Eccentric Muscle Contractions: Their Contribution to Injury, Prevention, Rehabilitation, and Sport

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Muscles operate eccentrically to either dissipate energy for decelerating the body or to store elastic recoil energy in preparation for a shortening (concentric) contraction. The muscle forces produced during this lengthening behavior can be extremely high, despite the requisite low energetic cost. Traditionally, these high-force eccentric contractions have been associated with a muscle damage response. This clinical commentary explores the ability of the muscle-tendon system to adapt to progressively increasing eccentric muscle forces and the resultant structural and functional outcomes. Damage to the muscle-tendon is not an obligatory response. Rather, the muscle can hypertrophy and a change in the spring characteristics of muscle can enhance power; the tendon also adapts so as to tolerate higher tensions. Both basic and clinical findings are discussed. Specifically, we explore the nature of the structural changes and how these adaptations may help prevent musculoskeletal injury, improve sport performance, and overcome musculoskeletal impairments. *J Orthop Sports Phys Ther 2003;33:557-571.*

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The greatest magnitude forces in muscle occur when an external force exceeds that produced by the muscle and the muscle lengthens, producing an eccentric contraction and negative work.[95,115] (Because work is force × displacement, it is a product of 2 vectors. When distance is in the opposite direction of the force generated, work is “negative.”) Because the muscle's force can be maximized when contracting eccentrically, damage to the contractile and cytoskeletal components of the muscle fiber itself,[64,65] weakness,[61] and a perception of soreness[12,13,43,135] often occur. It is curious that muscle, structured to absorb and perform mechanical work during eccentric lengthening, sustains muscle damage while performing a task it appears ideally suited to accomplish. However, muscle damage need not be an obligatory response following high-force eccentric contractions. In fact, the ability to produce high forces with eccentric contractions should perhaps more properly be perceived as a protective muscle adaptation and a stimulus for beneficial muscle (and tendon) responses, rather than as a common cause of damage.[21,64,115,173,177] Many have called for the use of chronic eccentric exercise in the preventative care or rehabilitation of patients.[49,86,110,111,115,116,140,146]

In this commentary we explore how muscles adapt both structurally and functionally to chronic high-force eccentric lengthening contractions and how this adaptation may help (1) to prevent musculoskeletal injury, (2) to improve sport performance, and (3) to overcome musculoskeletal impairments.

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ECCENTRIC CONTRACTIONS: MUSCLES OPERATING AS SHOCKS OR SPRINGS

Muscles act like shock-absorbing structures and springs when they absorb mechanical work while eccentrically lengthening. The forces resulting from these eccentric muscle contractions produce negative work.[112,115,116] Locomotor muscles function as shock absorbers during the descent of inclines or when decelerating the body segment (eg, going from stand to sit) and are ubiquitous in many other normal movements such as walking, jogging, maneuvering around obstacles, or regaining balance.[50,53,74,82,115] In fact, during normal locomotion, muscles are collectively doing near equal amounts of positive (shortening) and negative (lengthening) work.[74] While the energy that is absorbed during the muscle and tendon stretch is often dissipated as heat, elastic strain energy can also be stored and recovered if an immediate shortening concentric contraction follows.[4,12,25,51,59] When muscles are activated eccentrically immediately prior to shortening, they no longer act as shock absorbers; rather, they perform more like springs[115,116] (Figure 1).

During a stretch-shorten contraction (SSC), muscles are actively lengthened prior to a subsequent shortening phase.[34,99,101] The stretched components of the muscle-tendon unit store elastic recoil potential energy (or elastic strain energy), a portion of which may be subsequently recovered.[12,19,51,55,81,99,115,116] The storage and recovery of elastic strain energy during a SSC is an important determinant of performance, as the energy stored during a lengthening cycle can substantially amplify force and power production in the subsequent

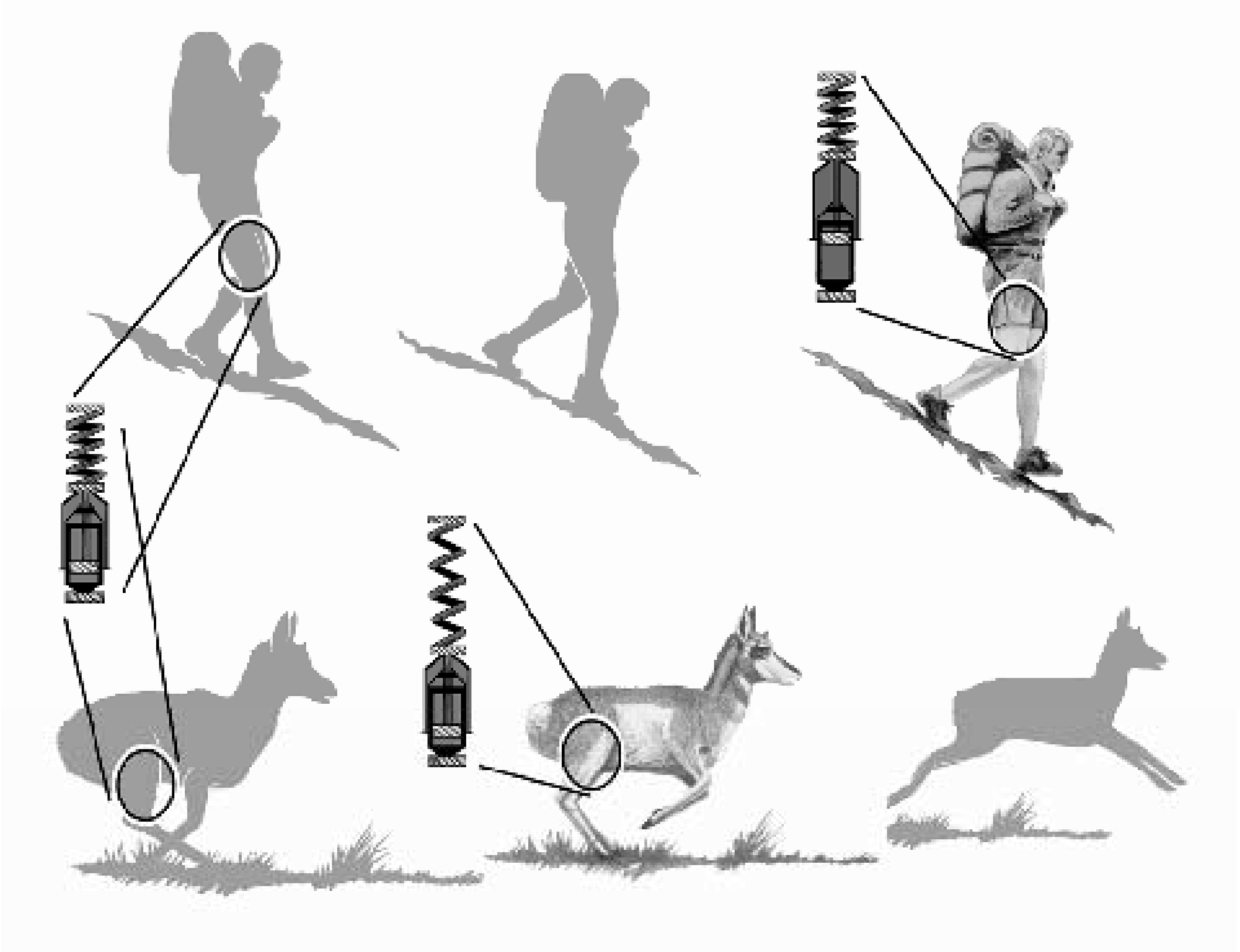

FIGURE 1. When an active muscle is lengthened during an eccentric contraction, it behaves, in the simplest sense, like a shock absorber in series with a spring. In hiking downhill (top panel), when the velocity of lengthening is relatively slow, the energy that stretches the active muscle is lost as heat. In this example, the knee extensors behave like a shock absorber as the knee moves from extension to flexion (the piston moves from the bottom of the cylinder to the top). In contrast, when mammals (including humans) run (lower panel), the knee and hip extensors are rapidly stretched; the absorbed energy is not immediately lost as heat, but is temporarily stored as elastic recoil potential (strain) energy. In this example, these muscles behave like a spring that can store (the elongated spring representing the hip and knee extensors when strained) and recover energy from stride to stride. The time course of stretch and recovery of elastic recoil energy is dependent on both the magnitude of the forces involved as well as the elasticity (spring stiffness) of the muscle. Reprinted with permission from Lindstedt et al.[115]

shortening cycle.[19,54,99,138,145,153] Some studies,[22,23] however, report that the restitution of elastic strain energy does not provide the increased power output, rather, an increased activation of the muscle enhances shortening work. In all likelihood, the increased power of shortening is a combination of both.[173] The ability to recover elastic strain energy is apparently energetically so advantageous that the most economical stride frequency in running may be set by this key property alone.[59,165] Apart from the role of tendons and collagen in energy storage, the muscle itself stores and recovers elastic strain energy, as elastic strain energy can occur in the absence of tendons.[32] In a sense, because the muscle is composed of both muscle fibers and tendinous materials, all of these structures must be collectively ''tuned'' to the spring properties for the muscle-tendon system to store and recover elastic strain energy during locomotion.[115]

ECCENTRICS IN MUSCLE INJURY: POSSIBLE PREVENTATIVE MECHANISMS

Muscle is a highly mutable tissue in that both its structure and function adapt to the demands placed on it.[24,142] Like all biological tissues, modifications to the relative level of physical stress to muscle produce predictable results.[130] One classic example of muscle responding to a high physical stress dosage with either an injury or a beneficial adaptation is high-force eccentric activity. For example, if naïve to hiking downhill (eccentric lengthening contractions), one can experience devastating delayed onset muscle soreness (DOMS) after an initial hike. There is clear evidence linking DOMS with muscle damage and inflammation,[90,122,134] suggesting that the muscle cell itself has been injured. Likewise, structural damage to the contractile[166] and cytoskeletal elements of the muscle fiber,[64,65] as well as impairment of the excitation-contraction coupling process,[175] are coupled to reduced force-producing abilities.[61] If one hikes downhill repeatedly, however, after relatively few hikes there is no soreness or muscle damage whatsoever, even with the same intensity exercise stimulus. Hence, the chronic use of eccentric contractions, in this case downhill hiking, results in a pronounced protective adaptation within the muscle termed the ''repeated-bout effect.''[42,134,136,156] Consequently, an eccentric activity that would cause serious, even debilitating, damage without adaptation no longer has any harmful effects if previously exposed.

