A look at the research on how men and women store and metabolize fat–and the implications for program design.

Gender Differences in Fat Metabolism

The incidence of obesity in the United States is on the rise. Approximately 20 percent (%) of U.S. adult females and 19% of U.S. adult males are currently obese (Centers for Disease Control and Prevention 2002). Why? Primarily because, for most individuals, energy intake constantly exceeds energy expenditure. According to Blair and Nichaman (2002), a decrease in regular physical activity—not an increase in energy intake—is responsible.

The rising incidence of obesity has sparked an increasing interest in the determinants of fat metabolism (the complete breakdown of fat into usable energy), both at rest and during exercise. Enhancing fat metabolism has become a key component in the battle of the bulge for many people. However, current research shows there may be gender differences in the way human beings store and metabolize fat. What does the research say about the differences between men and women and the mechanisms that may be involved? And how can you design programs that maximize caloric expenditure and fat metabolism for *all* your clients?

BY CHANTAL A. VELLA, MS, AND LEN KRAVITZ, PHD

Glossary of Terms

ADIPOCYTES: cells that act as storage sites for fat in the body.

ALBUMIN: a blood protein that is the main transporter of free fatty acids (FFAs) to the muscle cells.

ALPHA RECEPTORS: epinephrine receptors that act to inhibit lipolysis.

BETA RECEPTORS: epinephrine receptors that act to stimulate lipolysis.

EPINEPHRINE: the primary hormone that stimulates lipolysis. Epinephrine binds to receptors on various cells throughout the body and can either activate or inhibit hormone-sensitive lipase.

FAT METABOLISM: the complete biological breakdown, or oxidation, of fat into energy that the body can use.

FAT MOBILIZATION: the process of releasing fat from storage sites in the body.

FATTY ACID BINDING PROTEIN (FAPB), FATTY ACID TRANSLOCASE (FAT) AND FATTY ACID TRANSPORT PROTEIN (FATP): the three proteins located on the muscle cell membrane that bind FFA molecules and transport them across the cell membrane to the mitochondria.

GROWTH HORMONE (GH): a hormone that inhibits the uptake of glucose by active tissues and increases the mobilization of FFAs from adipose tissue.

HORMONE-SENSITIVE LIPASE (HSL): one of the two main enzymes that regulate the mobilization of FFAs. HSL is located directly in the adipocytes. When stimulated by the hormone epinephrine, it breaks apart triglycerides in the adipose tissue.

INTRAMUSCULAR TRIGLYCERIDES (IMTG): fat "droplets," or storage sites for fat within the muscles. IMTGs are an important source of fuel during moderate-to-high-intensity exercise.

LIPOLYSIS: the process by which triglycerides in fat cells, muscle cells and the blood are broken down and released into the bloodstream, where they are available for use as fuel for exercising muscles.

LIPOPROTEIN LIPASE (LPL): the other main enzyme that regulates the mobilization of FFAs. LPL is located on blood vessel walls throughout the body. Often referred to as the "gatekeeper," it is responsible for the breakdown of triglycerides in the bloodstream—for storage in adipose tissue or for use as fuel for active tissues.

TRIGLYCERIDES (TGS): the form in which fat is stored in the body. TGs are composed of three FFA molecules held together by a molecule of glycerol.

FAT STORAGE

Fat is stored in the body in the form of **triglycerides (TGs)**. TGs are made up of three **free fatty acid (FFA)** molecules held together by a molecule of glycerol (not a fat but a type of alcohol) (Robergs & Roberts 1997). Most body fat is stored in fat cells called **adipocytes.** Typically, about 50,000 to 60,000 kilocalories (kcal) of energy are stored as TGs in fat cells throughout the body (Coyle 1995). Fat can also be stored as "droplets" within skeletal muscle cells. These fat droplets, called **intramuscular triglycerides (IMTGs),** may hold 2,000 to 3,000 kcal of stored energy. In addition to the TGs stored throughout the body, some TGs travel freely in the blood. During exercise, TGs in fat cells, muscle cells and the blood can be broken down and used as fuel by the exercising muscles.

GENDER DIFFERENCES IN FAT STORAGE

Women generally have a higher percentage of body fat than men. A healthy range of body fat for women 35 to 55 years old is 25% to 32%, whereas a healthy range for men the same age is 10% to 18% (Heyward 2002). For this age group, a body fat percentage of over 25% for men or over 38% for women is considered an indication of obesity.

