size, but it has not been clarified how much muscle damage is necessary for such gain. The objectives of this paper are to 1) delineate what muscle damage is, 2) outline how muscle damage is induced in resistance training, 3) discuss how muscle damage is associated with increases in strength and/or

muscle cross sectional area, and 4) clarify how much "pain" is necessary for "gain" in muscle function and muscle volume.

2. What is Muscle Damage?

"Damage" is generally defined as "a loss due to injury," and muscle damage is often referred to as "muscle injury" [Armstrong et al. (1991)]. Safran et al. (1989) have stated that muscle injury can be divided into three major types based on clinical presentation. Type I injury is muscle soreness that occurs 24 to 48 hours after unaccustomed exercise, so-called delayed onset muscle soreness (DOMS). Type II injury is characterized by an acute disabling pain from a muscle tear, ranging from a tear of a few fibers with fascia remaining intact to a complete tear of the muscle and fascia. Type III injury includes muscle soreness or cramps that occur during or immediately after exercise. Although the exact underlying mechanism(s) of DOMS remains elu-

Fig. 1. Comparison between eccentric (ECC), concentric (CON) and isometric exercise (ISO) for changes in muscle soreness (a), isometric strength (b), and plasma CK activity (c) following 3 sets of 10 repetitions (5 s of maximal contraction every 15 s) of each exercise bout. *: significant ($P < 0.05$) difference between ECC and the other two exercises. (unpublished data)

sive, it is generally accepted that it involves microinjury to skeletal muscle fibers and extracellular matrix followed by inflammatory responses [Armstrong et al. (1991); Clarkson & Sayers (1999); Proske & Morgan, (2001); Pyne (1994); Stauber & Smith (1998)]. It has been documented that eccentric muscle actions result in greater injury to skeletal muscle than concentric or isometric muscle actions [Gibala et al. (2000); McCully & Faulkner (1985)]. **Fig.1** shows comparisons between eccentric, concentric, and isometric exercise for changes in muscle soreness, isometric strength, and plasma creatine kinase (CK) activity following 3 sets of 10 repetitions of each exercise bout. Compared to concentric and isometric exercise, the changes in isometric strength after eccentric exercise resulted in a larger and more protracted decrement in strength. Eccentric exercise also showed a larger increase in plasma CK activity, whereas the other two conditions did not increase. Similarly, muscle soreness was significantly increased after eccentric exercise only. Therefore, it seems plausible to state that DOMS (Type I injury) is closely associated with eccentric exercise-induced muscle damage.

3. Mechanism of Muscle Damage and Eccentric Muscle Action

Exercise-induced muscle injury has been reported to range from a disrupted sarcomere or sarcolemma to a "broken" myofiber, with the cause believed to be due primarily to eccentric (lengthening) muscle actions [Armstrong et al. (1991); Clarkson & Sayers (1999); Fridén & Lieber (2001); McCully & Faulkner (1985); Proske & Morgan (2001)]. Gibala et al. (1995) compared eccentric and concentric phases of muscle action

Fig. 2. Number of disrupted fibers within each category (focal: an area of disruption occupying 1-2 adjacent myofibrils and/or 1-2 continuous sarcomeres, moderate: an area of disruption occupying 3-10 continuous sarcomeres and/or 3-10 adjacent myofibrils, as well as a fiber containing >10 focal areas of disruption, extreme: an area of disruption spanning >10 continuous sarcomeres and 10 adjacent myofibrils) found in baseline, immediately post, and 48 hours post concentric (CON) or eccentric (ECC) exercise (8 sets of 8 repetitions at 80% of 1RM). *: significant ($P < 0.05$) difference. Data from the study by Gibala et al., 1995.

for changes in skeletal muscle ultrastructure after a single bout of resistance exercise of the elbow flexors (8 sets of 8 reps at 80% of concentric 1RM) performed by male subjects with no previous history of resistance training. As shown in **Fig.2**, myofibrillar disruption was found in the biopsy samples (30-54 fibers) taken from both eccentric and concentric arms; however, a significantly larger number of fibers (>80%) were disrupted in the eccentric arm compared to the concentric arm (>30 %). A significantly larger number of muscle fibers exposed to eccentric load showed extreme disruption when compared to that of the concentrically biased exercise.

