

RECOVERY FROM TRAINING: A BRIEF REVIEW

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ABSTRACT

Athletes spend a much greater proportion of their time recovering than they do in training. Yet, much attention has been given to training with very little investigation of recovery. The purpose of this review is to stimulate further research into this vital area of training. Recovery can be categorized in three terms: i) immediate recovery between exertions; ii) short-term recovery between repeats (e.g., between resistance sets or interval bouts); and iii) training recovery between workouts. The focus of this review is training recovery. Full training recovery is essential to optimal performance and improvement. This review includes an examination of extant research on recovery and a very brief review of some potential modalities and techniques for hastening recovery and the time course of recovery and responses to some treatments. Measures of recovery and practical considerations are discussed briefly. Much research is needed in this area, but there are obstacles to high quality research. Attention must be given to key issues in research on recovery, especially the individual response to recovery treatments.

KEY WORDS rest, training breaks, fatigue, recovery modalities, overtraining, recovery ergogenics

INTRODUCTION

Training in its simplest form represents acute challenges to the body intended to optimize chronic improvements in physiological capabilities. Research has advanced our knowledge of physiological, biomechanical, and the psychological aspects of physical training and performance. The majority of research has focused on training, although most exercise-induced adaptations take place during recovery. Recovery is one of the least understood and most underresearched constituents of the exercise-adaptation cycle. Even the most dedicated athlete spends much more time in recovery than in active training. We define recovery, from a practical perspective, to mean the ability to meet or exceed performance in

a particular activity. For example, if a person has done a challenging distance running workout, then that person's ability to run a personal best 10-km will be reduced for some period of time. Eventually that runner will be recovered, but certainly for the first 3–4 hours after a workout, no runner expects to perform at their best. This concept of recovery has been used by others (14,24,32).

In general, most coaches and athletes have assumed that increased training was the ultimate prescription for improvement. Endurance sports like swimming and running have, in some cases, carried this to an extreme. It is well accepted that over-load is necessary for improvement, whereas overtraining results in a breakdown at some level, thus impairing, rather than improving, performance. Overtraining is usually thought of strictly in terms of training, yet overtraining might also be expressed as underrecovering. If the recovery rate can be improved, greater training volumes would be possible without incurring the negative sequelae of overtraining. Improved recovery may result in establishment of a performance plateau at a higher level.

QUESTIONS ON RECOVERY

There are many open questions regarding recovery. Is short-term recovery, say, between sets, greatly different from recovery between successive workouts? How do the effects of training impact training recovery? Can recovery between sets, or days, be optimized for better training? How much individual variability is there among athletes in recovery? How does periodization impact recovery? Clearly varying the fatigue in the workout will change the recovery needs. What are the cellular and system (e.g., neural) aspects of recovery? Both recovery on the cellular level and recovery on the system level have to be complete for muscle cells to function in an integrated way. Likewise, different training stresses likely require different durations and possibly modalities for recovery. It seems reasonable to suppose that weight training requires a different type of recovery than distance running. What are the central and peripheral aspects of recovery? How does active exercise compared to passive rest influence recovery? What are some practical means for quantifying recovery? What modalities or techniques are useful in recovery? In fact, there are so many possible avenues of research that this review was limited to a few representative articles in each of the major aspects of recovery.

The purpose of this brief review is to examine the research knowledge regarding training recovery and potential means

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for improving recovery. This review will restrict itself to human studies only. Likewise, it would be overwhelming to try to review each possible aspect of recovery in any detail. Short sections are provided with a cursory review of several potential aspects of recovery only for the purpose of stimulating further thought and research on those topics. The hope is that this review will spur innovative research on the many aspects of recovery, specifically, of recovery between training bouts.

WHAT IS RECOVERY?

Immediate Recovery

A cursory review of the literature will find that the term “recovery” is used in 3 major ways. For purposes of this review, we are proposing 3 terms to encompass the broader field of recovery. The most immediate form of recovery we term, “immediate recovery.” Immediate recovery is the recovery which occurs between rapid, time-proximal finite efforts. For example, a race walker has one leg in immediate recovery between each stride. During the immediate recovery phase, the leg muscles must regenerate ATP and remove byproducts of bioenergetics. The more rapidly that each leg recovers, the faster the walker can complete a given race distance. If we make the race walker stride faster (while maintaining stride length), thereby reducing immediate recovery time, we find that the tolerable exercise duration (and distance covered) is reduced. This is commonly observed in that the higher the exercise intensity, the shorter the tolerable duration. At least one study has used the term “recovery” in this manner (25).

Short-Term Recovery

The next type of recovery, and perhaps the most common use of this term in exercise science, is what we are calling “short-term recovery.” Short-term recovery is the recovery between interval sprints or between weight training sets, for example. The duration of this recovery has been evaluated and various ratios of work-to-rest have been suggested. Several papers have used this meaning for the term recovery (8,10,33).

Short-term recovery of power from multi-bout sprint cycling perhaps parallels resynthesis of creatine phosphate (CP). Creatine supplementation has been reported to work only for recovery intervals of less than 6 minutes (33). This example demonstrates the importance of recovery duration. For longer recovery durations, the presence of increased creatine appears to convey no advantage.

