The Effect of Aging on Skeletal-Muscle Recovery From Exercise: Possible Implications for Aging Athletes

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Recovery from exercise is integral to the physical training process. There is a perception among older athletes that aging negatively affects the recovery process. Plausible arguments for an impaired recovery with aging are a greater susceptibility of older muscle to exercise-induced skeletal-muscle damage and a slower repair and adaptation response. Differences in the physical activity level of the research participants are rarely considered, however. This makes it difficult to differentiate the respective roles of declining physical activity and aging on the recovery process. Furthermore, the type of exercise used to induce damage and monitor recovery is often not indicative of a normal training stimulus for athletes. This review discusses the effects of aging on skeletal-muscle damage and recovery processes and highlights the limitations of many of these studies with respect to older athletes. Future research should use an exercise intervention representative of a normal training stimulus and take the physical activity level of the participants into account.

Keywords: muscle damage, repair, adaptation, habitual exercise

Over 21,000 competitors attended the world masters games in 2005 (International Masters Games Association, 2005). This number continues to grow and emphasizes the burgeoning interest in maintaining a high level of physical performance throughout the life span. Although the health benefits of maintaining regular physical activity with aging are well established, many of these older athletes would also be extremely interested in improving athletic performance. The aging process is accompanied by significant declines in physical functional capacity (Aniansson, Rundgren, & Sperling, 1980) and athletic performance (Balmer, Potter, Bird, & Davison, 2005; Maharam, Bauman, Kalman, Skolnik, & Perle, 1999; Moore, 1975). Among athletes who continue to train into old age there is a common belief that impaired recovery contributes to these changes in athletic performance (Reaburn, 2004). Most studies that have investigated the effect of aging on recovery from exercise, however, have used sedentary participants. This is incongruent because recovery from exercise is less important to sedentary individuals than to aging athletes.

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The physical training process aims to improve physical performance, and recovery is integral to this process. Consequently, an impaired rate of recovery in older athletes would be detrimental to the training process. The rate of recovery from exercise and how this might be related to declines in physical performance in an older athletic population, however, has been inadequately described. The purpose of this literature review is to explore the scientific evidence for an impaired recovery process in the skeletal muscle of aging athletes. Aging or older athletes can be defined as those who qualify to compete in veteran and master sporting events, and deterioration of sporting performance has been reported to begin at 35 years of age (Bortz & Bortz, 1996). Aging is a relative term and there is no clear delineation in the literature between descriptive terms such as senior, older, or veteran athlete, with these terms often used interchangeably (Burns, 1992). Therefore, in this review the terms aging, older, senior, and veteran athlete are taken to apply to athletes who have reached an age deemed to be after that at which normal peak performance would occur for a particular sport (usually between 30 and 40 years of age). This review of the literature will present arguments for and against greater damage and impaired recovery with aging from studies conducted using animals and humans that range from middle-aged to senescent. Because most human research has investigated participants older than 50 years, this might limit the generalizability of this research to athletes who are 30–50 years old, who might be experiencing some of the effects of aging but quite differently than an 80-year-old.

A virtual lack of any research investigating the recovery of functional performance in well-trained aging athletes presents a unique challenge to any review on this topic. The physical training model, which includes the breakdown and fatigue process (training) followed by the recovery process (rest; Kenttä & Hassmén, 1998; Smith & Norris, 2002), provides an essential reference point from which the mechanisms by which aging might adversely influence recovery and athletic performance can be discussed. Furthermore, because skeletal muscle provides the contractile element that powers sport performance, research that has addressed the effects of aging on the response of skeletal-muscle tissue to physical exercise might also provide insight into age-related differences in recovery. Although the recovery process incorporates both physiological and psychological parameters, this review will be limited to the literature pertaining to the physiology of aging, fatigue, and recovery.

**Fatigue and Recovery**

The main purpose of training is to unbalance the homeostasis of an individual’s functional systems, and the natural consequence of this is some degree of fatigue (Smith & Norris, 2002). Theoretically, this fatigue should lead to adaptations that will prepare the individual for future physical insult and, one hopes, enhance performance. Indeed, the principles of training include overload and recovery (Rushall & Pyke, 1990), and it is essential that a balance between these principles be achieved to provide an optimal training effect without impaired adaptation that could lead to underperformance.

Recovery from different training loads can take from less than 24 hr in response to an acute training session to more than 28 days in response to an excessive training stimulus (Smith & Norris, 2002). Previous research has investigated the recovery kinetics of various performance parameters such as power output after acute training...
loads (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995; Tomlin & Wenger, 2001; Yates, Kearney, Noland, & Felts, 1987), and substantial research has also been conducted investigating the effects of excessive training load with respect to overreaching and overtraining (Gleeson, 2002; Hartmann & Mester, 2000). There has been little research, however, to measure recovery from a standardized training load that replicates a normal acute training stress to determine whether recovery is impaired as a result of aging.