Damage to the excitation-contraction (E-C) coupling system and disruption at the sarcomere level are considered to be possible initial events responsible for the subsequent damage induced by eccentric muscle actions [Proske & Morgan (2001); Warren et al. (2002)] (**Fig.3**). It has been proposed by Armstrong et al. (1991) that repeated eccentric muscle actions induce initial damage to sarcomeres and/or sarcolemma followed by secondary damage when intracellular calcium concentration exceeds a limit ($\sim 0.1 \mu\text{mol/L}$). Note that initial damage is focal, and the number of muscle fibers advancing to secondary damage does not seem to be large [Fridén & Lieber (2001)].

It seems likely that one important factor in determining the magnitude of muscle damage is the level of force during muscle actions [Armstrong et al. (1991); Clarkson & Sayers (1999); Fridén & Lieber (2001)]. This is believed to be the reason why eccentric muscle actions, which are capable of producing higher

Fig. 3. Hypothetical mechanism of exercise-induced muscle damage by exercise with repetitive muscle actions or lengthening muscle actions. Sequence of events leading to initial and secondary muscle damage, and factors involved in the sequence are shown. This figure was created by the authors based on data drawn from other publications [Armstrong et al., (1991); Clarkson & Sayers, (1999); Fridén & Lieber, (2001); Proske & Morgan, (2001)].

force than isometric or concentric muscle actions, result in significantly greater muscle damage. Mechanical factors such as high force or strain appear to initiate the damage process by disturbing intracellular calcium homeostasis or mechanically disrupting the integrity of sarcomeres [Armstrong et al. (1991); Clarkson & Sayers (1999); Fridén & Lieber (2001), Proske & Morgan (2001)]. It has been shown that as little as 2 maximal eccentric actions of the elbow flexors can induce muscle damage [Nosaka et al. (2001b)], however, the larger the number of eccentric actions, the greater the magnitude of muscle damage (**Fig.4**). This would suggest that factors associated with repetitive eccentric muscle actions are also involved in the mechanism of muscle damage. It is interesting to note that eccentric muscle actions performed at greater muscle length (descending limb) induce more muscle damage than those at a shorter length (ascending limb). For example, eccentric exercise of the elbow flexors over an elbow joint angle range from 100-180 degrees resulted in greater damage than those performed from 50-130 degrees [Nosaka & Sakamoto (2001)]. It has been documented that sarcomere inhomogeneities may result in overstretching of some weak sarcomeres, causing them to "pop" during repeated eccentric actions, inducing disruption of sarcomeres and the plasma membrane [Proske & Morgan (2001)]. When cytosolic calcium concentration exceeds a certain threshold (0.1 mmol/L), calcium-sensitive degradation pathways are activated [Armstrong et al. (1991)] (**Fig.3**). Calpain, a nonlysosomal, calcium-activated neutral protease, has been shown to cleave cytoskeletal proteins such as

Fig. 4. Changes in maximal isometric strength from preexercise level (100%) following different numbers of maximal eccentric actions (2, 6, 12, 24, and 60) of the elbow flexors for 96 hours after exercise. Drawn from data of our previous publications [Nosaka et al., (2001a), (2001b); Nosaka et al., (2002)].

