Chapter 6

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 impact of genetics on metabolic train-ability, and derive a practical application from that discussion.
Introduction
To provide a training program that meets an individual's metabolic goals it is necessary to systematically apply the training principles. The way in which these principles are applied will determine the extent to which the aerobic and/or anaerobic systems of energy production are emphasized, which, in turn, will determine the training adaptations that occur.

Application of the Training Principles for Metabolic Enhancement
A general description of the training principles was included in Chapter 1. Each training principle has a special meaning in relation to the metabolic production of energy to support exercise.

Specificity
Any training program must begin with a determination of the goal (Fox and Mathews, 1974; McCafferty and Horvath, 1977). For example, a 50-yr-old male enrolled in a fitness program who wants to break 60 min at a local 10-km race will have a very different training program from a 16-yr-old high school student competing at the 400- and 800-m distances.

Once the goal is established, it is possible to determine an estimate of the relative contributions of the major energy systems by use of a graph such as the one shown in Figure 4.2. For the 50-yr-old male just mentioned, approximately 98% of the energy for his 60-min, 10-km run is derived from the O2 system, with the remaining 2% coming from the ATP-PC and LA systems. These percentages vary little even if an individual's time is considerably slower or even somewhat faster than 60 min. Thus, the O2 system should be emphasized in this individual's training regimen.

The high school middle-distance runner is a different story. To plan her training program, it is necessary to know her typical times at those distances. In general, she would be expected to be in the 1:00–3:00 range for both distances. (The American National 2000 record for a high school female was 0:50.74 for the 400-m run and 2:00.07 for the 800-m run.) Events in this range stress all three energy systems with a heavier reliance on the ATP-PC and LA anaerobic systems as her performance speed increases. Because a faster time is the goal, she should emphasize and work on the anaerobic systems.

Overload
Overload of the metabolic systems is typically achieved in one of two ways: first, by manipulating time and distance; and second, by monitoring lactic acid levels. 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 VO$_2$max and the use of % VO$_2$max reserve as an overload technique are therefore primarily discussed in the section on application of the training principles in the cardiovascular unit (see Chapter 14).

The Time or Distance Technique

The time or distance technique involves some version of 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, a steady state is maintained throughout the duration of the activity. Thus, the runner who completes an 8-mi training run at a 7:30-min·mi$^{-1}$ pace has done a continuous workout. Sometimes, if such a continuous steady-state aerobic training session is maintained for an extended period of time or distance, it is called a long slow distance (LSD) workout. If several periods of increased speed are randomly interspersed into 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 repetition of the work interval (Fox and Mathews, 1974). The energy system stressed is determined on the basis of the length of time of the work interval. Thus, a work time of less than 30 sec stresses the ATP-PC system; one between 30 sec and 3 min stresses the LA system. Anything over 3–5 min emphasizes the O$_2$ system. The target time is based on each individual’s ability at the selected distance. The length and type of recovery period employed depends on the energy system stressed; but the length is typically between 30 sec and 6 min, and the type may be rest-relief (which can include light aerobic activity and flexibility exercises) or work-relief (which means moderate aerobic activity.) Examples for an ATP-PC, LA, and O$_2$ interval set are presented in Table 6.1. Note that the three sets are not intended to be combined.

ATP-PC System In the ATP-PC set, the runner is doing 100-m sprints. Each repetition is to be run at 3 sec slower than her best time. A total of eight repetitions are to be completed with 0:54 of rest recovery between each repetition.

The amount of time required to restore half of the ATP-PC that has been used—that is, the half-life restoration period for ATP-PC—is approximately 30 sec, with full restoration occurring by 2 min (Fox and Mathews, 1974). Thus, this individual will restore over half her ATP-PC.

During the same recovery time myoglobin O$_2$ replenishment is also taking place. The amounts replenished and restored are influenced by the activity of the participant during the recovery phase, with the greatest restoration occurring with rest or light activity such as stretching and walking.

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 the capacity of that system. Any major involvement of the LA system is avoided by keeping the work intervals short so that little lactate accumulation occurs.

LA System To stress the lactic acid system requires work durations of 30 sec to 3 min. In this example, the runner is asked to perform five repetitions of 400 m in 1:20, with a work-relief recovery of 2:40 between repetitions. Lactic acid 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 min, with full clearance taking almost an hour, it is neither practical nor beneficial to allow for even half-life clearance of lactate between repetitions.

Tolerance to lactic acid is increased by incomplete recovery periods of 1 min 30 sec to 3 min. This
The amount of rest allows for the replenishment of ATP-PC and myoglobin O\textsubscript{2}, and it allows 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). It is intensity (from among the three overload factors of frequency, intensity, and duration) that is most important in improving the capacity of the LA system. Work-relief recovery is typically utilized at these work times, since active recovery does speed up lactate clearance.