Although we can monitor the physiological responses to training through techniques such as blood samples and muscle biopsies, these measures might not help in predicting performance levels because of the complexity of the biological adaptive processes (Urhausen & Kindermann, 2002). Performance depends on the quality and quantity of the training stimulus and the extent of recovery leading to optimal adaptations. The process of adaptation is the result of the interplay of work and recovery, and a single-factor model representing this relationship (Smith & Norris, 2002) provides a means for demonstrating the impact of potential differences in recovery because of aging on performance at any given time point after an acute bout of exercise (Figure 1). Fatigue and recovery patterns similar to this might be evident for a range of physiological variables and are likely to be influenced by state of training and the intensity and duration of the training stimulus.

There have been several comparisons of recovery from acute exercise between young and older individuals. For example, Klein, Cunningham, Paterson, and Taylor (1988) examined the contractile properties of the triceps surae muscle after electrically evoked muscle fatigue in a group of physically active young (19–32 years) and aging (64–69 years) men. They reported that although the maximum

![Figure 1](image-url)  
**Figure 1** — Theoretical model of the time course of adaptation after an exercise impulse. Bold line denotes normal model, and dashed lines represent proposed models for an aging athlete experiencing greater damage or slowed recovery (adapted from Smith & Norris, 2002).
voluntary contraction and vertical-jump height were reduced similarly in both groups after exercise, the rate of muscle relaxation after an electrically stimulated twitch contraction was significantly reduced in the aging group compared with the young participants for 1 hr after the fatiguing contractions, indicating impairment in functional recovery. This might be because of alterations in metabolic recovery as a result of aging. Smith et al. (1998) investigated the effects of creatine supplementation on quadriceps phosphocreatine recovery rate from knee-extension exercise in healthy young (31 ± 5 years) and older (58 ± 5 years) participants. They found that phosphocreatine recovery rate was initially (before oral creatine supplementation) lower in the older than in the young participants. In contrast, Kutsuzawa, Shioya, Kurita, Haida, and Yamabayashi (2001) reported no difference in phosphocreatine recovery rate between a group of young (28 ± 5 years) and older (61 ± 5 years) healthy sedentary participants after 3 min of handgrip exercise, suggesting that there was no difference between the groups for muscle-energy metabolism. The researchers did report, however, that the recovery rate of oxygenated hemoglobin was reduced in the older compared with the young group, suggesting an impaired oxygen supply to the muscles of the older participants. The reasons for the contrasting findings between the latter two studies is unclear but might indicate different effects of aging on fatigue and recovery processes in upper and lower body musculature.

In general, these investigations have found some evidence of differences between young and aging participants for acute recovery of physiological parameters from fatiguing exercise, suggesting that for a similar exercise stimulus, a longer recovery period might be required before older adults return to baseline levels. These studies have used sedentary or “recreationally active” participants, however, and consequently the applicability of these findings to well-trained older individuals is unclear.

**Damage and Repair**

The two logical arguments for delayed recovery in older athletes are that aging leads to a greater amount of exercise-induced damage or fatigue and that there is an impaired rate of repair or adaptation after an exercise bout (Figure 1). Although athletes demonstrate individual differences in fatigue and recovery (Smith & Norris, 2002), the common belief among athletes and coaches is that greater damage or slowed recovery is more prevalent for older individuals.

Several studies have used skeletal muscle from rats and mice to investigate the effects of age on muscle function and recovery after fatiguing or damaging contraction protocols (Brooks & Faulkner, 1990; Gonzalez & Delbono, 2001; McBride, Gorin, & Carlsen, 1995). Researchers have also undertaken comparisons in muscle damage and fatigue, as well as repair and recovery, for humans of different ages (Allman & Rice, 2001; Clarkson & Dedrick, 1988; Dedrick & Clarkson, 1990). There are conflicting findings from these studies that might be a result of factors such as the fatigue or damage protocol used or the age, gender, and training status of the study participants.
Greater Exercise-Induced Skeletal-Muscle Damage

It has been well documented that the skeletal muscles of aging rodents are more susceptible to contraction-induced damage than muscles of young rodents (Brooks & Faulkner, 1994, 1996; Zerba, Komorowski, & Faulkner, 1990). Few studies, however, have investigated the susceptibility of muscles of older humans to exercise-induced muscle damage. In studies that have used humans, there have been conflicting findings, with some studies reporting significant age-related differences in exercise-induced muscle damage (Manfredi et al., 1991; Ploutz-Snyder, Giamis, Formikell, & Rosenbaum, 2001; Roth et al., 2000), whereas other studies report no differences in muscle damage between young and older participants when assessed by decreases in isometric force (Clarkson & Dedrick, 1988) or via electron microscopy (Roth et al., 1999). In a recent study that attempted to address these incongruous findings, Lavender and Nosaka (2006a) compared indirect markers of muscle damage, such as change in muscle force and range of movement and perceived soreness, after lengthening contractions of the elbow flexors in young (19.4 ± 1.3 years) and older (70.5 ± 4.7 years) men. They reported that, in contrast to their hypothesis, younger men exhibited greater evidence of muscle damage than older men.