desmin, α -actinin, and vimentin, and induce degradation of the myofibrillar microstructure [Clarkson & Sayers (1999); Fridén & Lieber (2001)]. Damage to myofibrils is followed by an inflammatory response that is believed to be necessary for muscle regeneration [Clarkson & Sayers (1999); Pyne (1994); Stauber & Smith (1998)]. During this process, neutrophils and macrophages infiltrate damaged muscle fibers and degrade damaged proteins [Pyne (1994); Stauber & Smith (1998)]. However, it seems possible that this degradation process does not necessarily take place when damage is focal and not severe [Proske & Morgan (2001)]. Regeneration, in this scenario, would likely involve intracellular mechanisms. The capacity for muscle regeneration is evident even in cases where the entire myofiber is degraded. When regeneration occurs in this situation, satellite cells play an important role [Hawke & Garry (2001); Stauber & Smith (1998)]. Activation of satellite cells may be due in part to cytokines released by inflammatory cells as well as involvement of specific growth factors [Hawke & Garry (2001)]. Satellite cells, once activated, are committed to cellular regeneration by way of hypertrophic and hyperplastic responses [Hawke & Garry (2001)]. Eccentric muscle actions have been shown to be a potent indirect activator of satellite cells [Stauber & Smith (1998)].

It is important to note that the magnitude of muscle damage is dependent on the intensity of eccentric load. For example, eccentric exercise of the elbow flexors employing submaximal load (a dumbbell adjusted to 50% of the maximal isometric strength determined at an elbow joint angle of 90 degrees) induced less damage than maximal eccentric exercise in which maximal load was applied for the same range of motion during the dumbbell exercise [Nosaka & Newton (2002d)].

4. Markers of Muscle Damage and Muscle Soreness

Direct evidence of muscle damage is evident in histological changes in intracellular structure and the extracellular matrix [Armstrong et al. (1991); Warren et al. (1999)]. Intracellular damage is identified by an altered sarcomere structure that is characterized by a disturbance of the Z disk such as broadening, streaming, and total disruption [Armstrong et al. (1991); Fridén & Lieber (2001); Proske & Morgan (2001)]. Extracellular matrix expansion is notable in damaged muscle fibers, and has been found to contain fibronectin, collagens, proteoglycans, and glycoproteins [Stauber & Smith (1998)].

Among the many indirect markers of muscle damage, prolonged loss of force generating ability is considered to be the best indicator of muscle damage together with decreases in range of motion (ROM) [Clarkson et al. (1992); Warren et al. (1999)]. After 24 maximal eccentric actions of the elbow flexors in non-resistance trained subjects, maximal isometric strength decreased to approximately 50% of the pre-exercise level immediately following exercise, recovered to only 60% 5 days after exercise, and took more than a month for the complete recovery of strength [Clarkson et al. (1992); Nosaka & Clarkson (1996)] (**Fig.5**). ROM decreased approximately 20 degrees in the period immediately following to three days after exercise, and required more than two weeks for complete recovery [Clarkson et al. (1992); Nosaka & Clarkson (1996)]. Swelling of the exercised muscles, indicated by increases in upper arm circumference, or magnetic resonance (MR) or B-mode ultrasound images, is a typical occurrence with maximal changes being observed 4-6 days postexercise [Nosaka & Clarkson (1996)]. Increases in MR T2 relaxation time were conspicuous up to 4 weeks after exercise [Nosaka & Clarkson (1996)].

When muscle is damaged, there is disruption of the sarcolemma allowing muscle proteins such as creatine kinase (CK) and myoglobin (Mb) to be released from the cell into the blood stream via interstitium and lymph. It is a common finding that plasma CK activity and Mb concentration increase to abnormally high levels (e.g., CK: >10,000 IU/L, Mb: >1,000 IU/L) following eccentric exercise of the elbow flexors [Clarkson et al. (1992); Nosaka & Clarkson (1996)].

DOMS is also considered by many to be an indicator of muscle damage, as it appears following performance of unaccustomed exercise, especially that which includes an eccentric component [Armstrong et al. (1991); Clarkson et al. (1992); Proske & Morgan (2001); Pyne (1994); Stauber (1998)]. Sensations of

Fig. 5. Time course of changes in maximal isometric strength from preexercise level (0: 100%) to 60 days after 24 maximal eccentric actions for 10 different subjects (A-J). The mean value of the 10 subjects is also shown. Based on data of the study by Nosaka & Clarkson, 1996.