**O\textsubscript{2} System** Long work bouts, which are still only a portion of the competitive event (that is, 0.5–1-mi [800–1600 m] repeats for a 10-km runner), can be done to stress the O\textsubscript{2} system. The pace is typically close to average pace during competition and may exceed it. The smaller the proportion of the distance, the faster the pace is and the more repetitions there are. However, the intent is that the intervals be done aerobically. The example in Table 6.1 is for 1200 m. 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 full ATP-PC restoration prior to 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 cool-down) should rarely exceed 2–5 mi (3.2–8 km) with a frequency of 1–3 days per week (Costill, 1986). High-intensity interval training taxes the muscles and joints and care must be taken to avoid injury or overtraining. Refer to Chapter 2 for a review of the signs and symptoms of overtraining, if necessary. 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.

### Table 6.1

**Examples of Time-Distance Interval Training for Runners**

<table>
<thead>
<tr>
<th>Energy System</th>
<th>Competitive Distance</th>
<th>Best Time</th>
<th>Training Distance</th>
<th>Training Time</th>
<th>Repetitions</th>
<th>Recovery Time</th>
<th>Recovery Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP-PC</td>
<td>100 m</td>
<td>:15</td>
<td>100 m</td>
<td>0:18</td>
<td>8</td>
<td>(1:3) 0:54</td>
<td>Rest</td>
</tr>
<tr>
<td>LA</td>
<td>1500 m</td>
<td>5:16</td>
<td>400 m</td>
<td>1:20*</td>
<td>5</td>
<td>(1:2) 2:40</td>
<td>Work</td>
</tr>
<tr>
<td>O\textsubscript{2}</td>
<td>1500 m</td>
<td>5:16</td>
<td>1200 m</td>
<td>4:24#</td>
<td>3</td>
<td>(1:1/2) 2:12</td>
<td>Rest</td>
</tr>
</tbody>
</table>

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

† Based on 1–4 sec faster than average 400 m during 1500-m–1600-m race.

‡ Based on 1–4 sec slower than average 400 m during 1500-m–1600-m race.

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 resulting from a given workout. Currently there is no general agreement about the best way 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 (see Table 6.2). These training zones are based on the identification of the lactate thresholds (LT1 and LT2) obtained during incremental exercise. There is considerable overlap among the zones. The three lower zones (recovery, extensive aerobic, and intensive aerobic) involve predominantly low- to moderate-intensity aerobic activity, whereas each of the three higher zones (threshold, V\textsubscript{O\textsubscript{2}\text{max}}, and anaerobic) represent the transition from aerobic to anaerobic energy supply at progressively higher intensity until both aerobic and anaerobic energy production is maximized (Anderson, 1998; Bourdon, 2000).

Direct measurement of La– during training is not very practical, however, because of the necessity for special equipment and the cost of taking multiple blood samples. Several studies conducted by Weltman and his colleagues (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 was introduced in Chapter 2 and is presented and described fully in Chapter 14. The range of RPE values corresponding approximately to lactate values for each training zone are presented in Table 6.2.

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 6.1 presents such results. This individual reached his LT1 at 220 m-min\textsuperscript{-1}. At that speed...
he reported an RPE of 12. He reached LT2 at a speed of 260 m·min\(^{-1}\) with an RPE of 14. Combining these results with the guidelines from Table 6.2, we can see 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 \(\dot{V}O_2\text{max} \) at an RPE of at least 15. The test results presented in Figure 6.1 do not represent a maximal test for this individual. Maximal workouts should elicit an RPE of at least 17 for everyone. Thus, the individual can be given a workout and an RPE value and adjust his or her intensity accordingly. Another alternative method, if an individual has access to laboratory testing facilities, is to determine the relationship between \([\text{La}^-] \) and HR values. Then HR can be used to estimate the \([\text{La}^-] \) 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 140 b·min\(^{-1}\); extensive aerobic exercise bouts between 110 and 155 b·min\(^{-1}\); intensive aerobic exercise between 120 and 165 b·min\(^{-1}\); threshold workouts between 157 and 173 b·min\(^{-1}\); \(\dot{V}O_2\text{max} \) workouts close to 170 b·min\(^{-1}\); and maximal activity at least at 185 b·min\(^{-1}\).

Heart rate primarily reflects the functioning of the cardiovascular system, but lactate levels reflect the metabolic energy system. Fox, Bowers, and Foss (1988) estimate that if the HR-[\text{La}^-] \) relationship is not individually determined to ensure that 100% of the 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. 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.

Whichever system is used to prescribe an individual’s training session, it is important to provide a mixture of workout types to maximize the possibility for improvement and prevent boredom.

### Table 6.2
Training Zones Based on Lactate Thresholds and Values

<table>
<thead>
<tr>
<th>Recovery</th>
<th>Extensive Aerobic</th>
<th>Intensive Aerobic</th>
<th>Threshold</th>
<th>(\dot{V}O_2\text{max} )</th>
<th>Anaerobic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relation to LT1 and LT2</td>
<td>LT1</td>
<td>LT1 to halfway to LT2</td>
<td>&gt; LT1 but &lt; LT2</td>
<td>LT2</td>
<td>&gt; LT2</td>
</tr>
<tr>
<td>Lactate values mmol·L(^{-1})</td>
<td>&lt; 2.0</td>
<td>1.0–3.0</td>
<td>1.5–4.0</td>
<td>2.5–5.5</td>
<td>&gt; 5.0</td>
</tr>
<tr>
<td>Workout example</td>
<td>Low-intensity aerobic; e.g., 20–30 min</td>
<td>Long slow distance; e.g., 30 min to 2 hr</td>
<td>Tempo runs; e.g., 10–12 sec</td>
<td>Fartlek; e.g., 1-min bursts</td>
<td>High-intensity intervals; e.g., 6–8 reps</td>
</tr>
<tr>
<td>HR (b·min(^{-1}))</td>
<td>110–120</td>
<td>120–155</td>
<td>157–173</td>
<td>170</td>
<td>185</td>
</tr>
</tbody>
</table>

