

level of exercise intensity that a person can sustain during prolonged exertion. These findings help explain the tremendous increase in endurance associated with training. Fat is the most abundant energy source (50 times more abundant than carbohydrate). Improved fitness allows greater access to that immense storehouse of energy.

Greater Fat Utilization

The mobilization of fat does not ensure its metabolism. How does training influence the utilization of FFA as a source of energy for muscular contractions? Studies have shown that trained animals and humans are capable of extracting a greater percentage of their energy from FFA during submaximal exercise. How, then, does fitness influence fat utilization?

Móle, Oscai, and Holloszy (1971) provided convincing proof of the effect of training on FFA utilization. They found that the ability of rat muscle to oxidize the fatty acid

palmitate doubled following 12 weeks of treadmill training. The authors suggested that the shift to fat metabolism was a key factor in the development of endurance fitness and an important mechanism serving to spare carbohydrate stores and prevent low blood sugar during prolonged exertion. Thus, the physically fit person can derive a greater percentage of energy requirements from fat than the unfit subject can. At a given workload, fit subjects may obtain as much as

Lactic Acid

Lactic acid is produced when the breakdown of muscle glycogen to pyruvic acid exceeds the ability of the mitochondria to process the pyruvate. So, the pyruvic acid picks up hydrogen, becomes lactic acid, and begins to accumulate in the muscle and blood. The heart and skeletal muscle can use lactate as a source of energy, and the liver can oxidize it. But when the production of lactate exceeds its removal, the acid level in muscle and blood increases. The rising level of acid in the muscle reduces force production by interfering with muscle contractions and decreases endurance by lowering the efficiency of aerobic enzymes.

90 percent of their energy from fat. Free fatty acids are used during all forms of muscular activity, except for all-out bursts of effort, such as the 100-yard dash. Training even seems to improve the ability of the heart muscle to oxidize fat (Keul 1971).

When exercise begins, the initial source of energy from fat is from intramuscular fat, a supply that is enhanced with training. When prolonged activity depletes intramuscular fat, the body uses fat that comes from adipose tissue by way of the blood (Coggan and Williams 1995). Improved fitness increases the availability of fat through mobilization of FFAs, as well as from an increase in enzyme activity. Both contribute to the rate of FFA utilization.

Reduced Blood Lipids

Blood lipids—cholesterol and triglycerides—have been associated with the incidence and severity of coronary heart disease. They are related to other risk factors, including diet, overweight, and lack of exercise. Findings suggest that fitness training also influences the lipids.

Triglycerides

Dietary fat intake shows up in the blood as chylomicrons, large clumps of triglycerides. Most of the triglycerides are removed from the plasma in the capillaries adjacent to muscle and adipose tissue. The liver clears any remains from the circulation. Chylomicrons are responsible for the milky appearance of blood plasma following a meal (postprandial

lipemia). Besides containing 80 to 95 percent triglyceride, chylomicrons contain 2 to 7 percent cholesterol, 3 to 6 percent phospholipid, and 1 to 2 percent protein.

Dieting or participation in regular physical activity can reduce fasting serum triglyceride levels. The exercise-induced reduction occurs several hours afterward and lasts for about 2 days. With regular exercise, further reductions occur until subjects reach a lower level consistent with their exercise, diet, and other factors, including inherited blood lipid patterns.

Earlier in this chapter, we established the influence of exercise on postmeal fat in blood. The research supports the hypothesis that regular exercise enhances the removal and utilization of triglyceride by muscle cells, rather than allowing their deposit in adipose tissue or processing by the liver, possibly to synthesize more cholesterol.

A researcher trained sedentary rats for 12 weeks on a treadmill. Following the training, the muscles were analyzed for the activity of lipoprotein lipase (LPL), the enzyme responsible for the uptake of plasma triglyceride fatty acids (TGFA) from plasma chylomicrons and other sources in the blood. The researcher reasoned that an increase in LPL activity would accompany any increase in the uptake of TGFA by skeletal muscle during exercise. The results of the study confirmed the hypothesis. Regular endurance training led to a two- to fourfold increase in LPL activity, indicating that training increases the capacity of muscle fibers to take up and oxidize fatty acids originating in plasma triglycerides (Borensztajn 1975).

Because the fat is used before it can be deposited in adipose tissue, these findings have tremendous significance in the area of weight control. The implications for cardiovascular health are even more exciting, as is the realization that these benefits are associated with an entirely natural, enjoyable, and satisfying experience—aerobic training.

Cholesterol

Cholesterol ingested in the diet is absorbed in the small intestine, finds its way into the lymph system, and then is dumped into the blood. There it joins cholesterol produced by the body in the chylomicrons and in very-low-density lipoprotein particles (VLDL). Once in the plasma, the same enzymes that act on the chylomicrons attack the VLDL. Much of the triglyceride is removed (within 2 to 6 hours). The VLDL is degraded to low-density lipoprotein (LDL), which the liver removes over a period of 2 to 5 days. Because of the smaller size of the LDL particle and its high concentration of cholesterol, the LDL particle seems to be involved directly in the development of coronary artery disease (CAD). The LDL particles find their way into coronary arteries and contribute to the growth of

Maximal Fat Oxidation

To determine when **maximal fat oxidation** occurred during exercise, moderately trained subjects were exercised on a progressive laboratory bicycle test. Fat oxidation peaked at 55 to 72 percent of $\dot{V}O_2$ max, or 68 to 79 percent of maximal heart rate, values higher than those previously noted (Achten, Gleeson, and Jeukendrup 2002). This study, performed after subjects fasted for 10 hours, shows that fat can be a significant source of energy for rather vigorous exercise. Ingesting carbohydrate before performing exercise lowers the contribution of fat to energy expenditure. So, if your goal is oxidation of fat, exercise before breakfast or other meals. However, as mentioned earlier, if your goal is to burn more calories, you will need to find an intensity that you can sustain for a long period of time. As you become more fit, you may find joy in being able to jog or run at your second lactate threshold for an hour or more.

atherosclerotic plaques. Thus, LDL is believed to be a major culprit in the development of coronary artery disease.