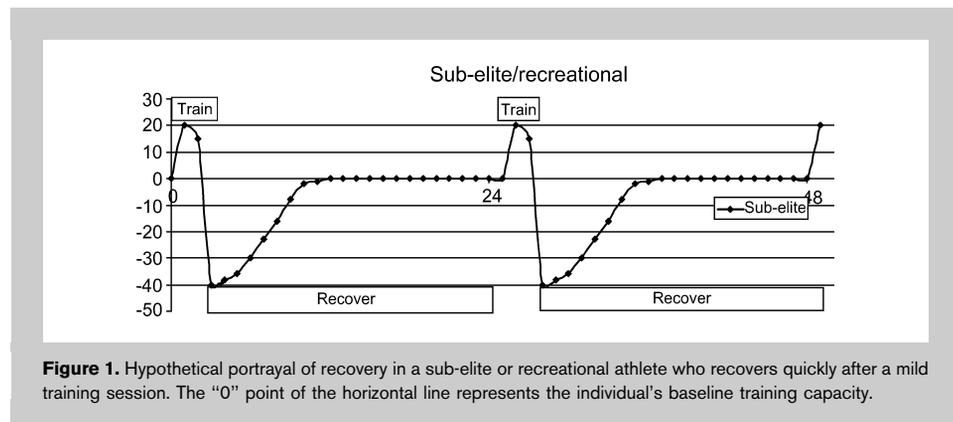
Training Recovery

The third type of recovery, and the one of interest in the present review, is what we are calling

“training recovery.” Training recovery is the recovery between successive work-outs or competitions. For swimmers, runners, weight trainers, football players, and others who sometimes do two-a-day workouts, the interval between training sessions is their recovery. Similarly, for some competitive sports with heats and finals the same day, training recovery would also include the recovery between successive same-day competitions. For most athletes who do one work-out a day, recovery is the period between the end of one work-out and the start of the next. An excellent illustration of training recovery has been published by Gomez et al. (15), regarding recovery from a 10-kilometer all out run.

Although all 3 of these types of recovery may be related in some way, our major interest lies in training recovery. Our belief is that training recovery holds promise for improving athletic performance. Figure 1 is a hypothetical illustration of training recovery in a nonelite athlete, perhaps a typical recreational exerciser. As can be seen, this person trains at a fairly low level and then recovers fully several hours before the following work-out. In Figure 2, we see a hypothetical rendering of what may happen in an elite athlete. In this case recovery is barely complete between workouts due to high volume or intensity. In other words, a complete 24-hour period is totally consumed by either working out or recovering. In our view, this may well be a plausible explanation for the plateau in performance, which is common among elite athletes.

Figure 3 illustrates what may happen hypothetically when the athlete over-trains. In this case, recovery between workouts is definitely incomplete, and successive workouts are begun with a less than optimal physiological (and perhaps psychological) condition. If the athlete has control of her/his own work-out, she/he may very well, consciously or unconsciously, reduce the stress of a following work-out to allow her/his body the opportunity to more fully recover. If an underrecovered athlete continues to try to complete workouts, over some period of time a breakdown occurs due to over-training. Therefore, the occurrence of overtraining is the simultaneous product of both the recovery and the work-out. The ultimate answer to overtraining lies in either reducing



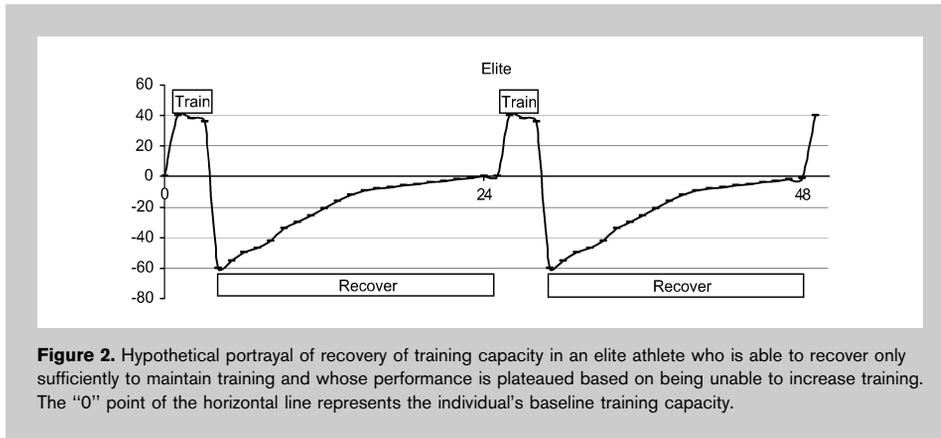


Figure 2. Hypothetical portrayal of recovery of training capacity in an elite athlete who is able to recover only sufficiently to maintain training and whose performance is plateaued based on being unable to increase training. The “0” point of the horizontal line represents the individual’s baseline training capacity.

the workload during training, or perhaps, in improving the quality of recovery.

Finally, Figure 4 is a hypothetical illustration of some effective methods for speeding the rate of recovery. In this situation, the athlete recovers more rapidly from a rigorous workout and therefore has the capacity to perform better. This results in the reestablishment of another performance plateau, but at a higher performance level.

In summary, the chief difference between immediate, short-term, and training recovery lies in the duration of the recovery. In each situation, recovery may be partial or complete. In addition, in each case, speeding recovery could be expected to improve total work capacity and consequently future performance.