It is possible that the effects of gender might complicate research into this area and contribute to these discordant findings (Tiidus, 2002). Investigations into the effect of aging on muscle damage caused by resistance exercise that have used similar training protocols found contrasting results between men and women for muscle damage determined by electron microscopy (Roth et al., 1999, 2000). Roth et al. (1999) reported that the amount of muscle damage was no different for younger (20–30 years) and older men (65–75 years), whereas 65- to 75-year-old women exhibited higher levels of muscle damage than 20- to 30-year-old women (Roth et al., 2000). This latter study, however, is in contrast to the findings of Clarkson and Dedrick (1988), who reported similar responses for indirect markers of muscle damage (soreness, serum creatine kinase) in both college-age (23.6 ± 3.3 years) and older (67.4 ± 5.3 years) women after a bout of lengthening muscle contractions. Roth et al. (2000) were at odds to explain their findings for greater exercise-induced muscle damage in older women, but not older men, and suggested a potential role of estrogen in protecting against muscle damage (Bar, 1990; Bar & Amelink, 1997; Bar, Amelink, Oldenburg, & Blankenstein, 1988). Therefore, it is possible that some of the age-related differences reported for various markers of muscle damage are influenced by factors such as participant gender and hormone levels (Riley, Robinson, Wise, & Price, 1999). The potential effects of estrogen on exercise-induced muscle damage remain controversial and have been highlighted in a recent review (Kendall & Eston, 2002).

An alternative argument for the conflicting findings between studies on the effects of aging on muscle damage involves the methods used to quantify the damage. Oftentimes indirect measures of muscle damage have been used. These include elevated concentration of myoglobin in the blood; increased serum activity of muscle enzymes such as lactate dehydrogenase and creatine kinase (CK); the presence of markers of oxidative damage (malondialdehyde); decrements in...
force, strength, or power; areas of inflammation observed through the use of computer tomography or magnetic resonance imaging; muscle soreness; swelling; and changes in range of motion (Nosaka, Newton, & Sacco, 2002). For example, CK activity in the blood is regularly reported as a reliable indirect marker of exercise-induced muscle damage (Child, Wilkinson, & Fallowfield, 2000; Kasper, Talbot, & Gaines, 2002; Lee et al., 2002). Using light and electron microscopy, however, Manfredi et al. (1991) reported significantly greater exercise-induced muscle damage (90% vs. 5–50% damaged fibers) in older (59–63 years old) than in young (20–30 years old) men, with no differences in plasma CK activity. Although serum CK activity might be a good qualitative indicator of damage to the sarcolemma, the results described by Manfredi et al. reveal that it is not a reliable indicator of the quantity of damage imposed on the contractile apparatus. In a recent review, Warren, Lowe, and Armstrong (1999) concluded that functional measures such as the ability to produce force or torque provided a better indication of muscle damage because of the high variability and measurement bias of methods such as serum activity of muscle enzymes and histology. Range of motion was also suggested as a valuable tool in studies on humans. These measurements have the added benefit of providing athletes with a measure of physical impairment that might be better related to exercise performance.

Another possible cause for the contradictory results in the study of aging and muscle damage might be the training status of the participants. Tiidus (2002) proposed that there are clear age-related differences in the susceptibility of skeletal muscle to exercise-induced muscle damage, supported by the lower number of lengthening contractions required to elicit structural and functional damage in older animals (Zerba et al., 1990). Tiidus also acknowledged, however, that training status could be an important factor in reducing the degree of postexercise damage in aging human muscle. Although several studies on animals have concluded that exercise-induced damage is greater in aging muscle (Brooks & Faulkner, 1990; McBride et al., 1995; Zerba et al.), none of these studies have taken into account training status or activity levels, thereby neglecting to consider that differences in muscle damage might be associated with decreases in habitual physical activity (Galloway & Jokl, 2000).