The changes within the muscle responsible for this acute adaptation are largely unknown. There are, however, suggestions that muscles adapt acutely to eccentric exercise by changing optimal length (ie, becoming more compliant via the addition of sarcomeres in series, allowing muscle fibers to operate at longer lengths while avoiding the descending limb of the length-tension curve)[28] or that groups of the more fragile, stress-susceptible fibers are reduced in number after the first bout while stronger fibers survive and provide a protective effect.[11,64] It has also been suggested that the acute effect lies outside the muscle and is mediated at a motor unit level (recruitment of surviving motor units) or at the neuromuscular junction.[42,176] One key finding is that even light eccentric training protocols that result in little to no muscle damage are sufficient to bring about this protection.[43] While the exact nature of the *acute* adaptation remains unclear, *chronic* exposure to eccentric muscle activity results in an active spring structure(s) adaptation (ie, the muscle stiffens) that occurs independent of the increases in both the muscle size and strength.[115,116,147]

To investigate the impact of chronic high-force eccentric training on muscle-tendon stiffness (ie, spring properties), we used an eccentric resistance exercise regimen that was progressively and gradually ramped up over 3 weeks to nearly 500 negative watts.[110,116] After 8 weeks, we noted the expected muscle size and strength increases. Additionally, we also explored how this training impacted the apparent muscle spring stiffness. We had subjects jump in a hopping motion (jump height was set to 107% of subject height) in place at a frequency that was reported by the subject to be the most ''comfortable.'' Farley et al[58] determined that at this preferred hopping frequency in humans (2.2 hops per second), the body behaved like a simple spring mass system, with deviations from this frequency reducing the storage and recovery of elastic energy. We have also noted that this comfortable frequency is the most economical, as the cost per jump doubles when the subjects are forced to jump at half this frequency.[115] Following eccentric training, every one of the subjects selected a higher hopping frequency than they did prior to training; the 12% mean overall increase was significant, as none of the control subjects (those exercising on a traditional concentric bike) changed their jumping frequency (Figure 2).

Reich et al[148] used a model of rats walking down a steep (36%) decline with an additional load of 15% of body weight, to eccentrically load rat locomotor muscles, to determine if this apparent increased stiffness resulted from changes in the muscle's contractile properties, eg, stiffness. (All tissues within the musculo-skeletal system exhibit stiffness. Young's modulus is the measure of the stiffness of a material [E = stress/strain]. Stress is calculated by force/area and strain by extension/original length. Reich[148] calculated both the Young's modulus of the muscle, which increased greater than 30% with eccentric training, and active-lengthening [eccentric] force production. The latter is reported here as active muscle stiffness, the force produced with a ramped stretch of 1.5% of the resting muscle length on a muscle 100

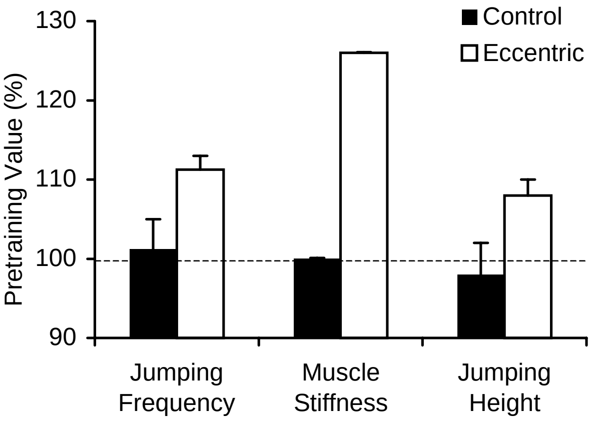

FIGURE 2. Chronic eccentric training results in shifts in muscle spring properties. Following 8 weeks of chronic eccentric training, the natural hopping frequency in humans (the frequency self-selected as being most comfortable) is increased over 12%.[116] Control subjects did an equal energetic intensity of concentric work during their training. Our work[148] with rats suggests that this shift in frequency may be due to a stiffer muscle spring. Following eccentric training, the triceps muscles of the rat produce 27% more force in response to an activated stretch. Finally, the height of a maximum vertical jump also increases following 6 weeks of eccentric training in humans. For this experiment, the active controls were engaged in weight lifting and plyometric exercises of the lower extremities.[116] Error bars = 1 SEM.

ms into tetanus.) After 8 weeks of training (30 minutes, 5 times per week), the triceps muscles of the eccentrically trained animals were significantly stiffer than those of the inactive controls.[147] The muscle had indeed demonstrated a mechanical adaptation by becoming approximately 26% stiffer when subjected to chronic eccentric use (Figure 2). These in vitro measurements of active muscle stiffness excluded the peripheral nerve and any tendinous attachments; hence, only muscle stiffness was recorded. Thus, we conclude that the apparent increases in muscle stiffness in the human subjects were also likely attributable to shifts in muscle stiffness independent of any shifts in other elastic elements. These results would seem to confirm those of others[103,120,144] in demonstrating that muscle stiffness changes in response to chronic eccentric muscle use.

WHAT IS THE SPRING STRUCTURE IN MUSCLE AND IS IT ADAPTABLE?

The elastic property of vertebrate myofibrils is thought to be due in large part to the enormous cytoskeletal protein (2.5–3.7 MDA) filament titin,[126,174] which spans an entire half-sarcomere from Z-disc to M-line. Titin functions as serially linked springs that develop tension when stretched.[117-119] There are multiple titin isoforms that vary in size and stiffness, which explains the elastic-stiffness diversity across vertebrate muscle.[36] Titin has multiple roles in striated muscle, ranging from sarcomere assembly to mechanical roles such as providing the forces needed to maintain proper sarcomere integrity during contractions[78,83] (for reviews see Granzier and Labeit[69] and Gregorio et al[71]). In addition, the differential expression of titin isoforms is thought to play a dynamic role in active force production.[37,163] Because of titin's structural properties, its most significant role may be as the muscle spring. First, as a muscle-stiffening spring, it may play a key role in the protective effect that occurs following eccentric exercise.[148] Supporting this idea is the fact that novel high-force eccentric contractions damage the cytoskeleton,[64] by including titin failure, and a bout of eccentric exercise results in diminished titin content.[168] As well, recent evidence suggests that small heat shock proteins that protect the cytoskeleton structures (eg, titin) increase dramatically after repeated eccentric bouts.[98]

If titin is functioning as a locomotor spring, then it should be tuned to the frequency of muscle use. We tested this hypothesis by examining titin isoform expression in muscles that are used cyclically at different frequencies. Because stride frequency varies predictably with body size among mammals, by examining the titin expressed in different-sized animals, we predicted shifts in titin isoform expression as a function of body size. Titin expression analyzed with SDS-PAGE in animals ranging in size from a shrew to an elephant[116,147] shows a predictable shift from the most compliant (largest) isoforms in the elephant to the stiffest (and smallest) isoforms in the shrew. These results suggest a strong link between stride frequency and titin "stiffness." While these results do not prove that titin is the muscle spring, they suggest that titin may be a significant and potentially tuned contributor to the muscle-tendon spring. If titin is functioning as a locomotor spring, then titin should adapt in response to changes in physiological demand due to exercise or disease. This notion has been recently reinforced as titin isoform expression has been reported as an adaptable property of striated muscle.[17]

ECCENTRIC CONTRACTIONS: STRUCTURAL AND FUNCTIONAL CHANGES TO LOCOMOTOR MUSCLE THAT ENHANCE SPORT ACTIVITIES

One of the primary goals for strength and conditioning coaches is to enhance the muscular force production of an athlete, as power output (force × velocity) often defines success in sport.[171] The pervasive role of eccentric muscular force enhancement prior to a power activity (eg, during a SSC) may be the most substantial during high-power sport activities such as running,[31,35] sprinting,[40,60,129] hopping,[40,115] and jumping.[22,115,153] The importance of the SSC in almost all sport activities (possible exceptions being bicycling and swimming) cannot be overstated. For example, during a baseball pitch or a high jump, a series of eccentric contractions in both the lower and upper extremities precedes concentric contractions. During the windup, cocking, and late cocking phases of a throwing motion, the trunk and lower extremities, coupled with the internal rotators of the shoulder, store elastic strain energy via eccentric lengthening prior to transitioning to the accelerating concentric shortening phase (Figure 3). Because SSC activities are ubiquitous in sport, plyometric exercises are popular training paradigms that have improved sport-related activities: upper-body ball put,[172] vertical jump,[127,131,169,178] and throwing.[75] Perhaps, if the magnitude of the force (as well as the elastic strain energy) during the eccentric phase can be maximized via training, power can also be maximized during the concentric phase.

We tested this hypothesis with a group of basketball players, examining the effects of high-force eccentric training on muscle power output.[116] After 6 weeks of high-force eccentric training in 1 subject group as compared to a weight-training ''active'' control group, there was a significant increase in vertical jump. While both groups had identical initial vertical jump heights at the start of the study, every one of the subjects in the eccentric-training group increased the vertical jump, with an overall mean increase of approximately 8% (5 cm) (Figure 2). Thus, high-force eccentric training can evoke gains in muscle power and size, possibly resulting in part from significant increases in the muscle spring stiffness.[115,116,144]

Muscle strength and power improvements seem to be a function of the muscle's ability to produce high forces.[115,116] Therefore, because much greater force (2 to 3 times greater) can be produced eccentrically than either isometrically or concentrically,[92,93] eccentric training has the capability of ''overloading'' the muscle to a greater extent and enhancing muscle mass, strength, and power,[27,84,87,88,92,100,109-111,115,116,172] when compared to concentric exercise (Figure 4).