Body fat distribution varies among individuals, and the way it is distributed is a determinant of cardiovascular risk. (See "Determining Body Type and Cardiovascular Risk" sidebar.) Some people carry more body fat in and around the abdominal area. This type of fat deposition—referred to as android, or apple-body, type—is most characteristic among males and is associated with a higher risk of cardiovascular disease. The body type most common among females is gynoid, or pear-body, type, characterized by fat stores in the hip and thigh region (Robergs & Roberts 1997). The scientific explanations for the dramatic differences in body fat distribution between men and women are largely unknown, although differences in hormones, hormone receptors and enzyme concentrations play a role. (These mechanisms are discussed in the "Gender Differences in Fat Mobilization" section.)

FAT MOBILIZATION AND METABOLISM

The **mobilization** of fat refers to the process of releasing fat from storage sites in the body. The **metabolism** of fat is the complete biological breakdown, or oxidation, of fat into energy that can be used by the body.

Two main enzymes regulate the mobilization of FFAs: **hormone-sensitive lipase (HSL)** and **lipoprotein lipase (LPL).**

The Role of HSL. HSL is located directly in the fat cells and is stimulated by the hormone epinephrine. When HSL is stimulated,

Determining Body Type and Cardiovascular Risk

A person's body type is recognized as an important predictor of risk for hypertension (high blood pressure), hyperlipidemia (high cholesterol), coronary heart disease, type 2 diabetes and premature death (American College of Sports Medicine 2000). People with more body fat in the abdominal area have a higher risk of developing the above conditions than people who are equally fat but have most of their fat in the hip and thigh regions.

There are two ways to determine body type and health risk: waist-to-hip ratio and waist circumference.

WAIST-TO-HIP RATIO. You can measure a client's waist-to-hip ratio in inches or centimeters. To calculate waist-to-hip ratio, divide the circumference of the waist by the circumference of the hips. Using a measuring tape, measure the smallest part of the waist (usually above the navel and below the chest) and then the largest part of the hips. Make sure the measuring tape is horizontal all the way around the body.

The standards for risk vary with age and sex. Ratios above 0.94 for young men and 0.82 for young women place the individual at very high risk of disease. For ages 60 to 69 years, ratios indicating very high risk are above 1.03 for men and 0.90 for women.

WAIST CIRCUMFERENCE. An expert panel on obesity and health risk recently developed the waist circumference measurement as an indicator of health risk. Measure the client's waist circumference as discussed above. A healthy waist circumference is below 40 inches (102 centimeters) for men and 34 inches (88 centimeters) for women (American College of Sports Medicine 2000).

it acts to break apart TGs in the adipose tissue and release the three FFA molecules and glycerol into the bloodstream. This process is called **lipolysis.**

Epinephrine, which is released by the sympathetic nervous system during exercise, is the primary stimulator of lipolysis (Rasmussen & Wolfe 1999). Epinephrine binds to specific receptors on the fat cell, which in turn activate HSL. (See "The Role of Epinephrine" below.) An individual's physiological state can affect the body's sensitivity to epinephrine. For example, during aerobic exercise, HSL responsiveness to epinephrine is enhanced because body temperature rises. (There is also a greater concentration of epinephrine in the bloodstream during exercise.) In addition, in an endurance-trained individual, HSL can be activated by a lower concentration of epinephrine than in a non-endurance-trained individual. Therefore, a metabolic training effect of aerobic exercise is an enhanced ability to mobilize and break apart TGs for energy use. In contrast, obesity blunts the HSL responsiveness to epinephrine, making a higher concentration necessary to activate HSL (Rasmussen & Wolfe 1999).

Once in the bloodstream, the FFA molecules bind to **albumin,** a blood protein that is the main transporter of FFA molecules. (FFA molecules require a protein carrier to transport them to cells and within the bloodstream.) Once the FFA molecules are transported to the muscle cell, they are released from albumin and carried across the muscle cell membrane by specific transporters.

Three main FFA transporters located on the muscle cell membrane take over at this point: **fatty acid binding protein (FABP), fatty acid translocase (FAT)** and **fatty acid transport protein (FATP)** (Turcotte et al. 1999). These proteins bind to the FFA molecules and transport them across the cell membrane to the mitochondria for complete oxidation. Aerobic training can increase the number of FFA transporters on the muscle cell membrane, thus enhancing the ability to metabolize fat.

The glycerol molecule released from the process of lipolysis is circulated to the liver for oxidation and is used either as an intermediate in the breakdown of glucose or to make more TGs (Robergs & Roberts 1997).

