

# 5

## Metabolic Training Principles and Adaptations



### After studying the chapter, you should be able to:

- Name and apply the training principles for metabolic enhancement.
- Describe and explain the metabolic adaptations that normally occur as a result of a well-designed and carefully followed training program.
- Discuss the influence of age and sex on the metabolic training adaptations.
- Discuss the detraining response that occurs in the metabolic system.

## INTRODUCTION

To provide a training program that meets an individual's metabolic goals, the training principles must be systematically applied. How these principles are applied will determine the extent to which the body uses the aerobic and/or anaerobic systems of energy production. Which energy systems are emphasized will, in turn, determine the training adaptations that occur. Individualized training programs designed to bring about metabolic adaptations may be used to enhance performance in endurance or anaerobic events or health-related fitness.

## APPLICATION OF THE TRAINING PRINCIPLES FOR METABOLIC ENHANCEMENT

The training principles were described generally in Chapter 1. Each training principle can be applied specifically in relation to the metabolic production of energy to support exercise.

### Specificity

In order to be specific and match the demands of the event, a training program must begin with determining the goal. For example, a 50-year-old male who is enrolled in a fitness program and wants to break 60 minutes in a local 10-km race will have a very different training program from a 16-year-old high school student competing in the 400- and 800-m distances.

With the goal established, the relative contributions of the major energy systems can be estimated using a graph such as the one shown in Figure 3.2 in Chapter 3. For the 50-year-old male described above, approximately 98% of the energy for his 60-minute, 10-km run is derived from the  $O_2$  system, with the remaining 2% from the adenosine triphosphate-phosphocreatine (ATP-PC) and lactic acid (LA) systems. These percentages vary little even if an individual's time is considerably slower or even somewhat faster than 60 minutes. Thus, this individual's training regimen should emphasize the  $O_2$  system.

For the high school middle-distance runner, it is a different story. Planning her training program requires knowing her typical times at those distances. In general, she would be expected to be in the 1:00–3:00 minute range for both distances. (The American National 2007 record for a high school female was 0:50.69 for the 400-m run and 2:00.07 for the 800-m run.) Events in this range rely on approximately 60% anaerobic and 40% aerobic metabolism, with a heavier reliance on the ATP-PC and LA anaerobic systems as performance speed increases. Because a faster time is the goal, her training should emphasize the anaerobic systems without neglecting the  $O_2$  system.

In general, only by stressing the primary energy system or systems used in the activity can improvement be expected. The one exception to this rule appears to be the development of at least a minimal level of cardiorespiratory fitness, often termed an *aerobic base* for all sports. An aerobic base does not need to be obtained through long distance running for all athletes. Other programs such as interval work may be more appropriate for sports that are basically anaerobic such as football (Kraemer and Gómez, 2001). Such a base should be achieved in the general preparatory phase (off-season) (see Figure 1.6 in Chapter 1). This base prepares the athletes for more intense and specific training for anaerobic sports and aids in recovery from anaerobic work. The most specific training for metabolic improvement should occur in the specific preparatory phase (preseason) (see Figure 1.6).

For those sports in which performance is not measured in time (such as basketball, football, softball, tennis, and volleyball), the sport's separate components must be analyzed to determine which energy system supports it. For example, the average football play lasts 4–7 seconds and the total action in a 60-minute game (although perhaps spread over 3 hr) may be only 12 minutes. Thus, football training must emphasize the ATP-PC system (the 4–7-sec range), not the  $O_2$  system (the 60-min time). In all cases, drills or circuits should be devised to stress the energy systems most important for a specific sport or positions within a sport.

Specificity also applies to the major muscle groups and exercise modality involved. Most biochemical training adaptations occur only in the muscles that have been trained repeatedly in the way in which they will be used. Thus, a would-be triathlete who emphasizes bicycling and running in his or her program but spends little time on swimming should be more successful (in terms of individual potential) competing in duathlons instead.

### Overload

Overload of the metabolic systems is typically achieved in one of two ways: first, by manipulating time and distance and second, by monitoring LA levels and adjusting work intensity accordingly. Maximal oxygen uptake, although a measure of aerobic power and a means of quantifying training load, is more a cardiovascular than a metabolic variable. Factors contributing to the improvement of  $\dot{V}O_{2\max}$  and the use of % $\dot{V}O_{2\max}$  reserve as an overload technique are therefore primarily discussed in the section on application of the cardiorespiratory training principles (see Chapter 13).

### The Time or Distance Technique

The *time or distance technique* involves performing continuous and/or interval training. As the name implies, continuous training occurs when an individual selects

a distance or a time to be active and continues uninterrupted to the end, typically at a steady pace. For example, a runner who completes an 8-mi training run at a 7:30-min·mi<sup>-1</sup> pace has done a continuous workout. If such a continuous steady-state aerobic training session is maintained for an extended period of time or distance, it is sometimes called a **long slow distance (LSD) workout**. If several periods of increased speed are randomly interspersed in a continuous aerobic workout, the term *fartlek* is used. Thus, a **fartlek workout**, named from the Swedish word meaning “speed play,” combines the aerobic demands of a continuous run with the anaerobic demands of sporadic speed intervals. The distance, pace, and frequency of the speed intervals can vary depending on what the individual wishes to accomplish that day.

**Interval training** is an aerobic and/or anaerobic workout that consists of three elements: a selected work interval (usually a distance), a target time for that distance, and a predetermined recovery or relief period before the next work interval. The target time for any given distance should be based on the time trials or past performance of the individual at that distance. The time period of the work interval determines the energy system that is stressed. A work time of less than 30 seconds stresses the ATP-PC system; one between 30 seconds and 2 minutes stresses the LA system. Anything over 2–5 minutes primarily stresses the O<sub>2</sub> system. The choice of length and type of recovery period also depends on the energy system to be stressed. Its length is typically between 30 seconds and 6 minutes, and the type may be rest-relief (which can include light aerobic activity and flexibility exercises) or work-relief (moderate aerobic activity.) Examples of ATP-PC, LA, and O<sub>2</sub> interval sets are presented in Table 5.1 (Fox and Mathews, 1974). Note that the three sets are not intended to be combined. In addition, other techniques for determining interval times can, of course, result in different target and recovery times for the same distance and ability level.

## ATP-PC System

In this example of the ATP-PC set, the runner is doing 100-m sprints. Each repetition is to be run at 3 seconds slower than her best time. A total of eight repetitions are to be completed with 0:54 of rest recovery (which may involve no or mild activity) between repetitions.

The amount of time required to restore half of the ATP-PC used—that is, the half-life restoration period for ATP-PC—is approximately 20–30 seconds, with full restoration taking at least 2 minutes to possibly 8 minutes (Fox and Mathews, 1974; Harris et al., 1976; Hultman et al., 1967). Thus, this individual will restore over half her ATP-PC.

During the same recovery time, myoglobin O<sub>2</sub> replenishment is also taking place. The amounts replenished and restored are influenced by the individual's activity during the recovery phase, with the greatest restoration occurring with rest during recovery (Dupont et al., 2004).

Because the ATP-PC stores recover so quickly, they can be called upon repeatedly to provide energy. Repeatedly stimulating the ATP-PC system should bring about an increase in its capacity. Any major involvement of the LA system is avoided by keeping the work intervals short so that little lactate accumulation occurs.

## LA System

Stressing the LA system requires work durations of 30 seconds to 2 minutes. In this example, the runner is asked to perform five repetitions of 400m in 1:20, with a work-relief recovery (which should include mild to moderate exercise) of 2:40 between repetitions. LA is produced in excess of clearance amounts during heavy work of this duration, resulting in an accumulation of lactate in the blood. Because lactate has a half-life clearance time of 15–25 minutes, with full clearance taking almost an hour, it is neither practical nor beneficial to allow for clearance of even half the accumulated lactate between repetitions.

**TABLE 5.1** Examples of Time-Distance Interval Training for Runners\*

Energy System	Competitive Distance	Best Time	Training Distance	Training Time	Repetitions	Recovery time	Recovery type
ATP-PC	100 m	0:15	100 m	0:18	8	(1:3) 0:54	Rest
LA	1500 m	5:16	400 m	1:20 <sup>†</sup>	5	(1:2) 2:40	Work
O <sub>2</sub>	1500 m	5:16	1200 m	4:24 <sup>‡</sup>	3	(1:1/2) 2:12	Rest

\*This is not intended to be one workout, although the 100- and 400-m training sets could constitute one workout and the 1200-m repeats another. Each would then total approximately 2 mi of intervals.

<sup>†</sup>Based on 1–4 seconds faster than average 400 m during 1500–1600-m race. ( $1500\text{m} \div 100\text{m} = 15$ ;  $5:16 = 316\text{sec} \div 15 = 0:21 \cdot 100\text{m}^{-1} \times 4 = 1:24 \cdot 400\text{m}^{-1}$ ;  $1:24 - 0:04 = 1:20$ .)

<sup>‡</sup>Based on 1–4 seconds slower than average 400 m during 1500-m–1600-m race. ( $0:21 \cdot 100\text{m}^{-1} \times 12 = 252\text{s} \cdot 1200\text{m}^{-1} = 4:12 + 0:12 = 4:24$ .)



Tolerance to LA is increased by incomplete recovery periods of 1 minute 30 seconds to 3 minutes. This amount of rest allows the replenishment of myoglobin  $O_2$  as well as most of the ATP-PC, thus allowing the high-intensity work in the next work interval to be partially supplied by the ATP-PC energy system before stressing the LA system again (Fox et al., 1969). Of the three overload factors of frequency, intensity, and duration, intensity is most important for improving the capacity of the LA system. Work-relief recovery is typically used at these work times, since active recovery does speed up lactate clearance.

## **$O_2$ System**

Long work bouts that are a portion of the competitive event (e.g., 0.5–1-mi [800–1600m] repeats for a 10-km runner) can be performed to stress the  $O_2$  system. The pace is typically close to average pace during competition and may exceed it. The smaller the proportion of the competitive distance that is performed with each repetition, the faster the pace and the more repetitions performed. The intent is for the intervals to be done aerobically, however. The example in Table 5.1 is for 1200m. Note that the time is longer than simply triple the 400-m time and that the recovery time is proportionally very short. The 2:12 recovery allows for a large portion of ATP-PC restoration before the start of the next repetition. Because this pace is already relatively low-intensity work, a rest or walking recovery is best.

The distance for an interval workout (excluding warm-up and cooldown) should rarely exceed 2–5 mi (3.2–8km) with a frequency of 1–3 d·wk<sup>-1</sup> (Costill, 1986; Rennie, 2007). High-intensity interval training taxes the muscles and joints, and one must be careful to avoid injury or overtraining. (Chapter 22 lists the signs and symptoms of overtraining.) Continuous work at lower intensities allows for greater frequency and longer durations, both of which lead to a greater volume of training. A higher training volume is particularly important to endurance athletes.