As you may have noted, there is an abundant use of the word “hypothetical” in this introduction of recovery. For us, this is what makes this area so interesting. There has been so little research in this area that much of what is known is extrapolated from studies originally targeted at another research question. In the remainder of this review, we will examine the extant research with the goal of identifying potential research lines for expanding our actual, as opposed to hypothetical, knowledge of training recovery.

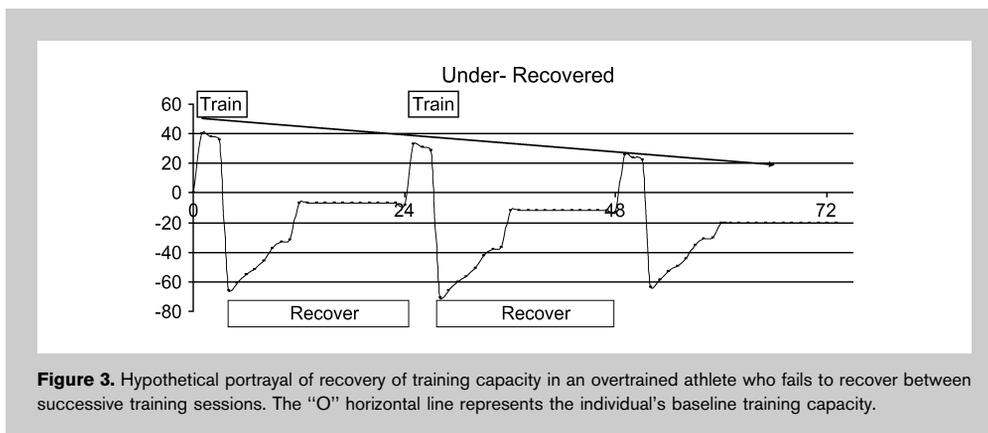


Figure 3. Hypothetical portrayal of recovery of training capacity in an overtrained athlete who fails to recover between successive training sessions. The “0” horizontal line represents the individual’s baseline training capacity.

FATIGUE AND TRAINING RECOVERY

Muscular Damage and Fatigue in Training Recovery

Extreme fatigue as either, or both, a central and peripheral phenomenon, and the muscle tissue damage often associated with a very hard work-out are at the opposite end of the physiological spectrum from full recovery. A good understanding of fatigue is essential to understanding and facilitating recovery. That is, recovery is intended to undo the fatigue/

damage incurred in training. An understanding of acute and chronic exercise-induced physiological adaptations, mechanical, and biochemical changes contributing to fatigue may help in designing optimal recovery modalities. One of the reasons recovery has not been well investigated is the absence of a clear understanding of fatigue (1,19). So, knowledge of fatigue helps to investigate recovery, and knowledge gained in the study of effective recovery may provide insights into the causes of fatigue. The same could be said about muscle damage. Understanding muscle tissue damage and the repair process is essential to mastering full training recovery. Therefore, this review will begin with a brief examination of the several theories explaining fatigue and exercise-induced adaptations and then will examine recovery and aids to muscular recovery.

Although there is little definitive information on fatigue, it has been hypothesized to have one, or both, of 2 main origins. In the central fatigue hypothesis, the muscles are believed capable of greater output but the central nervous system blocks continued extraordinary effort, perhaps as protection from injury (38). In peripheral fatigue, the muscle’s homeostasis has been perturbed, either metabolically through tissue damage, or some other way, to the point

that the muscle is biochemically or mechanically incapable of responding as effectively as it does when rested (21). Abbiss and Laursen (1) have recently published a good general review of both peripheral and central fatigue as it applies to cycling. It seems that their analysis of fatigue could be broadly applied to any sort of endurance training or competition. Clearly, a

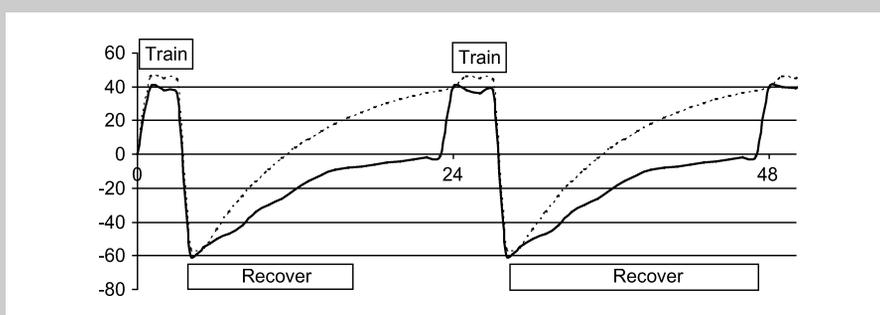


Figure 4. Hypothetical portrayal of recovery of training capacity in an elite or recreational athlete (solid line) and an athlete who is able to facilitate recovery sufficiently (dashed line) to reestablish a new higher performance plateau based on increased training. The “0” point of the horizontal line represents the individual’s baseline training capacity.

review of fatigue is beyond the scope of this paper, but suffice it to say that full training recovery is attempting to overcome all the effects of fatigue, whatever they may be.