Clarkson and Dedrick (1988), in their comparison of muscle damage between young and aging women, concluded that the muscle-damage process takes a similar course for young and older women and that the repair process is equally effective, with older muscle demonstrating the same ability to adapt to damage incurred through repeated bouts of exercise as young muscle. The researchers noted, however, that the older participants in their study were very active, regularly participating in sport, exercise, or strenuous work. In support of this finding, Ploutz-Snyder et al. (2001) resistance-trained a group of older women (63 ± 5 years) for 12 weeks and reported that after the training intervention, these women displayed no difference from younger untrained women (23 ± 4 years old) in muscle damage after a series of lengthening contractions as indicated by magnetic resonance imaging and loss of strength. Similarly, it has been recently reported (Fell, Haseler, Gaffney, Reaburn, & Harrison, 2006) that after a 30-min cycling time trial, serum CK concentrations increased in young and veteran endurance athletes matched for training load. There were no differences between the age groups, however, in changes in CK concentration or in muscle function after the exercise bout.
The influence of training status on muscle damage has also been demonstrated in a study that investigated damage from lengthening contractions in mice (Brooks, Opiteck, & Faulkner, 2001). Repeated weeks of preconditioning lengthening contractions in adult (7 months) and aging (22 months) mice resulted in smaller force deficits and less structural damage in the preconditioned muscles than in unconditioned muscles for both adult and older animals. The pooled data from animals in both age groups demonstrated that the force deficit in the conditioned dorsiflexor muscles was less than half that of unconditioned muscles (11.2% ± 4.6% vs. 27.4% ± 5.4%, respectively). In addition, the conditioned muscles displayed a significantly lower proportion of damaged fibers (2%) than the unconditioned muscles (10%). This protective effect has also been demonstrated in rats that have merely undergone passive stretches before being exposed to a lengthening-contraction protocol (Koh, Peterson, Pizza, & Brooks, 2003).

Exercise might cause a preconditioning effect in aging muscle that could attenuate subsequent exercise-induced damage because of the regeneration of muscle fibers that occurs after the exercise stimulus. Devor and Faulkner (1999) demonstrated that after exposure to damaging bupivacaine injections, regenerated muscle fibers were equally protected from damage induced by lengthening contractions in both young and older rats. Consequently, the role of training in providing a protective stimulus for skeletal muscle through continual remodeling might be substantial and cannot be discounted when investigating the influence of age on skeletal-muscle damage and repair.

Muscle damage caused by oxidative stress has also been found to be reduced in older animals as a result of regular training (Rosa et al., 2005). Rosa et al. examined the effect of lifelong aerobic training on exercise-induced oxidative stress in the skeletal, cardiac, and smooth muscle of mice. The researchers trained a group of mice from the age of 3 to 18 months for 1 hr each day at a treadmill speed that equated to 60% of their 3 months’ VO$_{2\text{max}}$. After an incremental exercise test to exhaustion, the levels of malondialdehyde, a marker of oxidative stress in the lipid membranes, were higher in the untrained older than in the young animals. Malondialdehyde was lower in the trained animals, however, than in either the untrained young or older groups in both skeletal and cardiac muscle, indicating a reduction in oxidative damage incurred during the incremental exercise test in this group. This was the case despite the fact that the trained animals exercised for longer and achieved a greater treadmill velocity than the young animals. This finding supports the proposal that appropriate regular exercise training in aging organisms can prevent cellular damage caused by oxidative stress (Ji, 2001; Powers, Ji, & Leeuwenburgh, 1999).

Age-associated differences in the degree of muscle damage after exercise in well-trained humans have yet to be clearly demonstrated in the literature. Difficulties associated with the accurate quantification of muscle damage (particularly in humans), confusion regarding the effects of gender on muscle-damage indices, the effect of training status (or in the case of aging, detraining status), and the often unrealistic exercise protocols make it difficult to unequivocally conclude that muscle tissue in aging athletes is more susceptible to exercise-induced damage. The factors described here clearly indicate that there is a need for further research in this area, with particular attention paid to the protocol used to elicit muscle damage, the measurement of the damage, and the training status of the participants.
Repair and Adaptation of Skeletal Muscle

The previous section presented arguments for and against increases in exercise-induced damage or fatigue with aging. The second possible cause of a delayed recovery in aging athletes is a longer repair and adaptation time for damaged or fatigued skeletal muscle. If the skeletal muscle of both young and aging athletes experiences similar amounts of exercise-induced damage but the kinetics of recovery are slowed with age, the time period before full performance recovery after exercise would be extended as we age. Moreover, if exercise-induced damage is in fact greater in aging skeletal muscle, this in itself might also contribute to further impair recovery processes such as glycogen repletion (O’Reilly et al., 1987).

Differences in the recovery kinetics of young and aging muscle can be investigated by comparing variables such as the time taken to replenish energy substrates or to repair structural and functional changes induced by exercise. Recovery from metabolic fatigue involves very different recovery mechanisms than recovery from contraction-induced muscle damage does and will consequently be addressed separately. The duration required for training adaptations to occur in response to an exercise stimulus might also be considered an indicator of these recovery and repair processes. The studies that have compared the recovery, repair, and adaptation processes in the skeletal muscle of young and aging animals and humans, however, has failed to clearly demonstrate age-associated differences (Jozsi et al., 2001; Pette & Skorjanc, 2001; Schulte & Yarasheski, 2001). Again, this might be the result of differences in the exercise protocols employed to elicit damage or fatigue of the exercising muscle, influencing recovery and repair processes.

