Gatorade Sports Science Institute®

Sports Science Exchange 72

VOLUME 12 (1999) ■ NUMBER 1

EXERCISE, MACRONUTRIENT BALANCE, AND BODY WEIGHT REGULATION

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KEY POINTS

- The prevalence of obesity has increased substantially in North America and in other developed countries during the past 20 years, a time in which the gene pool has not changed. This phenomenon argues strongly for the importance of dietary and physical activity patterns interacting with genetic polymorphism to explain the accumulation of excess body fat in a large portion of the American population.
- The rates of carbohydrate and protein oxidation are closely regulated by the magnitude of their respective intakes, but fat oxidation is not closely coupled to acute fat intake. Thus, the accumulation of excess body fat appears to be fundamentally a problem resulting from excessive fat intake relative to fat oxidation.
- If energy intake exceeds expenditure, it is possible for an individual to become obese even on a low-fat diet—not usually because of substantial synthesis of fat from carbohydrate, but rather because much of the dietary fat is stored as the body rapidly adjusts to oxidizing carbohydrates and protein to meet its energy needs, at the expense of fat oxidation.
- Humans are much more likely to overeat and to exhibit positive fat balance when the diet is high in fat.
- The effect of exercise on total daily energy expenditure results more from the increased energy expenditure during exercise than from its impact on resting metabolic rate or the thermic effect of food.
- Because exercise can increase total daily energy expenditure and fat oxidation, chronic exercise may substitute for expansion of the adipose tissue mass, allowing the physically active individual to achieve a balance in fat metabolism at a lower body fat mass.

INTRODUCTION

Obesity, especially that characterized by excessive deposition of abdominal body fat, is a risk factor for several untoward consequences, including cardiovascular disease and type-2 diabetes mellitus. Despite heightened public awareness in North America of the untoward health consequences of being overweight, the prevalence of obesity in adults, children, and adolescents is increasing (Kuczmarski et al., 1994). Obviously, with the increasing prevalence of obesity and its link to unfavorable outcomes, there is considerable interest in the prevention and treatment of obesity, with diet and physical activity being two clearly important factors. This paper will examine the impact of physical activity on energy and macronutrient balance and on regulation of body weight.

FACTORS CONTRIBUTING TO INCREASED RISK OF OBESITY

The laws of thermodynamics dictate that an energy surplus is at the root of all obesity; if energy intake (E_{IN}) exceeds energy expenditure (E_{OUT}), then energy storage will occur. While this may seem obvious, it is important to state this at the outset. With all the recent attention devoted to claims about weight loss resulting from diets varying in macronutrient composition (e.g., high-carbohydrate diets, low-carbohydrate diets, high-protein/high-fat diets), one must not forget that diet composition will only affect long-term changes in body composition when there is an imbalance in E_{IN} versus E_{OUT}.

Genetic vs Environmental Contributors

Specific risk factors associated with increased weight gain in adults have been identified in some populations such as Caucasians and Pima Indians (Ravussin & Swinburn, 1993). An obvious risk factor is excess energy intake relative to expenditure, and the highly palatable, energy dense food supply in North America is undoubtedly a contributor to the high prevalence of obesity. Other identified risk factors include: 1) a low rate of resting energy expenditure relative to the rate predicted on the basis of body size; 2) high 24-h respiratory quotient (RQ), indicative of a higher 24-h carbohydrate oxidation and lower fat oxidation; and 3) low level of spontaneous physical activity. There are insufficient data available at this time to determine whether these risk factors for weight gain are characteristic of other population groups.

There has been substantial research interest in recent years regarding the role of genetic factors in the etiology of obesity. The identification and sequencing of the ob gene, the peptide (leptin) that it encodes, and the discovery that a defect in this gene appears to be the single cause of obesity in the ob/ob mouse (Zhang et al., 1994) has generated considerable interest in the genetics of obesity. However, it is noteworthy that the prevalence of overweight and obesity in North America have increased substantially even within the last 20 years, a time in which the gene pool has not changed. This phenomenon argues strongly for the importance of behavioral factors such as dietary and physical activity patterns interacting with genetic factors to explain the accumulation of excess body fat in a large portion of the North American population.