ECCENTRIC EXERCISE IN PREVENTING MUSCULOSKELETAL INJURY AND IMPAIRMENT

Sarcopenia

The progressive loss of muscle mass with aging, sarcopenia, is a significant public health problem.[15,56,182] Decreases in muscle mass begin to occur as early as 25 years of age and progress to the point where by the age of 80 years, one half of the skeletal muscle has been lost.[114] Both cross-sectional and longitudinal data have established that muscle strength also declines by approximately 15% per decade in the sixth and seventh decades and by about 30% per decade thereafter.[57]

FIGURE 3. The sequential activated prestretching of muscle-tendon structures prior to the high-power acceleration phase of the throwing motion. (A) During the early cocking phase of the baseball pitch, the right leg is positioned to prestretch the knee extensors, hip extensors, and hip rotators (hip musculature not depicted) prior to ballistic shortening of knee and hip muscle-tendon structures. With muscle-tendon lengthening, high eccentric muscle forces (potential energy) can be converted into high muscle power outputs during the concentric-shortening phase. (B) Still in early cocking, the knee and hip extends, the torso begins to rotate to lengthen the abdominal muscles and the serratus anterior of the right scapula. (C) During the late cocking phase, a prestretch of the pectoralis, serratus anterior, and subscapularis is in preparation for the acceleration phase. Iliopsoas is also prestretched to transfer stability to the spine.

Resistance weight training for the elderly can counteract sarcopenia as strength, power, and muscle mass increases are possible.[39,62,63,89,94] The unique aspect of eccentric resistance training is that much greater forces, hence greater overload to the muscle, are possible as compared to traditional resistance weight training. In addition to the production of much higher forces, eccentric contractions have another unique attribute: the metabolic cost is greatly reduced.[1,2,20] This high-force, low-cost suite of attributes makes it ideal for energetically impaired patient populations. Thus, with high-force eccentric training, significant increases in muscle mass[91,109,110,115] and strength[73,84,88,92,100,109-111] have been reported. In elderly subjects (mean age, 78 years) suffering the consequences of sarcopenia, we have reported a large increases in isometric leg strength and significant increases in whole muscle mass (6%) and vastus lateralis muscle fiber cross-sectional area (60%) following 11 weeks of high-force eccentric ergometry[109] (Figure 4).

Muscle-Tendon Injuries

When forces within a muscle are used to decelerate a limb or body segment, the entire muscle-tendon system participates in dissipating, or temporarily storing, the energy. If the forces needed for deceleration exceed that of the muscle-tendon system, injury to the muscle, myotendinous unit, the tendon itself, and the osteotendinous insertion may occur.

For example, muscle strain injuries, especially to the hamstring musculature, are quite common in sports requiring explosive running such as football, track and field, and soccer.[67,184] Strain injuries to the adductor group, an especially common injury in hockey players,[155] occurs in injured weakened muscle following ballistic high-force eccentric contractions.[104,133] Athletes with a history of recurring hamstring and adductor muscle injuries have greater impairment of their eccentric strength (2-fold) as compared to concentric strength, suggesting that improvements in the former may minimize the risk of injury.[47,72] Others have suggested that eccentric resistance exercise may prevent injury to the muscle-tendon unit by improving the muscle's ability to absorb more energy before failing.[9,72,143,162] Increased stiffness in tendons,[29] greater force at failure, and an improved ability to absorb energy at the musculotendinous junction result following eccentric resistance training.[66,132] The exact mechanism of this adaptation is not defined. It is apparent, however, that if the tissue failure force threshold increases and the attenuation of loads is enhanced, a protective effect can occur. While we[115,116,148] propose that an increase in muscle spring stiffness might prevent a strain injury, others[28,146] postulate the opposite (ie, that an increased compliance of sarcomeres in series might mitigate muscle strain injuries). The adaptation of the muscle-tendon system to eccentric physical activity is also associated with changes to the myotendinous junction: increased size (hypertrophy), fibroblastic activity, and production of collagen and ground substance.[21,120,180,183]

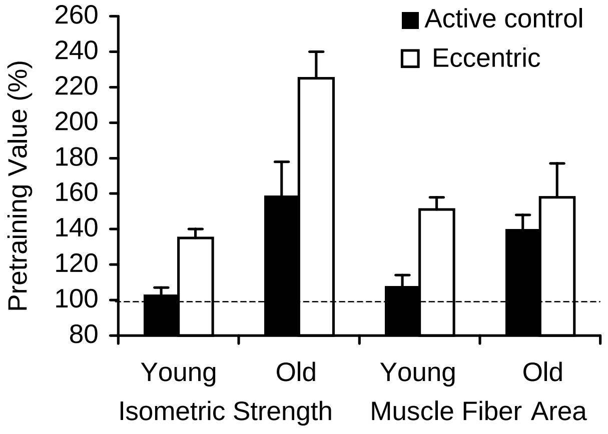

FIGURE 4. The high forces produced during eccentric contractions induced increases in isometric strength and muscle fiber (composites of type I and II fibers) area in both the young and old ($P<.05$).[109-111] Following 8 to 11 weeks of chronic (2-3 times per week) negative-work resistance training, strength increased by approximately 40% (young) and 120% (old) while the vastus lateralis muscle fiber area increased by approximately 50% to 60%. The active controls, who performed either isolated positive-work or traditional weight-lifting resistance exercises, either did not increase strength (young) or the magnitude of the increase was less (old) than the eccentrically trained. Error bars = 1 SEM.

Osteopenia

The magnitude of the increase in bone mass, like that in muscle mass and strength, seems to be a function of the magnitude of muscle forces and other loads to bone.[73,170] Specifically, the strength and density of bone is likely influenced by local strain of bone, which can occur with muscle exerting high forces on bone during resistance exercise.[38,106] Therefore, it is not resistance training per se, but the high forces and intensities possible with resistance training that promote increases in hip bone mineral density.[26,73,170] The suggestion that eccentric training, because it produces the highest muscle forces on bone during resistance exercise, should also result in the greatest bone adaptation is alluring.

Following 18 weeks of maximal effort, eccentric exercise on 1 leg and concentric exercise on the other leg (using a leg dynamometer), twelve 20- to 23-year-old women significantly increased midfemur bone mineral density by 3.9% following the eccentric training (a nonsignificant increase of 1.1% was noted in the concentrically trained leg).[73] This finding suggests that eccentric resistance to leg muscles provides a greater osteogenic stimulus than concentric resistance. Further, it suggests that the greater eccentric peak forces elicited from the eccentric group were the stimulus for this bone response, as the total resistance work over the 18 weeks was equivalent to that of the concentric training group. While the preliminary results are promising, they are in no way conclusive. Further investigation into the osteogenic potential of high-force eccentric exercise is warranted.

Fall Risk in the Elderly

Falls are the leading cause of accidental deaths among the elderly and many of these falls occur on stairs, where accidents during stair descent outnumber those of stair ascent by more than 3 to 1.[159] Eccentric muscle contractions, in contrast to concentric contractions, are relied upon almost exclusively to successfully descend stairs.[8,115,116,128] The ability to perform graded eccentric contractions in descending stairs is compromised much more in the elderly than is the ability to perform shortening contractions,[16,53,89] despite the fact that the absolute eccentric force-producing abilities are preserved in the aged.[90] This reduced ability of older adults to exert controlled steady forces during submaximal eccentric contractions has been suggested as the key factor contributing to the much greater frequency of falls during stair descent as compared with stair ascent.[33,53] In our previous work,[109] chronic exposure (11 weeks) to high-force eccentric leg ergometry in high fall-risk elderly individuals improved stair descent performance (20%) and balance (7%) and significantly decreased the risk of falling (Figure 5).

ECCENTRIC CONTRACTIONS IN THE MANAGEMENT OF MUSCULOSKELETAL IMPAIRMENTS

Tendinoses

Chronic tendon disorders often result from intensive repetitive activities, which are predominantly eccentric in nature. Due to higher-than-normal eccentric muscle forces transmitted via the tendon, the ability of the tendon to repair itself becomes impaired and the tendon deteriorates. This degenerative process, known commonly as tendinosis, at the Achilles, rotator cuff, lateral and medial elbow, posterior tibial, digital flexor, and patellar tendons, is associated with an abnormal angiofibroblastic healing response.[14,44,46,96,97,124,149]

Ironically, some have suggested that the very type of muscle activity (eccentric) that was in part responsible for the tendinosis should be emphasized in rehabilitation.[5-7,30,47,48,52,66,91,125,152,157,158,164] Ample evidence supports the notion that the tendon, like the muscle, can adapt favorably to physical stress, including that of high eccentric loads.[96,162,167,180,183] Specifically, tendons become stronger as fibroblast (tenoblast) activity increases and an appropriate collagen reaction accelerates. Macroscopic changes include a hypertrophied tendon, while microscopic adaptations are characterized by a thickening of the collagen fibers and fibrils and an increase in tropocollagen cross-links. The tendon fibers then align themselves optimally to manage the high stress levels transmitted from the muscle to the tendon.