The Role of LPL. LPL, the second enzyme that regulates the mobilization of FFAs, is located on blood vessel walls throughout the body. Both adipose tissue and the liver have large quantities of this enzyme. LPL acts on TGs within **lipoproteins,** special transporters in the bloodstream that carry cholesterol and TGs through the bloodstream to fat storage depots and body cells for fuel and cellular life-support needs. The TGs are broken down to FFA molecules and used as fuel by active tissues, or they diffuse into fat and liver cells, where they are resynthesized into TGs and stored. LPL located near muscle tissue breaks down TGs for fuel, whereas LPL located near adipocytes breaks down TGs to store as fat. For this reason LPL is often referred to as the "gatekeeper," since it controls the distribution of fat in the various storage depots of the body (Pollock & Wilmore 1990).

The Role of Epinephrine. Epinephrine is the primary hormone that stimulates lipolysis (Rasmussen & Wolfe 1999). Epinephrine binds to receptors on various cells throughout the body, such as adipocytes and muscle cells, and can either activate or inhibit HSL (Blaak 2001). As explained above, when HSL is stimulated, it acts to break apart TGs in the adipose tissue and release the three FFA molecules and glycerol into the bloodstream.

The two main types of epinephrine receptors are **alpha receptors** and **beta receptors.** Epinephrine can stimulate lipolysis through the beta receptors and inhibit lipolysis through the alpha receptors (Blaak 2001). The type of receptor available and its sensitivity to epinephrine will determine the response of HSL in any given tissue. Alpha and beta receptors can be located on the same cells; the response of HSL depends on which type is more abundant and available for binding with epinephrine.

The Facts About Adipose Tissue

Adipose tissue is a form of connective tissue composed of cells (adipocytes) separated by a matrix of collagenous and elastic fibers. Body fat accumulates in two ways: (1) by filling existing adipocytes, causing an increase in their size (hypertrophy) and (2) by forming new fat cells (a process called hyperplasia). Fat stores normally increase from birth to maturity by a combination of hypertrophy and hyperplasia. Obese adults typically have 60 to 100 billion fat cells, compared to 30 to 50 billion for nonobese adults (Pollock & Willmore 1990). Early research indicated that fat cell number increased markedly during the first year of life, gradually until puberty and then markedly again for a period of several years, with the maximum number of cells becoming fixed by adulthood. Current evidence suggests that fat cell size and number can increase at any age. The exact mechanism for hyperplasia is unknown; however it is hypothesized that fat cells have a certain capacity, and once that capacity is reached, a new cell will be formed (Pollock & Willmore 1990). Fat cells can increase or decrease in size but once a fat cell develops, it is permanent and can be removed only by liposuction.

GENDER DIFFERENCES IN FAT MOBILIZATION

Research has shown that, in both men and women, abdominal adipocytes are more sensitive than hip and thigh adipocytes to beta receptor stimulation by epinephrine (Braun & Horton 2001). This finding suggests that fat around the abdominal area is easier to mobilize than fat in the hip and thigh area. Moreover, women tend to have a greater number of alpha receptors in the hip and thigh region (Blaak 2001), suggesting that, in this area, fat storage would be favored over fat mobilization. This difference in the type and number of cell receptors may be one of the mechanisms contributing to the differences in fat distribution between men and women (Blaak 2001).

Another mechanism contributing to gender differences may be the concentration of LPL in various tissues. Women have higher LPL concentration and activity in the hip and thigh region than in the abdominal region (Pollock & Wilmore 1990). As explained above, LPL located near adipocytes will break down TGs to store as fat when the body is not using them for fuel, so the higher concentration of LPL in the hip and thigh area may help explain why women tend to store more fat in this region than men do.

Finally, the female hormone estrogen may have a positive effect on fat mobilization. Research has suggested that estrogen may aid in the mobilization of fat from adipose tissue. Although the mechanisms are not fully understood, several theories have been proposed.

Estrogen Inhibits LPL (Ashley et al. 2000). Remember that LPL is responsible for breaking down TGs in the bloodstream and storing them in adipose tissue whenever they are not needed as fuel for active tissues.

Estrogen Enhances Epinephrine Production. A higher concentration of epinephrine would increase the activity of HSL, the hormone responsible for adipose tissue lipolysis.