Training Recovery from Muscle Tissue Damage

Shlomit et al. (35) studied responses of 31 Israeli defense force troops after 50- and 80-km marches whilst carrying a 35-kg load. Of these, 29 completed 50-km and 16 completed 80-km marches. A summary of the results of this study are shown in Table 1.

Uric acid and protein carbonyl are considered to be plasma antioxidant markers, yet they changed in different directions in response to this exercise stress, and several others, including oxidative stress ascorbic acid, did not change. Most importantly, these investigators suggest that muscle damage was secondary to the chemical levels not primary to them. That is, elevated respiratory rates induced net increases in reactive oxidative species concentrations,

quantify the individual severity of the previous training session.

The Central Fatigue Hypothesis

The central fatigue hypothesis suggests that the brain acts as a protective mechanism to prevent excessive damage to the muscles. Central fatigue, if it is the chief mechanism in training recovery, may be more problematic than peripheral fatigue. In central fatigue, unspecified signals may change brain chemistry such to stop or decrease exercise or work. Manipulating both these signals from the periphery and then the central factors in an attempt to speed training recovery doubles the challenge of achieving training recovery.

Davis et al. (9) promote the idea that central fatigue is driven by serotonin (5-hydroxytryptamine). They hypothesize that carbohydrate or branch chain amino acid ingestion may mitigate 5-hydroxytryptamine increases thereby reducing fatigue. However, they conclude that there is little research support for this hypothesis.

Noakes (29) in a review argues that a central neural governor controls cardiac output by limiting the volume of skeletal muscle that can be activated during maximal exercise while the muscles are hypoxic. His conclusions are based on electromyography of muscular activity as well as cardiovascular function during maximal muscular exercise at varying conditions of hypoxia, normoxia, and hyperoxia.

St. Clair-Gibson et al. (37) make an interesting argument against peripheral disturbance as the sole cause of fatigue.

TABLE 1. Results from Shlomit et al. (36) showing physiological responses to 50- (n = 29) and 80-km (n = 16) marches.

	50-km (%)	80-km (%)
Uric acid	25	37
CPK	1400	940
Aspartate transaminase	380	430
Plasma		
Bilirubin	280	335
Creatine	120	130
Urea	160	160
Glucose	120	130
Protein Carbonyl decr.	75	80

% = % increase from pre to post.

They suggest that if depletion of energy stores were a prime cause of fatigue, and absolute, then cell death and rigor mortis should occur. Likewise, if peripheral metabolite accumulation were a principle cause of fatigue, then that would eliminate any possibility of any increase in exercise intensity late in exercise, such as that shown by Kay et al. (19). Following this line of thinking they suggest that the central governor mechanism is responsible for preventing such extreme and negative sequelae. In contrast, Enoka states (11) that different types of exercise produce different central factors as well as different muscle cell responses. Also, it might be argued that a peripheral safety mechanism would offer the advantages of both specificity and proximity.

Abbiss and Laursen (1) make the very reasonable suggestion, based on foundational work by Lambert, Noakes, and others, that peripheral feedback from the muscles is integrated in a nonlinear fashion with other centrally located senses (e.g., internal clock, memory of past exercise experience, motivation) to produce a central governor to protect the body from injury (19). This seems to agree with Boerio et al. (3), who investigated central and peripheral fatigue in 10 healthy active males. Maximal torque of the plantar flexors significantly decreased by almost 10% following 13 minutes of electrostimulation, and central activation was also reduced as interpreted from twitch interpolation. They interpreted their data to suggest that both central and peripheral fatigue were evoked by a single bout of electrostimulation.

Peripheral Fatigue Hypotheses

Over the years, the concept of fatigue has been explained in many ways according to the understanding of the phenomenon at that time. One of the earliest hypotheses regarding peripheral fatigue was that metabolic products accumulated and interfered with muscle cell function. In 1929, it was noticed that there was a correlation between the appearance of fatigue and accumulation of lactic acid. Since lactic acid accumulation was often associated with a decline in muscle function, it was assumed that the two were related and that lactic acid was possibly causative of fatigue. In 1978, Fabiato and Fabiato (13) supported the idea further by suggesting that acidosis might reduce force production of the contractile proteins. It is clear that metabolic products can directly and indirectly contribute to fatigue in many situations.

In addition to metabolic product accumulation, another obvious explanation for fatigue has been exhaustion of muscle glucose supplies. The evidence that glucose deficiency is essential in some types of fatigue is well-supported in the literature. For example, glycogen depletion is accepted to explain fatigue mainly in special situations such as high-volume muscle training. Glycogen synthesis, after depletion during exercise, occurs in 2 major stages (17). The initial stage is a short, 30–60-minute insulin-independent glycogen synthesis phase, in which glucose transporter proteins' (e.g., GLUT-4) relocation to the muscle cell membrane increases permeability of the membrane to glucose, and thereby

enhances muscle induction of glycogen. This rapid phase is followed by a slow phase in which muscle glycogen is captured by muscle cells in the presence of insulin. Ultimate recovery of low muscle glycogen concentration is affected by many factors including insulin concentration and sensitivity, timing, and availability. Immediate intake of carbohydrate rich foods ($1.0\text{--}1.85\text{g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$) after exercise, and, for up to 5 hours, protein and amino acid supplements (for glycogen and muscle tissue synthesis), and muscle contraction seem to be the major factors enhancing glycogen synthesis (17). These observations suggest that in situations in which glycogen depletion is implicated in fatigue, restoration of glycogen stores is essential to full recovery. Hence, administration of glucose food sources and glucose in combination with amino acids seems a logical strategy for enhancing recovery, which we will discuss later.