The primary impairments associated with tendinosis are pain and weakness, especially in the eccentric-strength component, which can take up to a year to resolve.[6,7] Hence, there is both anecdotal and experimental evidence that eccentric-resistance exercises are beneficial in the rehabilitation of tendinoses.[6,7,157,158] Patients with the diagnosis of chronic (18 months) Achilles tendinosis, who were managed unsuccessfully with a traditional physical therapy regimen, have responded favorably to high-force eccentric exercises, as reported by Alfredson.[6] Fifteen recreational running athletes (12 males; 3 females; mean age, 44 years) with Achilles tendon pain and decreased eccentric and concentric calf strength underwent an eccentric-resistance exercise program of progressively increasing loads. The eccentric-resistance group was compared to a similar control group (11 men; 4 women; mean age, 40) of patients with recalcitrant Achilles tendon pain treated with rest, nonsteroidal anti-inflammatory drugs

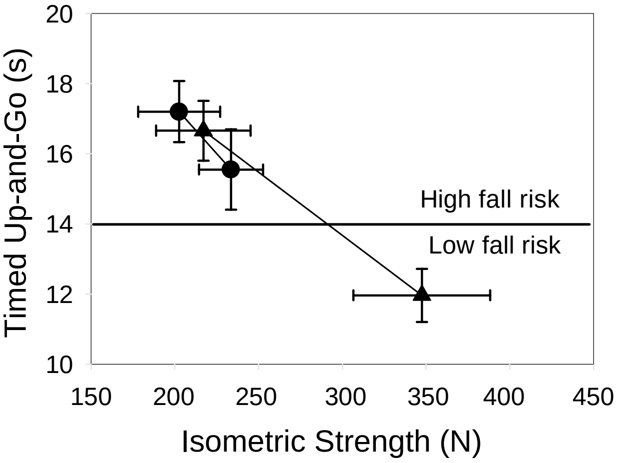

FIGURE 5. Performance of frail elderly (as measured both pretraining and posttraining) on a task dependent in part on leg strength (the "timed up and go" fall-risk assessment). Any improvement in leg strength in an elderly population likely has a clinical effect. Here the traditional resistance exercise group (circles) improved strength (15%) and time on the fall-risk assessment (1.7 seconds), though the improvement was not statistically significant (P = .511 and P = .071, respectively). The eccentric resistance group's (triangles) strength and fall-risk, however, though no different than the traditional group initially, did improve significantly ($P<.05$) in strength (60%) and with the timed up and go (4.7 seconds). In fact, the larger magnitude increase in strength was coupled to a shift in the eccentric group from a high fall risk to a low fall risk following training. Error bars = 1 SEM. Reprinted from LaStayo et al[109] with permission.

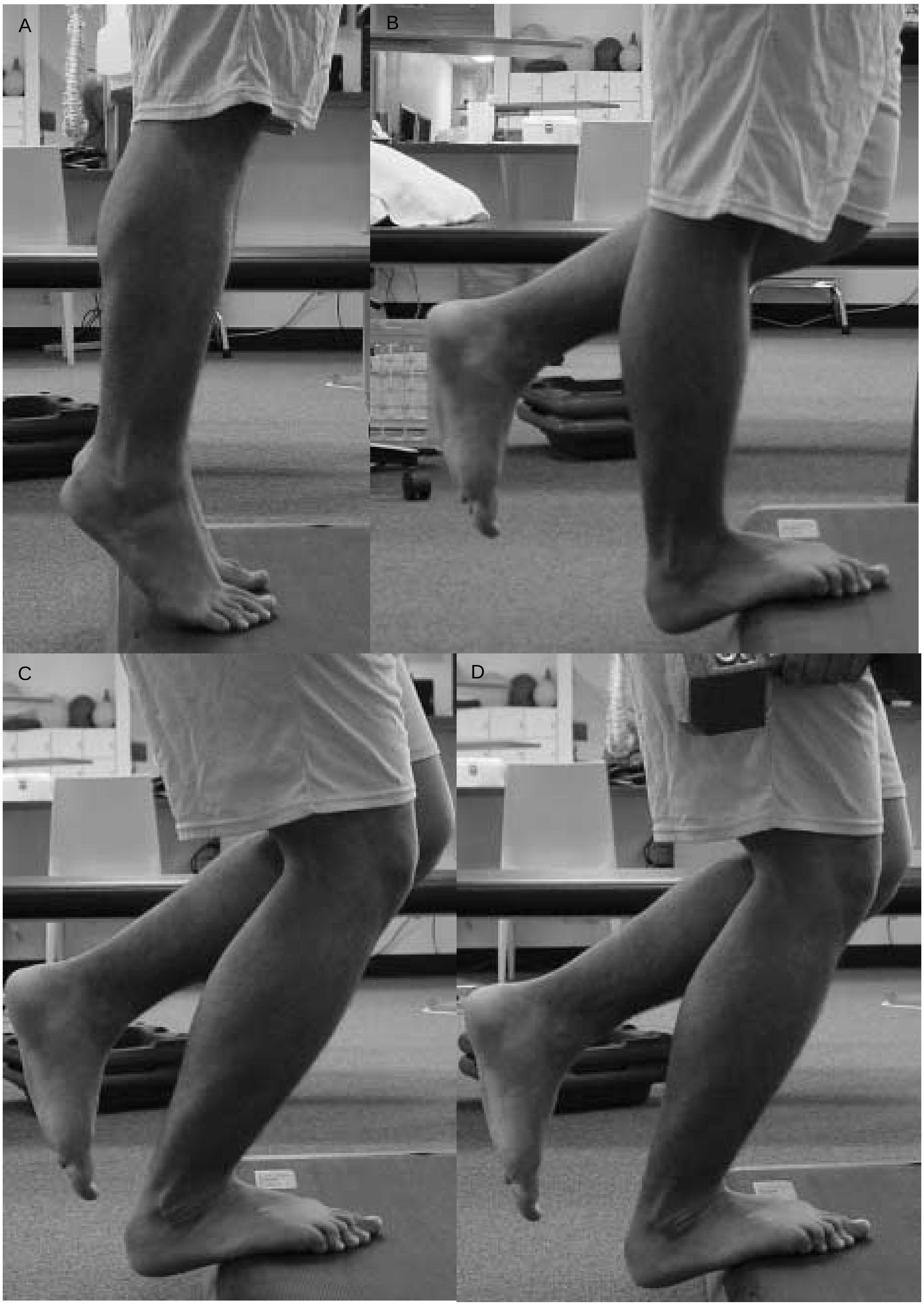

FIGURE 6. High calf muscle-tendon force training progression for patients with chronic Achilles tendinoses. (A) From an upright body position and standing bilaterally with all bodyweight on the forefoot, the involved ankle is lifted in a plantar-flexion position by the noninjured leg. (B) The involved calf muscle is loaded eccentrically as the ankle (only the involved side) is moved into dorsiflexion with the knee extended. (C) The eccentric calf muscle is loaded with the knee flexed. (D) Muscle-tendon forces are increased by adding weights into the exercise (either free weights, a weighted backpack, or with an exercise machine). Modified from Alfredson et al.[6]

(NSAID), orthotics, and physical therapy, which included an ordinary training program. The high-force eccentric exercise program consisted of calf raises twice a day, 7 days a week, for 3 sets of 15 repetitions. The subjects performed the concentric part of the exercise bilaterally (raising both heels), while using the impaired side only to do the eccentric lowering phase in a slow, controlled fashion. Once the exercises were possible with little or no discomfort, they were instructed to add resistance by using additional weight (Figure 6). After 12 weeks, all subjects in the eccentric-training program returned to preinjury levels of running activity, whereas subjects in the conventional resistance exercises group (that did not include high-force eccentric exercises) ultimately required surgery. These unambiguous findings may be interpreted with some skepticism, but clearly they suggest a clinical trend that high-force eccentric loading can be beneficial. Alfredson's[5] results are also strengthened by a 2-year follow-up, where 14 of the 15 runners in the high-force exercise group were still running pain free, while 1 went on to surgery. Similar findings have also been reported when using eccentrics as part of the resistance exercise program in patients with tendinoses at the knee and elbow.[30,48,91]

While the specific mechanisms as to why eccentric loading seems to optimize the rehabilitation of tendinoses have not been elucidated, it is implied that high muscle-tendon forces (eccentric) delivered in a controlled environment (rehabilitation setting) are needed for an optimal tendon adaptation. Again the irony presents itself in that the eccentric component is implicated in the initial injury (acute or chronic), yet high-force eccentric exercises are needed to maximize recovery. It is apparent, however, that the force generated during a concentric-eccentric exercise, or typical strengthening program, is not stimulating these beneficial tendon adaptations. The high forces produced eccentrically, while causing injury to tissues naïve to such forces, induce a beneficial tissue remodeling response when exposed to such forces chronically and progressively. That is, a program based on eccentric overload appears to be a suitable resistance exercise to elicit a remodeling response that meets the demands of functional and sport activities. These high eccentric muscle forces are only produced when an external force exceeds that of the muscle. To induce these high-magnitude forces, an external load capable of exceeding maximal isometric muscle force is required (Figure 7).

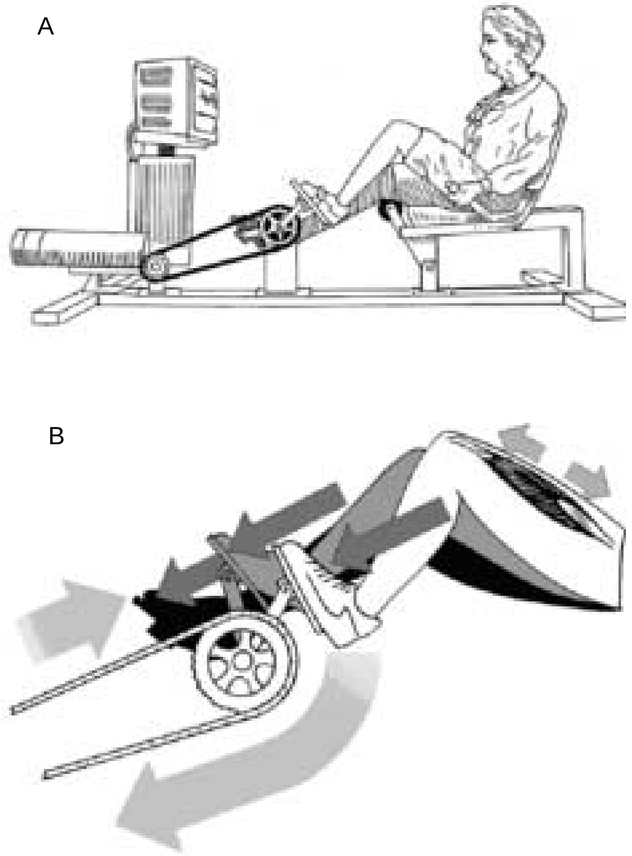

FIGURE 7. (A) Negative-work resistance exercise performed on an eccentric ergometer powered by a 3-hp motor driving the pedals in a backward rotation. The subjects vary their resistance to the reverse moving pedals to match a target on a computer monitor. The target is increased very gradually over a 2- to 3-week period to avoid muscle soreness, but by greater increments subsequently to optimize an individual's eccentric loading; (B) as the motor rotates the pedals at a set speed in a reverse direction (large rotating arrows), the subject attempts to slow down the reverse moving pedals by applying force (small arrows at foot). Negative work resistance training results when the magnitude of force produced by the motor exceeded that produced by the subject, thus the pedals continue backward, resulting in the eccentric lengthening of the quadriceps muscles, for example (small arrows at thigh). Reprinted from LaStayo et al[9] with permission.