**Long Slow Distance (LSD) Workout** A continuous aerobic training session performed at a steady-state pace for an extended time or distance.

**Fartlek Workout** A type of training session, named from the Swedish word meaning “speed play,” that combines the aerobic demands of a continuous run with the anaerobic demands of sporadic speed intervals.

**Interval Training** An aerobic and/or anaerobic workout that consists of three elements: a selected work interval (usually a distance), a target time for that distance, and a predetermined recovery period before the next repetition of the work interval.

## **The Lactate Monitoring Technique**

Assessing blood lactate concentration ( $[La^-]$ ) is the second common technique for monitoring overload. Ideally, this technique involves the direct measurement of blood lactate levels from a given workout. Currently, there is no general agreement about how best to use blood lactate values to design and monitor training programs. Nomenclature also varies greatly. Generally, however, six categories of workouts or training zones are useful (Table 5.2). These training zones are based on the lactate thresholds (LT1 and LT2) obtained during incremental exercise. The zones overlap considerably. The three lower zones (recovery, extensive aerobic, and intensive aerobic) involve predominantly low- to moderate-intensity aerobic activity, whereas the three higher zones (threshold,  $\dot{V}O_{2max}$ , and anaerobic) represent the transition from aerobic to anaerobic energy supply at progressively higher intensities until both aerobic and anaerobic energy productions are maximized (Anderson, 1998; Bourdon, 2000).

Although it is done for elite athletes, direct measurement of  $La^-$  during training for others is not very practical because of the special equipment needed and the cost of taking multiple blood samples. Several studies (Stoudemire et al., 1996; Weltman, 1995) have demonstrated a reasonably stable relationship between blood lactate values and Borg’s rating of perceived exertion (RPE) 6–20 scale. Borg’s RPE scale is described fully in Chapter 13. The range of RPE values corresponding approximately to lactate values for each training zone is presented in Table 5.2.

A better method to individualize the use of RPE is to test the individual in a laboratory and record both RPE and lactate values at each progressive work rate. Figure 5.1 presents an example of such results. This individual reached his LT1 at 220m·min<sup>-1</sup>. At that speed, he reported an RPE of 12. He reached LT2 at a speed of 260m·min<sup>-1</sup> with an RPE of 14. Combining these results with the guidelines from Table 5.2 means that his recovery workouts should be performed at an RPE of 9–12; extensive aerobic workouts at 12 or 13; intensive aerobic at 13–14; threshold workouts at 14; and  $\dot{V}O_{2max}$  at an RPE of at least 15. The test results presented in Figure 5.1 do not represent a maximal test for this individual. Maximal workouts should elicit at least an RPE of 17 for everyone. Thus, an individual can be given a workout and an RPE value and adjust his or her intensity accordingly. An alternative method, when a laboratory testing facility is available, is to determine the relationship between  $[La^-]$  and HR values. Then, HR can be used to estimate the  $[La^-]$  level during training sessions, and the intensity modified accordingly (Dwyer and Bybee, 1983; Gilman and Wells, 1993).

In our example, now reading the rectilinear plot as heart rate, the individual would perform recovery activity between approximately 110 and 140b·min<sup>-1</sup>; extensive

**TABLE 5.2** Training Zones Based on Lactate Thresholds and Values

	Recovery	Extensive Aerobic	Intensive Aerobic	Threshold	$\dot{V}O_{2\max}$	Anaerobic
Relation to LT1 and LT2	<LT1	LT1 to halfway	>LT1 but <LT2	LT2	>LT2	Maximal
Lactate values $\text{mmol} \cdot \text{L}^{-1}$	<2.0	1.0–3.0	1.5–4.0	2.5–5.5	>5.0	>7.0
RPE	<11–12	11–15	12–15	14–17	17–20	17–20
Workout example	Low-intensity aerobic; e.g., 20–30 min continuous	LSD; e.g., 30 min to 2 hr continuous	Tempo runs; e.g., 10–12 sec slower than 10-km race pace	Fartlek; e.g., 1-min bursts	High-intensity intervals; e.g., 6–8 reps 0:30–3:00	Interval repetitions at maximum; e.g., 2–4 reps 0:45–1:30

**Sources:** Based on information from Anderson, O.: The rise and fall of tempo training. *Running Research*. 14(8):1, 4–5 (1998); Bourdon, P.: Blood lactate transition thresholds: Concepts and controversies. In C. J. Gore (ed.), *Physiological Tests for Elite Athletes*. Champaign, IL: Human Kinetics, 50–65 (2000).

aerobic exercise bouts between 110 and 155  $\text{b} \cdot \text{min}^{-1}$ ; intensive aerobic exercise between 120 and 165  $\text{b} \cdot \text{min}^{-1}$ ; threshold workouts between 157 and 173  $\text{b} \cdot \text{min}^{-1}$ ;  $\dot{V}O_{2\max}$  workouts close to 170  $\text{b} \cdot \text{min}^{-1}$ ; and maximal activity at least at 185  $\text{b} \cdot \text{min}^{-1}$ .

Heart rate reflects primarily the functioning of the cardiovascular system, but lactate levels reflect the metabolic energy system. Fox et al. (1988) estimate that if the HR-[La<sup>-</sup>] relationship is not individually determined to ensure that all individuals are working at or above their “anaerobic threshold,” heart rate would have to be

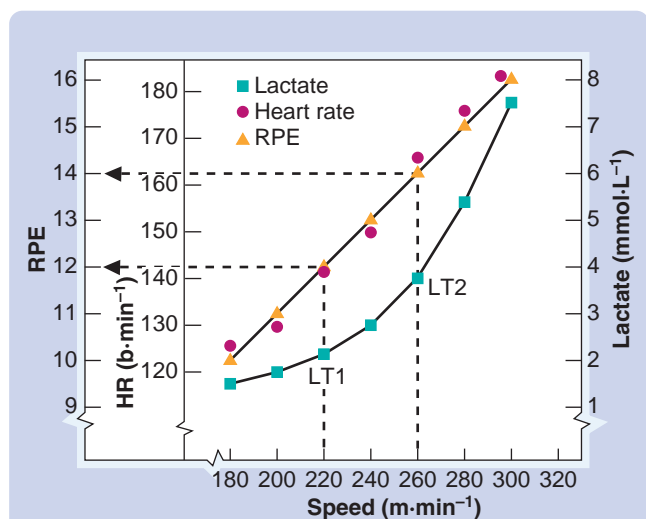
greater than 90% of the maximal heart rate or equal to or greater than 85% of the heart rate reserve in order to use it for anaerobic exercise prescription. Experimental data reported by Weltman (1995) confirm that techniques for exercise prescription involving percentages of heart rate maximum or heart rate reserve do not reflect specific blood lactate concentrations. Thus, unless individually correlated with lactate values, heart rate cannot be used for anaerobic exercise prescriptions.

Regardless of the system used to prescribe an individual’s training session, a mixture of workout types should be used to maximize the possibility for improvement and prevent boredom.

## Rest/Recovery/Adaptation

Adaptation is evident when a given distance or workload can be covered in a faster time with an equal or lower perception of fatigue or exertion and/or in the same time span with less physiological disruption (lower [La<sup>-</sup>] values) and faster recovery. The key to adaptation for energy production in muscles appears to be allowing for sufficient recovery time between hard-intensity workouts. Periodized training programs that alternately stress the desired specific energy system on a hard day and allow it to recover on an easy day lead to optimal adaptation (Bompa, 1999; McCafferty and Horvath, 1977; Weltman et al., 1978). Too many successive hard days working the same muscles and same energy system can lead to a lack of adaptation because of overtraining, and too many successive easy days can lead to a lack of adaptation because of undertraining.

Recently, there has been much interest in the use of modalities to enhance recovery between training sessions or within tournament structures requiring multiple competitions. Overall, the few studies conducted



**FIGURE 5.1.** Heart Rate, Lactate, and Perceived Exertion Responses to Incremental Exercise as a Basis for Exercise Prescription.

LT1 occurred at 220  $\text{m} \cdot \text{min}^{-1}$  and LT2 at 260  $\text{m} \cdot \text{min}^{-1}$  for the individual whose data are plotted. See text for the explanation of how these points are used to prepare an exercise prescription.

so far have not found massage, hyperbaric oxygen therapy (exposure to whole-body pressure >1 atmosphere while breathing 100% oxygen), stretching, or electro-myostimulation (the transmission of electrical impulses through surface electrodes to stimulate motor neurons and induce muscle contraction) advantageous. There appears to be no harm in using water immersion, and this modality may be of benefit if the temperature of the water is thermoneutral to cold. Studies have not supported the use of contrast (alteration of hot and cold) water immersion. Nonsteroidal anti-inflammatory drugs have potential negative health outcomes (cardiovascular, gastrointestinal, and renal) and may negatively affect muscle repair and adaptation to training. Insufficient data are available to evaluate compression garments (stockings, sleeves, tights, and tops) (Barnett, 2006; Wilcock et al., 2006).

## Progression

Once adaptation occurs, the workload should be progressed if further improvement is desired. Progression can involve increasing the distance or workload, decreasing the time, increasing the number of repetitions or sessions, decreasing the length of the relief interval, or changing the frequency of the various types of workouts per week. The key to successful progression is an increase in intensity and total **training volume**. The progression should be gradual. A general rule of thumb is that training volume—the total amount of work done, usually expressed as mileage or load—should not increase more than 10% per week. For example, for an individual currently cycling 60 mi·wk<sup>-1</sup>, the distance should not be increased by more than 6 mi the following week. Steploading, as described in Chapter 1, should be used.

Often in fitness work the challenge is to prevent an individual from doing too much too soon. For example, a 50-year-old man remembers being a high school star athlete and wants to regain that feeling and physique—now! Fitness leaders must gently help such participants be more realistic and should err, if at all, on the side of caution in exercise prescription and progression.

Metabolic adaptation appears to plateau in approximately 10 days to 3 weeks if training is not progressed (Hickson et al., 1981). The ultimate limit may be set by genetics.

**Training Volume** The total amount of work done, usually expressed as mileage or load.

**Training Taper** A reduction in training load before an important competition that is intended to allow the athlete to recover from previous hard training, maintain physiological conditioning, and improve performance.

## Individualization

The first step in individualizing training is to match the sport, event, fitness, or health goal of the participant with the specific mix of energy system demands. The second step is to evaluate the individual. The third step is to develop a periodization sequence for general preparation, specific preparation, competitive, and transition phases. The fourth step is to develop a format: the number of days per week for each type of training or energy system to be stressed. The fifth step is to determine the training load (distance, workload, repetitions, or the like) based on the individual's evaluation and adjusted according to how he or she responds and adapts to the program. Interpreting and adjusting to an individual's response is the art of being a coach or fitness leader.