Neural Fatigue

Gandevia (14) supports the notion of a central neural fatigue. Gandevia suggests that during sustained muscle contraction, the discharge of motor neurons declines below the level necessary to produce maximal force. Most importantly, Gandevia reports that the brain's motor cortex shows evidence of reduced output during fatigue (14).

Kay et al. (19) studied fatigue in a group of 11 physically active men and women who performed 60 minutes of self-paced cycling in a warm and humid environment. After each 10 minutes of exercise, subjects performed 1-minute all out sprints. Power output fell to 87% and integrated electromyography output fell to 77% by sprint number 5, but increased to 94% and 90%, respectively, for the last sprint. This ability to produce almost the full power output on the last sprint was attributed to possible changes in neuromuscular recruitment, central or peripheral control, or the nature of the integrated electromyography. In this study, they also mention the potential contribution of heat storage to fatigue, since fatigue seems to be exacerbated in hot conditions. As Kay et al. (19) point out, neural processes are temperature sensitive, and muscle metabolic properties may also be influenced. In our lab we have observed this "early fatigue" phenomenon for exercise in the heat. However, when the heat storage resolves, the fatigue remains. Though it is apparent that exercise in high temperatures contributes to fatigue, it is unclear what role temperature might play in recovery. We will review some cryotherapy studies later in the paper.

Taper as Mitigation of Fatigue in Training Recovery

Tapering, defined as the insertion of reduced work combined with increased recovery, represents a form of training recovery that is common to swimming and distance running. The idea that tapering is effective in improving performance argues for the value of adequate training recovery. The premise of tapering is that with additional rest, performance can improve. The key objective in taper is to allow for an optimal level of recovery while avoiding detraining. In one review paper, Mujika and Padilla (26) suggest that taper is

best accomplished by reducing the training volume whilst maintaining the intensity, often referred to as the quality of training. They suggest that performance can improve between 0.5% and 6.0% due to the improved status of the cardiorespiratory, metabolic, hematological, hormonal, neuromuscular, and psychological systems. They show a broad time for effective taper, suggesting that between 4 and 28 days is optimal. They also conclude that a “fast exponential” taper is optimal, wherein a rapid reduction in volume occurs over 4 days, as opposed to a more gradual reduction in volume. At least some swim coaches see tapering not as an improvement in “permanent” performance (i.e., ability to perform the task), but rather that taper only improves immediate performance as compared to a nontapered immediate performance (i.e., for a particular single event or bout).

One might ask, if an elite performer can improve by as much as 6% by tapering, why not taper very often? The reason this is not typically done is that both athletes and coaches fear that athletes lose substantial training during tapering, and thus, tapering too often will hinder overall performance. Obviously this would be difficult to study, and we know of no systematic investigations of repeated taper. Nevertheless, taper presents a common sport paradigm wherein increased recovery improves performance. If some means can be determined to improve recovery between training bouts, then the quality of training should be improved and improved performance should result (see Figure 4).

Time Course of Training Recovery

Recovery of muscle function is chiefly a matter of reversing the major cause of fatigue or damage. Since the causes of fatigue may be many and varied, depending on the nature of the exercise, there are numerous approaches to restoring homeostasis in the muscle cell. The following sections review the time course of recovery from different exercise exposures.

Sayers and Clarkson (32) studied recovery over hours and days in 98 males and 94 females. Fatigue was induced by 50 maximal voluntary eccentric contractions. Maximal voluntary contraction (MVC) ability was not fully recovered (restored to baseline levels) at 132 hours after exercise. In one group, MVC was not recovered for at least 33 days, and in one subject, MVC had not recovered when last tested after 89 days of recovery. This study is also noteworthy because it used the ability to achieve the same performance matching as a marker for recovery. Also, it provides a good example of training recovery. The use of extensive eccentric exercise, however, may not be realistic. Unfortunately, in a study with good sample size, there was no mention of training status.

Gomez et al. (15) studied recovery after a 10-km foot race of 10 experienced distance runners using peak torque, and the total work performed over the last 17 repetitions of a 50-repetition knee flexion protocol. Immediately postrace, $30^{\circ}\cdot\text{sec}^{-1}$ torque, average power, and 17-rep outputs were significantly reduced. They found that only vertical jump

ability and the total work for the last 17 reps of the 50-rep test were not recovered after 48 hours, although the other measures had recovered.

McLester et al. (24) conducted a series of studies in our laboratory to determine training recovery after bouts of resistance training. In trained males we established a 10-rep maximum (10-RM). Subjects performed 3 sets of 10 repetitions of 8 exercises, all to momentary muscular failure. Then in counterbalanced order, we had them try to replicate the same workout after 24, 48, 72, or 96 hours of recovery. As expected, the variability among participants was substantial. None of these participants was able to reproduce their 10-RM at 24 hours. This suggests that they were not fully recovered. After 48 hours of recovery, 40% of our subjects were recovered. After 72 hours of recovery, and after 96 hours of recovery, 80% were recovered. When the sets were increased to 7 sets of each to failure, recovery was delayed as would be expected. When older (50 to 65 years of age) trained men repeated the 3-set protocol, recovery was delayed, compared to younger exercisers. For example, in 70% of the cases, participants were unable to replicate their baseline performance even after 96 hours, suggesting recovery was not complete.