Physical Inactivity and Obesity

Intuitively, one would expect that individuals who are sedentary would exhibit greater risk for excess body fat accumulation compared to their physically active counterparts. Inverse relationships between measures of physical activity (usually self-reports) and indices of obesity (usually body mass index, or BMI) in the U.S. population have been reported (Eck et al., 1992). Among African-Americans, Melby et al. (1991) found that adults who exercised two or more times per week had significantly lower values for body mass index (weight/height^2) and waist circumference than did adults who exercised once per week or less. In a recent report of a cohort of over 5,000 men and women in Finland followed for a 10-year period, a

decreased level of leisure-time physical activity was associated with large gains in body mass in comparison with those individuals who increased their level of physical activity (Haapanen et al., 1997). While there are strong associations between physical inactivity and obesity, there are insufficient data to conclude that a low level of physical activity is a cause of obesity. It is equally possible that a low level of physical activity is a consequence of obesity. Also, if low energy expenditure due to physical activity were accompanied by low energy intake such that energy balance were maintained, obesity would not result. Note, however, that access to the highly palatable, energy dense diet available in North America and many other parts of the world would make positive energy and fat balance much more likely in the face of low energy expenditure associated with physical activity.

Diet and Macronutrient Oxidation

Body Fat Mass and Fat Balance. Flatt (1995) and Schutz (1995) have suggested that obesity is fundamentally a problem caused by a low rate of fat oxidation relative to fat ingestion, leading to expansion of the adipose tissue mass. They theorized that the increased adiposity, which may or may not be coupled with increased resistance of fat cells to the actions of insulin, increases plasma free fatty acids, which in turn enhances cellular lipid oxidation. When the fat mass has increased sufficiently, fat balance will be reestablished, and the fat mass will stabilize (Figure 1). These changes allow the body to reach a new equilibrium, a point at which the rate of fat oxidation and intake are equal, albeit at a cost of obesity. Similarly, when fat intake is less than fat oxidation, the body fat mass will decline to bring about a lower fat oxidation.

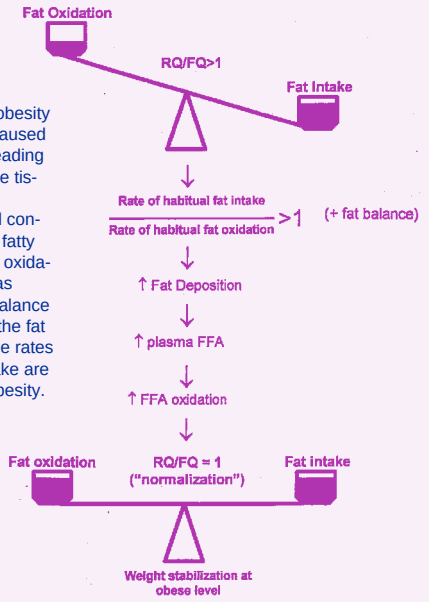

Figure 1. Fundamentally, obesity appears to be a problem caused by a positive fat balance leading to expansion of the adipose tissue mass. The increased adiposity leads to elevated concentrations of plasma free fatty acids, which enhance lipid oxidation. When the fat mass has increased sufficiently, fat balance will be reestablished, and the fat mass will stabilize when the rates of fat oxidation and fat intake are equal, albeit at a cost of obesity.

Macronutrient Intake and Fat Balance. While the focus in this paper is on exercise and macronutrient oxidation rather than diet, it is important within the context of weight regulation to briefly examine the impact of energy intake and diet composition on macronutrient balance. It has been previously shown that carbohydrate oxidation is strongly influenced by carbohydrate intake, such that increased carbohydrate consumption leads to marked increases in postprandial carbohydrate oxidation (Acheson et al., 1988). Long-term overfeeding of carbohydrate results in a dramatic increase in daily carbohydrate oxidation and suppression of fat oxidation. In a similar fashion, an elevation in protein intake results in increased amino acid oxidation rather than storage. The small increases and decreases in body protein content result in corrective responses by way of increases and decreases in amino acid oxidation. Thus, the body maintains protein balance by closely matching protein oxidation to intake, in a manner similar to carbohydrate intake and oxidation. Because protein contributes proportionately far less to energy expenditure during exercise than carbohydrate or fat, little attention will be devoted to protein in this paper.

The addition of fat to a mixed meal does not significantly increase the rate of fat oxidation during the postprandial period (Schutz et al., 1989). It appears that under conditions in which up to 40-50 g of fat are included in a mixed meal, the partitioning of dietary fat is toward storage rather than immediate oxidation. In fact, consumption of a mixed meal containing both fat and carbohydrate will result in a decrease in fat oxidation relative to the preprandial condition.