DOMS are subjective, however, they are still often used to diagnose the presence of muscle damage. It has been shown that when DOMS is evaluated by a visual analog scale (a 50-mm line, 0: no pain, 50: extremely painful) following palpation, flexion and stretching of the elbow flexors, poor correlations are observed with other indicators of muscle damage such as maximal isometric strength, ROM, upper arm circumference, and plasma CK activity [Nosaka et al. (2002)]. Therefore, it appears that DOMS is a poor indicator of eccentric exercise-induced muscle damage, and the magnitude of DOMS does not necessarily reflect the magnitude of muscle damage [Nosaka & Newton (2002d); Nosaka et al. (2002); Warren et al. (1999)]. During strength training, it may be wise to judge the magnitude of muscle damage by a measure of muscle function, and, to date, the best criteria remains changes in isometric muscle strength following exercise.

Although there are several indirect markers of muscle damage such as muscle strength, ROM, swelling, blood borne intramuscular muscle proteins, muscle soreness, MR, and ultrasound images, none of them match the time course of morphological changes (**Fig.6**). This may indicate that multiple factors are involved in determining the extent of morphological injury, and each marker may highlight different aspects of overall damage.

5. Eccentric Exercise-induced Muscle Damage and Adaptation

The influence of eccentric exercise on strength-trained subjects appears to be different from that on untrained subjects. Gibala et al. (2000) reported that 45% of the fibers were disrupted in the eccentric arm and 27% in the concentric arm, which was not different from the nonexercised arm for trained subjects.

Fig. 6. Magnitude of change in several markers of muscle damage (muscle soreness, swelling, strength, range of motion, MRI and ultrasound images, and morphological changes in muscle fibers) following 24 maximal eccentric actions of the elbow flexors performed by untrained subjects. When a maximal change appears, it is considered as 100% for measures showing increases, or -100% for measures showing decreases. Based on data of several studies [Clarkson et al., (1992); Nosaka & Clarkson, (1996); Nosaka & Sakamoto, (2001); Pyne, (1994); Warren et al., (1999)].

Numerous studies have reported that a single bout of eccentric exercise affords protection against muscle damage following subsequent bouts of similar exercise, and this adaptation is usually referred to as the repeated bout effect [Clarkson et al (1992); Clarkson & Sayers (1999); McHugh et al. (1999)]. The mechanism involved with this adaptation is poorly understood; however, it has been proposed that neural, connective tissue, cellular adaptations, or a combination of these are associated with it [Clarkson & Sayers (1999); McHugh et al. (1999)]. The repeated bout effect has been shown to last at least several weeks, and up to six to nine months in the case of maximal eccentric exercise of the elbow flexors [Clarkson et al. (1992); Nosaka et al. (2001a)]. It has been reported that the initial bout of eccentric exercise does not have to cause appreciable damage to confer the repeated bout effect; however, the intensity of the initial eccentric exercise must be close to maximal when subsequent bouts involve high intensity eccentric actions [Nosaka & Newton (2002a)].

This adaptation is of importance when considering muscle damage sustained during resistance training. We tend to think that muscle damage is always induced with resistance training; however, it seems unlikely that the same magnitude of muscle damage as the initial bout of training is induced as training progresses. During eccentric training (3 sets of 10 reps) of the elbow flexors using a load approximating 10RM, the largest decreases in maximal isometric strength and increases in plasma CK activity were seen after the

Fig. 7. Changes in maximal isometric strength (upper graph) and plasma CK activity (lower graph) over 8 weeks of eccentric training of the elbow flexors (3 sets of 10 reps at 80% of maximal isometric strength). The training was performed once per week, and the changes before (pre), immediately after (0), and 4 days (4) after each training session are shown. Based on data of a study by Nosaka & Newton, 2002a.