Jones et al. (18) replicated the McLester study and examined training recovery reliability. In that study, 10 college-aged resistance trained males performed 3 sets to volitional failure using a 10RM load for 6 exercises. Recovery was evaluated by the number of repetitions performed following recovery periods of 48, 72, 96, and 120 hours in counterbalanced order. When all 6 exercises were pooled, 80% of participants returned to baseline strength levels after the same recovery duration. However, individual muscle group reliability varied from 20% to 70%. Instability in a participant's performance was at least partly due to rest, nutrition, prior activity, and other factors. When considering summed repetitions for 6 exercises in our first study (24), we found it took 72 hours for 80% of the participants to return to baseline, but in this study we found that by 48 hours 70% of participants had returned to baseline performance. The groups were different and the 6 exercises examined were also slightly different, but the disagreement between studies is difficult to explain other than that a large number of factors influence recovery. It appears that acute recovery also varies within a given person from one training session to another. In a third study in our lab, Church et al. (unpublished) found that 48 hours was sufficient for most lifters to recover.

METHODS FOR POTENTIALLY ENHANCING RECOVERY

Active versus Passive

There have been a few recent studies published on training recovery. Active and passive recovery have been investigated in both short-term and training recovery. In a training recovery study, Bosak et al. (5) compared the effects of active and passive recovery in $n = 12$ trained runners after a 5-km run. In a prior study on training recovery, Bosak et al. (4) had

demonstrated that our sample of recreational runners were unable to fully recover in 24 hours, but were recovered by 72 hours. Consequently we compared active training recovery to passive recovery at 72 hours (5). We found that active and passive recovery yielded similar performances, incidentally providing some evidence of test-retest reliability of this paradigm. But, we noted that variability did occur among the participants and some runners did benefit more from a particular recovery scheme. It seems unwise to suggest, based on one study, that active recovery, on average, confers no benefits. Even if that is true for 5k running, as Enoka has pointed out (11), it may not be true for other forms of training.

Diet, Ergogenics, and Training Recovery

One interesting explanation of muscle damage consequent to training is that reactive oxygen species are the primary cause of muscle cell damage, rather than mechanical trauma. Regardless of whether this hypothesis is true, many investigators are concerned with the impact of free radicals on human physiology. This leads to interest in the role of potential antioxidants on muscle status and in our case, training recovery.

Poor diets may cause dietary deficiencies which could contribute to early fatigue in some athletes. Researchers have also found that endurance athletes suffer more iron deficiency than control (27). Iron is lost in sweat, feces, and urine in endurance athletes at a rate of $1.75 \text{ mg}\cdot\text{d}^{-1}$ and $2.3 \text{ mg}\cdot\text{d}^{-1}$ ($1 \text{ mg}\cdot\text{d}^{-1}$ and $1.4 \text{ mg}\cdot\text{d}^{-1}$ in population reference values) in males and females, respectively (40). The benefits of normal levels of iron are extensive, but the one that most directly related to exercise performance is that iron is a key component of the oxygen-carrying protein, hemoglobin. Oxygen transport capacity of blood is a major determining factor of maximal oxygen uptake ($\dot{V}O_{2\text{max}}$). Although iron supplementation is beneficial for anemic athletes, it is still debatable whether or not supplements can benefit non-anemic athletes (28). Clearly adding iron to the diets of athletes in the presence of the associated colinked nutrients is relatively inexpensive.

Creatine has been studied as an aid to recovery. Branch (7) found it to be effective for short duration (<30 seconds) recovery, but not for swimming or running events lasting over 3 minutes. This is in contrast to the review by Misisic and Kelley (25), who found that it was not effective in enhancing repeated anaerobic performance. Other ergogenics such as ginseng have not proven consistently helpful either (10). This is important because some ergogenics claim their mode of action is in speeding recovery.

Rehydration and Training Recovery

As Maughan and Shirreffs say so well, restoration of body fluids following an intense competition or training bout is a key part of the total training recovery process (23). As many researchers have demonstrated, restoration of fluids necessitates restoration of electrolytes. For this reason, most rehydration experts recommend inclusion of sodium in concentrations of $50 \text{ mmol}\cdot\text{L}^{-1}$ or greater, along with some

potassium in rehydration beverages. Carbohydrates should be included in rehydration beverages to improve palatability and to aid in the immediate restoration of muscle glycogen stores. It is also generally accepted that the volume of fluid replaced must exceed that amount lost during exercise, because the body is not 100% efficient in retaining that fluid. It is generally accepted that inclusion of appropriate concentrations of carbohydrates and reasonably large volumes of fluid will speed gastric emptying.

Massage Therapy

Massage has gained popularity among athletes as a training recovery modality. This may be in part because it feels good, is not prohibited by any sport governing body, and has no known side effects. Despite these advantages there is little to suggest it is effective in speeding training recovery.