## Maintenance

Once a specific level of endurance adaptation has been achieved, it can be maintained by the same or a reduced volume of work. How the volume is reduced is critical. When training intensity is maintained, reductions of one third to two thirds in frequency and duration have been shown to maintain aerobic power ( $\dot{V}O_{2\max}$ ), endurance performance (at a given absolute or relative submaximal workload), and lactate accumulation levels at submaximal loads. This maintenance may last for at least several months. One day per week may be sufficient for short periods of time (such as during a 1-wk vacation) if intensity is maintained (Chaloupka and Fox, 1975; Neuffer, 1989; Weltman, 1995). Conversely, a reduction in intensity brings about a decrease in training adaptation.

It also seems to be important that the mode of exercise is consistent with or closely simulates an athlete's activity because many training adaptations are specific to the muscles involved. Thus, cross training—the use of different modalities to reduce localized stress but increase the overall training volume—is likely to be more beneficial for the cardiovascular system than the metabolic system.

The level of maintenance training necessary for the anaerobic energy systems to keep operating at maximal levels is unknown. Sprint performances are known to deteriorate less quickly than endurance performances, however, with a decrease in training (Wilmore and Costill, 1988).

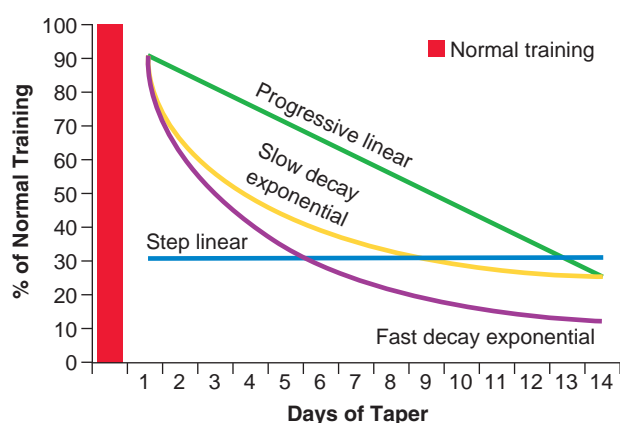
Many fitness participants are primarily in a maintenance mode after the initial several months or the first year of participation. The appropriate level for maintenance should be based on the individual's goals. For athletes, maintenance should occur primarily during the competitive training cycle.

A special kind of maintenance called tapering is often used by athletes in individual sports such as swimming, cycling, and running. A **training taper** is a reduction in training load before an important competition that

is intended to help the athlete recover from previous hard training, maintain physiological conditioning, and improve performance. Athletes often fear that if they taper more than just a few days, their competitive fitness and performance will suffer. However, studies consistently show that if intensity is maintained while training volume is reduced, physiological adaptations are retained and performance either stays the same or improves after a taper (Costill et al., 1985; Houmard et al., 1990; Johns et al., 1992; Mujika and Padilla, 2003; Shepley et al., 1992). Figure 5.2 presents a schematic of the four different possible types of taper. Two of the variations are linear—one a gradual decrease in training load and the other making one large step down in training load and remaining at that level throughout the taper. Two of the variations are exponential, with either a large initial drop (fast decay) or slow initial drop (slow decay). Exponential techniques have been shown to be more effective than linear ones, and fast decay more beneficial than slow decay. Optimal tapering strategies include

1. Using a progressive, fast decay exponential taper.
2. Maintaining training intensity.
3. Reducing training duration by 60–90%.
4. Maintaining training frequency at more than 80% in highly trained athletes and 30–50% for moderately trained individuals.
5. Individualizing the training taper duration between 4 and 28 days.

The art of tapering involves balancing the goals of minimizing fatigue but not compromising previously acquired adaptations and fitness level. Done properly, tapering



**FIGURE 5.2.** Representation of Taper Types: Progressive Linear, Step Linear; Slow Decay Exponential, and Fast Decay Exponential.

**Source:** Modified from Mujika, I., & S. Padilla: Scientific bases for precompetition tapering strategies. *Medicine and Science in Sports and Exercise*. 35(7):1182–1187 (2003). Reprinted with permission.

strategies can result in an improved performance of approximately 3% (range 0.5–6.0%) (Mujika and Padilla, 2003).

## Retrogression/Plateau/Reversibility

Coaches and fitness leaders anticipate and react to, rather than apply, the training principles of retrogression, plateau, and reversibility. At one or more times in the process of training, an individual may fail to improve with progression and will either stay at the same level (plateau) or show a performance or physiological decrement (retrogression). When such a pattern of nonimprovement occurs, it is important to check for other signs of overtraining. Changing the training emphasis or including more easy days may be warranted. Remember that reducing training load does not necessarily lead to detraining. Of course, not all plateaus can be explained as overtraining, sometimes there is no explanation.

If an individual ceases training completely, for whatever reason, detraining or reversibility of the achieved adaptations will occur.

## Warm-up and Cooldown

Information about the effects of warm-up on metabolic function is sparse, but several generalizations can be made. An elevated body temperature—and more specifically, an elevated muscle temperature ( $T_m$ )—increases the rate of metabolic processes in the cells. This increase occurs largely because enzyme activity is temperature dependent, exhibiting a steady rise from 0°C to approximately 40°C before plateauing and ultimately declining. At the same time, at elevated temperatures, oxygen is more readily released from the red blood cells and transported into the mitochondria. Therefore, one consequence of an increased  $T_m$  is a greater availability of oxygen to the muscles during work. When more oxygen is available sooner, there is less reliance on anaerobic metabolism, and less lactate accumulates at any given heavy workload. At lighter endurance workloads, a greater utilization of fat for energy is possible earlier in the activity. This early use of fat spares carbohydrate and allows a high-intensity effort to be continued longer. Other metabolic benefits of an increased  $T_m$  include increased glycogenolysis, glycolysis, and ATP-PC degradation (Bishop, 2003). These beneficial metabolic effects of a warm-up appear to occur in children and adolescents as well as adults (Bar-Or, 1983).

As with the other training principles, to be effective, the warm-up needs to match the intensity and duration of the intended activity. In addition, the structure of the warm-up should depend on the participant's fitness level, the environmental conditions, and specific constraints of the situation in which the warm-up will occur. Several guidelines are available for devising a warm-up to achieve these metabolic benefits and possibly improved performance (Bar-Or, 1983; Bishop, 2003; Franks, 1983).



## CLINICALLY RELEVANT

FOCUS ON  
APPLICATION

## The Effect of Warm-up on Metabolism

**T**welve female ballet dancers (age =  $13.7 \pm 10$  yr; HT =  $1.61 \pm 0.05$  m; WT =  $45 \pm 7$  kg; and  $\dot{V}O_{2\max} = 46 \pm 2$  mL  $\cdot$  kg $^{-1}$   $\cdot$  min $^{-1}$ ) at the Italian National Academy of Dance were tested to determine the effects of active warm-up on energy cost and energy source. As in classical dance practice, the warm-up consisted of 3 minutes of free jogging (25–35%  $\dot{V}O_{2\max}$ ), 15 minutes of stretching (10–20%  $\dot{V}O_{2\max}$ ), 2 minutes of *pre barre*, and 2:40 of *plié* dance exercises. The ballet dance exercise consisted of a sequence of 25 *tours piques* on full *pointe* performed on diagonal continuously for 30 seconds to music.

The total energy requirement ( $\dot{V}O_{2\text{ equivalent}}$ ) was determined by adding the amount of measured  $\dot{V}O_2$  during exercise above resting ( $\dot{V}O_{2\text{ aerobic}}$ ) to the  $\dot{V}O_2$  from the fast component of recovery ( $\dot{V}O_{2\text{ alactic}}$ ) and the energy equivalent of peak

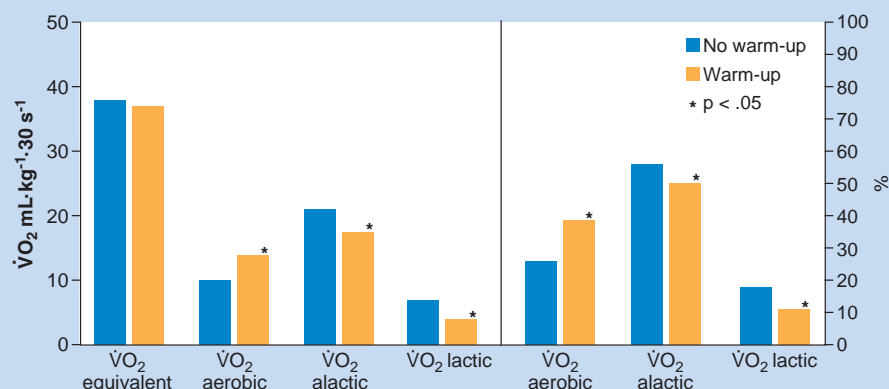
lactate accumulation ( $\dot{V}O_{2\text{ lactic}}$ ).

The metabolic demand was 1.6 times the dancers'  $\dot{V}O_{2\max}$  both with and without warm-up, so the energy cost of the activity (graph below,  $\dot{V}O_2$  equivalent) did not change. The anaerobic systems were taxed more in both conditions. Relatively (right side of the graph), the anaerobic systems ( $\dot{V}O_{2\text{ alactic}} + \dot{V}O_{2\text{ lactic}}$ ) provided 74% of the energy without warm-up and 61% with warm-up.

The decreases in anaerobic energy sources (both alactic and lactic, absolute and relative) following warm-up were significant. Conversely, the aerobic component increased significantly.

**Source:**

Guidetti, L., G. P. Emerenziani, M. C. Gallotta, & C. Baldari: Effect of warm-up on energy cost and energy sources of a ballet dance exercise. *European Journal of Applied Physiology*. 99:275–281 (2007).



1. For a short-term (<10 sec) high-intensity activity, the goal is to increase  $T_m$  but allow time for resynthesis of ATP-PC immediately before the activity. Research suggests a warm-up performed at approximately 40–60%  $\dot{V}O_{2\max}$  (~60–70% HRmax) for 5–10 minutes followed by a 5-minute recovery period is optimal.
2. Explosive tasks (such as long or high jumping) at full speed should be used sparingly during warm-up even if they are to be performed during exercise or competition. However, a brief, task-specific burst of activity may be beneficial, or the action can be patterned at lower levels.
3. For an intermediate or long-term high-intensity activity, the goal is to elevate baseline oxygen consumption without either causing fatigue or imposing a high thermal load. Research suggests a warm-up performed at approximately 60–70%  $\dot{V}O_{2\max}$  (~70–80% HRmax) for 5–10 minutes, followed by  $\leq 5$  minutes recovery for moderately trained individuals.
4. The warm-up for endurance activities generally can occur at a lower intensity (25–30%  $\dot{V}O_{2\max}$  or <35%

HRmax) than for short or intermediate high-intensity activities. The warm-up may be built into an endurance workout if the participant begins at a low intensity and progresses nonstop into higher levels of work.