Source: Based on information from Anderson (1998) and Bourdon (2000).
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 \([\text{La}^-]\) values) and faster recovery. The key to adaption for energy production in muscles appears to be allowing for sufficient recovery time between hard-intensity workouts. Cyclic training programs that alternately stress the desired specific energy system (hard day) and allow it to recover (easy day) induce optimal adaptation (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.

Progression

Once adaptation occurs, the workload should be progressed if further improvement is desired. Progression can be done by 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 the increment in training volume—which is the total amount of work done, usually expressed as mileage or load—should not exceed more than 10% per week. For example, if an individual is currently cycling 60 mi per week, 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. The 50-yr-old man remembers when he was a high school star athlete and wants to regain that feeling and physique now! The fitness leader must gently be more realistic and err, if at all, on the side of caution in exercise prescription and progression.

The limit of metabolic adaptation appears to be achieved 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.

Individualization

The first step in individualizing training is, as mentioned earlier, to match the sport, event, or fitness goal of the participant with the specific mix of energy system demands. The second step is to evaluate each 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, that is, the number of days devoted per week for each type of training or energy system stressed. The fifth step is to determine the training load (distance, workload, repetitions, or the like) on the basis of the individual’s evaluation and modified by 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 appears that this level can be maintained by the same or by a reduced volume of work. However, the way that the volume is reduced is critical. If the training intensity is maintained, reductions of one-third to two-thirds in frequency and duration have been shown to maintain aerobic power (\(\text{VO}_2\text{max}\)), 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 (say during a one-week vacation) if intensity is maintained (Chaloupka and Fox, 1975; Neufer, 1989; Weltman, 1995). Conversely, a reduction in intensity brings about a reduction in training adaptation.

It also seems to be important that the mode of exercise is consistent or closely simulated, because, as mentioned previously, many of the training adaptations that occur are specific to the muscles involved. Thus, cross training—the utilization of different modalities to reduce localized stress but increase overall training volume—is likely to be more beneficial for the cardiovascular system than for the metabolic system.

The level of maintenance training necessary for the anaerobic energy systems to operate at maximal levels is unknown. However, sprint performances deteriorate less quickly in response to a decrease in training than do endurance performances (Wilmore and Costill, 1988).

Many fitness participants are primarily in a maintenance mode after the initial several months or first year of participation. The appropriate level for maintenance should be determined by individual goals.
Maintenance should occur primarily during the competitive training cycle of an athlete.

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 volume prior to important competitions that is intended to allow the athlete to recover from previous hard training, maintain physiological conditioning, and improve performance. Athletes often fear that if they taper for 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 is either equaled or improved after a taper of 7 to 21 days (Costill, et al., 1985; Houmard, et al., 1990; Johns, et al., 1992; Shepley, et al., 1992).

The one group of individuals who probably do not respond to these maintenance and taper guidelines with joy are those who are injured. Maintaining a high level of intensity in the training modality is one of the most difficult tasks for an injured individual. Research continues in an attempt to solve this problem, such as trying to determine whether work in the water can be substituted for land-based work. In the meantime, the injured individual who cannot continue to train at his or her preinjury level should probably accept the fact that he or she will experience a loss of adaptation.

**Retrogression/Plateau/Reversibility**

The training principle of retrogression, plateau, and reversibility is not one that a coach or fitness leader applies as much as anticipates and reacts to. At one or more points in the process of training, an individual will fail to improve with progression and will either stay at the same level (**plateau**) or show a performance or physiological decrement (**retrogression**). When a pattern of nonimprovement occurs, it is important to check for other signs of overtraining. A shift in training emphasis or the inclusion of more easy days may be warranted. Remember that a reduction in training 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 will occur. This **reversibility** of training adaptations in skeletal and metabolic potential occurs within days to weeks after training ceases. A reduction in both maximal and submaximal performance ultimately follows. It should come as no surprise that those metabolic factors that show the greatest improvement with training—that is, those involved with aerobic energy production (see training adaptations later in this chapter)—also show the greatest reversal. Within 3 to 6 weeks after the cessation of activity, pretraining levels are typically reached again, especially if the training program was of short duration. Individuals with a long and established training history, however, tend to show an initial rapid decline in some aerobic variables but then level off at higher-than-pretraining levels. Anaerobic metabolic variables show 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).

Detraining does not occur as rapidly as detraining and is not easier or more rapid than 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 (Bar-Or, 1983).