It is evident then, that alterations in carbohydrate intake produce rapid and substantial changes in carbohydrate oxidation that serve to maintain carbohydrate balance. However, alterations in fat intake produce little, if any, immediate changes in fat oxidation, so that there is little effort to maintain fat balance acutely. Thus, weight changes following challenges to energy balance are due primarily to disruptions in fat balance, which accounts for most of the imbalance produced in total energy. Even if bodily carbohydrate stores were doubled, body mass would not typically increase by more than 1-2 kg. Because carbohydrate and protein balance are maintained over a short period of time, any surplus of dietary energy beyond a few days must necessarily be accommodated by expansion of the fat stores.

It is important to recognize that most fat storage in humans eating mixed diets is not caused by net synthesis of fat from carbohydrate sources (Horton et al., 1995). Rather, the positive fat balance that occurs with overfeeding appears to result from greater storage of dietary fat relative to whole-body fat oxidation. *If energy intake exceeds expenditure, it is possible for an individual to become obese even on a low-fat diet—not usually because of substantial synthesis of fat from carbohydrate, but rather because most of the dietary fat is stored as the body rapidly adjusts to oxidizing carbohydrates and protein to meet its energy needs, at the expense of fat oxidation.*

Integration of Macronutrient Intake and Oxidation. Because the human body acutely adjusts the rates of oxidation of carbohydrate and protein to match their intakes, the amount of fat oxidized in a given time period will be the difference between total energy expenditure and energy expenditure due to protein and carbohydrate oxidation. When energy balance and stable body composition prevail, the fuel mix oxidized (as represented by the respiratory quotient, or RQ) equals the macronutrient composition of the diet, as reflected by the food quotient (FQ), which is calculated as the ratio of carbon dioxide produced to oxygen consumed during the oxidation of foods representative of the habitual diet. During days of energy surplus on a regular mixed diet, the fuel mix oxidized will be higher in carbohydrate, and fat oxidation will decrease (RQ > FQ), with the surfeit of energy largely stored as fat. When food intake is inadequate to meet the body's energy demands, the average RQ is lower than the FQ, which reflects the increased oxidation of both dietary and endogenous fat. To lose body fat, the average RQ/FQ must be less than 1.0. While dietary restriction can accomplish this, it should be apparent that increasing energy expenditure and fat oxidation via regular exercise could also yield an RQ/FQ< 1.0. The role of exercise in weight loss and maintenance of fat balance at a low percent body fat will be discussed in subsequent sections.

In exploring diet composition and food intake, Flatt (1995) has suggested that carbohydrate balance regulates eating behavior and macronutrient selection. This theory is based on observations of mice, in which negative carbohydrate balance on one day is followed by increased carbohydrate intake on the following day, presumably to restore hepatic glycogen levels to some regulated level. Accordingly, weight stability in which the RQ/FQ =1.0 is achieved over time based on modulation of food intake. If this were true of humans, in those individuals with a high daily rate of carbohydrate oxidation and a low daily rate of fat oxidation, the drive to adjust carbohydrate intake to match carbohydrate oxidation over the course of several days will necessarily result in overfeeding when the meals are of high-fat content. In other words, carbohydrate balance is achieved by regulating carbohydrate intake, at the expense of positive fat balance when the diet is rich in fat. This concept is supported by the results of some studies of humans in whom dietary carbohydrate was displaced by fat (Tremblay et al., 1989), but not by the results of others (Stubbs et al., 1993). In the latter two studies, dietary manipulation of glycogen stores had little impact on subsequent ad-libitum food intake. However, energy and fat balance were profoundly positive

when men were given ad-libitum access to high-energy, high-fat diets for 7 days, whereas energy balance was slightly negative for these same men fed low-fat, high-carbohydrate diets (Stubbs et al., 1993). Stubbs et al. (1993) suggested that carbohydrate balance is maintained, not by modulation of carbohydrate intake, but rather by adjustments of RQ to FQ. In a recent study of overfeeding and underfeeding assessed for 12 consecutive days in a room calorimeter, this same research group (Jebb et al., 1996) concluded that metabolic fuel selection is dominated by the need to maintain carbohydrate balance, which results in inappropriate counterregulatory alterations in fat oxidation during a surplus of energy. An important finding from these studies and others (Tremblay et al., 1989) that must not be overlooked is that humans are much more likely to overeat and to exhibit positive fat balance when given opportunity for ad-libitum feeding from a palatable diet high in fat.