JOINT AND LIGAMENT INJURIES: PREVENTION AND MANAGEMENT CONSIDERATIONS RELATED TO ECCENTRIC MUSCLE CONTRACTIONS

The high forces resulting from eccentric muscle activity can assist either in stabilizing or disrupting a joint. When a passive ligamentous restraint is disrupted, as in an anterior cruciate ligament (ACL) injury, muscle activity (particularly the eccentric component) is the only remaining way to prevent excessive translation of the joint. Ironically, these high eccentric muscle forces can also, however, amplify this unintended excessive motion in an unstable joint.[45]

The best-established method of stabilizing a ligament-impaired joint during a potentially destabilizing activity is to recruit a powerful muscular synergist to restrain the joint. At the knee, the hamstring's eccentric activity provides a posterior pull on the tibia to help offset the anterior force of the quadriceps.[154] In addition to their role as tibiofemoral stabilizers, the hamstrings are activated eccentrically prior to the initial contact of the limb,

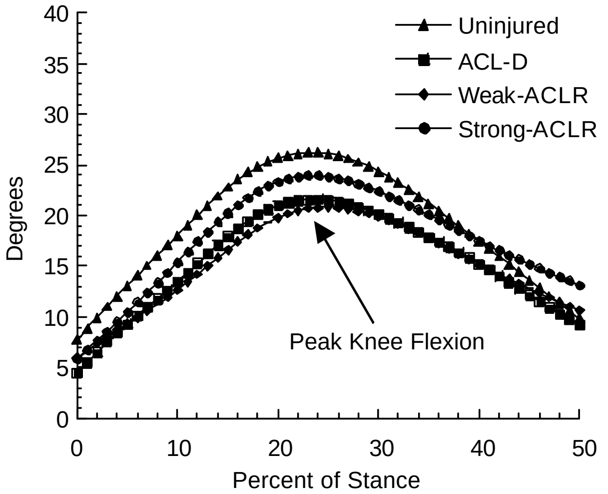

FIGURE 8. Knee flexion angles during the early stance phase of walking of subjects who were either (1) uninjured, (2) anterior cruciate ligament deficient (ACL-D), or (3) had undergone an anterior cruciate ligament reconstruction (ACLR) and were further classified as strong or weak. The weak-ACLR group strongly resembles the ACL-D patients who have lower knee flexion angles (creating a stiffer knee). The strong-ACLR group appeared to have sufficient eccentric quadriceps control during weight acceptance to resume normal knee flexion movement patterns. Reprinted from Lewek et al[113] with permission from Elsevier Science.

decelerating the forward progress of the leg in preparation for contact. Prior to initial contact during gait, both the medial and lateral hamstrings are activated earlier in subjects who are ACL deficient as compared to control subjects.[161] This protective hamstring activity can also be observed in healthy subjects prior to initial contact during higher-level activities, such as cutting, stopping, and landing maneuvers.[137] In fact, some have suggested that gender-related differences in muscular ability to decrease tibial translation explain, in part, the higher incidence of ACL injuries in women.[179] This necessary process of ''presetting'' the hamstrings for improved stability has been suggested as the reason that few studies have reported hamstring weakness following ACL injury.[102]

Individuals after ACL injury, particularly those whose knees are unstable (noncopers), excessively stiffen the knee via muscular cocontraction in an attempt to increase mechanical stability, which is a crude and ultimately undesirable method of stabilizing the joint. In several studies of movement after ACL rupture, we have demonstrated that the noncopers characteristically truncated knee flexion ranges during the weight acceptance phase of stance when the quadriceps acted eccentrically. The strategy was one of stiffening to damp the shock absorption of the joint and has been exemplified in all tasks we have studied (walking, jogging, stepping, and hopping). This exaggerated, suboptimal response increases contact force and shock (joint compression force) and can be detrimental. Some have even postulated that impaired load-attenuating abilities might be predictive of an increase in joint and ligament injuries.[79] Therefore, a balance between knee stability and shock absorption via appropriate hamstring and eccentric quadriceps actions during the loading phase is required.

Patients with ligament tears often exhibit eccentric muscle weakness in periarticular muscles.[123,154] Specifically, patients with ACL deficiency often have quadriceps muscles that are incapable of producing comparable eccentric forces to uninjured limbs.[45,139] While it is difficult to draw unequivocal conclusions from the conflicting evidence following surgical reconstruction of the torn ACL, it appears that eccentric hamstring strength is impaired after ACL reconstruction.[105,108,123] As well, quadriceps weakness following ACL reconstruction is also variable[105,108] and somewhat dependent on the numerous rehabilitation and testing protocols used, making it difficult to draw any consensus from the literature. Clearly, however, eccentric muscle dysfunction does exist around unstable (and painful[141]) joints, and therefore must be reversed. Following ACL reconstruction, those with weak quadriceps abnormally stiffen the knee (with knee angles and moments similar to those of subjects with ACL-deficient knees) during early stance while walking and jogging[113] (Figure 8). This diminished force-generating capability of the quadriceps impairs the ability to appropriately absorb shock at the knee via submaximal eccentric contractions.

While the consensus is that eccentric muscle retraining is essential, the question as to the mode and optimal dosage of eccentric exercise has not been answered. Certainly for the return of muscle mass, strength, and for muscle spring adaptations, chronic high-force eccentric exercise for 6 to 12 weeks is a potent option. Submaximal eccentric muscle-loading regimes may also be ideally suited to help overcome the force-attenuating and SSC impairments noted in these patients, however, this too remains untested.

CONCLUSION

The traditional thinking that eccentric contractions result in an obligatory damage response may be overstated. While the high forces produced in muscles working eccentrically can certainly cause damage and injury, muscle and tendon appear very capable of adapting to such high forces if the muscle experiences this stimulus progressively and repeatedly. The adaptive mechanisms are not uniformly defined, but it is apparent that muscle can increase in size and strength and its spring quality can change following chronic exposure to eccentric contractions. The muscle-tendon structure also responds favorably to an eccentric-resistance exercise protocol. These adaptations, which need to be explored further in well-defined, basic, randomized epidemiological studies, play a part in (1) the enhancement of high-power sport activities, (2) the prevention, and (3) the rehabilitation of sport injuries and nonsport musculoskeletal impairments, especially those that afflict the elderly. Despite the dearth of studies comparing traditional strength training to exclusively eccentric training, the beneficial effects of the high negative-work exercise regimes are apparent. In this paper we have explored the potential to capitalize on the ability to perform eccentric contractions (1) chronically (due to the low energetic cost), even with the frail elderly, and (2) with extremely high muscle forces (in excess of the maximum isometric force), which is only possible during eccentric, not isometric nor concentric, contractions. If an exercise is designed to simply recover, eccentrically, the forces generated concentrically, then that exercise does not take advantage of the unique high force-producing properties of eccentric contractions.

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REFERENCES

1. Abbott BC, Bigland B. The effects of force and speed changes on the rate of oxygen consumption during negative work exercise. *J Physiol.* 1953;120:319-325.
2. Abbott BC, Bigland B, Ritchie JM. The physiological cost of negative work. *J Physiol.* 1952;117:380-390.
3. Albert M. Introduction. In: Alpter M, eds. *Eccentric Muscle Training in Sports and Orthopaedics.* 2nd ed. New York, NY: Churchill Livingstone; 1995:1-11.
4. Alexander RM, Bennet-Clark HC. Storage of elastic strain energy in muscle and other tissues. *Nature.* 1977;265:114-117.
5. Alfredson H, Lorentzon R. Chronic Achilles tendinosis: recommendations for treatment and prevention. *Sports Med.* 2000;29:135-146.
6. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med.* 1998;26:360-366.
7. Alfredson H, Pietila T, Ohberg L, Lorentzon R. Achilles tendinosis and calf muscle strength. The effect of short-term immobilization after surgical treatment. *Am J Sports Med.* 1998;26:166-171.
8. Andriacchi TP, Andersson GB, Fermier RW, Stern D, Galante JO. A study of lower-limb mechanics during stair-climbing. *J Bone Joint Surg Am.* 1980;62:749-757.
9. Archambault JM, Wiley JP, Bray RC. Exercise loading of tendons and the development of overuse injuries. A review of current literature. *Sports Med.* 1995;20:77-89.
10. Armstrong RB. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Med Sci Sports Exerc.* 1984;16:529-538.
11. Armstrong RB, Ogilvie RW, Schwane JA. Eccentric exercise-induced injury to rat skeletal muscle. *J Appl Physiol.* 1983;54:80-93.
12. Asmussen E. Positive and negative muscular work. *Acta Physiol Scand.* 1953;28:364-382.
13. Asmussen E, Bonde-Petersen F. Apparent efficiency and storage of elastic energy in human muscles during exercise. *Acta Physiol Scand.* 1974;92:537-545.
14. Astrom M, Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. *Clin Orthop.* 1995;151-164.
15. Bassey EJ, Fiatarone MA, O'Neill EF, Kelly M, Evans WJ, Lipsitz LA. Leg extensor power and functional performance in very old men and women. *Clin Sci (Lond).* 1992;82:321-327.
16. Bean J, Herman S, Kiely DK, et al. Weighted stair climbing in mobility-limited older people: a pilot study. *J Am Geriatr Soc.* 2002;50:663-670.
17. Bell SP, Nyland L, Tischler MD, McNabb M, Granzier H, LeWinter MM. Alterations in the determinants of diastolic suction during pacing tachycardia. *Circ Res.* 2000;87:235-240.
18. Benn C, Forman K, Mathewson D, et al. The effects of serial stretch loading on stretch work and stretch-shorten cycle performance in the knee musculature. *J Orthop Sports Phys Ther.* 1998;27:412-422.
19. Biewener AA, Roberts TJ. Muscle and tendon contributions to force, work, and elastic energy savings: a comparative perspective. *Exerc Sport Sci Rev.* 2000;28:99-107.
20. Bigland-Ritchie B, Woods JJ. Integrated electromyogram and oxygen uptake during positive and negative work. *J Physiol.* 1976;260:267-277.
21. Birch HL, McLaughlin L, Smith RK, Goodship AE. Treadmill exercise-induced tendon hypertrophy: assessment of tendons with different mechanical functions. *Equine Vet J Suppl.* 1990;30:222-226.
22. Bobbert MF. Dependence of human squat jump performance on the series elastic compliance of the triceps surae: a simulation study. *J Exp Biol.* 2001;204:533-542.
23. Bobbert MF, Gerritsen KGM, Litjens MCA, van Soest AJ. Explanation of differences in jump height between countermovement and squat jumps. *15th Congress of the International Society of Biomechnics.* Jyvaskyla, Finland: Gummerus Printing; 1996: 110-111.
24. Booth FW, Baldwin KM. Muscle plasticity: energy demand and supply processes. In: Rowell LB, Shepherd JT, eds. *Handbook of Physiology.* Shepard, NY: Oxford University Press; 1996:1075-1123.
25. Bosco C, Tihanyi J, Komi PV, Fekete G, Apor P. Store and recoil of elastic energy in slow and fast types of human skeletal muscles. *Acta Physiol Scand.* 1982;116:343-349.
26. Braith RW, Mills RM, Welsch MA, Keller JW, Pollock ML. Resistance exercise training restores bone mineral density in heart transplant recipients. *J Am Coll Cardiol.* 1996;28:1471-1477.
27. Brandenburg JP, Docherty D. The effects of accentuated eccentric loading on strength, muscle hypertrophy, and neural adaptations in trained individuals. *J Strength Cond Res.* 2002;16:25-32.
28. Brockett CL, Morgan DL, Proske U. Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Med Sci Sports Exerc.* 2001;33:783-790.
29. Buchanan CI, Marsh RL. Effects of long-term exercise on the biomechanical properties of the Achilles tendon of guinea fowl. *J Appl Physiol.* 2001;90:164-171.
30. Cannell LJ, Taunton JE, Clement DB, Smith C, Khan KM. A randomised clinical trial of the efficacy of drop squats or leg extension/leg curl exercises to treat clinically diagnosed jumper's knee in athletes: pilot study. *Br J Sports Med.* 2001;35:60-64.
31. Cavagna GA, Komarek L, Mazzoleni S. The mechanics of sprint running. *J Physiol.* 1971;217:709-721.