**Warm-Up and Cool-Down**

The information about warm-up is sparse in relation to the effects on metabolic function, but several generalizations seem to be warranted. An elevated body temperature—and more specifically, an elevated muscle temperature—increases the rate at which the metabolic processes in the cells can proceed. This rate increase occurs in large part 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, oxygen is more readily released from red blood cells and transported into the mitochondria at elevated temperatures (Van de Graaff and Fox, 1989). One consequence of increased body temperature is a decreased oxygen deficit at the initiation of exercise (Gutin et al., 1976). Another consequence is a greater availability of oxygen to the muscles during work. When more oxygen is available sooner, less reliance is placed on anaerobic metabolism, and less lactate accumulates at any given heavy workload. At lighter endurance workloads a greater utilization of fats for the production of energy is possible earlier in the activity. This early use of fats serves to spare carbohydrate and extend the time a given high-intensity effort can be continued. These beneficial metabolic effects of a
warm-up appear to exist for children and adolescents as well as adults (Bar-Or, 1983).

In devising a warm-up to achieve these metabolic benefits, several considerations need to be taken into account (Franks, 1983).

1. The activity should involve large muscle groups that will elevate the body temperature from 37° to 38° or 39°C. This temperature elevation generally occurs simultaneously with the onset of sweating.

2. The activity should last approximately 5–20 min and end no longer than 15 min before the hard phase of the workout or competitive performance. The harder the planned workout, the longer the warm-up phase should be. The less time elapsing between the warm-up and the performance, the better it is for an athlete.

3. The intensity of the warm-up should be such that it does not fatigue the participant. Highly fit individuals can utilize longer, more intense warm-ups than low fit individuals. Generally speaking, the warm-up for endurance activities can occur at a lower intensity (25–30% \( \text{VO}_2\text{max} \) or less than 35% \( \text{HRmax} \)) than for sprinting or anaerobic activities (45–50% \( \text{VO}_2\text{max} \) or 50–60% \( \text{HRmax} \)). Higher values in the range of 60–80% \( \text{VO}_2\text{max} \) (70–85% \( \text{HRmax} \)) may be needed by highly conditioned athletes to elevate core temperature. In any case, it is probably best to stay below the lactate threshold.

4. 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. Identical warm-ups of explosive tasks (such as long or high jumping) at full speed should be used sparingly. The action can be patterned at lower levels.

6. An intermittent or interval type warm-up has been found to be more beneficial for children than a continuous warm-up (Bar-Or, 1983).

The primary metabolic value of a cool-down lies in the fact that lactate is dissipated faster during an active recovery. As described in Chapter 4, the lactate removal rate is maximized if the cool-down activity is of moderate intensity (a little higher than the individual tends to self-select) and continues for approximately 20 min.

In general, all of the 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 on the basis of age or sex, although individual differences should always be kept in mind.

### Metabolic Adaptations to Exercise Training

When the training principles just discussed are systematically applied and rigorously followed, a number of adaptations occur relative to the production and utilization of energy. The extent to which the adaptations occur depends on the initial fitness level of the individual and his or her genetic potential. Figure 6.2 is an expanded version of Figure 3.4 showing the metabolic pathways you studied earlier. Numbers have been inserted following the naming of some factors to indicate sites where these adaptations occur. The following discussion will follow that numerical sequence. Refer to Figure 6.2 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 3, Figure 3.16, and Table 3.3). Although little is known about the impact of training on the hypothalamic-releasing factors and adrenocorticotrophic hormone (ACTH), a definite pattern is seen for 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 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 glucagon is lower and the suppression of insulin less; the rise in norepinephrine and epinephrine is less; the rise in growth hormone is less; and the rise in cortisol is less during submaximal exercise in trained individuals than in untrained individuals (Galbo, 1983). 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 GLUT-4 number and concentration in skeletal muscle (Sato, et al., 1996). This results in a greater uptake of glucose under the influence of insulin. Thus, at any resting insulin level, whole body glucose clearance is enhanced. This occurs in both young and elderly 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. The precise mechanisms for this apparent paradox are unknown at this point in time (Coggan, 1996).

Both endurance and sprint training bring about an increase of muscle and liver glycogen reserves. In addition, at the same absolute submaximal workload (that is, the same rate of oxygen consumption) muscle and liver glycogen depletion occurs at a slower rate in the trained individual than in the untrained individual (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 are seen in lower RER values (Figure 6.3a on page 165). 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 for longer periods of time 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.

Figure 6.2
Metabolic Training Adaptations
The circled numbers indicate sites where training changes occur. The boxes indicate processes.
Fat (2)

The trained individual is able to use his or her carbohydrate stores more slowly than the 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. an increased mobilization or release of free fatty acids from the adipose tissue;
2. an increased level of plasma free fatty acids during submaximal exercise;
3. an increase in fat storage adjacent to the mitochondria within the muscles; and
4. an increased capacity to utilize fat at any given plasma concentration.

The increased reliance on fat as a fuel is said to have a glycogen-sparing effect and is responsible for lowered RER values (Figure 6.3a) at the same absolute and same relative (% VO2max) work intensities. Because glycogen supplies last longer, there is a delay in fatigue and 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).