Regardless of how closely carbohydrate balance regulates carbohydrate intake, it is appears that overfeeding occurs much more readily on a high-fat diet. Such a diet would clearly increase risk for obesity, especially among individuals who also exhibit a relatively low daily rate of fat oxidation. An excess of calories and fat in the diet does not acutely increase fat oxidation, so a positive fat balance occurs that leads to fat storage. It follows that a lower average rate of fat oxidation could be a predisposing factor to body fat accumulation if dietary fat were widely available, whereas a higher rate of fat oxidation could impart some protection against obesity. However, the key to understanding body fat accumulation or loss, is fat balance not fat oxidation by itself or fat intake by itself. For example, a weight-stable endurance athlete who consumes a diet containing 70% of the energy from carbohydrate and only 15% from fat will exhibit a relatively high 24-h RQ. (This is necessarily so because the weight-stable athlete will exhibit macronutrient balance over time in which the RQ and the FQ are the same.) The high 24-h RQ reflects a low rate of oxidation of fat relative to carbohydrate, but it does not indicate that the athlete is at risk for fat accumulation. In this case, the low rate of fat oxidation is matched by an equally low rate of fat intake, so there is no net fat deposition. The reverse can also be true. An individual who consumes 40% of dietary energy as fat is not destined to become obese if fat balance is maintained by an equally high proportion of fat oxidation. It should be understood that focusing on fat oxidation alone or fat intake alone is insufficient to understand fat balance.

EXERCISE AND ENERGY EXPENDITURE

Despite the lack of unequivocal data regarding low levels of physical activity as a cause of obesity, there are several ways by which physical activity can have an impact on energy balance. Of the three components of daily energy expenditure (resting metabolic rate (RMR), the thermic effect of feeding (TEF), and the thermic effect of physical activity (TEPA), the obvious component of energy expenditure that is altered by changes in physical activity is TEPA. The energy expended in physical activity will vary with the characteristics of both the exercise (i.e., frequency, intensity, and duration) and the subject (body weight, aerobic capacity, etc.).

Energy expenditure does not return to baseline levels immediately following physical activity. The magnitude and duration of this postexercise energy expenditure is controversial (Poehlman et al., 1991). Exercise intensity affects the magnitude of the postexercise elevation of metabolic rate more than does exercise duration (Sedlock et al., 1989). The intensity and duration of the types of exercise sessions engaged in by most nonathletes (40-70% VO_2max for 15-40 min) typically result in a return of energy expenditure to baseline values within 5-40 min following exercise; this accounts for only 21-125 additional kJ of energy expended beyond the exercise bout itself (Freedman-Akabas et al., 1985). In individuals capable of performing high-intensity, long-duration exercise, the postexercise energy expenditure may be higher and could be a significant contributor to total daily energy requirements. Less is known about the effects on postexercise energy expenditure following resistance exercise, but recent data suggest that high-intensity weightlifting exercise may elevate energy expenditure above baseline levels for several hours (Melby et al., 1993). However, it appears doubtful that individuals untrained in such intense resistive exercise would be capable of sustaining the exercise intensity necessary to produce a prolonged elevation of postexercise energy expenditure.

Changes in chronic physical activity may influence the other components of energy expenditure, specifically the TEF and RMR. The thermic effect of food is caused by the digestion, absorption, and assimilation of macronutrients (obligatory thermogenesis) as well as by additional energy expenditure probably resulting from increased activity of the sympathetic nervous system; TEF is generally considered to account for about 10% of daily energy expenditure. It is likely that the effect of exercise on TEF is fairly small, with the benefits of exercise on weight control resulting more from the increased energy expenditure during exercise, rather than from its impact on this specific component of 24-h energy expenditure.

Resting metabolic rate (RMR) is the energy cost of maintaining life processes in an awakened state, and it typically accounts for 65-75% of total daily energy expenditure. It is unclear whether or not changes in physical activity alter RMR independently of changes in fat-free mass. Conflicting data have been reported. In several cross-sectional investigations, an elevated RMR was seen in endurance-trained individuals compared to sedentary, untrained subjects, independent of differences in body composition (Poehlman, 1989). Arciero et al. (1993) compiled data on more than 500 healthy men and women and reported that peak VO_2 was a significant predictor of RMR, independent of body weight and body composition. Other studies have found that highly fit, trained subjects have RMR values no different from those of sedentary controls (Broeder et al., 1992). The reasons for the discrepant findings are unknown, but they may be related to differences between studies in the time interval between the last bout of exercise and measurement of RMR and to the level of energy intake during the days immediately preceding measurement of RMR. Furthermore, it is impossible to determine cause-and-effect relationships from cross-sectional analyses.