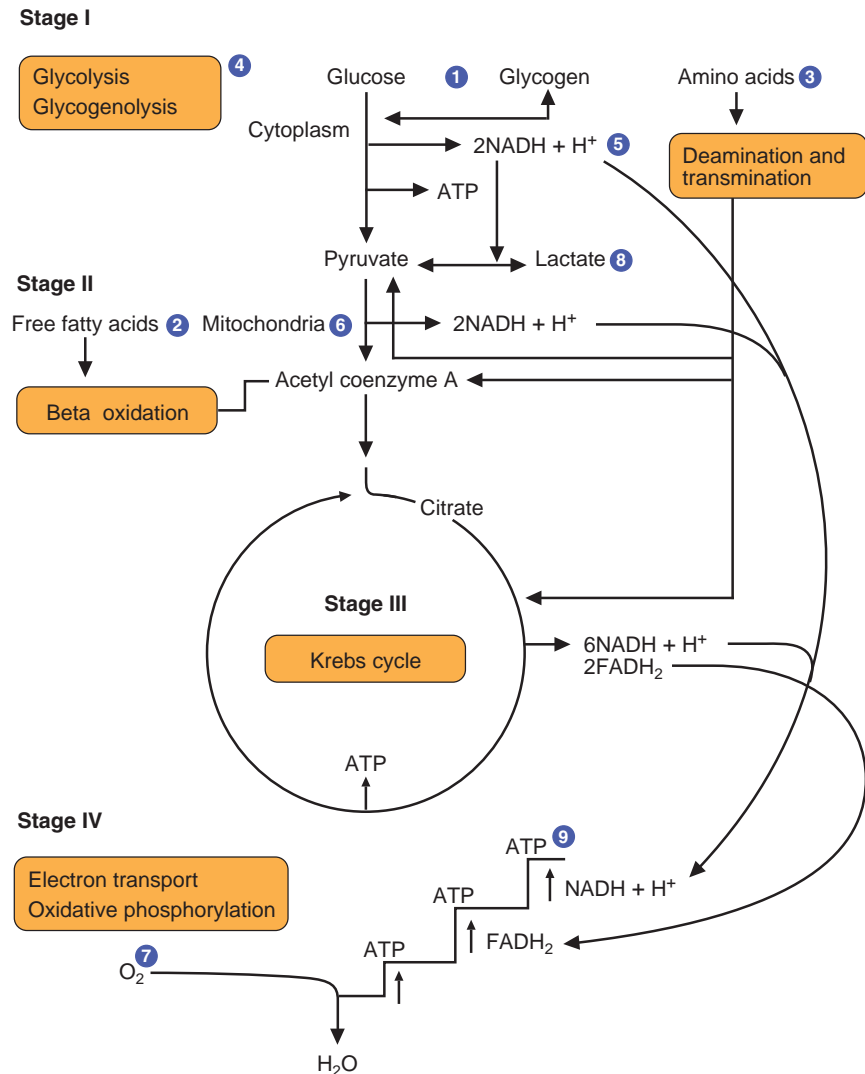
5. Very fit individuals can use longer, more intense warm-ups than less fit individuals. Higher intensity may be needed by well-conditioned athletes to elevate their core temperature. In any case, it is probably best to stay below the lactate threshold and to avoid impacting glycogen stores negatively.
6. An intermittent or interval-type warm-up has been found to be more beneficial for children than a continuous warm-up.

The primary metabolic value of a cooldown lies in the fact that lactate is dissipated faster during an active recovery. As described in Chapter 3, the lactate removal rate is maximized if the cooldown activity is of moderate intensity (a little higher than an individual tends to self-select) and continues for approximately 20 minutes.



**FIGURE 5.3. Metabolic Training Adaptations.**

The circled numbers indicate sites where training changes occur. The boxes indicate processes.



In general, all training principles appear to apply to both sexes and, except where noted, to all ages. At the very least, there is insufficient evidence for modifying any of the general concepts based on age or sex, although individual differences should always be kept in mind.

**CHECK YOUR COMPREHENSION**

The metabolic changes described in the Focus on Application: Clinically Relevant box on the previous page are considered beneficial. Why? Check your answer in Appendix C.

**METABOLIC ADAPTATIONS TO EXERCISE TRAINING**

When the training principles discussed above are systematically applied and rigorously followed, a number of

adaptations occur relative to the production and utilization of energy. The extent to which adaptations occur depends on the individual's initial fitness level and genetic potential. Figure 5.3 is an expanded version of Figure 2.4 in Chapter 2, showing the metabolic pathways you studied earlier. Numbers have been inserted on the graph following the names of some factors to indicate sites where these adaptations occur. The following discussion will follow that numerical sequence. Refer to Figure 5.3 as you read.

**Substrate or Fuel Supply**  
**Regulatory Hormones**

Primary among the metabolic adaptations to a training program are changes that occur in the hormones responsible for the regulation of metabolism (see Chapters 2 and 21, Figure 2.17, and Table 2.3). Although little is known about the impact of training

on the hypothalamic-releasing factors and adrenocorticotrophic hormone, a definite pattern occurs with the five hormones directly involved in carbohydrate, fat, and protein substrate regulation. That pattern is one of a blunted response in which the amount of hormone secreted during submaximal aerobic activity is reduced. This pattern occurs whether the load is absolute or relative and in both the fast-responding and slow-responding hormones. Thus, the rise in epinephrine and norepinephrine is less in the trained state. As a result, the rise in glucagon (stimulated by epinephrine) is lower and there is less suppression of insulin (caused by norepinephrine). Similarly, the rise in growth hormone and cortisol is less during submaximal exercise in trained individuals than in untrained individuals (Galbo, 1983; Talanian et al., 2007). Because of these smaller disruptions at submaximal levels, more work can be done before maximum is reached.

## Carbohydrate (1)

The rate-limiting step for glucose utilization in muscles is glucose transport, and glucose transport is primarily a function of GLUT-4 transporters. Exercise training increases the GLUT-4 number and concentration in skeletal muscle, especially slow twitch oxidative fibers (Daugaard et al., 2000; Sato et al., 1996; Seki et al., 2006). This results in a greater uptake of glucose under the influence of insulin. Thus, at any resting insulin level, the whole-body glucose clearance is enhanced. This occurs in both young and older healthy individuals as well as in individuals with non-insulin-dependent diabetes (Dela, 1996). Despite this increase in the number of GLUT-4 transporters, endurance exercise training reduces glucose utilization during both absolute and relative, moderate-intensity submaximal exercise. This occurs because the translocation of GLUT-4 decreases during exercise. As a result, trained muscles take up and utilize less glucose than untrained muscles during moderate exercise. (Mougios, 2006).

Both endurance and sprint training increase muscle and liver glycogen reserves. In addition, at the same absolute submaximal workload (the same rate of oxygen consumption), muscle and liver glycogen depletion occurs at a slower rate in trained individuals than in untrained individuals (Abernethy et al., 1990; Gollnick et al., 1973; Holloszy, 1973; Holloszy and Coyle, 1984; Karlsson et al., 1972). Thus, the trained individual uses less total carbohydrate in his or her fuel mixture. These changes result in lower respiratory exchange ratio (RER) values (Figure 5.4A). Because glycogen is the primary source of fuel for high-intensity work, a larger supply of glycogen used less quickly enables an individual to participate in fairly intense activities at submaximal levels longer before fatigue occurs. On the other hand, sprint training can

also increase the rate of glycogenolysis at higher levels of work, giving the exerciser a fast supply of energy when needed for short bursts of maximal or supramaximal activity.

## Fat (2)

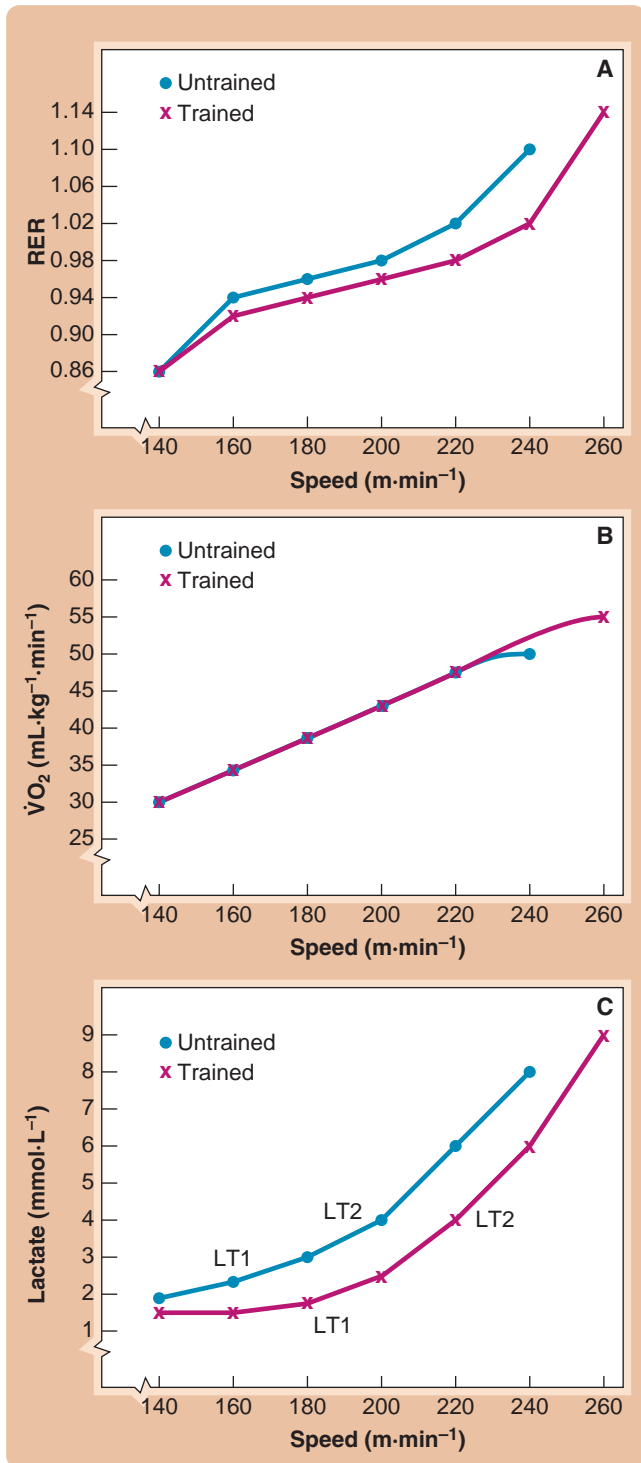
A trained individual can use his or her carbohydrate stores more slowly than an untrained individual because of the changes that occur in fat metabolism. Both trained and untrained individuals have more than adequate stores of fat. However, the rate of free fatty acid oxidation is determined not by the storage amount but by the concentration of free fatty acids in the bloodstream and the capacity of the tissues to oxidize the fat. Training brings about several adaptations in fat metabolism, including the following:


1. increased mobilization or release of free fatty acids from the adipose tissue
2. increased level of plasma free fatty acids during submaximal exercise
3. increase in fat storage adjacent to the mitochondria within the muscles
4. increased capacity to utilize fat at any given plasma concentration

The rise in the capacity of the muscle to oxidize lipids is larger than the rise in its capacity to oxidize glycogen. This in conjunction with the lower plasma glucose uptake, because of the decreased translocation of the GLUT-4 receptors, leads to the larger contribution of fat to energy production. The increased reliance on fat as a fuel is said to have a *glycogen-sparing effect* and is responsible for lowered RER values (Figure 5.4A) at the same absolute and same relative (% $\dot{V}O_{2\max}$ ) work intensities. Because glycogen supplies last longer, fatigue is delayed allowing greater endurance at submaximal work levels. Both endurance and sprint training have glycogen-sparing effects (Abernethy et al., 1990; Gollnick et al., 1973; Holloszy, 1973; Holloszy and Coyle, 1984; Mougios, 2006; Stisen et al., 2006; Talanian et al., 2007).