Until the mid-1970s, diet, weight loss, and drugs were believed to be the major weapons in the fight against cholesterol. Studies on the effect of exercise on cholesterol typically reported a modest reduction, but only when the exercise was vigorous and of long duration; for example, 3 or more miles (4.8 km or more) of running per day. But remember that the blood transports cholesterol in several ways. A single measure of serum cholesterol does not indicate how the cholesterol is distributed among the several lipoprotein fractions, nor does it indicate the effects of exercise.

Dr. Wood (1975) of the Stanford Heart Disease Prevention Program compared the lipoprotein patterns of sedentary and active middle-aged men (35 to 59 years old). The active group consisted of joggers who averaged at least 15 miles (24.1 km) per week for the preceding year. As expected, the triglycerides were “strikingly” lower for the active group, while total cholesterol was only “modestly” lower. But when the lipoprotein pattern was analyzed, the joggers exhibited a significantly lower level of dangerous LDL and an elevated level of high-density lipoprotein (HDL). These findings were significant, because there is a direct relationship between LDL and heart disease and an inverse relationship between HDL and heart disease (as HDL goes up, the incidence of heart disease goes down). HDL seems to carry cholesterol away from the tissues for removal by the liver. Dr. Wood noted that the lipoprotein pattern of the active men was similar to that of young women, who have the lowest risk of heart disease in the adult population. Evidence on raising HDL with drug therapy shows that this process also fails to reduce CAD risk. This suggests that physical activity and diet related to increased HDL levels may cause the reduced risk of heart disease, not the increase in HDL cholesterol (Voight et al. 2012).



- Get active and improve your fitness to help reduce your risk of heart disease.

When researchers studied the effects of 7 weeks of training on the serum lipids and lipoproteins of medical students, triglycerides fell by 27 percent. Furthermore, they found a marked reduction of LDL and VLDL cholesterol, an increase in HDL cholesterol, and no changes in body weight to confuse the results (Lopez et al. 1974). Studies in our lab (Sharkey et al. 1980) agree with those reported by Dr. Wood and many others (Leon et al. 2002). They indicate how training helps shift cholesterol from the dangerous LDL to the favorable HDL and how activity and fitness help reduce the risk of coronary artery disease.

How's that for an extra benefit of fitness? Fitness not only allows increased caloric expenditure and enhanced fat utilization, but also directly affects the blood lipids and reduces risk of heart disease. In our view, this effect is one of the most important benefits of exercise and fitness. If all this doesn't convince you to become active and improve your aerobic fitness, well, we'll just have to keep trying. For example, evidence indicates that it may be possible to lower serum cholesterol levels enough to reverse the process of atherosclerosis, removing fatty buildup from the lining of the coronary arteries (Ornish 1993). If it proves to be true that diet and exercise, perhaps with the help of drug therapy, can accomplish this reversal, it will be possible to prevent or cure, not just treat, many cases of the nation's number-one killer.

Increased Lean Tissue

Finally, let us remind you that muscle is the furnace that burns fat. Whereas dieting leads to loss of muscle, a lower metabolic rate, and reduced ability to exercise and burn fat, fitness training has the capacity to maintain or increase muscle mass and to burn more calories and more fat. Aerobic training such as running leads to a small increase in lean body weight. Training with more resistance, such as in cycling, can cause more noticeable changes in muscle. And, of course, muscular fitness (resistance) training leads to impressive changes in muscle mass. Fortunately, activity and training can rapidly reclaim muscle lost in dieting.

Whereas aerobic exercise doesn't have a great effect on the resting metabolic rate, resistance training has been shown to increase strength and metabolic rate and maintain metabolically active tissue in older adults (Campbell et al. 1994). It has been established that for every pound (.45 kg) of muscle that is added, basal metabolic rate increases by about 10 calories a day, which is about the equivalent of 1 pound of fat per year. Moreover, resistance training lowers visceral fat, the fat associated with a higher risk of heart disease (Treuth, Hunter, and Kekes-Szabo 1995).

Leptin

Leptin, a hormone produced by fat cells, helps regulate body weight and metabolism. Named after the Greek word *leptos*, which means thin, leptin influences hunger, feeding behavior, body temperature, and energy expenditure. Leptin stimulates lipid metabolism and increases energy expenditure, and it may limit excess energy storage. Leptin levels are correlated to the amount of fat stored in the body, although not all obese people have increased levels. Studies indicate that exercise lasting 1 hour or more reduces serum-leptin concentrations (Hulver and Houmar 2003). Exercise, diet, and sleep influence leptin levels. High-fat diets reduce the stimulatory effect of leptin on fatty-acid metabolism in skeletal muscle (Steinberg 2003). Sleep restriction is associated with decreased leptin levels, increased hunger, and greater appetite (Spiegel et al. 2004). Over the past 40 years, self-reported sleep duration in the United States has decreased 2 hours per night. Does lack of sleep contribute to overweight?