From earlier studies, it was suggested that the combination of high energy expenditure and energy intake (high energy flux or turnover) could elevate RMR in endurance-trained athletes, even when they are in energy balance (Poehlman et al., 1989). Data from our laboratory support this hypothesis (Bullough et al., 1995). In trained versus untrained subjects, RMR was elevated under acute conditions of high exercise energy expenditure and high energy intake, but this elevation was attenuated as the time interval increased from the last exercise bout to the measurement of RMR. These data suggest that RMR may be chronically elevated in individuals who engage in daily, high-intensity, prolonged exercise and that this is caused by an effect of acute exercise rather than by an adaptation to chronic exercise. It should be noted that the amount of exercise performed by nonathletes for the purpose of weight control is typically of much lower intensity and duration and would likely have little, if any, impact on RMR.

In summary, the thermic effect of physical activity changes directly with changes in physical activity. Whether or not other components of energy expenditure are affected by changes in physical activity is controversial. The major impact of physical activity on energy expenditure occurs during the activity itself. Note also that the major impact of physical activity on the risk of obesity results from its effects on total energy expenditure and thus energy balance, rather than to the proportion of fat versus carbohydrate oxidized during acute exercise bouts.

EXERCISE AND MACRONUTRIENT OXIDATION

Arguably, an increase in physical activity is the most viable approach for those attempting to increase energy expenditure and fat oxidation in order to decrease body fat stores. There are two different sources of fat for oxidation during exercise—plasma free fatty acids mobilized from adipose tissue and fatty acids stored in intramuscular triacylglycerol. Adipose tissue contains an abundance of triacylglycerol, easily enough to fuel an 800 km walk in even a lean person. Obviously, for use during exercise, adipocyte triacylglycerol must undergo lipolysis and transport to the mitochondria in skeletal muscle where oxidation occurs.

Acute Exercise

Much of the information regarding fat mobilization and oxidation during exercise at various intensities comes from research using isotopic tracers combined with indirect calorimetry. During low-intensity exercise (e.g., walking at an oxygen uptake of 25% VO_2max), there can be up to a five-fold increase compared to resting conditions in the rate of appearance of FFA (R_a FFA) in the plasma (Klein et al., 1994). For an individual in a fasted state who exercises at this intensity, the oxidation of the plasma FFAaccounts for a majority of the energy needed for exercise, with much smaller contributions from blood glucose and intramuscular triacylglycerol. As the exercise intensity increases from 25% to about 65% VO_2max (a jogging/running pace that can be sustained for two or more hours), muscle glycogen contributes substantially to meeting the energy demand. Tracer studies have shown that the rate of FFAappearance in the plasma decreases with this increase in exercise intensity, but total fat oxidation as measured by indirect calorimetry (i.e., VO_2 and RER measurements) actually increases (Romijn et al., 1993). The most logical explanation for this phenomenon is that intramuscular triacylglycerol provides the additional fatty acids to account for this increase in fat oxidation (Martin, 1997). As the exercise intensity increases to about 85% VO_2max, muscle glycogen serves as the primary fuel. Plasma concentrations of FFAactually decrease as the rate of appearance of FFA (R_a FFA) decreases even further (Romijn et al., 1993). The explanation for the declining R_a FFAis not clear. One might surmise that because the concentration of plasma catecholamines increases dramatically with high-intensity exercise, lipolysis should also be occurring at a high rate, leading to an increase in the R_a FFA. Tracer studies using labeled glycerol suggest that, indeed, a substantial rate of lipolysis occurs during high-intensity exercise (Klein et al., 1996), but for unexplained reasons most of the adipocyte FFAfails to enter the plasma (i.e., the R_a of glycerol is high, but the R_a FFAis low). Coyle (1995) suggested that the reduced blood flow to adipose tissue during high-intensity exercise may result in inadequate plasma albumin concentrations needed to mobilize the adipocyte FFA. Regardless of the reason, it is apparent that during high-intensity exercise, both plasma FFA and intramuscular triacylglycerol contribute less to meeting the energy demands than does skeletal muscle glycogen. Note that these studies examining fuel utilization at exercise intensities of 25%, 65%, and 85% VO_2max have used trained endurance athletes. Presumably, at each of the same exercise intensities untrained subjects would exhibit greater reliance on carbohydrate and less reliance on fat when compared to trained subjects.