Estrogen Stimulates the Production of Growth Hormone. Growth hormone (GH) inhibits the uptake of glucose (carbohydrate) by active tissues and increases the mobilization of FFAs from adipose tissue (Robergs & Roberts 1997). GH works by inhibiting insulin production from the pancreas and stimulating HSL (Ashley, Kramer & Bishop 2000). (Insulin is the main hormone that promotes the transport of *glucose,* rather than fat, into muscle cells to be used as energy; it is also a potent inhibitor of HSL.) Estrogen may enhance fat metabolism by increasing the production of GH and inhibiting the production of insulin, which would, in turn, decrease glucose metabolism and increase FFA utilization (Ashley, Kramer & Bishop 2000).

Estrogen Causes a Vasodilation in Blood Vessels. Estrogen increases the production of nitric oxide. This hormone, produced by cells that line the blood vessels, causes a relaxation of the smooth muscle that surrounds them, leading to vasodilation, or widening, of the blood vessels. It is not yet known if this vasodilation is specific to adipose tissue perfusion (flow of blood into the adipose tissue). If so, the higher blood flow to this tissue would increase the interaction between epinephrine and adipose tissue beta receptors, thereby enhancing FFA transport from adipose tissue to active muscles during exercise (Braun & Horton 2001).

GENDER DIFFERENCES IN FAT METABOLISM AT REST

The level of fat metabolism at rest is positively correlated with the size of fat cells in the body, with larger fat cells having a higher lipolytic activity (Blaak 2001). Earlier research hypothesized that women might have a higher resting fat metabolism than men because women typically store more body fat than men do. However, recent research has found that resting fat metabolism (adjusted for differences in lean body mass) is actually *lower* in women than in men (Nagy et al. 1996; Toth et al. 1998). Although the mechanisms are unclear, this finding suggests that a lower resting fat metabolism may contribute to the increased fat storage in women.

What Exercise Intensity Burns the Most Fat?

During low-intensity exercise, the majority of energy (kilocalories) comes from fat. As exercise intensity increases, the percent of energy derived from fat decreases. However, the absolute amount of energy derived from fat actually increases! As exercise intensity increases, so does total energy expenditure (caloric expenditure). Even though a smaller *percentage* of the energy expenditure is coming from fat, more kilocalories are burned from fat because there is a *greater absolute energy expenditure.* Therefore, expressing energy derived from fat as a percentage of energy expenditure without considering the total energy expenditure is misleading.

Another consideration is the effect exercise has on energy expenditure after the exercise bout is completed. Following high-intensity exercise, the rate of metabolism is elevated for slightly longer than it is after lower-intensity exercise, and more energy is expended as the body returns to homeostasis (resting condition). With regular aerobic exercise, this postexercise energy expenditure will contribute positively to weight loss goals.

GENDER DIFFERENCES IN FAT METABOLISM DURING EXERCISE

IMTGs, the fat storage sites within the muscles, are an important source of fuel during moderate-intensity exercise. It's estimated that up to 50% of fat oxidized during moderate to intense exercise (60/65% to 80% VO_2max) is derived from IMTGs (Robergs & Roberts 1997; Coyle 1995). Most of the rest of the oxidized fat comes from adipose tissue, and the least comes from TGs in the bloodstream.

IMTG lipolysis is similar to adipose tissue lipolysis. During exercise, increasing levels of epinephrine activate HSL to begin the breakdown of IMTGs. The FFA molecules released from IMTGs are located within muscle cells; therefore IMTGs can be transported directly to the mitochondria for oxidation (Robergs & Roberts 1997).

The majority of the research shows that, compared to men, women derive a greater proportion of their energy expenditure from fats during low-to-moderate-intensity exercise. Research is still discerning the possible mechanisms leading to this gender difference.

GENDER DIFFERENCES IN FUEL SELECTION

Most of the current research is finding that, during low-to-moderate-intensity exercise, women derive a greater proportion of energy from fat than men do. The studies outlined here used different protocols but arrived at similar conclusions.

Respiratory Exchange Ratio

One of the most common methods used to determine fuel selection is the respiratory exchange ratio (RER). The RER is a numeric index of carbohydrate and fat utilization based on a ratio of carbon dioxide produced to oxygen consumed. A lower RER is an indication of a greater fat metabolism, whereas a higher RER is an indication of a greater carbohydrate metabolism.

Current studies show that during low-to-moderate-intensity exercise, women maintain a lower RER than men do.

Tarnopolsky et al. (1990). Male and female subjects were matched for training status and performance experience. Throughout a 90-minute run at 65% VO_2max, females had significantly lower RER values than males, indicating an increased reliance on fats as fuel. The calculated energy expenditure (EE) from fat was 428.4 kcal for the women (42% of total EE) and 242.1 kcal for the men (20% of total EE).