first training bout [Nosaka & Newton (2002a)]. No significant increases in plasma CK activity or additional decreases in strength and ROM were seen following subsequent training bouts (2nd -8th) performed once per week for eight weeks [Nosaka & Newton (2002a)] (**Fig.7**). This data has relevance for resistance-trained individuals who have been training for a period of time and have become accustomed to eccentric loading. These individuals do not seem to sustain severe muscle damage even following a large volume of high-intensity eccentric exercise. A recent study from our laboratory showed that decreases in strength and ROM and increases in plasma CK activity were significantly diminished for trained subjects compared to untrained subjects (unpublished data). Interestingly, both groups reported a similar magnitude of DOMS following exercise.

It is also of interest to note that additional eccentric exercise performed in the early recovery phase does not exacerbate muscle damage and has minimal effect on the recovery process [Nosaka & Newton (2002b), (2002c)]. Another of our recent studies showed that a subsequent bout of eccentric exercise with concurrent electrical stimulation, which induced larger decreases in force (22% of pre) than the initial bout (45% of pre) did not exacerbate muscle damage; however, a slightly retarded recovery of muscle function was observed [Nosaka & Newton (2002b)].

Fig. 8. Comparison between eccentric (ECC), concentric (CON), and mix (MIX) training for changes in muscle fiber area (Type I, Type IIa, and Type IIx) before (pre), immediately after 3 weeks of immobilization (post-Immo), and after 12 weeks of exercise training (post-Tr.). *: significant ($P < 0.05$) difference between ECC training and CON training. Based on the data of the study by Hortobágyi et al., 2000.

6. Effects of Eccentric Exercise in Resistance Training

It has been demonstrated that eccentric training results in greater gains in muscle strength and size than concentric training. Hortobágyi et al. (1996) reported that 12 weeks of isokinetic eccentric training of the knee extensors increased type IIa fiber area 10 times that of the same amount of isokinetic concentric training. Hortobágyi et al. (2000), in a subsequent study, compared the effects of 12 weeks of eccentric, concentric, and mixed (eccentric and concentric) isokinetic training on strength, muscle fiber size, and myofibrillar gene expression after 3 weeks of knee immobilization. Eccentric and mixed training resulted in an expedited and greater recovery of strength as well as greater increases in Type IIa and IIx fibers, and eccentric training was even more effective for increases in Type IIa fiber area than mixed training (Fig.8). Higbie et al. (1996) also showed that quadriceps cross-sectional area increased to a greater extent when training was eccentric (6.6%) rather concentric (5.0%). It is noteworthy that the concentric training in these studies increased muscle size significantly, although the magnitude of the effect was lower than when eccentric training was employed.

Mitogen-activated protein kinases (MAPKs) are known to be involved in the intracellular signaling that is associated with increases in protein synthesis induced by mechanical stress. Martineau & Gardiner (2001) demonstrated that eccentric muscle actions increased MAPK activation shown by p54 c-Jun NH2-terminal kinase (JNK) and p44 extracellular regulated

Fig. 9. Effect of contraction type (passive static stretch, passive cyclical stretch, concentric, isometric, and eccentric contractions) on p54 c-Jun NH2-terminal kinase (JNK) and p44 extracellular regulated kinase (ERK) phosphorylation compared to control (1). Based on data of the study by Martineau & Gardiner, 2001.

kinase (ERK) phosphorylation to a greater extent than concentric or isometric actions. Eccentric muscle actions appear to be effective for increasing the number of myofibrils and muscle hypertrophy, and this may be partly explained by the activation of MAPK due to the increased tension on a fewer number of myofibers during this type of contraction [Martineau & Gardiner (2001)] (Fig.9). Although eccentric muscle actions seem to be effective for inducing muscle hypertrophy, hyperplasia and increases in connective tissue, it seems likely that muscle damage is necessary only for the latter two of these adaptations (Fig.10). It appears that hyperplasia and increases in connective tissue are produced mainly by eccentric exercise that induces muscle damage. All training modes (concentric, isometric, and eccentric) seem capable of producing muscle hypertrophy. However, the magnitude of the training effect seems to be larger for eccentric training, possibly due to the mechanical stress of eccentric training generally being larger than that of concentric and isometric training. It is important to note that eccentric training does not necessarily induce muscle damage, and muscle is capable of hypertrophy in the absence of muscle damage.