It is well recognized then, that for exercise of high intensity compared to low intensity, a greater portion of the oxygen consumed is used for carbohydrate and less for fat oxidation during the exercise bout. Owing to this phenomenon, it is not uncommon for some fitness practitioners to suggest that low-intensity exercise is better than high-intensity for regulation of body weight and body composition, because the former produces greater fat oxidation. However, such advice fails to consider the greater energy expenditure with exercise of greater intensity, as well as the impact of high-intensity activity on postexercise substrate utilization. For example, Phelan et al. (1997) examined the effects of low- and high-intensity exercise of similar total energy output on energy expenditure during and after exercise and on substrate oxidation in eight active eumenorrheic females. They found that, as expected, carbohydrate oxidation was significantly greater for the high-intensity protocol (75% VO_2max) than for the low-intensity (50% VO_2max) activity. Total fat oxidation (exercise plus 3-h recovery) was greater during the low-intensity exercise treatment. However, the calculated rate of fat oxidation was 23.8 % higher 3 h after the cessation of high-intensity compared to low-intensity exercise. Other recent studies provide further evidence that acute glycogen-depleting exercise enhances fat oxidation during recovery. Schrauwen et al. (1997) tested the effect of glycogen-lowering exercise versus no exercise on fat oxidation during a 36-h postexercise period, during which time subjects were fed a high-fat (60% fat energy) diet. They found that subjects were capable of rapidly adjusting fat oxidation to fat intake when glycogen stores were lowered by exhaustive exercise, but during the 36-h when they consumed the high-fat diet without exercise, fat balance was substantially positive. Also, Tuominen et al. (1997) determined the rate of lipid oxidation under basal conditions and during a euglycemic, hyperinsulinemic clamp, 44 h following a bout of glycogen-depleting exercise compared to a control condition of no exercise in 14 males. There was a two-fold higher rate of fat oxidation in the basal state and almost a three-fold increase during hyperinsulinemia following the exercise compared to the no-exercise condition. Data from these studies demonstrate that glycogen-lowering exercise enhances fat oxidation for up to 44 h after exercise, which has important implications for fat balance. These data indicate that the recovery period must also be considered when determining the impact of different exercise intensities on total energy expenditure and fat and carbohydrate utilization.

Exercise Training

Exercise training increases the ability to use both fat and carbohydrate, with fat oxidation predominant at low and moderate exercise intensity, and carbohydrate utilization the dominant fuel for high intensity exercise. Brooks and Mercier (1994) have reviewed exercise macronutrient utilization based on the interaction between exercise intensity-induced responses and exercise training-induced adaptations. They describe the crossover point as the power output at which energy derived from carbohydrate predominates over energy derived from lipid, with further increases in exercise intensity producing incremental increases in carbohydrate utilization and concomitant decreases in fat oxidation. Exercise training produces a rightward shift in the crossover point, so that at low and moderate exercise intensity the training adaptations enable the individual to oxidize more lipid than can the untrained individual. During high-intensity exercise, the carbohydrate-related adaptations in the trained individual allow the utilization of large amounts of glucose needed for high power output. After exercise training, the same amount of submaximal work can be performed with a greater contribution of fat oxidation to meet the energy requirement (Gollnick, 1985). Increased fat oxidation in trained subjects is greatly facilitated by morphological and enzymatic adaptations in skeletal muscle following training, combined with increased availability of lipid substrate.

Resistance Exercise

Few studies have focused on the impact of acute resistance exercise on fat oxidation. It is well recognized that during weightlifting exercise, phosphocreatine and skeletal muscle glycogen are the major sources of fuel for ATP synthesis. However, there remains the possibility that during rest periods between sets of exercise and during the postexercise recovery period fat may contribute to energy needs. Melby et al. (1993) found the RQ to be lower 15 hours following a strenuous bout of weightlifting, which likely reflects greater fat oxidation at this point. Thus, it appears that fat oxidation is enhanced during recovery from resistance exercise as well as after other types of high-intensity exercise; this serves to spare available carbohydrate for glycogen resynthesis.