Horton et al. (1998). During 2 hours of exercise at 40% VO_2max, women had significantly lower RER values than men. The percent of fat metabolized during exercise averaged 43.7% for the men and 50.9% for the women.

Blatchford, Knowlton & Schneider (1985). At both 45 and 90 minutes of treadmill walking at 35% VO_2max, untrained women had significantly lower RER values than untrained men. Both groups gradually increased the percent of fat metabolized during exercise, with the 90-minute values being 59% for the men and 73% for the women.

Froberg & Pedersen (1984). Women subjects exercised for a significantly longer period of time than age- and training-matched male subjects at 80% VO_2max. The women also had significantly lower RER values during exercise than the men. The researchers concluded that the superior performance in women was due to a greater reliance on fats as fuel and a sparing of muscle glycogen.

Muscle Glycogen Depletion

Muscle glycogen concentration is another common technique used to determine fuel utilization during exercise. Muscle glycogen is the storage form of carbohydrate located within the muscle cells.

Tarnopolsky et al. (1990). This study, which compared the RER values of trained males and females during a 90-minute run at 65% VO_2max, also compared the muscle glycogen depletion patterns. Although muscle glycogen levels were similar between males and females before the exercise bout, postexercise biopsy data indicated that glycogen depletion was 25% greater in males than in females. This was in agreement with the lower RER data reported for females, indicating a greater reliance on fats as fuel during submaximal exercise.

Epinephrine Concentrations

Studies examining the hormonal responses to exercise have reported that epinephrine concentrations during submaximal exercise

Gender Comparisons in the Current Research

	WOMEN	MEN
healthy body fat (for physically active people ages 35-55)	25%-32%	10%-18%
obesity (ages 35-55)	> 38%	> 25%
body type	gynoid (pear)	android (apple)
estrogen	may enhance fat mobilization and spare glycogen	not applicable
lipoprotein lipase	higher concentration in hip & thigh area	lower concentration in hip & thigh area
hormone-sensitive lipase stimulation	greater sensitivity to lipolytic action of epinephrine	lower sensitivity to lipolytic action of epinephrine
resting fat metabolism (adjusted for lean body mass)	lower	higher
fuel use during exercise of low to moderate intensity	higher fat metabolism, lower glycogen depletion	lower fat metabolism, higher glycogen depletion
intramuscular triglyceride storage	higher concentration	lower concentration
epinephrine during submaximal exercise	lower concentration	higher concentration
fatty acid transporters	higher number	lower number
blood free fatty acid & glycerol concentrations during submaximal exercise	higher concentration	lower concentration

are higher in men than in women. Assuming a lower RER response in women during exercise, these findings indicate that women may be more sensitive to the lipolytic actions of epinephrine and therefore able to metabolize fat more effectively.

Tarnopolsky et al. (1990). This study, which found lower RER values during, and less glycogen depletion after, submaximal exercise in females than in males, also found lower epinephrine concentrations in females.

Horton et al. (1998). During exercise at 40% VO_2max, epinephrine levels (as well as RER values) were significantly lower in women than in men, again suggesting that women have a greater sensitivity to the lipolytic action of epinephrine.

FFA and Glycerol Concentrations

As adipose tissue lipolysis increases, concentrations of FFAs and glycerol increase in the plasma (fluid portion of the blood). With this in mind, several investigators have studied the gender differences in plasma FFA and glycerol concentrations in response to submaximal exercise.

Blatchford, Knowlton & Schneider (1985). When males and females matched for training status exercised at 35% VO_2max, there were significant gender differences in FFA and glycerol concentrations. At both 45 and 90 minutes of exercise, plasma FFA values were higher in females than in males. In addition, plasma glycerol levels were significantly higher in females than in males at 45 minutes of exercise.

Horton et al. (1998). This study, which found lower epinephrine and RER values in women exercising at 40% VO_2max, also found significantly higher plasma FFA and glycerol concentrations in women.

Tarnopolsky et al. (1990). On the other hand, Tarnopolsky and colleagues, who reported significantly lower RER values in females during submaximal exercise, found that these lower RER values were *not* accompanied by an increase in plasma concentration of FFAs or glycerol. The researchers hypothesized that the increase in fat metabolism in women was due to a higher utilization of IMTGs (which do not increase plasma concentrations of FFAs or glycerol), as opposed to a greater adipose tissue lipolysis.