Protein (3)

Despite the fact that proteins are the least important energy substrate, changes do occur as a result of endurance training, which enhance their role. 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 indicating an accelerated removal for gluconeogenesis. In ultraendurance events, this increased gluconeogenesis effect would be beneficial in 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 investigating the activity of the glycolytic enzymes have been contradictory, with most showing little (if any) change but some showing increased activity. 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 but that others respond better to endurance training. Three key enzymes have shown significant training changes: glycogen phosphorylase, phosphofructokinase, and lactic dehydrogenase.

Glycogen Phosphorylase Glycogen phosphorylase catalyzes the breakdown of glycogen stored in the muscle cells so that it may be used as fuel in glycolysis. An increase in this enzyme’s activity has been found with high-intensity sprint training. The ability to break down glycogen quickly is important in near-maximal, maximal, and supramaximal exercise.

Phosphofructokinase (PFK) PFK is the main rate-limiting enzyme of glycolysis. Results from both endurance and sprint training studies are inconsistent but tend to suggest an increase in activity with adequate levels of training. Increased PFK activity leads to a faster and greater quantity of ATP being produced glycolytically.

Lactic Dehydrogenase (LDH) LDH catalyzes the conversion of pyruvate into lactate. It exists in several discrete forms, including a cardiac muscle form that has a low affinity for pyruvate (thus making the formation of lactate less likely) and a skeletal muscle form that has a high affinity for pyruvate (thus making the formation of lactic acid 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 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 Herman sen, 1973; Holloszy and Coyle, 1984; Sjödin, et al., 1982).
Shuttles (5)

You will recall that the hydrogen ions removed in glycolysis must be transported across the mitochondrial membrane by a shuttle since that membrane is impermeable to NADH + H⁺. No training changes have been found to occur in the glycerol-phosphate shuttle enzymes that predominate in skeletal muscle. Conversely, large increases in the enzymes of the cardiac muscle’s malate-aspartate shuttle both in the cytoplasm and mitochondria have been found, 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 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 to a greater degree than the interfibrillar mitochondria. The stimulus for these increases appears to be 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, a runner would exhibit an increase in mitochondrial size and number only in the legs, whereas a cross-country skier would show 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 there are, the greater is 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 for trained and untrained individuals; however, the greater mitochondrial protein content means an overall greater enzyme activity to utilize the pyruvate. 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 in supplying energy for submaximal exercise than for maximal exercise (Abernethy, et al., 1990; Gollnick, et al., 1986; Holloszy, 1973; Holloszy and Coyle, 1984).

Oxygen Utilization (7)

Maximal Oxygen Uptake

Maximal oxygen uptake (VO₂max) increases with training (Figure 6.3b). Even though this is a measure of the amount of oxygen utilized at the muscle level, VO₂max 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 VO₂max includes the fact that individuals can have essentially the same mitochondrial content but very different VO₂max values. Conversely, individuals with equivalent VO₂max values can have quite different mitochondrial enzyme levels. Additionally, small training changes can occur in one (mitochondrial activity or VO₂max) 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 VO₂max) (Holloszy and Coyle, 1984).

Submaximal Oxygen Cost

The oxygen cost (VO₂ in mL·min⁻¹ or mL·kg⁻¹·min⁻¹) of any given absolute submaximal workload is the same before and after training, assuming that no skill is involved where efficiency would change (Gollnick, et al., 1986; Holloszy and Coyle, 1984) (Figure 6.3b). 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, for an individual who is just learning the front crawl stroke, the oxygen cost would actually go down. It would decrease not because of a change in the exercise 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 ventilation and heart rate costs and by increasing the percentage of carbohydrate. The first two changes do typically occur 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 stride length. High-intensity interval training has produced improvements in running economy, but the evidence is not strong (Bailey and Pak, 1991; Conley, et al., 1981; Sjödin, et al., 1982). Decreased efficiency or economy, on the other hand, can occur. If so, it should be interpreted as a symptom of overtraining (Fry, et al., 1991).

If an individual increases his or her VO\(_{2}\)max yet the oxygen cost of any given (absolute) workload remains the same, then the % VO\(_{2}\)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 is a consequence of the biochemical adaptations in the muscle rather than changes in oxygen delivery.

The myoglobin concentration in 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 a trained individual. The primary reason for this reduction is that oxidative phosphorylation is activated sooner owing to the increased number of mitochondria that are sensitive to low levels of ADP and P\(_i\). This result is advantageous to the exerciser, because less lactic acid will be produced and less creatine phosphate will be depleted (Holloszy, 1973; Holloszy and Coyle, 1984). The magnitude of oxygen drift will also be 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).

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**Figure 6.3**

Metabolic Responses of Endurance Trained versus Untrained Individuals to Incremental Exercise to Maximum
Oxygen Free Radicals, Exercise Intensity, and Exercise Training Adaptations

Cancer, atherosclerosis, cataracts, Alzheimer's disease, diabetes, loss of memory, 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 is created. The resultant structure is called a free radical. Free radicals have a drive to return to a balanced stable state and attempt to do so by either taking an electron from, giving an electron to, or sharing an electron with another atom. Often a chain reaction is set up that results in damage (oxidative stress) to lipids (especially the lipid bilayer of cell membranes), proteins (in enzymes, immune cells, joints, and muscles), and DNA (breaking stands or shifting bases, thus influencing the genetic code). These are the changes that ultimately can lead to the disease conditions listed earlier (Alessio and Blasi, 1997; Jenkins, 1993; Keith, 1999).