32. Cavagna GA, Mazzanti M, Heglund NC, Citterio G. Storage and release of mechanical energy by active muscle: a non-elastic mechanism? *J Exp Biol.* 1985;115:79-87.
33. Cavanagh PR, Mulfinger LM, Owens DA. How do the elderly negotiate stairs? *Muscle Nerve Suppl.* 1997;5:S52-55.
34. Cavagna GA, Saibene FP, Margaria R. Effect of negative work on the amount of positive work performed by an isolated muscle. *J Appl Physiol.* 1965;20:157.
35. Cavagna GA. Storage and utilization of elastic energy in skeletal muscle. *Exerc Sport Sci Rev.* 1977;5:89-129.
36. Cazorla O, Freiburg A, Helmes M, et al. Differential expression of cardiac titin isoforms and modulation of cellular stiffness. *Circ Res.* 2000;86:59-67.
37. Cazorla O, Wu Y, Irving TC, Granzier H. Titin-based modulation of calcium sensitivity of active tension in mouse skinned cardiac myocytes. *Circ Res.* 2001;88:1028-1035.
38. Chambers TJ, Evans M, Gardner TN, Turner-Smith A, Chow JW. Induction of bone formation in rat tail vertebrae by mechanical loading. *Bone Miner.* 1993;20:167-178.
39. Charette SL, McEvoy L, Pyka G, et al. Muscle hypertrophy response to resistance training in older women. *J Appl Physiol.* 1991;70:1912-1916.
40. Chelly SM, Denis C. Leg power and hopping stiffness: relationship with sprint running performance. *Med Sci Sports Exerc.* 2001;33:326-333.
41. Chen TC, Hsieh SS. Effects of a 7-day eccentric training period on muscle damage and inflammation. *Med Sci Sports Exerc.* 2001;33:1732-1738.
42. Clarkson PM, Nosaka K, Braun B. Muscle function after exercise-induced muscle damage and rapid adaptation. *Med Sci Sports Exerc.* 1992;24:512-520.
43. Clarkson PM, Tremblay I. Exercise-induced muscle damage, repair, and adaptation in humans. *J Appl Physiol.* 1988;65:1-6.
44. Clement DB, Taunton JE, Smart GW. Achilles tendinitis and peritendinitis: etiology and treatment. *Am J Sports Med.* 1984;12:179-184.
45. Colby S, Francisco A, Yu B, Kirkendall D, Finch M, Garrett W, Jr. Electromyographic and kinematic analysis of cutting maneuvers. Implications for anterior cruciate ligament injury. *Am J Sports Med.* 2000;28:234-240.
46. Cooper B, Oberdorfer M, Rumpf D, Malakhova O, Rudman R, Mariotti A. Trauma modifies strength and composition of retrodiscal tissues of the goat temporomandibular joint. *Oral Dis.* 1999;5:329-336.
47. Croisier JL, Forthomme B, Namurois MH, Vanderthommen M, Crielaard JM. Hamstring muscle strain recurrence and strength performance disorders. *Am J Sports Med.* 2002;30:199-203.
48. Curwin S, Stanish WD. *Tendinitis: Its Etiology and Treatment.* Lexington, MA: Collamore Press; 1984.
49. Dean E. Physiology and therapeutic implications of negative work. *A review. Phys Ther.* 1988;68:233-237.
50. DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. *J Appl Physiol.* 2000;88:1804-1811.
51. Dickinson MH, Farley CT, Full RJ, Koehl MA, Kram R, Lehman S. How animals move: an integrative view. *Science.* 2000;288:100-106.
52. el Hawary R, Stanish WD, Curwin SL. Rehabilitation of tendon injuries in sport. *Sports Med.* 1997;24:347-358.
53. Enoka RM. Neural strategies in the control of muscle force. *Muscle Nerve Suppl.* 1997;5:S66-69.
54. Ettema GJ, Huijing PA, van Ingen Schenau GJ, de Haan A. Effects of prestretch at the onset of stimulation on mechanical work output of rat medial gastrocnemius muscle-tendon complex. *J Exp Biol.* 1990;152:333-351.
55. Ettema GJ. Mechanical efficiency and efficiency of storage and release of series elastic energy in skeletal muscle during stretch-shorten cycles. *J Exp Biol.* 1996;199(Pt 9):1983-1997.
56. Evans WJ. Exercise, nutrition and aging. *J Nutr.* 1992;122:796-801.
57. Evans WJ. What is sarcopenia? *J Gerontol A Biol Sci Med Sci.* 1995;50:5-8.
58. Farley CT, Blickhan R, Saito J, Taylor CR. Hopping frequency in humans: a test of how springs set stride frequency in bouncing gaits. *J Appl Physiol.* 1991;71:2127-2132.
59. Farley CT, Glasheen J, McMahon TA. Running springs: speed and animal size. *J Exp Biol.* 1993;185:71-86.
60. Farley CT. Maximum speed and mechanical power output in lizards. *J Exp Biol.* 1997;200(Pt 16):2189-2195.
61. Faulkner JA, Brooks SV, Opiteck JA. Injury to skeletal muscle fibers during contractions: conditions of occurrence and prevention. *Phys Ther.* 1993;73:911-921.
62. Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA, Evans WJ. High-intensity strength training in nonagenarians. Effects on skeletal muscle. *JAMA.* 1990;263:3029-3034.
63. Fielding RA. The role of progressive resistance training and nutrition in the preservation of lean body mass in the elderly. *J Am Coll Nutr.* 1995;14:587-594.
64. Friden J, Lieber RL. Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiol Scand.* 2001;171:321-326.
65. Friden J, Sjostrom M, Ekblom B. Myofibrillar damage following intense eccentric exercise in man. *Int J Sports Med.* 1983;4:170-176.
66. Fyfe I, Stanish WD. The use of eccentric training and stretching in the treatment and prevention of tendon injuries. *Clin Sports Med.* 1992;11:601-624.
67. Garrett WE, Jr. Muscle strain injuries. *Am J Sports Med.* 1996;24:S2-8.
68. Garrett WE, Jr., Safran MR, Seaber AV, Glisson RR, Ribbeck BM. Biomechanical comparison of stimulated and nonstimulated skeletal muscle pulled to failure. *Am J Sports Med.* 1987;15:448-454.
69. Granzier H, Labeit S. Cardiac titin: an adjustable multi-functional spring. *J Physiol.* 2002;541:335-342.
70. Granzier HL, Wang K. Passive tension and stiffness of vertebrate skeletal and insect flight muscles: the contribution of weak cross-bridges and elastic filaments. *Biophys J.* 1993;65:2141-2159.
71. Gregorio CC, Granzier H, Sorimachi H, Labeit S. Muscle assembly: a titanic achievement? *Curr Opin Cell Biol.* 1999;11:18-25.
72. Hasselman CT, Best TM, Seaber AV, Garrett WE, Jr. A threshold and continuum of injury during active stretch of rabbit skeletal muscle. *Am J Sports Med.* 1995;23:65-73.
73. Hawkins SA, Schroeder ET, Wiswell RA, Jaque SV, Marcell TJ, Costa K. Eccentric muscle action increases site-specific osteogenic response. *Med Sci Sports Exerc.* 1999;31:1287-1292.
74. Heglund NC, Cavagna GA. Mechanical work, oxygen consumption, and efficiency in isolated frog and rat muscle. Am *J Physiol.* 1987;253:C22-29.
75. Heiderscheit BC, McLean KP, Davies GJ. The effects of isokinetic vs. plyometric training on the shoulder internal rotators. *J Orthop Sports Phys Ther.* 1996;23:125-133.
76. Heikkinen E, Suominen H, Vihersaari T, Vuori I, Kiiskinen A. Effects of physical training on enzyme

activities of bones, tendons and skeletal muscle in mice. In: Howard H, Poortmans JR, eds. *Metabolic Adaptation to Prolonged Exercise.* Basel, Switzerland: Birkausen Verlag; 1975:262-267.