**Glycogen Recovery.** Exercise can deplete muscle glycogen stores, which can negatively affect muscle function and athletic performance (Snyder, 1998). Although there is evidence that aging muscle might have lower resting levels of both high-energy phosphates (Moller, Bergstrom, Furst, & Hellstrom, 1980; Tarnopolsky, 2000) and glycogen (Cartee, 1994), it is likely that in humans this is largely the result of a more sedentary lifestyle, because training has been shown to restore resting levels toward that of young muscle (Cartee; Meredith et al., 1989). Greater levels of depletion or impaired restoration of glycogen after exercise in aging muscle would be detrimental to recovery and subsequent exercise performance.

With respect to glycogen recovery, both of the proposed mechanisms for a delayed recovery in older skeletal muscle—greater damage and slower recovery—might actually work together in a cumulative manner. Exercise-induced muscle damage has been shown to impair postexercise muscle glycogen resynthesis (Costill et al., 1990). Glycogenesis impairment after lengthening contractions has been attributed to transient decreases in GLUT-4 glucose-transport-protein content (Asp, Kristiansen, & Richter, 1995), but the same mechanism was not associated with impaired recovery of muscle glycogen postmarathon (Asp, Rohde, & Richter, 1997). In the latter study GLUT-4 translocation to the sarcolemma might have been affected by the marathon, but this variable was not measured. Any impairment of glucose transport and glycogenogenesis postexercise could contribute to delayed recovery and decrease subsequent exercise capacity if the exercise is performed before complete recovery of glycogen stores (Asp, Daugaard, Kristiansen, Kiens, & Richter, 1998).
Muscle GLUT-4 levels have also been suggested to decrease with aging (Hall, Mazzeo, Podolin, Cartee, & Stanley, 1994). A combination of aging and damaging exercise therefore might imply postexercise glycogenesis to a greater extent than would be seen with either of these factors individually. Cox, Cortright, Dohm, and Houmard (1999) found, however, that even short-term exercise training (7 days) could improve glucose uptake via increased muscle GLUT-4 concentration equally in both young (18–30 years old) and older (50–70 years old) sedentary participants, with no differences between groups either before or after training. This has also been demonstrated in rodents in which GLUT-4 concentrations were shown to be ~50% higher in adult rats (10 months) with access to running wheels than in controls (Gulve, Rodnick, Henriksen, & Holloszy, 1993). Although the same study did not demonstrate the equivalent training-induced adaptations in GLUT-4 levels in an older group of exercised rats (25 months), the lesser adaptation was attributed to a markedly reduced daily training volume in the older animals than in the younger adult animals (<50%). Thus, although it is possible that this finding suggests a reduced adaptability in older age, it might simply reflect a decreased training load (Cartee, 1994).

Musculoskeletal-Tissue Recovery and Repair. The concern for aging athletes is that the time taken for muscle to repair and recover after fatiguing exercise or exercise-induced damage might be longer than for young muscle, slowing and potentially limiting the adaptation response (Figures 1 and 2). Studies on the skeletal muscle of rodents have revealed significantly delayed repair and recovery from both contraction-induced injury and immobilization (Brooks & Faulkner, 1990; McBride et al., 1995; Rader & Faulkner, 2006a, 2006b; Zarzhevsky et al., 1999).

**Figure 2** — Hypothetical training response in young and veteran athletes demonstrating the progressive overreaching response in the veteran athlete because of an impaired rate of recovery from fatigue after training sessions.
Brooks and Faulkner (1990) used 15 min of lengthening contractions to induce similar reductions from the preexercise values for maximum isometric tetanic force (~34%) and fiber number (~80%) in both young (2–3 months) and older (26–27 months) mice at 3 days postexercise. Although the injured muscles of young mice had fully recovered by 28 days postexercise, however, the muscles of the older mice remained incompletely recovered and isometric force was still reduced at 60 days postexercise. More recently Rader and Faulkner compared the recovery rate from 225 electrically stimulated lengthening contractions in the plantar-flexor muscles of adult (4–13 months) and old (26–29 months) male (Rader & Faulkner, 2006b) and female (Rader & Faulkner, 2006a) mice. They reported severe injury to the muscle that was not different between the age groups in the first 3 days after the contraction protocol. Nonetheless, although force deficits remained in both age groups at 1 month postexercise, the young animals had recovered by 2 months, whereas the old mice showed incomplete recovery of isometric force and muscle mass, leading the authors to conclude that the changes in the old animals might be permanent. This delayed recovery from lengthening contractions has also been reported by McBride et al. (1995), who found that the tibialis anterior of aging (32 months) rats took 14 days to return to preexercise functional levels, compared with only 5 days for young-adult (6 months) muscle. These researchers also found that the adult muscle was protected from a second exercise bout performed 14 days after the initial bout (the repeated-bout effect; Byrnes et al., 1985). In the aging muscle, however, there was a significant functional reduction in muscle force-generating capacity after the second exercise bout, although the force decrement was less than after the first bout.