Free radicals can be produced from sources that originate outside the body, such as x-rays, UV rays from sunlight, air pollutants (ozone, nitric oxide from car exhaust), cigarette smoke, toxic chemicals (some pesticides), and physical injury (from contact sports or concussions). They may also be produced from sources that originate within the body, specifically as part of normal immune function and as a normal by-product of the production of energy (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 location of this continuous electron leak is at 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 reactive oxygen species, such as hydrogen peroxide, H₂O₂. Hypoxia leads to a freeing of metals (Fe, Cu, 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.

Despite the increased production of free radicals resulting from exercise, it is unlikely that exercise results in substantial damages to the normal healthy individual. The body has a number of natural defenses, and antioxidants ingested from food provide additional defenses. Each cell contains a variety of antioxidant scavenger enzymes, predominantly superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX). 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, although tolerance does appear to be better following moderate-intensity rather than maximal-intensity exercise. This applies to 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 100–400 IU of vitamin E may be warranted (Jenkins, 1993; Keith, 1999).

Sources:
Alessio & Blasi (1997); Jenkins (1993); Keith (1999).
Lactate Accumulation (8)

Lactic acid 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. It is a matter of some debate whether the rate of production decreases or the rate of clearance increases more as a result of training.

Factors that lead to a decrease in lactate production include:
1. fuel shifts;
2. enzyme activity changes; and
3. blunted neurohormomonal 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. Pyruvate dehydrogenase activity increases, converting more pyruvate to acetyl CoA. LDH enzyme shifts from the skeletal muscle form, which favors lactic acid production, to the cardiac muscle form, which has a lower affinity for pyruvate. In addition, glycolysis is inhibited following training by several factors, two of which are related to the increased utilization of fat during submaximal exercise. The first is a high concentration of free fatty acid in the cytoplasm, and 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, hence, decrease the possible production of lactic acid. 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 lactic acid (Holloszy, et al., 1986; Holloszy and Coyle, 1984).

There are also several changes that lead to higher rates of clearance. These can be classified into two main factors:
1. enhanced lactate transport; and
2. enhanced lactate oxidation.

Lactate transport is enhanced by a combination of increased substrate affinity, increased intrinsic activity, and increased density of the mitochondrial membrane and cell membrane MCT1 lactate transporters. At the same time, mitochondrial size, number, and enzyme concentrations are elevated. Taken together, these enable muscle cells to increase both the extracellular and intracellular lactate shuttle mechanisms. There is an overall uptake of lactate by muscles, and, consequently, more lactate can be oxidized more rapidly during exercise. Concomitantly, blood flow to the liver is enhanced, which aids in lactate removal overall (Bonen, 2000; Brooks, 2000; Brooks, et al., 1999; Gladden, 2000; Pilegaard, et al., 1994). These adaptations mean that the change in the rate of clearance probably is more important than the change in the rate of production (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 (% VO₂max) after training.

As a consequence of the change in the ratio of lactate clearance to production, a higher workload (both in absolute and relative terms) is required to achieve lactate levels in the 2- to 4-mmol·L⁻¹ range (Figure 6.3c). This means that an individual can improve performance by exercising at a higher relative intensity for a given period of time and yet delay the onset of fatigue since the lactate thresholds (LT1 and LT2) have been raised (Allen, et al., 1985; Henritze, et al., 1985; Holloszy and Coyle, 1984; Skinner and Morgan, 1985; Williams, et al., 1967; Yoshida, et al., 1982).

At maximal aerobic/anaerobic endurance exercise, the level of lactic acid 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 a result that is more psychological than physiological, in that the trained individual is more motivated and is better able to tolerate the pain caused by lactic acid (Galbo, 1983) and is 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 seconds of rest between sets, 3 days per week), 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 from 8 mmol·L⁻¹ to 6 mmol·L⁻¹. 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 lactate levels (8 mmol·L⁻¹ versus 7.5 mmol·L⁻¹). These results indicate that more work could be done before the same accumulation of lactate was obtained after training 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 10 exercises 3 times per session, doing 8–10 repetitions with 30 seconds of rest between exercises, 3 days per week. 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 VO2max nor cycle ergometer VO2peak. However, the experimental subjects cycled 33% longer at 75% VO2peak 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⁻¹) increased by 12%. 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-six ATP 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 the trained than in the untrained individual, especially if muscle mass increases. Whether this amount is large enough to markedly increase anaerobic capacity is questionable. At the same absolute workload 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. Hence, the rate of turnover of ATP and PC increases. 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 O₂ systems are presented in Table 6.3 contrasting the trained and untrained male. It can be seen that the LA system changes much more with training than does the ATP-PC system, but that the greatest change is in the O₂ system (Bouchard et al., 1991, 1982).