Martin et al. (22) studied 10 trained cyclists with 20 minutes of recovery. Elevated blood lactate was induced by 3 Wingate tests with a 2-minute rest between bouts. Massage had no impact compared to passive rest; however, active recovery sped lactic acid clearance down to 41% of that immediately after exercise compared to about 62% of the post exercise lactate for passive rest or massage (i.e., active recovery lowered lactic acid by a third more over 20 minutes). This approach raises the question of the role of lactate clearance in training recovery. Whereas lactate clearance may be useful in short-term or immediate recovery, it probably is not a useful marker for training recovery.

Robertson et al. (31) used a Wingate performance test to measure recovery along with lactic acid clearance in assorted athletes ($n = 9$). After six 30-second Wingates with 30-second recovery, participants received either 20 minutes of massage or control (passive rest). There was no effect on lactate clearance or on performance except that the fatigue index (FI) was better for massage at 30% versus 34%. However, in this study, fatigue index was calculated as a percent change of first 5 seconds and last 5 seconds. Fatigue index can be an uncertain marker because it is somewhat dependent upon pacing, even in such a short test. Likewise, how the resistance is applied can influence the first 5-second average.

Ice massage is a common medical therapy technique for soft tissue injuries. Howatson and Van Someren (16) subjected 9 recreationally trained males to 3 sets of 10 single-arm biceps curls with a 7-second eccentric phase to induce soreness. Ice massage or sham ultrasound was given immediately and at 24, 48, and 72 hours. Plasma CK, 1-RM, and DOMS were checked at pre- and immediately post-, and at 24, 48, and 72 hours after exercise in a random crossover design. Only creatine kinase (CK) at 72 hours was reduced in the ice massage treatment, from $800 \pm 680 \text{ u}\cdot\text{L}^{-1}$ to $197 \pm 56 \text{ u}\cdot\text{L}^{-1}$. This study suggests that under these conditions ice massage does not appear to be an effective training recovery method, but specificity of recovery may again be a necessary caveat.

Weerapong et al. (41) have recently published a comprehensive review of massage, including a section on massage

and recovery. Despite a few studies suggesting there could be a positive effect of massage, there appear to be no well-designed studies that have shown a strong effect of massage on recovery. Weerapong et al. (41) did report that some studies have shown that massage effectively reduced delayed onset muscle soreness, while others have not seen any effect. One good point made in the review is that the potential psychological benefit of massage on recovery should not be discounted.

Analgesics in Training Recovery

Anti-inflammatory analgesics have been used by coaches and athletes to relieve pain and inflammation consequent to hard training. The hypothesis seems to be that the anti-inflammatory effects would minimize edema, and the analgesic effects would allow more motion and quicker return to training, in both cases due to their differing impacts on prostaglandins (30).

Semark et al. (34) tested the impact of prophylactic application of flurbiprofen in 25 rugby and field hockey athletes in a single-blind placebo-controlled experiment. DOMS was induced by 7×10 drop jumps. Thigh girth, lactic acid levels after 30-m spring test, CK, muscle soreness, and sprint performances were measured pre- and 12, 24, 48, and 72 hours postexercise. The analgesic did not induce any significant differences in performance or any other variable and appeared to have no impact on inflammatory processes.

Lanier (20) in a review of nonsteroidal anti-inflammatory drugs (NSAIDs) concluded that NSAIDs were useful in speeding training recovery of muscle function but noted that prophylactic use of NSAIDs may be more effective than therapeutic uses.

Some investigators have reported reduced muscle soreness (20) and creatine kinase activity (30) through the use of non-steroidal anti-inflammatory drugs or analgesics. However, Trappet et al. (39) suggested that ibuprofen and acetaminophen in over-the-counter doses suppressed post-eccentric-exercise protein synthesis. An increase, rather than a reduction, in protein synthesis would seem to be more useful in training recovery.

Cryotherapy and Training Recovery

Eston and Peters (12) studied cold water immersion as a recovery therapy in 15 females in a between-group design. DOMS was induced by 8×5 contractions at 0.58 radians·sec⁻¹ of the elbow flexors. The cryotherapy treatment group immersed their exercise arm in 15°C water for 15 minutes immediately after exercise and 6 more times spaced 12 hours apart. Relaxed elbow angle, and CK activity were lower for the cryotherapy group on days 2 and 3 postexercise, but muscle tenderness, edema, and isometric strength were not different up to 3 days following the exercise.

From our lab, Bosak et al. (6) compared 5km racing performance after 24 hours of training recovery with and without cold water immersion in 12 well-trained runners. Repeated-measure treatments were counterbalanced and

separated by 6–7 days of normal training. Run times for the cold water immersion were not significantly different ($p = 0.09$) from baseline, but the control run times were significantly ($p = 0.03$) slower than baseline, though these differences were not large. The ratings of perceived exertion at the end of the run were lower for cold water immersion than for control. Seven individuals responded negatively to cold water immersion running and 9 individuals responded negatively to control, running slower than baseline. Three individuals responded positively to cold water immersion and 3 to the control by running faster during second day performance.

Cold treatment does seem to have some effect on some aspects of recovery, though its effects on performance varies among individuals.