The delayed force recovery of skeletal muscle that has been observed in aging animals is concordant with the findings of several earlier studies in humans (Dedrick & Clarkson, 1990; Klein et al., 1988). Dedrick and Clarkson found that lengthening contractions in college-age women (23.6 ± 3.3 years) elicited the greatest strength loss immediately after the exercise, and thereafter strength demonstrated a progressive return to preexercise values after 3 days. In contrast, older women (67.4 ± 5.3 years) in the same study experienced a continued decline in strength until the second day after exercise, and their strength had not returned to preexercise levels after 5 days of recovery (38% below preexercise values). Muscle function as assessed by reaction time and movement time after damaging exercise was not affected in either the older or young participants, suggesting that skill-related aspects of neuromuscular function might not be affected by age or muscle damage. The type of contraction protocol used by Dedrick and Clarkson, however, might not be representative of normal training-induced fatigue or muscle damage.

Using a fatiguing but not necessarily damaging exercise protocol in humans, Klein et al. (1988) found there were no significant differences between young (19–32 years) and older (64–69 years) adults for loss of force during a fatigue test or the recovery of force during 1 hr of recovery in the triceps surae muscle. These researchers, however, reported that in the older participants there was a decrease in the rate of relaxation of twitch force and an increased half-relaxation time. In addition, functional performance, as measured by maximal vertical-jump height, remained significantly reduced (~9%) after 1 hr of recovery. Hence, although neither loss nor recovery of force resulting from the fatiguing exercise was age dependent, dynamic muscle function was impaired, suggesting that recovery of power takes longer for older participants. This might have a negative impact on functional sport capabilities after fatiguing contractions in the skeletal muscle of aging athletes.
Recovery of maximal contraction force after fatiguing, but not damaging, exercise might be well preserved with age. Allman and Rice (2001) investigated the recovery profiles of force, contractile speed, surface electromyography, muscle activation via twitch interpolation, and muscle compound action potentials in the elbow flexors of young (24 ± 2 years) and older (84 ± 2 years) men after fatiguing isometric contractions. They found that when parameters were normalized to the prefatigue value there were no differences between age groups for fatigue dynamics or recovery of maximal voluntary contraction force.

Only one study has compared recovery in previously trained young and aging participants. McLester et al. (2003) investigated functional recovery 24, 48, 72, and 96 hr after an acute bout of resistance exercise in resistance-trained participants. A significant difference was observed between young (18–30 years) and older (50–65 years) participants in the number of repetitions performed at 72 hr postexercise. These results suggest a more rapid recovery in the younger group, providing evidence for the hypothesized slower recovery model in older athletes (Figure 1).

The research discussed in this section has provided contrasting evidence regarding the effect of age on the rate of muscle recovery and repair from fatiguing exercise. Several methodological differences could account for these conflicting findings. One such difference is the use of exercise protocols that range from severe muscle-lengthening contractions designed to cause widespread damage to contraction protocols aimed at eliciting metabolic fatigue through nondamaging exercise. It might be that recovery from more damaging exercise protocols such as that used by Dedrick and Clarkson (1990) is slower as a result of age, whereas there are no age-related differences in recovery from less damaging metabolic fatigue (Allman & Rice, 2001). Another factor that might limit comparison between studies is the different relative ages of the research participants. Several studies have examined the effects of aging on physiological variables and athletic performance (Galloway, Kadoko, & Jokl, 2002; Pimentel, Gentile, Tanaka, Seals, & Gates, 2003) and found that there might be critical ages to which muscle function can be maintained if undertaking appropriate training protocols but beyond which rapid decrements in function are unavoidable. Finally, the training status of participants in studies that have examined aging and muscle function is also likely to influence the discordant findings reported in the literature. These range from studies that examined participants or animals with sedentary lifestyles to active (but rarely athletic) individuals. Thus, there are clearly difficulties in comparing indices of muscle recovery in very old, inactive rats after severe lengthening contractions with recovery from training-induced fatigue in older humans.