77. Heikkinen E, Vuori I. Effect of physical activity on the metabolism of collagen in aged mice. *Acta Physiol Scand.* 1972;84:543-549.
78. Helmes M, Trombitas K, Granzier H. Titin develops restoring force in rat cardiac myocytes. *Circ Res.* 1996;79:619-626.
79. Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes. Decreased impact forces and increased hamstring torques. *Am J Sports Med.* 1996;24:765-773.
80. Hiemstra LA, Webber S, MacDonald PB, Kriellaars DJ. Knee strength deficits after hamstring tendon and patellar tendon anterior cruciate ligament reconstruction. *Med Sci Sports Exerc.* 2000;32:1472-1479.
81. Hof AL, Geelen BA, Van den Berg J. Calf muscle moment, work and efficiency in level walking; role of series elasticity. *J Biomech.* 1983;16:523-537.
82. Hoffer JA, Caputi AA, Pose IE, Griffiths RI. Roles of muscle activity and load on the relationship between muscle spindle length and whole muscle length in the freely walking cat. *Prog Brain Res.* 1989;80:75-85; discussion 57-60.
83. Horowits R, Podolsky RJ. The positional stability of thick filaments in activated skeletal muscle depends on sarcomere length: evidence for the role of titin filaments. *J Cell Biol.* 1987;105:2217-2223.
84. Hortobagyi T, Barrier J, Beard D, et al. Greater initial adaptations to submaximal muscle lengthening than maximal shortening. *J Appl Physiol.* 1996;81:1677-1682.
85. Hortobagyi T, Denahan T. Variability in creatine kinase: methodological, exercise, and clinically related factors. *Int J Sports Med.* 1989;10:69-80.
86. Hortobagyi T, DeVita P. Favorable neuromuscular and cardiovascular responses to 7 days of exercise with an eccentric overload in elderly women. *J Gerontol A Biol Sci Med Sci.* 2000;55:B401-410.
87. Hortobagyi T, Devita P, Money J, Barrier J. Effects of standard and eccentric overload strength training in young women. *Med Sci Sports Exerc.* 2001;33:1206-1212.
88. Hortobagyi T, Hill JP, Houmard JA, Fraser DD, Lambert NJ, Israel RG. Adaptive responses to muscle lengthening and shortening in humans. *J Appl Physiol.* 1996;80:765-772.
89. Hortobagyi T, Tunnel D, Moody J, Beam S, DeVita P. Low- or high-intensity strength training partially restores impaired quadriceps force accuracy and steadiness in aged adults. *J Gerontol A Biol Sci Med Sci.* 2001;56:B38-47.
90. Hortobagyi T, Zheng D, Weidner M, Lambert NJ, Westbrook S, Houmard JA. The influence of aging on muscle strength and muscle fiber characteristics with special reference to eccentric strength. *J Gerontol A Biol Sci Med Sci.* 1995;50:B399-406.
91. Jensen K, Di Fabio RP. Evaluation of eccentric exercise in treatment of patellar tendinitis. *Phys Ther.* 1989;69:211-216.
92. Johnson BL. Eccentric and concentric muscle training for strength development. *Med Sci Sports.* 1972;4:111-115.
93. Jones DA, Rutherford OM. Human muscle strength training: the effects of three different regimens and the nature of the resultant changes. *J Physiol.* 1987;391:1-11.
94. Judge JO, Whipple RH, Wolfson LI. Effects of resistive and balance exercises on isokinetic strength in older persons. *J Am Geriatr Soc.* 1994;42:937-946.
95. Katz B. The relation between force and speed in muscular contraction. *J Physiol.* 1939;96:45-64.
96. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. *Sports Med.* 1999;27:393-408.
97. Khan KM, Cook JL, Maffulli N, Kannus P. Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin. *Br J Sports Med.* 2000;34:81-83.
98. Koh TJ. Do small heat shock proteins protect skeletal muscle from injury? *Exerc Sport Sci Rev.* 2002;30:117-121.
99. Komi PV, Bosco C. Utilization of stored elastic energy in leg extensor muscles by men and women. *Med Sci Sports.* 1978;10:261-265.
100. Komi PV, Buskirk ER. Effect of eccentric and concentric muscle conditioning on tension and electrical activity of human muscle. *Ergonomics.* 1972;15:417-434.
101. Komi PV. Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *J Biomech.* 2000;33:1197-1206.
102. Kramer J, Nusca D, Fowler P, Webster-Bogaert S. Knee flexor and extensor strength during concentric and eccentric muscle actions after anterior cruciate ligament reconstruction using the semitendinosus tendon and ligament augmentation device. *Am J Sports Med.* 1993;21:285-291.
103. Kubo K, Kanehisa H, Kawakami Y, Fukunaga T. Elastic properties of muscle-tendon complex in long-distance runners. *Eur J Appl Physiol.* 2000;81:181-187.
104. Kujala UM, Orava S, Jarvinen M. Hamstring injuries. Current trends in treatment and prevention. *Sports Med.* 1997;23:397-404.
105. Kvist J, Karlberg C, Gerdle B, Gillquist J. Anterior tibial translation during different isokinetic quadriceps torque in anterior cruciate ligament deficient and nonimpaired individuals. *J Orthop Sports Phys Ther.* 2001;31:4-15.
106. Lanyon LE. Functional strain in bone tissue as an objective, and controlling stimulus for adaptive bone remodelling. *J Biomech.* 1987;20:1083-1093.
107. Larsson L, Grimby G, Karlsson J. Muscle strength and speed of movement in relation to age and muscle morphology. *J Appl Physiol.* 1979;46:451-456.
108. Lass P, Kaalund S, leFevre S, Arendt-Nielsen L, Sinkjaer T, Simonsen O. Muscle coordination following rupture of the anterior cruciate ligament. Electromyographic studies of 14 patients. *Acta Orthop Scand.* 1991;62:9-14.
109. LaStayo PC, Ewy GA, Pierotti DD, Johns RK, Lindstedt S. The positive effects of negative work: increased muscle strength and decreased fall risk in a frail elderly population. *J Gerontol A Biol Sci Med Sci.* 2003;58:M419-424.
110. LaStayo PC, Pierotti DJ, Pifer J, Hoppeler H, Lindstedt SL. Eccentric ergometry: increases in locomotor muscle size and strength at low training intensities. *Am J Physiol Regul Integr Comp Physiol.* 2000;278:R1282-1288.
111. LaStayo PC, Reich TE, Urquhart M, Hoppeler H, Lindstedt SL. Chronic eccentric exercise: improvements in muscle strength can occur with little demand for oxygen. Am *J Physiol.* 1999;276:R611-615.
112. Leadbetter WB. Cell-matrix response in tendon injury. *Clin Sports Med.* 1992;11:533-578.