### Work Output

Work output—measured as watts or kilocalories per kilogram of body weight on a bicycle test such as the 10-sec or 30-sec Wingate Anaerobic Test and/or a 90-sec test—shows improvements with training. This result is evidenced by higher scores of athletes when compared to nonathletes and by higher posttraining than pretraining scores in all populations. Furthermore, sprint or power type athletes typically show higher anaerobic values and greater adaptations than endurance athletes. Elite sprinters and power athletes score higher than do 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 for a longer period of time than an untrained individual. The trained individual can also accomplish more total work to a higher absolute workload before achieving his or her maximum than an untrained individual can. 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 6.4, are specific to the training employed.

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**Table 6.3**

<table>
<thead>
<tr>
<th>System</th>
<th>Power</th>
<th>Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>kcal·min⁻¹</td>
<td>kcal·min⁻¹</td>
</tr>
<tr>
<td></td>
<td>UT TR UT TR</td>
<td>UT TR UT TR</td>
</tr>
<tr>
<td>Phosphagens (ATP-PC)</td>
<td>72 96 300 400</td>
<td>11 13 45 55</td>
</tr>
<tr>
<td>Anaerobic glycolysis (LA)</td>
<td>36 60 150 250</td>
<td>48 72 200 300</td>
</tr>
<tr>
<td>Aerobic glycolysis plus Krebs cycle plus ETS/OP (O₂)</td>
<td>7–19 32–37 30–80 135–155</td>
<td>360–1270 10,770–19,140 1,500–5,300 45,000–80,000</td>
</tr>
</tbody>
</table>

*Source: Modified from Bouchard, Taylor, & Dulac (1991); Bouchard, et al. (1982).*
Table 6.4
Metabolic Training Adaptations

1. Fuel Supply
   a. Carbohydrate
      (1) ↑ GLUT-4 transporter number and concentration.
      (2) ↓ glucose utilization.
      (3) ↑ Muscle and liver glycogen reserves.
      (4) ↓ Rate of muscle and liver glycogen depletion at absolute submaximal loads, that is, 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 same absolute and 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.
   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 the Krebs cycle, electron transport, and oxidative phosphorylation due to greater mitochondrial protein amount.

3. O₂ Utilization
   a. ↑ VO₂max with aerobic endurance training but not dynamic resistance training.
   b. = VO₂ cost at absolute submaximal workload.
   c. ↑ Myoglobin concentration.
   d. ↓ Oxygen deficit.
   e. ↓ Oxygen drift.

4. Lactic Acid Accumulation
   a. ↑ MCT1 lactate transporters.
   b. ↑ Intracellular and extracellular lactate shuttle activity.
   c. ↓ La⁺ accumulation at same absolute workload and % VO₂max relative intensity for endurance activity.
   d. ↓ La⁺ accumulation at same absolute load but = La⁺ accumulation at 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 same absolute workload.
   d. = Depletion of PC and degradation of ATP at same relative workload.
   e. ↑ ATP-PC turnover.

↑, increase; ↓, decrease; =, no change.
The Influence of Age and Sex on Metabolic Training Adaptations

With the exception of \( \text{VO}_2\text{max} \) (which is discussed in the cardiovascular-respiratory unit), there is a scarcity of research data on most of the metabolic variables across the age spectrum. What scattered evidence is available indicates that training and detraining changes in children, adolescents, and the elderly are similar to changes for adults in the 20- to 50-yr range. This is especially true if the changes are considered relative to baseline values (that is, as a percentage of change) and not 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).

Data concerning metabolic adaptations in females of all ages are also minimal, again with the exception of \( \text{VO}_2\text{max} \) (Shepard, 1978; Tlusty, 1969; Wells, 1991). Studies have shown the following adaptations in females as a result of appropriate specific training:

1. Fuel utilization shifts in favor of fat.
2. Lactic acid levels decrease during submaximal work and increase at maximal effort.
3. Enzyme levels change.
4. Submaximal \( \text{VO}_2 \) consumption remains stable or decreases slightly.

Focus on Research

Substrate Training Adaptations in Children


As has been described in the text, it has been clearly established in adults that the primary substrate utilized to fuel exercise depends on the modality, intensity, and duration of the activity as well as the training status of the exerciser. The “crossover” concept states that at some point, as the intensity increases during incremental exercise, the predominant fuel source will shift 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 boys and girls (ages 7–12) volunteered and were divided into a training group (N = 10) and a control group (N = 13). All were tested for \( \text{VO}_2\text{peak} \) on a cycle ergometer and then at five power outputs designed to elicit approximately 35%, 45%, 55%, 65%, and 75% of \( \text{VO}_2\text{peak} \). 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-min work bouts separated by 1–2 min of rest at roughly 50% \( \text{VO}_2\text{peak} \), three times per week.

The results, presented in the accompanying graph, 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.

![Graph showing substrate training adaptations in children](image-url)
In short, both males and females respond to the same training with the same adaptations. This is not to say that sex differences are obliterated in equally trained males and females in the metabolic variables—just that both sexes are trainable and probably to the same extent.

Detraining also appears to operate in the same way and to the same extent for males and females.

The Impact of Genetics on Metabolic Trainability

Genetics impacts human physiological variables two ways. The first impact is directly on the expression of that variable. For example, at least 50% of an individual’s endurance capacity, as measured by the maximal amount of work done on a cycle ergometer in 90 min, can be accounted for by heritability (Bouchard, 1986). Other examples have been discussed for both anaerobic and aerobic metabolism in previous chapters.