Aging and Adaptive Potential

The adaptive potential of muscle tissue (and other associated systems) limits the gains that can be realized through training. The rate that a tissue can repair from, and prepare for, future exercise-induced stress will govern how frequently and how intensely the tissue can be stressed. If this rate is reduced by aging, the adaptive potential will decrease as a result of reduced capacity for frequent and intense physical training. The repeated-bout effect provides an excellent model that demonstrates the ability of skeletal muscle to adapt to what was a previously damaging exercise protocol. Age-related impairment of the repeated-bout effect is suggestive of slower or inadequate adaptation to a prior exercise bout.
Slower adaptation in response to various conditioning exercises has been reported with aging in rodents (Brooks et al., 2001; McBride et al., 1995). Adaptation (protection from injury) from an initial bout of lengthening muscle contractions was less in the muscle of aging rats (32 months) than in that of young (6 months) rats when exposed to the same exercise 2 weeks later (McBride et al.). In comparison, Clarkson and Dedrick (1988) used two bouts of lengthening contractions 7 days apart to induce muscle damage and investigate the effect of age on repair and adaptation in humans. In contrast to the findings of McBride et al., they found that the damage and repair processes, as measured by force production, range of movement, muscle soreness, and serum CK activity, were similar for aging and young participants. A recent study, however, has challenged these findings. Lavender and Nosaka (2006b) reported that the protective effect conferred by an initial bout of lengthening contractions of the elbow flexors was less for sedentary older men (70.5 ± 4.1 years) than for young men (20.4 ± 2.0 years). It is difficult to reconcile the discordant findings from these studies. Factors such as state of training, the amount of damage from the initial exercise bout, the different gender (and species) of the study participants, the type of exercise protocol employed, and the time between repeated exercise bouts might all contribute to the inconsistent findings.

Although repeated exercise bouts might provide a level of protection for muscle, the adaptation might be slower in older muscle. Brooks et al. (2001) reported that although a period of preconditioning exercise training could provide protection from skeletal-muscle injury induced by lengthening contractions, this protective effect took 6 weeks to develop in older mice compared with only 4 weeks in young mice. Hence, although the preconditioning exercise helped reduce the degree of subsequent exercise-induced muscle damage similarly for both the young and the older animals, the time taken to condition the muscles of the older mice was significantly longer. This finding suggests that older muscle might experience slower but equivalent adaptation to exercise compared with young muscle and also provides evidence for the hypothesized prolonged recovery duration in older athletes because of slower adaptation mechanisms. It cannot be discounted, however, that the longer conditioning period could be a result of reduced habitual activity in older rodents (Mondon, Dolkas, Sims, & Reaven, 1985) before the initial bout of lengthening contractions.

Less damaging treadmill running exercise has also been found to protect against force deficit caused by lengthening contractions equally in the muscles of both young (3 months) and older (23 months) rats (Gosselin, 2000). The researchers reported that 10 weeks of treadmill training reduced the loss of isometric force after lengthening contractions in older trained rats compared with older control rats, from 28% to 13%, and that this was comparable to the adaptation reported with training in the young rats (26% to 13%). Similar evidence of adaptation to muscle contraction has been demonstrated by Pette and Skorjanc (2001). They reported that aging rats (23–25 months) demonstrated the same alterations in muscle fiber-type composition, myosin heavy-chain isoform pattern, and enzyme activities as young adult rats (4–5 months) in response to 50 days of chronic (10 hr/day) low-frequency stimulation. Unfortunately, neither of these studies investigated the time course taken for these adaptations to occur in the young and older animals. Nonetheless, these two studies indicate the potential for muscle to adapt to physical training similarly in older and younger animals.
From the research presented, it seems likely that exercise training leads to muscle adaptation and can impart a protective effect on skeletal muscle in the aging. The impact this has on functional recovery, however, is still to be elucidated. The optimization of recovery for a subsequent bout of exercise is important to athletes, because alterations in the rate of recovery can affect subsequent performance and adaptation. Consequently, the effect of aging on recovery from exercise and subsequent performance needs to be considered.

Recovery and Performance

A major goal of the training process regardless of age is to maintain or improve functional performance in the chosen sport. Although the training model aims to elicit progressive overload leading to positive functional adaptations and improved performance, the concern for older athletes is that if recovery processes are impaired there is a greater risk of inadequate recovery, whereby continued training actually results in progressive overreaching. Progressive overreaching involves a gradual decline in functional performance despite maintenance of training load, because of insufficient recovery (Figure 2).