Combined Treatments

Many techniques and treatments have been tested in all 3 types of recovery in athletes. Research performed in our laboratory (2) assessed the impact on training recovery of concurrent use of ibuprofen, a protein supplement, vitamins C and E, and cryotherapy in 22 competitively trained athletes using a counterbalanced crossover repeated measure design to examine the treatment effect on performance, CK, muscle soreness, and ratings of perceived exertion (RPE). A non-eccentric exercise protocol consisting of three 30-second Wingate tests were performed in AM and PM sessions to mimic two-a-day exercise sessions or heats and finals. The treatment improved recovery of mean power and mean power per unit body weight without significantly impacting the other variables: CK, RPE, and muscle soreness (2).

A second study investigated the gender differences in response to these same simultaneous recovery procedures in 11 male and 11 female participants. The antioxidant effects of estrogen and the lower resting levels of CK in females suggested a potential gender difference in response. Using the same design as in the previous study, similar responses of both genders in performance and pain were found. In the treatment condition RPE increased in females but this may have been attributed to a trend towards a slight increase in females' performance under this condition. The difference in the change in CK levels between genders approached significance ($p = 0.059$).

MARKERS FOR TRAINING RECOVERY

In our studies of recovery we have strongly favored performance measures. The obvious advantage of performance as a marker for recovery is that it is the most important variable. The disadvantage of performance is that a "blunt force" approach does not indicate the underlying physiology. Second, repeated maximal performance efforts may not be practical for competitive athletes during the competitive season. Gomez et al. (15) used performance measures not of the same type as the fatiguing exercise. Fatigue was induced by a 10-km run and recovery was assessed through leg peak

torques, average power output, and work performed at the end of a 50-rep leg flexion protocol.

An alternative to performance markers of recovery are biochemical or muscle-status markers. Many projects, including those from our lab, have used CK as a marker for muscle damage. Reduced muscle damage suggests less training strain and faster recovery. An issue with CK is that it is difficult to measure, and the measure seems unstable. Many athletes have elevated CK levels consequent to normal training. Interrupting training long enough to normalize CK would be even more disruptive, in many cases, than performance testing. Other markers to identify muscle damage include myoglobin, calpain, myosin heavy chain and Soricher et al. (36) recommend skeletal troponin I.

Lanier (20) suggested that strength recovery was one of the best markers of recovery from muscle injury. She also reports that there are gender and age differences, as well as considerable individual variability in CK response to the same exercise bouts, reducing the utility of CK as a marker for muscle injury, and thus recovery status. Magnetic resonance imagery is also suggested as a means for quantifying muscle injury. Of course these observations are useful only if one is open to including muscle injury as a common occurrence in training.

Many of these measurements are not practical for some laboratory investigations and are unlikely to be useful in the near future for repeated field measures in athletes.

ISSUES IN STUDIES OF RECOVERY

Future studies of recovery must utilize trained participants. The issue of recovery may be of interest in untrained participants; however, most untrained individuals are probably limited by immediate factors unrelated to training recovery. Trained participants are different physiologically and psychologically from untrained participants, and thus, results are often not transferable between groups. The fatiguing protocol for studies of training recovery should be specific to the sport in question. Previous protocols intended to induce muscle damage have been chiefly eccentric exercise. Although some athletic endeavors have large eccentric components, many do not. Care must be used in applying the results of eccentric protocols to training recovery studies.

There seems to be some evidence supporting a central governing theory of fatigue. This raises questions concerning training recovery. If therapy is applied to the central governor (as suggested by Davis et al. (9), for example), will that impact recovery? That is, will the central governor overcome any extant disturbance of homeostasis in the muscles themselves, or will it be impossible to reset the central governor before the muscle cells are fully cleaned and fueled? Also, what role would motor neural fatigue play in this paradigm? Fatigue is certainly a major part of training. Coaches may well choose to deliberately reduce recovery in an effort to induce a super-compensation response in their athletes. However, effective super-compensation depends on recovery from a long hard

period of training. But, this can only occur when training recovery is adequate.

Enoka's review (11) supports the notion of specific recovery for specific training. In his review, he supports the central fatigue hypothesis and highlights that variations in intensity, duration and muscle type impacts the role of the various factors responsible for fatigue. Since we all acknowledge the specificity of training, does it not also seem rational to suspect a specificity of recovery? If we take this view, this means that all recovery research must be interpreted in light of the exact nature of the fatigue-inducing training from which we are trying to recover. Clearly, specificity of recovery would make our research challenge much more complex.

One of the key issues in training recovery is the identification of key markers of recovery. Performance capability is an ecologically valid measure of recovery but not always practical. Other markers are useful indicators of the mechanisms of recovery, but are often impractical and improvement in these other markers seems much less useful if performance is not improved.

Because we have so little foundational research in this area, we recommend testing combined treatments in training recovery studies. The chief advantage to testing combined treatments is the economy. Historically, few treatments have been shown to aid recovery. Testing these one by one is very laborious. In contrast, the chief disadvantage to this approach is that one treatment could cancel another one which alone may be efficacious. It would seem that the probability of that would be quite low.

PRACTICAL APPLICATIONS

Recovery from training is one of the most important aspects of improving athletic performance. Effective training recovery strategies have not been fully elucidated, and may prove to be specific to the individual athlete and to the point in the competitive season. Coaches may be wise to experiment with different techniques for their athletes, noting which are most effective with which athletes. Likewise, research investigators are encouraged to note and provide individual as well as group mean responses to training recovery strategies.

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