## Protein (3)

Although proteins are the least important energy substrate, changes do occur as a result of endurance training that enhance their role in metabolism. Adaptations in protein metabolism include an increased ability to utilize the branched chain amino acid leucine and an increased capacity to form alanine and release it from muscle cells. This increased production of alanine is accompanied by decreased levels in the plasma, probably



**FIGURE 5.4.** Metabolic Responses of Endurance Trained Versus Untrained Individuals to Incremental Exercise to Maximum. 

indicating an accelerated removal for gluconeogenesis. In ultraendurance events, this increased gluconeogenesis effect is beneficial for maintaining blood glucose levels (Abernethy et al., 1990; Holloszy and Coyle, 1984; Hood and Terjung, 1990).

## Enzyme Activity

The key to increasing the production of ATP is enzyme activity. Since every step in each metabolic pathway is catalyzed by a separate enzyme, the potential for this training adaptation to influence energy production is great. However, it appears that not all enzymes respond to the same training stimulus nor change to the same extent.

### Glycolytic Enzymes (4)

The results of studies on the activity of the glycolytic enzymes have historically been contradictory, but a pattern is emerging. Glycolysis is involved in both the aerobic and anaerobic production of energy, and it may be that high-intensity training is required for some glycolytic enzymes to adapt while others respond better to endurance training. Strength and sprint training appear to increase glycolytic enzyme activity (Mougios, 2006). These changes are generally less than the activity increases seen in aerobic enzymes, and the functional significance in terms of actual performance remains questionable (Ross and Leveritt, 2001). Three key enzymes have shown significant training changes: glycogen phosphorylase, phosphofructokinase (PFK), and lactate dehydrogenase (LDH).

### Glycogen Phosphorylase

Glycogen phosphorylase catalyzes the breakdown of glycogen stored in the muscle cells for use as fuel in glycolysis. An increase in this enzyme's activity has been found with high-intensity sprint training consisting of either short- (<10sec) or long- (>10sec) sprint intervals (Mougios, 2006; Ross and Leveritt, 2001). The ability to break down glycogen quickly is important in near-maximal, maximal, and supramaximal exercises.

### Phosphofructokinase

PFK is the main rate-limiting enzyme of glycolysis. Results of endurance and sprint training studies are inconsistent but tend to suggest an increase in enzyme activity with adequate levels of training especially consisting of long duration sprint repetitions or a combination of long and short sprint efforts (Ross and Leveritt, 2001). Increased PFK activity leads to a faster and greater quantity of ATP being produced glycolytically.

### Lactate Dehydrogenase

LDH catalyzes the conversion of pyruvate into lactate. It exists in several discrete forms, including a cardiac muscle form (LDH 1) that has a low affinity for pyruvate (thus, making the formation of lactate less likely) and a skeletal muscle form (LDH 5) that has a high affinity for pyruvate (thus, making the formation of LA more likely). Endurance training tends to have two effects on LDH. It lowers the overall activity of LDH, and it causes a shift from the

skeletal muscle to the cardiac muscle form. Thus, lactate is less likely to be produced in the skeletal muscle, and pyruvate is more likely to enter the mitochondria for use as an aerobic fuel. Both of these changes are beneficial to endurance performance (Abernethy et al., 1990; Gollnick and Hermansen, 1973; Holloszy and Coyle, 1984; Sjödin et al., 1982). Strength and sprint training (consisting of both short and long sprint intervals) show opposite effects from endurance training, increasing the overall amount of LDH and favoring the LDH skeletal muscle form because of the changes in fast twitch muscle hypertrophy (Mougios, 2006; Ross and Leveritt, 2001).

## Shuttles (5)

The hydrogen ions removed in glycolysis must be transported across the mitochondrial membrane by a shuttle because that membrane is impermeable to  $\text{NADH} + \text{H}^+$ . No training changes have been found in the glycerolphosphate shuttle enzymes that predominate in the skeletal muscle. Conversely, training leads to large increases in the enzymes of the cardiac muscle's malate-aspartate shuttle in both the cytoplasm and the mitochondria, thus increasing shuttle activity. This increase enhances aerobic metabolism in the heart (Holloszy and Coyle, 1984).

## Mitochondrial Enzymes (6)

Changes in the mitochondrial enzymes of beta-oxidation, the Krebs cycle, electron transport, and oxidative phosphorylation are coupled with changes in the mitochondria themselves. Both the size and the number of the mitochondria increase with training. Thus, mitochondria occupy a proportionally larger share of the muscle fiber space. The sarcolemmal mitochondria are affected more than the interfibrillar mitochondria. The stimulus for these increases appears to be a contractile activity itself, rather than any external stimulus such as hormonal changes, since only those muscles directly involved in the exercise training show these changes. For example, runners have an increase in mitochondrial size and number only in the legs, whereas cross-country skiers have mitochondrial increases in both arms and legs.

Within limits, the extent of the augmentation in the mitochondria seems to be a function of the total amount of contractile activity. That is, the more contractions, the greater the change in the mitochondria. It does not seem to matter whether the increase in contractile activity is achieved by completing more contractions per unit of time (speed work) or by keeping the rate of contractions steady but increasing the duration (endurance training). Resistance training does not appear to enhance mitochondria.

With larger mitochondria, more transport sites are available for the movement of pyruvate into the mitochondria. The enzymatic activity per unit of mitochondria

appears to be the same in trained and untrained individuals; however, the greater mitochondrial protein content means an overall greater enzyme activity to utilize the pyruvate that has been transported there. Interestingly, although most mitochondrial enzymes increase in activity, not all do; nor is the rate of change the same for all. The overall effect of the increased enzyme activity and the increased availability of pyruvate is an enhanced capacity to generate ATP by oxidative phosphorylation. This augmented capacity is more important for supplying energy for submaximal exercise than for maximal exercise (Abernethy et al., 1990; Gollnick et al., 1986; Holloszy, 1973; Holloszy and Coyle, 1984; Mougios, 2006; Wibom et al., 1992).

## Oxygen Utilization (7)

### Maximal Oxygen Uptake

Maximal oxygen uptake ( $\dot{V}\text{O}_{2\text{max}}$ ) increases with training (Figure 5.4B). Even though this is a measure of the amount of oxygen utilized at the muscle level,  $\dot{V}\text{O}_{2\text{max}}$  is determined more by the cardiovascular system's ability to deliver oxygen than by the muscle's ability to use it. Evidence for the subsidiary role of muscle in determining  $\dot{V}\text{O}_{2\text{max}}$  includes the fact that individuals can have essentially the same mitochondrial content but very different  $\dot{V}\text{O}_{2\text{max}}$  values. Conversely, individuals with equivalent  $\dot{V}\text{O}_{2\text{max}}$  values can have quite different mitochondrial enzyme levels. Additionally, small training changes can occur in one factor (mitochondrial activity or  $\dot{V}\text{O}_{2\text{max}}$ ) without concomitant changes in the other—although, typically, both will increase. These differences probably explain why some runners are more economical (use less oxygen at a given pace than others do) and others possess a greater aerobic power (have a higher  $\dot{V}\text{O}_{2\text{max}}$ ) (Holloszy and Coyle, 1984).

### Submaximal Oxygen Cost

The oxygen cost ( $\dot{V}\text{O}_2$  in  $\text{mL}\cdot\text{min}^{-1}$  or  $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) of any given absolute submaximal workload is the same before and after the training, assuming that no skill is involved where efficiency would change (Gollnick et al., 1986; Holloszy and Coyle, 1984) (Figure 5.4B). For example, if an individual has a smooth, coordinated front crawl stroke but has not participated in lap swimming, the oxygen cost of covering any given distance at a set pace will remain the same as this person trains. However, in an individual who is just learning the front crawl stroke, the oxygen cost could actually go down. It decreases not because of a change in the oxygen requirements but because extraneous inefficient movements that add to the oxygen cost are eliminated as skill is improved (Daniels et al., 1978; Ekblom, 1968; Gardner et al., 1989).



Running economy depends both on the energy needed to move at a particular speed (external energy) and on the energy used to produce that energy (internal energy). Specifically, internal energy is associated with oxygen delivery (ventilation and heart rate in particular), thermoregulation, and substrate metabolism (remember that it takes more oxygen to utilize fat as a fuel than to burn carbohydrate). Theoretically, internal energy demand can be lowered by decreasing the ventilation and the heart rate costs and by increasing the percentage of carbohydrate utilized. The first two changes do occur typically with training, but the last one does not. Indeed, the trained individual utilizes a higher percentage of fat at any given submaximal load than does an untrained individual. The primary possibility for improving external energy demand is stride length. However, studies have shown that experienced runners freely select the optimal stride length, so additional improvements in training status do little to change the stride length. The influence of training on running economy is controversial. Cross-sectional studies generally have reported no economy advantage in trained runners over untrained individuals. Longitudinal studies using a running training modality have shown mixed results (Beneke and Hütler, 2005). High-intensity interval training has produced improvements in running economy, but the evidence is not strong (Bailey and Pate, 1991; Conley et al., 1981; Sjodin et al., 1982). Recent evidence suggests that running economy may be improved by the addition of resistance training to the normal aerobic endurance regime of runners. Both traditional weight training and plyometric training have been shown to be effective. It has been speculated that a combination of improved running mechanics and neuromuscular function results in a decreased oxygen consumption, but many questions remain unanswered (Jung, 2003; Millet et al., 2002; Saunders et al., 2006; Spurrs et al., 2003; Turner et al., 2003). Decreased efficiency or economy can also occur. If so, it should be interpreted as a symptom of overtraining (Fry et al., 1991).