The second way in which genetics impacts physiology is on the adaptation to training of any variable. Research in which multiple pairs of monozygous (MZ) twins have been trained under laboratory conditions has shown that for many of the variables examined the impact of genetics on trainability is stronger than the heritability of the trait itself in sedentary individuals. Furthermore, the influence of genetics is sometimes stronger the closer the individual is to achieving his or her maximal potential.

Although many of the energy system variables whose training adaptations have just been described have not been investigated to date, specific information is available on several (Bouchard, 1986).

Submaximal Substrate or Fuel Utilization

Adaptations in substrate availability and utilization that occur as a result of aerobic endurance training are largely genetically based. In particular, data show that the change in epinephrine-stimulated lipolysis or breakdown of fat from isolated adipose cells that results from training is genetically dependent. The genetic influence on the training-induced ability to mobilize fat is also reflected in the ability to utilize fat during light submaximal exercise. Forty-four percent of the variance in the training adaptation of the respiratory exchange ratio can be attributed to genetics. Likewise, 42% of the variance of submaximal oxygen cost at the same absolute load depends on genetics (Bouchard, et al., 1992).

Maximal Work Output and Oxygen Consumption

Approximately 70% of the variance in the training increase in work output in 90 sec (primarily lactic anaerobic metabolism) can be accounted for by genetics. For training changes in V\textsubscript{O\textsuperscript{2}}\text{max} and 90-min maximal work capacity, the amount of variance accounted for by genetics is approximately 75% and 80%, respectively. Both the training adaptations and the percentage of that work increase that can be accounted for by genetics are smaller for a 10-sec (alactic anaerobic) task: only about 30%. Maximal aerobic endurance capacity is about 60% more trainable than V\textsubscript{O\textsuperscript{2}}\text{max}. Males show higher trainability in maximal aerobic endurance capacity than females (Bouchard, 1986). Genetically dependent training changes in both glycolytic and oxidative muscle enzyme activity underlie these changes (Bouchard, 1986; Hamel, et al., 1986).

Genetic Variability

One of the most interesting aspects of the genetic impact on trainability is the wide range of adaptation. For example, following a 20-week endurance training program in which the mean improvement in endurance performance was about 50% of the pretraining work output, the range of improvement was from 16 to 97% (Bouchard, et al., 1992). Similarly wide ranges have been shown for other variables, suggesting that most of the individual differences in responding to the same training program are genetically determined (Bouchard, 1993; Bouchard, et al., 1988). Thus, it appears that there are both low and high responders. And there may be as much as a threefold to tenfold variation between high and low responders. Indeed, some people (probably less than 5% of the population) may be nonresponders. In addition, there are also early and late responders. Early responders show metabolic adaptations very quickly, within the first 5–7 weeks of a training program. Late responders may show little initial progress but after 8 weeks or so appear to make a major breakthrough. These variations, of course, are the basis for the individualization training principle (Bouchard, et al., 1988; Hamel, et al., 1986).

Unfortunately, there is no way at the present time to determine an individual’s genetic training potential for either the aerobic or the anaerobic system. Thus, the physical educator, coach, or fitness leader must be alert for these variations and counsel those under their tutelage accordingly (Bouchard, 1993).
**Summary**

1. The most important considerations in 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 adaptation, alternate hard and easy days.
   d. For progression, reoverload 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 your evaluation.
   f. For maintenance, emphasize intensity.
   g. For retrogression, plateau, and detraining, evaluate the training adaptations and modify as indicated.
   h. For warm-up and cool-down, include activities that will actually elevate or reduce body temperature, respectively.

2. Properly prescribed training programs bring about adaptations in fuel supply, enzyme activity, oxygen utilization, lactate accumulation, and ATP production, storage, and turnover.

3. Most of the variation between individuals in metabolic training adaptation is genetically based. Long-term, lactic anaerobic, and aerobic metabolic training adaptations are influenced more by genetics than are short-term, alactic anaerobic adaptations.

**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. Discuss the impact of genetics on the metabolic adaptations to exercise training in terms of the following factors:
   a. Submaximal substrate or fuel utilization
   b. Maximal work output and oxygen consumption
   c. Variability of response

4. Derive a practical application from this discussion, and provide a realistic example.

For further review and additional study tools, go to thePoint.lww.com/Plowman and view the Student Study Guide for Exercise Physiology for Health, Fitness, and Performance by Sharon A. Plowman and Denise L. Smith.

**Passport to the Internet**

Visit the following Internet sites to explore further topics and issues related to training principles and adaptations. To visit an organization’s web site, go to www.physiologyplace.com and click on “Passport to the Internet.”

**Runners World Online**  
Runners World is one of the leading magazines for dedicated runners. This online resource provides training advice and general information about running.

**President’s Council on Physical Fitness and Sports**  
The President’s Council on Physical Fitness and Sports (PCPFS) serves as a catalyst to promote, encourage, and motivate Americans of all ages to become physically active and participate in sports. Surf this site for hands-on information as well as the latest research on these efforts.

**References**