Exercise and Fat Mass

The fat oxidation associated with exercise (both during exercise and during recovery) appears to have significant implications for body weight regulation. Flatt (1995) suggests that exercise is a substitute for expansion of the adipose tissue mass, allowing the physically active individual to achieve fat balance at a lower body fat mass. In other words, exercise increases daily fat oxidation to become commensurate with fat intake, obviating the need to expand the body fat mass. He also suggests that the reverse is true, i.e., obesity is a substitute for exercise in bringing about fat balance and weight maintenance at the higher body weight. This is somewhat of an oversimplification of a complex issue because there are individuals who obviously do not exercise and yet maintain low body fat mass, and some obese individuals are regular exercisers. However, it does suggest the importance of exercise in elevating daily energy expenditure and fat oxidation so that in the face of a highly palatable diet, there would be less likelihood of establishing a positive fat balance that leads to excessive body fat storage.

EXERCISE AND ENERGY COMPENSATION

With the preceding discussion on the effects of exercise on macronutrient oxidation, it still must be understood that exercise contributes to weight loss only if it creates negative energy bal-

ance. Negative energy balance will not occur if energy intake increases to meet the energy demands brought on by exercise and/or if spontaneous physical activity declines. Factors that influence the degree of such energy compensation that may occur in response to exercise are not well understood, but they clearly have an important impact on energy balance.

In cross-sectional studies, physically active individuals generally exhibit higher energy intakes, but exercise may also invoke an immediate postexercise anorexia, especially at higher intensities of exercise (King et al., 1994). Unfortunately, such studies are often limited by the inability to accurately assess food intake in humans. There is yet much to learn regarding the effects of acute and chronic exercise on the intake of energy and macronutrients.

Surprisingly little is known about the impact of acute exercise on 24-h spontaneous physical activity. It seems reasonable that a vigorous bout of exercise might induce a degree of fatigue that could limit the amount of physical activity performed during the remaining hours of the day, as was seen in a group of elderly subjects (Goran & Poehlman, 1992). However, studies in lean and obese women (Meyer et al., 1991) found no reduction of spontaneous physical activity outside of the training hours in response to increases in exercise energy expenditure. Furthermore, in lean males and obese boys, exercise training appeared to actually stimulate physical activity during the nonexercising part of the day (Blaak et al., 1992; Meyer et al., 1991). Perhaps compensation in normal daily physical activities in response to exercise occurs more readily in older adults than in young adults. On the other hand, older subjects with physical limitations to strength, flexibility, agility, and stamina may benefit from strength training exercise that causes increases in spontaneous activity due to improvements in the strength and agility needed to carry out routine daily tasks. Currently, it is unclear what is the optimal mode and intensity of exercise that both maximizes exercise energy expenditure and minimizes compensatory decreases in spontaneous activity. Future research should address this important question.

EXERCISE VERSUS DIETING FOR WEIGHT LOSS

Either alone or in combination, many studies have addressed the contribution of diet and exercise to body weight loss. From these studies, it is clear that dieting produces greater weight loss than does exercise (Saris, 1995) and that exercise in combination with dieting often adds little to the weight loss achieved by dieting alone. These findings have led some investigators to discount the importance of exercise (Garrow, 1995). However, it is not surprising that exercise compared to dietary energy reduction would be less effective at promoting weight loss. It is unrealistic to expect that a sedentary person who initiates an exercise program can achieve the same magnitude of energy deficit that can be achieved by a low-energy diet. Thus, a greater and more rapid weight loss should occur with dieting compared to exercise. However, this does not mean that exercise has only a marginal impact on weight loss over an extended period of time. The trained individual is able to exercise at higher intensities for longer durations, which increases energy expenditure, and the trained person also has a greater capacity for fat oxidation. These training adaptations that occur over an extended period may enable the exercising individual to gradually lose fat mass and to successfully maintain weight loss initially achieved by dieting.

SUMMARY

The accumulation of excess body fat appears to be fundamentally a problem resulting from inadequate fat oxidation relative to intake. An increase in body fat mass increases the concentration of free fatty acids in the blood, and this increased availability of lipid fuel is associated with increased fat oxidation. The increase in fat utilization that accompanies obesity helps re-establish a new equilibrium between fat intake and fat oxidation that serves to stabilize body weight, albeit at the cost of obesity. Because exercise can increase total daily energy expenditure and fat oxidation, chronic exercise can help prevent expansion of the adipose tissue mass, allowing the physically active individual to achieve fat balance at a lower body fat mass.

ACKNOWLEDGMENT

Parts of this manuscript appear in: Melby, C.L., Commerford, S.R., & Hill, J.O. (1998). Exercise, Macronutrient Balance, and Weight Regulation. In: D.R. Lamb and R. Murray (eds.) *Perspectives in Exercise Science and Sports Medicine. Vol. 11. Exercise, Nutrition, and Weight Control*, Carmel, IN: Cooper Publishing Group.