These findings on gender differences in plasma FFA and glycerol concentrations suggest that in women, a higher percentage of fat metabolism during exercise may be due to an increase in beta receptor sensitivity (which would stimulate lipolysis), a decrease in alpha receptor sensitivity or an increase in the utilization of IMTGs.

In addition, it has been reported that IMTG stores are higher in women than in men (Blaak 2001; Braun & Horton 2001). This finding suggests the possibility that higher IMTG oxidation may contribute to the increased fat oxidation and glycogen sparing in women during exercise. It has also been reported that women have a higher expression of FFA transport proteins (FABP, FAT, FATP) in skeletal muscle cells (Blaak 2001). An increase in these transport proteins would augment the number of FFA molecules entering the muscle cells, thereby increasing the FFAs available for oxidation in the mitochondria or storage in IMTGs.

THE BOTTOM LINE

Distinct differences exist in the mobilization, metabolism and storage of fat between genders. Most of the current research is finding that in women, the proportion of energy derived from fat is greater during exercise of low to moderate intensity. Additional research is needed to determine the exact mechanisms involved and also to ascertain why the increase in fat metabolism is evident during exercise but not at rest. Differences in percent body fat, distribution of body fat, hormonal responses to exercise and hormone receptor type and sensitivity may all play a role.

DESIGNING PROGRAMS TO ENHANCE FAT METABOLISM

Although the current research suggests differences in fat utilization between males and females, closer inspection of the data also shows much variation within each gender. The following exercise guidelines may help enhance fat metabolism for both genders. For optimal cardiorespiratory program design, try the following suggestions with your clients and progressively individualize the exercise plan according to results.

The foundational research on the development and maintenance of cardiorespiratory fitness recommends performing endurance exercise 3 to 5 days per week, using an exercise mode that involves the major muscle groups (in a rhythmic nature) for a prolonged time period. The American College of Sports Medicine (ACSM) recommends an exercise intensity of between 55% and 90% of maximum heart rate (or 40% to 85% of oxygen uptake reserve), with a continuous duration of 20 to 60 minutes per session (American College of Sports Medicine 2000). Inherent in these recommendations is the concept of individualizing the program for each person's fitness level, health, age, personal goals, risk factor profile, medications, behavioral characteristics and individual preferences. The ACSM recommendations appropriately serve as the framework for the guidelines that follow.

This review of literature leads us to believe that the concept of **periodization,** which has become so popular in resistance training, may also be helpful in designing cardiorespiratory training programs aimed at increasing fat metabolism. Periodization training is based on an inverse relationship between intensity and volume (Stone et al. 1999). With cardiorespiratory exercise, intensity can be individualized by using percent heart rate max, percent VO_2max or rating of perceived exertion; volume can be individualized by session duration and frequency.

The following periodization suggestions can help you design individual cardiorespiratory training programs with the goal of optimizing fat metabolism.

1. Regularly Incorporate Low-Intensity, Long-Duration Workouts. *Rationale:* Although the majority of the research shows that, compared to men, women derive a greater proportion of their energy expenditure from fats during exercise of low to moderate intensity, low-intensity, long-duration exercise will also improve fat utilization in men. (The actual numbers may vary widely among individuals.)

2. Incorporate Some Higher-Intensity, Shorter-Duration Workouts. You can do this with either continuous training or interval training. *Rationale:* Although the percent of energy derived from fat decreases as exercise intensity increases, the absolute amount of energy derived from fat actually increases—for males and females. (See "What Exercise Intensity Burns the Most Fat" sidebar.)

3. Cross Train. Incorporate various modes of training (Kravitz & Vella 2002). *Rationale:* The theory of cross training implies that using different modes of exercise keeps the body from getting overly fatigued and prevents muscle overuse, because the same muscles are not constantly worked in the same movement patterns. Cross training helps reduce the occurrence of musculoskeletal system stress, thereby aiding in the prevention of muscle soreness and injuries. Therefore, theoretically, a person will be able to safely do more work more frequently, resulting in higher total energy expenditure and fat utilization.

4. Regularly Vary the Intensity and Volume of Workouts. Help clients find ways to vary their cardiorespiratory workouts—within each week, weekly, biweekly or across any combination of these timelines. For example, some weeks, have clients perform two low-intensity cardiorespiratory workouts at different times in the same day—perhaps one short and one slightly longer in duration. This will considerably increase the weekly volume of endurance exercise. Other weeks, have clients do fartlek training, in which you spontaneously vary the intensity and volume within each workout. *Rationale:* Varying the workouts provides a new stimulus to the body's cardiorespiratory system and lowers the risk of overuse exercise fatigue.

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