7. Conclusion

Strength training appears to induce muscle damage, especially in the initial phase of training when unaccustomed eccentric actions are performed. Muscle damage is best indicated by loss of muscle function, especially that reflected by decrements in maximal isometric strength. Although muscle soreness is common following training, it does not necessarily reflect the

Fig. 10. Hypothetical effect of concentric, isometric, and eccentric training on increases in muscle cross-sectional area (CSA). Increases in CSA are due to hypertrophy, hyperplasia (increases in muscle fiber number), increase in connective tissue (CT), or a combination of these factors. (Modified from figures drawn by Ishii 1994.)

magnitude of muscle damage. Eccentric exercise should be included in resistance training because it seems to have the potential to enlarge muscle size and to increase not only eccentric strength but also concentric and isometric strength. Since muscle damage and muscle soreness are induced to a greater extent with eccentric muscle actions than with either isometric or concentric actions, it is commonly believed that muscle damage is necessary for size and strength gain. However, it seems unlikely that muscle damage and/or muscle soreness are essential for muscle adaptation. Further research is necessary to delineate how much muscle damage is necessary for muscle adaptation in strength training.

References:

- Armstrong, R.B., Warren, G.L., & Warren, J.A. (1991). Mechanisms of exercise induced muscle fibre injury. *Sports Medicine* 12, 184-207.
- Clarkson, P.M., Nosaka, K., & Braun, B. (1992). Muscle function after exercise induced muscle damage and rapid adaptation. *Medicine and Science in Sports and Exercise* 24, 512-520.
- Clarkson, P. M., & Sayers, S. P. (1999). Etiology of exercise-induced muscle damage. *Canadian Journal of Applied Physiology* 24, 234-248.
- Fridén, J., & Lieber, R. L. (2001). Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiologica Scandinavica* 171, 321-326.
- Gibala, M.J., Interisano, S.A., Tarnopolsky, M.A., Roy, B.D., MacDonald, J.R., Yarasheski, K.E., & MacDougall, J.D. (2000). Myofibrillar disruption following concentric and eccentric resistance exercise in strength trained men. *Canadian Journal of Physiology and Pharmacology* 78, 656-661.
- Gibala, M.J., MacDougall, J.D., Stauber, W.T., & Elorriaga, A. (1995). Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. *Journal of Applied Physiology* 78, 702-708.
- Hawke, T.J., & Garry, D.J. (2001). Myogenic satellite cells: physiology to molecular biology. *Journal of Applied Physiology* 91, 534-551.
- Higbie, E.J., Cureton, K. J., Warren III, G. L., & Prior, B.M. (1996). Effects of concentric and eccentric training on muscle strength, cross-sectional area, and neural activation. *Journal of Applied Physiology* 81, 2173-2181.
- Hortobágyi, T., J. Barrier, D. Beard, Braspeninx, J., Koens, P., DeVita, P., Dempsey, L., & Lambert, J. (1996). Greater initial adaptation to submaximal muscle lengthening than maximal shortening. *Journal of Applied Physiology* 81, 1677-1682.
- Hortobágyi, T., Dempsey, L., Fraser, D., Zheng, D., Hamilton, G., Lambert, J., & Dohm, L. (2000). Changes in muscle strength, muscle fibre size and myofibrillar gene expression after immobilization and retraining in humans. *Journal of Physiology* 524, 293-304.
- Ishii, N. (1994). Resistance training and muscle hypertrophy. In *Training Science Association (Eds.) Resistance Training*. (pp.19-31). Tokyo; Asakura Publishing Co. Ltd. (in Japanese)