If an individual increases his or her  $\dot{V}O_{2\max}$  and yet the oxygen cost of any given (absolute) workload remains the same, then the % $\dot{V}O_{2\max}$  at which that individual is doing the given workload will go down. The task will be relatively easier for the individual, and endurance performance will be greatly enhanced. This improvement results from the biochemical adaptations in the muscle rather than changes in oxygen delivery.

The myoglobin concentration in the muscles increases with endurance training in the muscles directly involved in the activity. As a consequence, the rate of oxygen diffusion through the cytoplasm into the mitochondria increases, making more oxygen available quickly.

## Oxygen Deficit and Drift

The oxygen deficit at the onset of activity is smaller, but is not eliminated, in trained individuals. The primary

reason for this reduction is that oxidative phosphorylation is activated sooner because of the greater number of mitochondria that are sensitive to low levels of ADP and  $P_i$ . This result is advantageous to the exerciser because less LA is produced and less creatine phosphate depleted (Holloszy, 1973; Holloszy and Coyle, 1984; Krstrup et al., 2004).

The magnitude of oxygen drift is also less after training. This change may be caused by concomitant reductions in epinephrine, norepinephrine, lactate, and body temperature rise during any given submaximal workload (Casaburi et al., 1987; Hagberg et al., 1978).

## Lactate Accumulation (8)

LA is produced when the hydrogen atoms carried on  $NADH + H^+$  are transferred to pyruvic acid in a reaction catalyzed by lactic dehydrogenase. Lactate accumulates when the rate of production exceeds the rate of clearance. Although it is known that a trained individual accumulates less lactate at the same absolute workload than an untrained individual, whether the rate of lactate production decreases or the rate of clearance increases more with training is under debate.

Factors that lead to a decrease in lactate production following training include:

1. fuel shifts
2. enzyme activity changes
3. blunted neurohormonal responses

Pyruvate is the end product of carbohydrate metabolism (glycolysis). Less carbohydrate is utilized at an absolute submaximal workload after training; therefore, less pyruvate is available for conversion into lactate. At the same time, pyruvate dehydrogenase activity increases following training, causing more pyruvate to be converted to acetyl CoA. LDH enzyme shifts from the skeletal muscle form, which favors LA production, to the cardiac muscle form, which has a lower affinity for pyruvate. In addition, following training, glycolysis is inhibited by several factors, two of which relate to the increased utilization of fat during submaximal exercise. The first is a high concentration of free fatty acid in the cytoplasm; the second is a high level of citrate (the first product in the Krebs cycle). Both factors cause the rate-limiting enzyme PFK to slow down glycolysis and thus decrease the possible production of LA. Finally, a smaller increase in the concentration of epinephrine and norepinephrine has been found at the same absolute and relative workloads in trained individuals. This decreased sympathetic stimulation may also decrease the activation of glycogenolysis and the potential production of LA (Gollnick et al., 1986; Holloszy and Coyle, 1984; Messonnier et al., 2006).

**FOCUS ON  
APPLICATION****Oxygen-free Radicals, Exercise, and Exercise  
Training Adaptations**

**C**ancer, atherosclerosis, cataracts, Alzheimer disease, diabetes, loss of memory, Parkinson disease, and aging may all, in part, be caused by free radical damage (Keith, 1999). What are free radicals? How can they cause so many different problems? What is the link between oxygen-free radicals, exercise, and exercise training adaptations?

Under normal conditions, electrons orbiting in the shells of a molecule are in pairs. If a single electron is added or removed, instability occurs. The resulting structure is called a free radical. Free radicals have a drive to return to a balanced stable state and attempt to do so by taking an electron from, giving an electron to, or sharing an electron with another atom. Reactive oxygen species (ROS) contain free radicals and reactive forms of oxygen. ROS have several positive roles in the body including involvement in the immune system, cellular signaling, enzyme activation, facilitation of glycogen replenishment, and muscle fiber contractile force. However, ROS also have numerous negative effects. If the production of free radicals exceeds that of components (called antioxidants) that suppress them and their harmful effects, oxidative stress occurs. Often, a chain reaction occurs that results in damage to lipids (especially the lipid bilayer of cell membranes), proteins (in enzymes, immune cells, joints, and muscles), DNA (breaking strands or shifting bases, thus influencing the genetic code), and, in excess, decreases muscular contractile force. These changes ultimately can lead minimally to muscular fatigue or more seriously

to the diseases listed earlier (Alessio and Blasi, 1997; Finaud et al., 2006; Jenkins, 1993; Keith, 1999).

ROS can be produced from sources originating outside the body, such as X-rays, UV rays in sunlight, air pollutants (ozone and nitric oxide in car exhaust), cigarette smoke, toxic chemicals (some pesticides), and physical injury (from contact sports or concussions). They may also result from sources within the body, specifically as part of normal immune function or as a normal by-product of the production of energy (Finaud et al., 2006; Keith, 1999).

Acute exercise is involved in several ways. During the aerobic production of ATP, single electrons leak from electron transport in Stage IV in the mitochondria. The principal locations of this continuous electron leak are complexes I and III and involve coenzyme Q. The higher the rate of metabolism (as in moving from rest to submaximal to maximal exercise), the more free radicals are produced. Possibly as much as 4–5% of the oxygen consumed is converted to free radicals. Anaerobic energy production provides an abundance of hydrogen ions that can react with an oxygen-free radical to form a ROS, such as hydrogen peroxide,  $H_2O_2$ . Hypoxia leads to freeing of metals (Fe, Cu, and Mg) that are needed to catalyze free radical production. Exercise-induced hyperthermia may trigger free radical proliferation. Any damage to muscle fibers leads to increased immune response (Finaud et al., 2006).

Despite the increased production of free radicals resulting from

exercise, it is unlikely that exercise results in substantial damages to a normal healthy individual. The body has a number of natural defenses, and antioxidants ingested in food provide additional defenses. Each cell contains a variety of antioxidant scavenger enzymes, predominantly superoxide dismutase, catalase, and glutathione peroxidase. Antioxidant vitamins, minerals, and phytochemicals include vitamin E, vitamin C, beta carotene (precursor of vitamin A), selenium, and flavonoids.

Acute exercise has been shown to selectively enhance the antioxidant enzymes. In addition, most exercise training studies have shown increased antioxidant levels following exercise. This is seen following both aerobic endurance exercise and dynamic resistance exercise. Part of the training adaptation may be due to increases in the cytochromes in electron transport, which reduces electron leakage (Alessio and Blasi, 1997).

All individuals, but especially those doing high-intensity training, should make sure that their diets contain large amounts of antioxidant-rich foods, primarily fruits and vegetables. Prunes, raisins, blueberries, strawberries, oranges, spinach, broccoli, beets, onions, corn, eggplant, nuts, and whole grains are particularly beneficial. Supplementation with antioxidants may be warranted if the dietary intake is inadequate (Finaud, 2006; Jenkins, 1993; Keith, 1999).

**Sources:**

Alessio and Blasi (1997); Finaud et al. (2006); Jenkins (1993); Keith (1999).

Several other changes lead to higher rates of clearance in trained individuals. These can be classified into two main factors:

1. enhanced lactate transport
2. enhanced lactate oxidation

Lactate transport is enhanced by a combination of increased substrate affinity (the ability of the lactate to bind to the transporter), increased intrinsic activity of the enzymes involved, and increased density of the mitochondrial membrane and cell membrane MCT1 lactate transporters (Juel et al., 2004; Thomas et al., 2005). At the same time, mitochondrial size, number, and enzyme concentrations are increased. Taken together, these changes enable muscle cells to increase both the extracellular and intracellular lactate shuttle mechanisms. There is an overall uptake of lactate by muscles, and as a result, more lactate can be oxidized more rapidly during exercise. Concomitantly, blood flow to the liver is enhanced, which aids in overall lactate removal (Bonen, 2000; Brooks, 2000; Brooks et al., 2000; Gladden, 2000; Pilegaard et al., 1994). These adaptations in the rate of lactate clearance are probably greater than the changes in the rate of lactate production although both contribute to the reduction in lactate accumulation (Brooks, 1991; Donovan and Brooks, 1983; Mazzeo et al., 1986). The result is a decreased concentration of lactate in the muscles and blood at the same relative workload ( $\% \dot{V}O_{2\max}$ ) after training.

As a consequence of the change in the ratio of lactate clearance to production after training, a higher workload (both in absolute and relative terms) is required to reach lactate levels in the 2- to 4-mmol·L<sup>-1</sup> range (Figure 5.4C). This means that an individual can exercise at a higher relative intensity for a given period of time and yet delay the onset of fatigue because the lactate thresholds (LT1 and LT2) have been raised (Allen et al., 1985; Henritze et al., 1985; Holloszy and Coyle, 1984; Londeree, 1997; Skinner and Morgan, 1985; Williams et al., 1967; Yoshida et al., 1982).

At maximal aerobic/anaerobic endurance exercise, the level of LA accumulation is higher as a result of training. The higher level probably results from the greater glycogen stores and increased activity of some of the glycolytic enzymes other than LDH (Abernethy et al., 1990; Gollnick et al., 1986). It may also be more a psychological than a physiological adaptation, in that the trained individual is more motivated and can better tolerate the pain caused by LA (Galbo, 1983) when working at a higher absolute load.

Resistance training has been shown to affect lactate response to both weight-lifting exercise and dynamic aerobic exercise. For example, after 10 weeks of strength training (3 sets of 7 exercises at 8–12 repetitions with 60–90 sec of rest between sets, 3 d·wk<sup>-1</sup>), college females

significantly improved their squat 1-RM. When blood lactate values were compared before and after training at the same absolute load (70% and 50% of pretraining 1-RM), there was a significant reduction in blood lactate from 8 to 6 mmol·L<sup>-1</sup>. When the same relative load was compared (70% and 50% of pretraining 1-RM versus 70% and 50% of posttraining 1-RM), there was no significant difference in blood lactate levels (8 mmol·L<sup>-1</sup> versus 7.5 mmol·L<sup>-1</sup>). These results indicate that after training more work could be done before the same accumulation of lactate occurred than before training. Interestingly, the heart rate responses did not vary among the three testing conditions (before, after absolute loads, and after relative loads), but RPE responses paralleled the changes in blood lactate (Reynolds et al., 1997).

In a 12-week study, young adult males trained using a circuit of three sets of 10 exercises, doing 8–10 repetitions with 30 seconds of rest between exercises, 3 d·wk<sup>-1</sup>. As anticipated, the experimental group significantly improved in both 1-RM upper and lower body strength and leg peak torque, while the controls did not. Neither group changed their treadmill  $\dot{V}O_{2\max}$  nor cycle ergometer  $\dot{V}O_{2\text{peak}}$ . However, the experimental subjects cycled 33% longer at 75%  $\dot{V}O_{2\text{peak}}$  after training, and blood lactate concentrations were significantly reduced at all submaximal intensities tested. Lactate threshold (defined as an absolute value of 3.3 mmol·L<sup>-1</sup>) increased by 12% following resistance training. These results support the generalization that a higher intensity of endurance exercise can be accomplished before reaching the same level of blood lactate concentration, whether the training modality is dynamic aerobic endurance or dynamic resistance activity (Marcinik et al., 1991).