A recent study by Pimental et al. (2003) found that there was no notable decline in training volume, cardiovascular fitness, or athletic performance up to 50 years of age in well-trained runners. Beyond the age of 50, however, all these variables begin to decline rapidly. What cannot be ascertained from this research is whether the decrements in performance and cardiovascular fitness are caused by declines in training volume or vice versa. This raises the question as to whether older athletes voluntarily reduce their training volume because of age-related declines in rate of recovery, or, alternatively, when they attempt to maintain training volumes, their athletic performance declines because of inadequate recovery (progressive overreaching). This conundrum might be further influenced by evidence that mechanisms of perceived effort and pain might be altered with aging (Allman & Rice, 2003; Gibson & Farrell, 2004). A different perceived effort or rating of pain or soreness in response to a similar bout of exercise in an aging athlete might affect the athlete’s performance in a subsequent exercise bout. Allman and Rice (2003) reported that older (84 ± 1 year) men had higher ratings of perceived exertion in the early stages of a matched fatigue task than their younger (25 ± 1 year) counterparts. Lavender and Nosaka (2006a) reported greater soreness in young sedentary than in older sedentary men after exercise designed to damage the elbow flexors. In contrast to Lavender and Nosaka, recent research (Fell, unpublished observations) has shown that during normal training, aging athletes report greater changes in perceived muscle soreness, fatigue, and recovery than training-matched young athletes, despite no measured difference in physical fatigue or recovery between the groups. The different findings between the latter two studies might reflect the different muscles used (elbow flexors vs. lower limb muscles), the type of exercise performed, or the training status of the participants. If athletes use these perceptual feedback mechanisms to monitor training load and recovery (Hooper, Mackinnon, Howard, Gordon, & Bachmann, 1995), however, any age-related change in the response of these variables to exercise might influence decisions about training frequency, volume, and intensity.
The evidence reported in this section indicates that the mechanisms that might contribute to impair recovery from exercise have not been adequately demonstrated in well-trained aging athletes. Consequently, the ways in which the perception of recovery might be altered in aging athletes and how this could affect training warrant further investigation.

**Proposed Research Questions**

This review has identified a number of studies that observed performance decrements and increases in indirect indicators of muscle damage after an acute bout of exercise and a potential for delayed recovery or slowed adaptation in aging compared with young participants. Hence, it is plausible that the decrements in performance are a result of increased levels of muscle damage incurred from an acute bout of exercise and/or a reduced rate of repair from incurred damage. It is less certain, however, whether any difference in damage or recovery is the result of reduced training intensity or volume or aging per se. Consequently, any future research investigating the effects of aging on muscle damage and fatigue or repair and recovery needs to tightly control for training status and history.

Several different study designs might be used to better elucidate the role of habitual physical activity in protecting skeletal muscle from the recovery deficits that have been observed with aging. Few studies have examined the effect of an acute bout of accustomed exercise (as opposed to exercise designed to specifically damage the muscle) on skeletal-muscle morphology and metabolism. Therefore, a study using older and young trained and untrained participants who are matched for physical activity is warranted. The use of muscle biopsies before and after a single bout of exercise that is not designed to specifically damage the muscle would allow measurements of variables such as gene expression, mitochondrial function, antioxidant status, and muscle morphology. This approach might help resolve some of the issues surrounding the role of physical activity in recovery in older athletes.

An alternative approach might be to investigate whether the susceptibility to overreaching is increased in an older cohort of well-trained athletes. Overreaching is considered a state of training in which attempts to gain improvements in performance too quickly, by training too often or at too high an intensity, can lead to short-term decrements in performance. Studies examining overreaching deliberately increase training load over a brief period (approximately 4 weeks) to monitor changes in variables such as mood state or performance. These variables are often used as indicators of overreaching and, arguably, overtraining (Halson & Jeukendrup, 2004). If the exercise-induced damage is greater or the recovery process slower in an older cohort of athletes, we might assume that this group would be more likely to demonstrate the signs and symptoms of overreaching than a training-matched younger group.

One difficulty with any study investigating well-trained aging athletes is the potential impact that any period of inactivity that might have occurred during the life span can have on the mechanisms of recovery. It might be challenging to find a cohort of older athletes who have experienced no substantial reductions in training activity at any time throughout their lives. To counter this limitation a study using animals that are trained throughout the lifespan provides a means of controlling for
physical activity levels. The effect of aging on exercise-induced muscle damage and functional recovery can then be genuinely compared without the confounding influence of age-related declines in habitual physical activity.

**Conclusions**

The importance of recovery to the physical training process has been presented with reference to the potential mechanisms by which aging might influence these recovery and repair processes. The main hypothesis presented suggests that aging skeletal muscle experiences greater exercise-induced fatigue or damage and has a slower rate of repair and recovery from this fatigue or damage. This leads to prolonged recovery duration, although it is plausible that this might be partially a result of age-associated reductions in physical activity as much as of any age-specific impairment.

This review of the literature has identified deficiencies with respect to the research relating to recovery from hard physical training and competition in aging athletes. Many of the studies that have compared muscle recovery in young and aging humans or animals have used highly damaging protocols of lengthening contractions that might not be representative of typical physical training- and competition-induced fatigue or damage in athletes. The main limitation of the research investigating the effects of aging on muscle damage and muscle recovery has been the potential confounding influence of decreased habitual activity levels that often occur in conjunction with increasing age (Lowe, Warren, Snow, Thompson, & Thomas, 2004). Training older individuals acutely or throughout the life span results in significant functional benefits and potential protection from exercise-induced muscle damage (Close, Kayani, Vasilaki, & McArdle, 2005). Therefore, any future research into the effect of age on fatigue and recovery should attempt to closely control for the training status of participants and employ exercise models representative of typical athletic training or competition.

**References**