- Martineau, L.C., & Gardiner, P.F. (2001). Insight into skeletal muscle mechanotransduction : MAPK activation is quantitatively related to tension. *Journal of Applied Physiology* 91, 693-702.
- McCully, K.K., & Faulkner, J.A. (1985). Injury to skeletal muscle fibers of mice following lengthening contractions. *Journal of Applied Physiology* 59:119-126.
- McHugh, M. P., Connolly, D.A.J., Eston, R.G., & Gleim, G.W. (1999). Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. *Sports Medicine* 27, 157-170.
- Nosaka, K., & Clarkson, P.M. (1996). Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Medicine and Science in Sports and Exercise* 28, 953-961.
- Nosaka, K., & Newton, M. (2002a). Concentric or eccentric training on eccentric exercise-induced muscle damage. *Medicine and Science in Sports and Exercise* 34, 63-69.
- Nosaka, K., & Newton, M. (2002b). Is recovery from muscle damage retarded by a subsequent bout of eccentric exercise inducing larger decreases in force? *Journal of Science and Medicine in Sport* 5, 204-216.
- Nosaka, K., & Newton, M. (2002c). Repeated eccentric exercise bouts do not exacerbate muscle damage and repair. *Journal of Strength and Conditioning Research* 16, 117-122.
- Nosaka, K., & Newton, M. (2002d). Differences in the magnitude of muscle damage between maximal and submaximal eccentric loading. *Journal of Strength and Conditioning Research* 16, 202-208.
- Nosaka, K., Newton, M., & Sacco, P. (2001a). How long does the protective effect on eccentric exercise-induced muscle damage last? *Medicine and Science in Sports and Exercise* 33, 1490-1495.
- Nosaka, K., Newton, M., & Sacco, P. (2002). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports* 12, 1-10.
- Nosaka, K. & Sakamoto, K. (2001). Effect of elbow joint angle on the magnitude of muscle damage to the elbow flexors. *Medicine and Science in Sports and Exercise* 33, 22-29.
- Nosaka, K., Sakamoto, K., Newton, M., & Sacco, P. (2001b). The repeated bout effect of reduced-load eccentric exercise on muscle damage of the elbow flexors. *European Journal of Applied Physiology* 85, 34-40.
- Proske, U., & Morgan, D.L. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, and adaptation and clinical applications. *Journal of Physiology* 537, 333-345.
- Pyne, D.B. (1994). Exercise-induced muscle damage and inflammation: a review. *Australian Journal of Science and Medicine in Sport* 26, 49-58.
- Safran, M.R., Seaber, A.V., & Garrett Jr, W.E. (1989). Warm-up and muscular injury prevention. *Sports Medicine* 8, 239-249.
- Stauber, W.T., & Smith, C.A. (1998). Cellular responses in exertion-induced skeletal muscle injury. *Molecular and Cellular Biochemistry* 179, 189-196.
- Warren, G.L., Ingalls, C.P., Lowe, D.A., & Armstrong, R.B. (2002). What mechanisms contribute to the strength loss that occurs during and in the recovery from skeletal muscle injury? *Journal of Orthopaedic & Physical Therapy* 32, 58-64.
- Warren, G.L., Lowe, D.A., & Armstrong, R.B. (1999). Measurement tools used in the study of eccentric contraction-induced injury. *Sports Medicine* 27, 43-59.

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- "Effect of elbow joint angle on the magnitude of muscle damage to the elbow flexors." *Medicine and Science in Sports and Exercise* 33:22-29, 2001
- "The repeated bout effect of reduces-load eccentric exercise on muscle damage of the elbow flexors." *European Journal of Applied Physiology* 85:34-40, 2001.

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