## ATP Production, Storage, and Turnover

### ATP-PC (9)

Although exercise training increases the potential for the production of larger quantities of ATP by oxidative phosphorylation, it does not change the efficiency of converting fuel to ATP or ATP to work. Thirty-two actual ATP molecules are still produced from glucose in skeletal muscle, and the potential energy per mole of ATP is still between 7 and 12 kcal (Abernethy et al., 1990; Gollnick et al., 1986; Gollnick and Hermansen, 1973; Holloszy, 1973; Karlsson et al., 1972; Skinner and Morgan, 1985).

However, the amount of ATP and PC stored in the resting muscle is higher in trained than in untrained individuals, especially if muscle mass increases. Whether this amount is large enough to markedly increase anaerobic capacity is questionable. The resting PC/ATP ratio does not differ among sprint-trained runners, endurance-trained runners, and untrained individuals (Johansen and Quistorff, 2003). At the same absolute workload,

**TABLE 5.3** Estimated Maximal Power and Capacity for Untrained (UT) and Trained (TR) Males

System	Power				Capacity			
	$\text{kcal} \cdot \text{min}^{-1}$		$\text{kJ} \cdot \text{min}^{-1}$		$\text{kcal} \cdot \text{min}^{-1}$		$\text{kJ} \cdot \text{min}^{-1}$	
	UT	TR	UT	TR	UT	TR	UT	TR
Phosphagens (ATP-PC)	72	96	300	400	11	13	45	55
Anaerobic glycolysis (LA)	36	60	150	250	48	72	200	300
Aerobic glycolysis plus Krebs cycle plus ETS/OP ( $\text{O}_2$ )	7–19	32–37	30–80	135–155	360–1270	10,770–19,140	1500–5300	45,000–80,000

**Source:** Modified from Bouchard, C., A. W. Taylor, J. A. Simoneau, & S. Dulac: Testing anaerobic power and capacity. In J. D. MacDougall, H. A. Wenger, & H. J. Green (eds.), *Physiological Testing of the Elite Athlete* (1st edition). Hamilton, ON: Canadian Association of Sport Sciences Mutual Press Limited, 61–73 (1982); Bouchard, C., A. W. Taylor, & S. Dulac: Testing maximal anaerobic power and capacity. In J. D. MacDougall, H. W. Wenger, & H. J. Green (eds.), *Physiological Testing of the High-performance Athlete*. (2nd edition). Champaign, IL: Human Kinetics, 175–221 (1991).

there is less depletion of the PC and degradation of ATP levels after training. At the same relative workload, PC depletion and ATP degradation do not change with training. However, the activity of the enzymes responsible for the breakdown of ATP to ADP and the regeneration of ADP and ATP increase. Therefore, the rate of turnover of ATP and PC increases and may be as much as double that of untrained individuals in both sprint- and endurance-trained runners (Johansen and Quistorff, 2003). Taken together, the ATP-PC-LA changes indicate an increased anaerobic power and capacity with sprint type training (Medbø and Burgers, 1990). Values for the ATP-PC, LA, and  $\text{O}_2$  systems are presented in Table 5.3. The values for the untrained were previously presented in Table 3.1 but are now contrasted with trained males. The LA system changes much more with training than the ATP-PC system, but the greatest change is in the  $\text{O}_2$  system (Bouchard et al., 1982, 1991).

### Work Output

Work output—measured as watts or kilocalories per kilogram of body weight on a bicycle test such as the 10 or 30-second Wingate Anaerobic Test and/or a 90-second test—improves with training. This is evidenced by higher scores of athletes than nonathletes and by higher post-training than pretraining scores in all populations. Furthermore, sprint or power type athletes typically have higher anaerobic values and greater adaptations than endurance athletes. Elite sprinters and power athletes score higher than less successful competitors (Bar-Or,

1987; Beld et al., 1989; Horswill et al., 1989; Patton and Dugan, 1987).

Aerobically, a trained individual can continue any given submaximal workload longer than an untrained individual. The trained individual can also accomplish more total work and a higher absolute maximum than an untrained individual. Overall, the trained individual has a metabolic system capable of supporting enhanced performance, both at submaximal and at maximal levels. These changes, summarized in Table 5.4, depend on the type of training used.

## THE INFLUENCE OF AGE AND SEX ON METABOLIC TRAINING ADAPTATIONS

With the exception of  $\dot{V}\text{O}_{2\text{max}}$  (discussed in the cardiovascular-respiratory unit), there is little research data on most of the metabolic variables across the age spectrum. The scattered evidence that is available indicates that training and detraining changes in children, adolescents, and older adults are similar to changes in adults in the 20 to 50 years range. This is especially true when changes are considered relative to baseline values (i.e., as a percentage of change) rather than as absolutes (Adeniran and Toriola, 1988; Bar-Or, 1983; Clarke, 1977; Eriksson, 1972; Gaisl and Wiesspeiner, 1986; Massicotte and MacNab, 1974; Rotstein et al., 1986; Rowland, 1990).

There is also minimal data on metabolic adaptations in females of all ages, again with the exception of  $\dot{V}\text{O}_{2\text{max}}$  (Shepard, 1978; Thlusty, 1969; Wells, 1991).



## FOCUS ON RESEARCH

## Substrate Training Adaptations in Children

**Duncan, G. E., & E. I. Howley:** Metabolic and perceptual responses to short-term cycle training in children. *Pediatric Exercise Science*. 10:110–122 (1998).

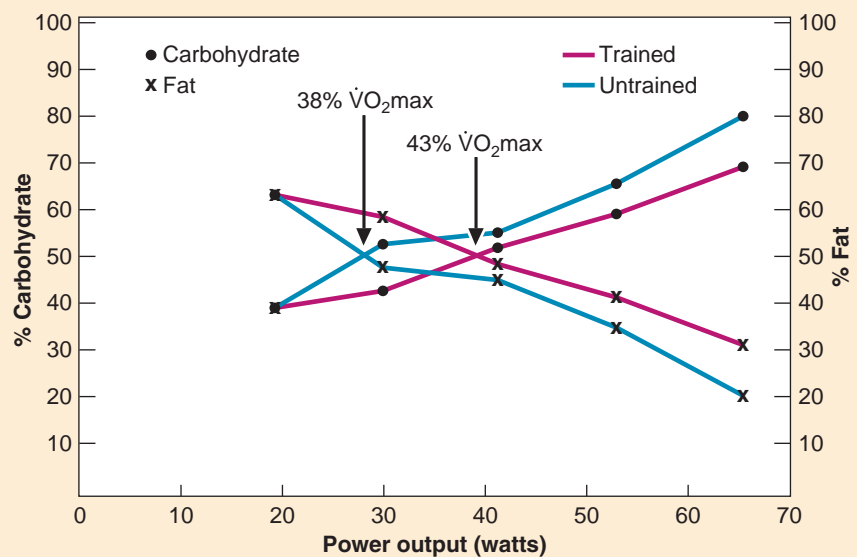
As has been described, it is clear that in adults the primary substrate utilized to fuel exercise depends on the modality, intensity, and duration of the activity as well as the individual's training status. The "crossover" concept states that at some point, as the intensity increases during incremental exercise, the predominant fuel source shifts from fat to carbohydrate. The crossover point is the power output at which this occurs.

This study by Duncan and Howley shows that the same processes occur in children. Twenty-three volunteer boys and girls (ages 7–12 yr) were divided into a training group ( $N = 10$ ) and a control group ( $N = 13$ ). All were tested for  $\dot{V}O_{2\text{peak}}$  on a cycle ergometer and then at five power outputs designed to elicit approximately 35, 45, 55, 65, and

75% of  $\dot{V}O_{2\text{peak}}$ . RER values were determined by open-circuit spirometry for the five-stage submaximal test before and after 4 weeks of training. Training consisted of three 10-minute work bouts separated by 1–2 minutes of rest at roughly 50%  $\dot{V}O_{2\text{peak}}$ , three times per week.

The results, presented in the graph below, clearly show that as the intensity of the submaximal exercise increased, so did the

percentage of carbohydrate utilized as fuel, both before and after the training. Furthermore, the crossover point was delayed or shifted to the right in the training group (data for the control group are not shown). This means that the trained children could work harder while using fat as the predominant fuel. Training apparently has the same carbohydrate-sparing benefit for children as for adults.



Studies have shown the following adaptations in females as a result of appropriate specific training:

1. Fuel utilization shifts in favor of fat.
2. LA levels decrease during submaximal work and increase at maximal effort.
3. Some glycolytic enzyme and most oxidative enzymes increase in activity.
4. Submaximal  $\dot{V}O_{2\text{peak}}$  consumption remains stable or decreases slightly.
5. Anaerobic power and capacity increase (Slade et al., 2002; Wells, 1991; Weltman et al., 1978).

In short, both males and females respond to the same training with the same adaptations. Sex differences in the metabolic variables are not obliterated in equally trained males and females, but both sexes are trainable and probably to the same extent.

## DETRAINING IN THE METABOLIC SYSTEM

*Detraining* is the partial or complete loss of training-induced anatomical, physiological, and performance adaptations in response to an insufficient training stimulus (Mujika and Padilla, 2000a). If an individual ceases training or reduces training beyond that of a planned taper, detraining or reversibility will occur. This reversibility of training adaptations in metabolic potential occurs within days to weeks after training ceases. A reduction in both maximal and submaximal performance ultimately follows. Those metabolic factors that improve the most with training—that is, those involved with aerobic energy production—also show the greatest reversal. Within 3 to 6 weeks after the cessation of training, the individual typically returns to pretraining levels, especially if the training program was of short duration. Individuals with a long and established