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SUPPLEMENT

VOLUME 12 (1999) ■ NUMBER 1

PRACTICAL IMPLICATIONS

1. Lifestyle factors, including changes in diet and physical activity patterns, are important in controlling obesity.
2. The accumulation of excess body fat appears to be a problem resulting from inadequate fat oxidation relative to intake.
3. If energy intake exceeds expenditure, it is possible for an individual to become obese even on a low-fat diet. On a eucaloric or hypercaloric diet, most of the dietary fat is stored as the body preferentially burns carbohydrates and protein to meet its energy needs, at the expense of fat oxidation.
4. Weight loss results from an energy deficit (energy intake < energy expenditure). Regardless of macronutrient content (e.g., high-carbohydrate, low-fat diet vs. low-carbohydrate, high-fat diet), weight loss will occur only if the diet produces an energy deficit, not because of purported optimal ratios of macronutrients.
5. The effect of exercise on total daily energy expenditure results primarily from the increase in energy expenditure during exercise, not because of a sustained effect on energy expenditure during the remainder of the day.
6. Endurance exercise of varying intensities, as well as resistance exercise (weight lifting), are useful for weight loss and weight maintenance as long as the total net energy cost of the exercise contributes to an energy deficit.

TABLE 1.

Theoretical impact on energy balance of adding 5 h./wk. of low-intensity exercise to an individual's normal daily activities.

Length of Program	Energy Deficit	Body Weight Loss
	NO ENERGY COMPENSATION	
12 weeks	70.32 MJ (16,800 kcal)	1.9-2.2 kg
20 weeks	117.20 MJ (28,000 kcal)	3.1-3.7 kg
52 weeks	304.71 MJ (72,800 kcal)	8.1-9.7 kg
	25% ENERGY COMPENSATION	
12 Weeks	52.74 MJ (12,600 kcal)	1.4-1.7 kg
20 weeks	87.90 MJ (21,000 kcal)	2.3-2.8 kg
52 weeks	228.53 MJ (54,600 kcal)	6.1-7.3 kg
	50% ENERGY COMPENSATION	
12 weeks	35.16 MJ (8,400 kcal)	0.8-1.1 kg
20 weeks	58.60 MJ (14,000 kcal)	1.4-1.8 kg
52 weeks	152.36 MJ (36,400 kcal)	4.0-4.7 kg

Table 1 illustrates the theoretical impact on energy balance produced by a chronic increase in physical activity. Suppose a moderately overweight, sedentary woman initiates a program of exercise in which she expends 1425 kJ/h (340 kcal/h) in fairly low-intensity physical activity. Because the energy necessary for internal work (i.e., resting metabolic rate or RMR) would have been approximately 251 kJ (60 kcal) during that same hour had she not been active, the net increase is 1174 kJ (280 kcal). If she engaged in exercise for 5 h/wk, this would increase the thermic effect of physical activity by 5870 kJ/wk (1400 kcal/wk) or an average increase in energy expenditure of 840 kJ/d (200 kcal/d). This increase in chronic physical activity represents a perturbation of the steady-state of energy and macronutrient balance. In order to maintain the steady state, the system must completely compensate for the effects of the increased physical activity. This can occur if total energy intake increases to match the increased energy expenditure due to activity and if the composition of fuel consumed is changed to match the composition of fuel oxidized. Most available data suggest that this is not the case and that energy compensation for alterations in physical activity is incomplete. If there was no energy compensation for the exercise, i.e., (no change in diet or spontaneous daily activity) the subject would be in a negative energy balance of 840 kJ/d (200 kcal/d). At 25% compensation, the negative energy balance would be 630 kJ/d and at 50% compensation would be only 420 kJ/d (100 kcal/d). Unless energy compensation is complete, the caloric deficit will eventually affect body weight and body composition. Note that in Table 1 the projected weight loss due to participation in an exercise program for 12, 20, or 52 wk with varying degrees of energy compensation is, in fact, only estimated. In actuality, weight loss is accompanied by decreases in RMR and in the energy cost of physical activity, which can attenuate the magnitude of the energy deficit over time. Also, the amount of weight lost over time will depend on the amount of fat versus lean tissue lost. Because this caloric imbalance cannot be maintained indefinitely, the final effect of the activity on energy requirements cannot be determined until the system reaches a new steady-state.