113. Lewek M, Rudolph K, Axe M, Snyder-Mackler L. The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. *Clin Biomech (Bristol, Avon).* 2002;17:56-63.
114. Lexell J, Taylor CC, Sjostrom M. What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year-old men. *J Neurol Sci.* 1988;84:275-294.
115. Lindstedt SL, LaStayo PC, Reich TE. When active muscles lengthen: properties and consequences of eccentric contractions. *News Physiol Sci.* 2001;16:256-261.
116. Lindstedt SL, Reich TE, Keim P, LaStayo PC. Do muscles function as adaptable locomotor springs? *J Exp Biol.* 2002;205:2211-2216.
117. Linke WA, Granzier H. A spring tale: new facts on titin elasticity. *Biophys J.* 1998;75:2613-2614.
118. Linke WA, Ivemeyer M, Olivieri N, Kolmerer B, Ruegg JC, Labeit S. Towards a molecular understanding of the elasticity of titin. *J Mol Biol.* 1996;261:62-71.
119. Linke WA, Rudy DE, Centner T, et al. I-band titin in cardiac muscle is a three-element molecular spring and is critical for maintaining thin filament structure. *J Cell Biol.* 1999;146:631-644.
120. Liu SH, Yang RS, al-Shaikh R, Lane JM. Collagen in tendon, ligament, and bone healing. A current review. *Clin Orthop.* 1995;265-278.
121. Lynn R, Morgan DL. Decline running produces more sarcomeres in rat vastus intermedius muscle fibers than does incline running. *J Appl Physiol.* 1994;77:1439-1444.
122. MacIntyre DL, Reid WD, Lyster DM, Szasz IJ, McKenzie DC. Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise. *J Appl Physiol.* 1996;80:1006-1013.
123. MacLean CL, Taunton JE, Clement DB, Regan W. Eccentric and concentric isokinetic moment characteristics in the quadriceps and hamstrings of the chronic isolated posterior cruciate ligament injured knee. *Br J Sports Med.* 1999;33:405-408.
124. Maffulli N, Ewen SW, Waterston SW, Reaper J, Barrass V. Tenocytes from ruptured and tendinopathic achilles tendons produce greater quantities of type III collagen than tenocytes from normal achilles tendons. An in vitro model of human tendon healing. *Am J Sports Med.* 2000;28:499-505.
125. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surg Sports Traumatol Arthrosc.* 2001;9:42-47.
126. Maruyama K, Matsubara S, Natori R, Nonomura Y, Kimura S. Connectin, an elastic protein of muscle. Characterization and Function. *J Biochem (Tokyo).* 1977;82:317-337.
127. Matavulj D, Kukolj M, Ugarkovic D, Tihanyi J, Jaric S. Effects of plyometric training on jumping performance in junior basketball players. *J Sports Med Phys Fitness.* 2001;41:159-164.
128. McFadyen BJ, Winter DA. An integrated biomechanical analysis of normal stair ascent and descent. *J Biomech.* 1988;21:733-744.
129. Mero A, Komi PV. Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. *Eur J Appl Physiol Occup Physiol.* 1986;55:553-561.
130. Mueller MJ, Maluf KS. Tissue adaptation to physical stress: a proposed "Physical Stress Theory" to guide physical therapist practice, education, and research. *Phys Ther.* 2002;82:383-403.
131. Newton RU, Kraemer WJ, Hakkinen K. Effects of ballistic training on preseason preparation of elite volleyball players. *Med Sci Sports Exerc.* 1999;31:323-330.
132. Noonan TJ, Garrett WE, Jr. Injuries at the myotendinous junction. *Clin Sports Med.* 1992;11:783-806.
133. Noonan TJ, Garrett WE, Jr. Muscle strain injury: diagnosis and treatment. *J Am Acad Orthop Surg.* 1999;7:262-269.
134. Nosaka K, Clarkson PM. Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Med Sci Sports Exerc.* 1996;28:953-961.
135. Nosaka K, Clarkson PM. Muscle damage following repeated bouts of high force eccentric exercise. *Med Sci Sports Exerc.* 1995;27:1263-1269.
136. Nosaka K, Clarkson PM, McGuiggin ME, Byrne JM. Time course of muscle adaptation after high force eccentric exercise. *Eur J Appl Physiol Occup Physiol.* 1991;63:70-76.
137. Nyland JA, Shapiro R, Caborn DN, Nitz AJ, Malone TR. The effect of quadriceps femoris, hamstring, and placebo eccentric fatigue on knee and ankle dynamics during crossover cutting. *J Orthop Sports Phys Ther.* 1997;25:171-184.
138. Olson JM, Marsh RL. Activation patterns and length changes in hindlimb muscles of the bullfrog Rana catesbeiana during jumping. *J Exp Biol.* 1998;201 (Pt 19):2763-2777.
139. Osternig LR, James CR, Bercades DT. Eccentric knee flexor torque following anterior cruciate ligament surgery. *Med Sci Sports Exerc.* 1996;28:1229-1234.
140. Overend TJ, Versteegh TH, Thompson E, Birmingham TB, Vandervoort AA. Cardiovascular stress associated with concentric and eccentric isokinetic exercise in young and older adults. *J Gerontol A Biol Sci Med Sci.* 2000;55:B177-182.
141. Owings TM, Grabiner MD. Motor control of the vastus medialis oblique and vastus lateralis muscles is disrupted during eccentric contractions in subjects with patellofemoral pain. *Am J Sports Med.* 2002;30:483-487.
142. Pette D. Historical Perspectives: plasticity of mammalian skeletal muscle. *J Appl Physiol.* 2001;90:1119-1124.
143. Plancher KD, Litchfield R, Hawkins RJ. Rehabilitation of the shoulder in tennis players. *Clin Sports Med.* 1995;14:111-137.
144. Pousson M, Van Hoecke J, Goubel F. Changes in elastic characteristics of human muscle induced by eccentric exercise. *J Biomech.* 1990;23:343-348.
145. Prilutsky BI, Herzog W, Leonard TR, Allinger TL. Role of the muscle belly and tendon of soleus, gastrocnemius, and plantaris in mechanical energy absorption and generation during cat locomotion. *J Biomech.* 1996;29:417-434.
146. Proske U, Morgan DL. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *J Physiol.* 2001;537:333-345.
147. Reich TE, Lindstedt SL, LaStayo PC, Keim P. Eccentrics and springs: individual molecule to whole organism. *Experimental Biology 2003.* San Diego, CA: 2003.
148. Reich TE, Lindstedt SL, LaStayo PC, Pierotti DJ. Is the spring quality of muscle plastic? *Am J Physiol Regul Integr Comp Physiol.* 2000;278:R1661-1666.
149. Riley GP, Harrall RL, Constant CR, Chard MD, Cawston TE, Hazleman BL. Glycosaminoglycans of

human rotator cuff tendons: changes with age and in chronic rotator cuff tendinitis. *Ann Rheum Dis.* 1994;53:367-376.

150. Rudolph KS, Axe MJ, Buchanan TS, Scholz JP, Snyder-Mackler L. Dynamic stability in the anterior cruciate ligament deficient knee. *Knee Surg Sports Traumatol Arthrosc.* 2001;9:62-71.
151. Rudolph KS, Eastlack ME, Axe MJ, Snyder-Mackler L. 1998 Basmajian Student Award Paper: Movement patterns after anterior cruciate ligament injury: a comparison of patients who compensate well for the injury and those who require operative stabilization. *J Electromyogr Kinesiol.* 1998;8:349-362.
152. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. *Am J Sports Med.* 2002;30:287-305.
153. Seyfarth A, Blickhan R, Van Leeuwen JL. Optimum take-off techniques and muscle design for long jump. *J Exp Biol.* 2000;203(Pt 4):741-750.
154. Shirakura K, Kato K, Udagawa E. Characteristics of the isokinetic performance of patients with injured cruciate ligaments. *Am J Sports Med.* 1992;20:754-760.
155. Sim FH, Simonet WT, Melton LJ, 3rd, Lehn TA. Ice hockey injuries. *Am J Sports Med.* 1987;15:30-40.
156. Smith LL, Fulmer MG, Holbert D, et al. The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *Br J Sports Med.* 1994;28:267-271.
157. Stanish WD, Curwin S, Rubinovich M. Tendinitis: the analysis and treatment for running. *Clin Sports Med.* 1985;4:593-609.
158. Stanish WD, Rubinovich RM, Curwin S. Eccentric exercise in chronic tendinitis. *Clin Orthop.* 1986;65-68.
159. Startzell JK, Owens DA, Mulfinger LM, Cavanagh PR. Stair negotiation in older people: a review. *J Am Geriatr Soc.* 2000;48:567-580.
160. Stauber WT. Eccentric action of muscles: physiology, injury, and adaptation. *Exerc Sport Sci Rev.* 1989;17:157-185.
161. St Clair Gibson A, Lambert MI, Durandt JJ, Scales N, Noakes TD. Quadriceps and hamstrings peak torque ratio changes in persons with chronic anterior cruciate ligament deficiency. *J Orthop Sports Phys Ther.* 2000;30:418-427.
162. Stone MH. Connective tissue and bone response to strength training. In: Komi PV, ed. *Strength and Power in Sport. The Encyclopedia of Sports Medicine.* Oxford, UK: Blackwell; 1991:279-290.
163. Sutko JL, Publicover NG, Moss RL. Titin: an elastic link between length and active force production in myocardium. *Circulation.* 2001;104:1585-1587.
164. Svernlov B, Adolfsson L. Non-operative treatment regime including eccentric training for lateral humeral epicondylalgia. *Scand J Med Sci Sports.* 2001;11:328-334.
165. Taylor CR. Force development during sustained locomotion: a determinant of gait, speed and metabolic power. *J Exp Biol.* 1985;115:253-262.
166. Thompson JL, Balog EM, Fitts RH, Riley DA. Five myofibrillar lesion types in eccentrically challenged, unloaded rat adductor longus muscle—a test model. *Anat Rec.* 1999;254:39-52.
167. Tipton CM, Matthes RD, Maynard JA, Carey RA. The influence of physical activity on ligaments and tendons. *Med Sci Sports.* 1975;7:165-175.
168. Trappe TA, Carrithers JA, White F, Lambert CP, Evans WJ, Dennis RA. Titin and nebulin content in human skeletal muscle following eccentric resistance exercise. *Muscle Nerve.* 2002;25:289-292.
169. Verhoshanski Y, Chornonson G. Jump exercises in sprint training. *Track Field Q.* 1967;9:1909.
170. Vincent KR, Braith RW. Resistance exercise and bone turnover in elderly men and women. *Med Sci Sports Exerc.* 2002;34:17-23.
171. Voight ML, Draovitch P. Plyometrics. In: Alberts M, ed. *Eccentric Muscle Training in Sports and Orthopedics.* New York, NY: Churchill Livingstone; 1991:45.
172. Vossen JF, Kramer JF, Burke DG, Vossen DP. Comparison of dynamic push-up training and plyometric push-up training on upper-body power and strength. *J Strength Cond Res.* 2000;14:248-253.
173. Walshe AD, Wilson GJ, Ettema GJ. Stretch-shorten cycle compared with isometric preload: contributions to enhanced muscular performance. *J Appl Physiol.* 1998;84:97-106.
174. Wang K, McClure J, Tu A. Titin: major myofibrillar components of striated muscle. *Proc Natl Acad Sci USA.* 1979;76:3698-3702.
175. Warren GL, Ingalls CP, Lowe DA, Armstrong RB. What mechanisms contribute to the strength loss that occurs during and in the recovery from skeletal muscle injury? *J Orthop Sports Phys Ther.* 2002;32:58-64.
176. Wernig A, Salvini TF, Irintchev A. Axonal sprouting and changes in fibre types after running-induced muscle damage. *J Neurocytol.* 1991;20:903-913.
177. White AT, Johnson SC. Physiological aspects and injury in elite Alpine skiers. *Sports Med.* 1993;15:170-178.
178. Wilson GJ, Murphy AJ, Giorgi A. Weight and plyometric training: effects on eccentric and concentric force production. *Can J Appl Physiol.* 1996;21:301-315.
179. Wojtys EM, Ashton-Miller JA, Huston LJ. A gender-related difference in the contribution of the knee musculature to sagittal-plane shear stiffness in subjects with similar knee laxity. *J Bone Joint Surg Am.* 2002;84-A:10-16.
180. Woo SL, Ritter MA, Amiel D, et al. The biomechanical and biochemical properties of swine tendons—long term effects of exercise on the digital extensors. *Connect Tissue Res.* 1980;7:177-183.
181. Wretman C, Lionikas A, Widegren U, Lannergren J, Westerblad H, Henriksson J. Effects of concentric and eccentric contractions on phosphorylation of MAPK(erk1/2) and MAPK(p38) in isolated rat skeletal muscle. *J Physiol.* 2001;535:155-164.
182. Young A, Stokes M, Crowe M. Size and strength of the quadriceps muscles of old and young women. *Eur J Clin Invest.* 1984;14:282-287.
183. Zamora AJ, Marini JF. Tendon and myo-tendinous junction in an overloaded skeletal muscle of the rat. *Anat Embryol (Berl).* 1988;179:89-96.
184. Zarins B, Ciullo JV. Acute muscle and tendon injuries in athletes. *Clin Sports Med.* 1983;2:167-182.