**TABLE 5.4 Metabolic Training Adaptations**

1. Fuel Supply
  - a. Carbohydrate
    - (1) ↑ GLUT-4 transporter number and concentration; ↓ exercise induced translocation
    - (2) ↓ glucose utilization
    - (3) ↑ Muscle and liver glycogen reserves
    - (4) ↓ Rate of muscle and liver glycogen depletion at absolute submaximal loads, i.e., glycogen-sparing
    - (5) ↑ Velocity of glycogenolysis at maximal work
  - b. Fat
    - (1) ↑ Mobilization, transportation, and beta-oxidation of free fatty acids
    - (2) ↑ Fat storage adjacent to mitochondria
    - (3) ↑ Utilization of fat as fuel at the same absolute and the same relative workloads
  - c. Protein
    - (1) ↑ Ability to utilize the BCAA leucine as fuel
    - (2) ↑ Gluconeogenesis from alanine
2. Enzyme Activity
  - a. ↑ Selected glycolytic enzyme activity: glycogen phosphorylase and probably phosphofructokinase
  - b. ↓ LDH activity with some conversion from the skeletal muscle to cardiac muscle form with endurance training but ↑ with strength/sprint training
  - c. ↑ Activity of the malate-aspartate shuttle enzymes but not the glycerol-phosphate shuttle enzymes
  - d. ↑ Number and size of mitochondria
  - e. ↑ Activity of most, but not all, of the enzymes of beta-oxidation, the Krebs cycle, electron transport, and oxidative phosphorylation due to greater mitochondrial protein amount
3.  $O_2$  Utilization
  - a. ↑  $\dot{V}O_{2\max}$  with aerobic endurance training but not with dynamic resistance training
  - b. =  $\dot{V}O_{2\max}$  cost at absolute submaximal workload unless neuromuscular skill aspects improve
  - c. ↑ Myoglobin concentration
  - d. ↓ Oxygen deficit
  - e. ↓ Oxygen drift
4. LA Accumulation
  - a. ↑ MCT1 lactate transporters
  - b. ↑ Intracellular and extracellular lactate shuttle activity
  - c. ↓  $La^-$  accumulation at the same absolute workload and % $\dot{V}O_{2\max}$  relative intensity for endurance activity
  - d. ↓  $La^-$  accumulation at the same absolute workload but =  $La^-$  accumulation at the same relative intensity for resistance exercise
  - e. ↑ Workload to achieve lactate thresholds
  - f. ↑  $[La^-]$  at maximum
5. ATP Productions, Storage, and Turnover
  - a. = ATP from gram of precursor fuel substrate
  - b. ↑ ATP-PC storage
  - c. ↓ Depletion of PC and degradation of ATP at the same absolute workload
  - d. = Depletion of PC and degradation of ATP at the same relative workload
  - e. ↑ ATP-PC turnover

↑, increase; ↓, decrease; =, no change.

training history, however, tend to have an initial rapid decline in some aerobic variables but then level off at higher-than-pretraining levels. Anaerobic metabolic variables have less incremental increase with training and less loss with detraining. This fact may explain why

sprint performance is more resistant to inactivity than endurance performance. Complete bed rest or immobilization accelerates detraining (Coyle et al., 1984; Neufer, 1989; Ready and Quinney, 1982; Wilmore and Costill, 1988).

## CLINICALLY RELEVANT

FOCUS ON  
RESEARCH

## Detraining and Retraining

**Nichols, J. F., D. Robinson, D. Douglass, & J. Anthony:** Retraining of a competitive master athlete following traumatic injury: A case study. *Medicine and Science in Sports and Exercise*. 32(6):1037–1042 (2000).

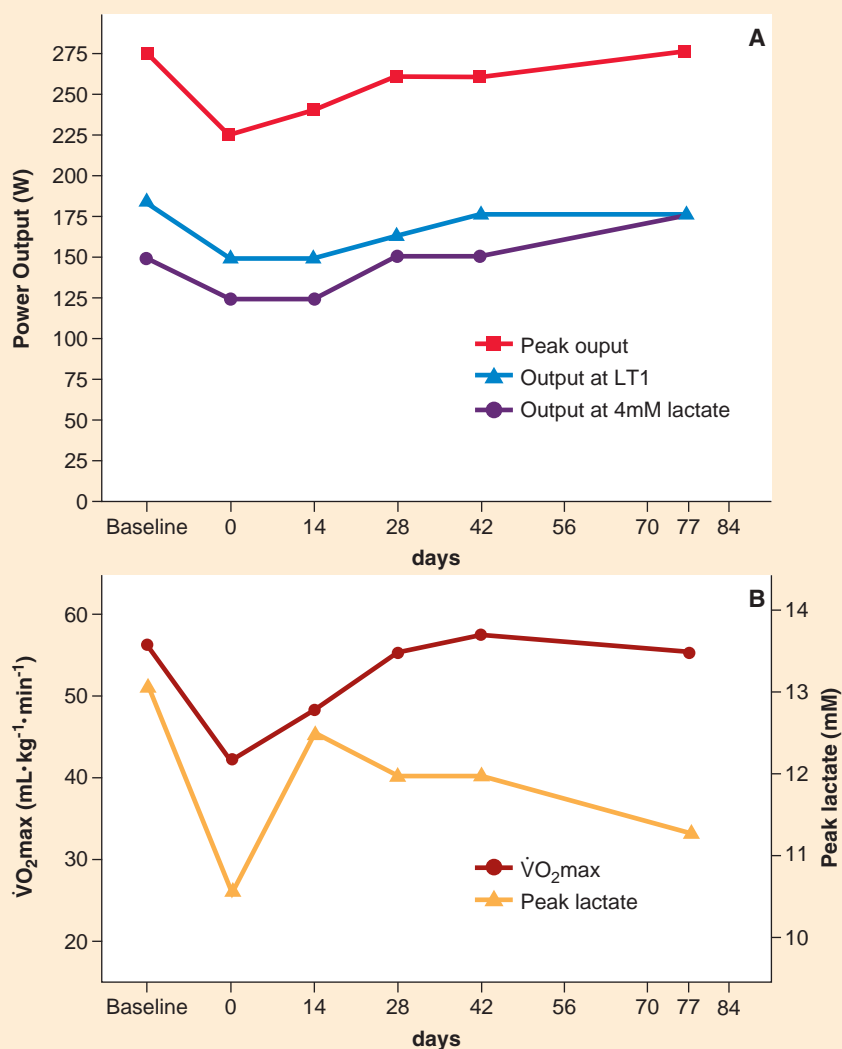
This case study of detraining and retraining was conducted on a 49.5-year-old female elite competitive cyclist. Two days after undergoing testing for a research project, the cyclist sustained a right clavicular fracture during a criterion race. She continued a modified training regime until 22 days after the injury, when she developed loss of motion in the shoulder and pain and numbness in the hand. Surgery was performed 26 days after the injury for brachial plexus impingement. Thus, the detraining period began approximately 4 weeks after the injury. Retraining began the 32nd day after surgery (0 on x-axis). Results for her metabolic variables are presented in the graphs below. The baseline represents the values obtained 2 days before the injury. Retesting was done approximately every 2 weeks (0, 14, 28, 42, 56 and 70 days) for 6 weeks and then again at week 11 of retraining (77 days).

$\dot{V}O_2\text{max}$  decreased approximately 25% during detraining, but by week 11, it was within  $2\text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$  of preinjury values. The improvement was steady over the first 6 weeks of retraining.

Power output decreased 18.2% at peak, 16.7% at LT1, and 18.9%

at 4 mM lactate. Peak lactate decreased by a comparable 19.1%. Although peak power output increased during the first 2 weeks, power output at LT1 and 4 mM lactate did not. This probably occurred because high-intensity work was not included in the initial retraining

weeks as is appropriate given the progression principle. By week 11, peak power output and power output at LT1 had returned to baseline values. However, at that point neither peak power output at 4 mM nor peak lactate had regained preinjury levels.



The following specific detraining effects have been documented in detraining studies (Mujika and Padilla, 2000a,b, 2001):

1. Fuel supply:
  - a. Both short-term (<4wk) and long-term (>4wk) detraining are characterized by an increased RER during exercise, indicating a shift toward an increased reliance on carbohydrates as an energy substrate and a decreased reliance on lipid metabolism.
  - b. A reduced (17–33% in 6–10 d) muscle GLUT-4 transporter content occurs with detraining. Possibly as a result, insulin-mediated glucose uptake also decreases rapidly with detraining. Accompanying this is an increase in epinephrine and norepinephrine during submaximal exercise.
  - c. Decreases in intracellular glycogen storage have also been reported with as little as 1 week of detraining. Reductions of approximately 20% have been reported in 4 weeks in highly trained athletes, and the level may completely revert to the pretraining level shortly after that.
2. Enzyme activity:
  - a. Oxidative enzyme activity decreases between 25 and 45% with both short-term and long-term detraining in highly conditioned individuals and after short-term training programs.
  - b. Small, nonsystematic changes in glycolytic enzymes may occur.
3.  $O_2$  utilization:
  - a.  $\dot{V}O_{2\max}$  has been reported to decline 4–14% with short-term (<4wk) detraining and 6–20% with long-term detraining (>4wk). The decline in trained individuals tends to be progressive and directly proportional to the  $\dot{V}O_{2\max}$  during the first 8 weeks and then to stabilize at levels higher or equal to that of sedentary individuals. Recently trained individuals may decline a lesser amount in the initial weeks, but with long-term detraining, they tend to revert to pretraining levels.
4. LA accumulation:
  - a. Increased blood lactate levels during submaximal exercise occur in both the short- and long-term with detraining.
  - b. Detraining leads to a decrease in maximal lactate values.
  - c. LT1 and LT2 occur at a lower percentage of  $\dot{V}O_{2\max}$  with detraining.
5. ATP production, storage, and transport:
  - a. Mitochondrial ATP production rate decreases 12–28% during 3 weeks of detraining after 6 weeks of training in previously sedentary individuals but still remains above pretraining levels.

Retraining does not occur as rapidly as detraining and is not easier or more rapid than the initial training (Wilmore

and Costill, 1988). Although direct experimental evidence is scanty, the consequences of detraining and retraining appear to be similar for adults, children, and adolescents of both sexes (Bar-Or, 1983).

## SUMMARY

1. The most important considerations for applying each training principle to achieve metabolic adaptations are as follows:
  - a. For specificity, match the energy system of the activity.
  - b. For overload, manipulate time and distance or lactate level.
  - c. For rest/recovery/adaptation, alternate hard and easy days.
  - d. For progression, re-overload if additional improvement is desired.
  - e. For individualization, evaluate the individual according to the demands of the activity and develop a periodization training sequence, system, and load on the basis of this evaluation.
  - f. For maintenance, emphasize intensity.
  - g. For retrogression, plateau, and reversibility, evaluate the training adaptations and modify as indicated.
  - h. For warm-up and cooldown, include activities that will actually elevate or reduce body temperature, respectively.
2. Properly prescribed training programs bring about positive adaptations in fuel supply, enzyme activity, oxygen utilization, lactate accumulation, and ATP production, storage, and turnover.

## REVIEW QUESTIONS

1. Name and briefly describe the eight training principles. Select a sport or fitness activity and show how each of the training principles can be specifically applied to that activity.
2. Describe and explain the metabolic adaptations to exercise training for each of the following factors:
  - a. Substrate or fuel supply
  - b. Enzyme activity
  - c. Oxygen utilization
  - d. Lactate accumulation
  - e. ATP production, storage, and turnover
3. Describe and explain the effect of detraining on each of the following factors:
  - a. Substrate or fuel supply
  - b. Enzyme activity
  - c. Oxygen utilization
  - d. Lactate accumulation
  - e. ATP production, storage, and turnover



For further review and additional study tools, visit the website at <http://thepoint.lww.com/Plowman3e>

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