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# The Effects of Exercise on the Storage and Oxidation of Dietary Fat

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## Abstract

Obesity has become a worldwide problem of pandemic proportions. By definition, obesity is the accumulation of excess body fat and it represents the long-term results of positive energy and fat balance. The failures in the regulatory mechanisms leading to the development of obesity are still not well understood, but there is growing evidence that exercise is an important element in obesity prevention. Exercise promotes energy/fat balance while providing beneficial alterations to obesity/overweight-related comorbidities and mortality. Also, exercise, in large part, influences whether the fate of dietary fat is storage or oxidation. Many factors including intensity, duration and type (aerobic vs anaerobic) of exercise, energy expended during exercise and individual fitness level impact the amounts of fat oxidised at any given time. Evidence suggests that moderate-intensity exercise yields the most cumulative (during and post-exercise) fat grams used for substrate in the average individual. All intensities of exercise, however, promote fat oxidation during the post-exercise period. We suggest that it is the effects of exercise on 24-hour fat balance that are most important in understanding the role of exercise in the prevention of fat accumulation and obesity.

## 1. Background

Obesity in the US is a significant health problem. Obesity, defined as a body mass index (BMI) >30 kg/m$^2$, now characterises >30% of the adult population; and with the inclusion of those adults who are considered overweight, defined as a BMI >25.0 kg/m$^2$, the value approaches 65%.[1] Additionally, a study released in October 2003[2] reported that the prevalence of BMIs of ≥30 has doubled between the years 1986–2000, and that the prevalence of severe (BMI >40) and super (BMI >50) obesity, have increased 4- and 5-fold, respectively. While alarming, an even more shocking statistic is that the prevalence of paediatric overweight and obesity have also skyrocketed. Ogden et al.[3] reported a 15.5% incidence of overweight in a population of adolescents aged 12–19 years.

The deleterious effects of this obesity epidemic on individuals includes increased risk of diabetes, cardiovascular disease, hypertension, digestive disease, joint disorders, cancer[4] and reduced quality of life. Obesity is estimated to account for approximately 300 000 deaths annually[5] and to have an estimated monetary cost that already approached $US100 billion in 1995.[6]

Millions of Americans experience illnesses that can be improved or prevented through weight loss and/or regular physical activity. These two factors, weight loss and physical activity, are highly interrelated. Obesity, although tracked by BMI, is defined as the state of excess body fat. It develops through an accumulation of fat during periods of positive

energy balance. Positive energy balance occurs when energy intake exceeds energy expenditure (equation 1):

energy IN – energy OUT = energy STORED (Eq. 1)

However, evidence suggests that fat balance (dietary fat – fat oxidised = fat stored) might be a more important determinant in weight gain. Fat balance includes trafficking of fat to tissues (liver, muscle, adipose) and the subsequent partitioning of fat to oxidation or storage. Positively altering the trafficking and partitioning of fat can determine how individuals are able to maintain bodyweight and body composition. A review of trafficking of fat in the body, its partitioning between storage and oxidation, and the role of exercise in changing these parameters is discussed throughout the article.

### 1.1 Exercise in Weight Loss and Weight Maintenance

Several non-invasive and non-pharmacological treatments aimed to ameliorate the obesity epidemic and its associated comorbidities have been implemented to drive the energy balance equation to negative balance with varying degrees of success. These include attempts of caloric restriction and increased exercise administered either independently or in combination.[7-9]

With respect to initial weight loss, dietary modification (caloric restriction) alone has been shown to have a dramatic impact in the short term. Miller et al.[10] reviewed literature over a period of 25 years with respect to caloric restriction. This meta-analysis revealed that obese people lost approximately 11kg in a 15-week period. Unfortunately, the same literature showed that the average individual regained approximately 35% of this loss after 1 year and more thereafter.

Exercise alone is generally less efficacious compared with dietary restriction in attaining weight loss; however, it does play a major role in weight regain.[11,12] Pavlou et al.[13] examined the interaction effect of exercise with four randomised treatments of low caloric diets on weight loss. All treatment groups lost weight in the initial 8-week period and, as indicated in the paragraph above, exercise had only a small effect on the initial weight loss. In addition, all non-exercisers regained most of the weight they had lost. Jakicic et al.[14] suggest that regardless of the continuous or discontinuous nature of the exercise, the subjects who achieved a minimum of 2000 kcal/week of exercise demonstrated a greater weight loss and maintenance than those who did not.[14,15] It should be pointed out, however, that one weakness of studies in which exercise and energy restriction are prescribed is that those who are likely to exercise are also those who are likely to maintain a restricted caloric intake. While the cited studies controlled for this caloric intake and exercise relationship, compliance may confound the results.

Many factors dictate the success of these paradigms (dietary or exercise interventions) for weight loss and maintenance, one of which seems to be the intrusive nature of the intervention on our lives. Saris[16] points out that an average 40-year-old female who starts exercising (e.g. fast walking; ~7 kcal/kg/hour) 1 hour/day for 5 days would burn extra calories equalling a weight loss of only about 0.3kg of bodyweight in a month. Decreasing daily energy balance by 200 kcals with caloric restriction (e.g. reducing dietary fat by 22g) appears to be easier for most people than exercising for >60 minutes (e.g. fast walking). Although exercise contributes to producing the negative energy balance needed for weight loss, it appears that reduced caloric intake plays a more manageable role in the attainment of negative energy balance.[10]

Lost in this debate over weight loss, however, is the strong evidence that exercise has very significant and independent health benefits. The Surgeon General's Report on Physical Activity and Health[17] summarises these benefits as:

- lowered mortality;
- decreased risk for cardiovascular disease and some cancers;
- enhanced insulin sensitivity and subsequent reduced risk of developing type II diabetes mellitus;
- reduced risk of osteoporosis;

- favoured body composition alterations, thus, lowered rates of obesity and associated comorbidities.

While weight loss/maintenance is the goal for this section and many individuals, the associated benefits of physical activity should not be overlooked and perhaps even be the priority.[17,18]

### 1.2 Energy Balance and Fat Balance

Energy balance (equation 1) plays a key role in weight maintenance. Long-term weight maintenance occurs when an individual balances their energy intake with their energy expenditure. The prevention of obesity or return to obesity can also be viewed in terms of fat-balance because obesity is, by definition, the accumulation of excess fat (equation 2). Fat balance is more complicated because input can come from both diet and fat synthesised internally.

(dietary + *de novo* fat) − fat oxidised = fat STORED (Eq. 2)

*De novo* lipogenesis converts carbohydrate into fatty acids using the acetyl-coenzyme A carboxylase regulated pathway. Hellerstein[19] comprehensively reviews this anabolic pathway, therefore, it will only be briefly mentioned in this article. An attempt at balancing acute and/or initial chronic overfeeding is shown by an increasing 24-hour respiratory exchange ratio (RER) with carbohydrate overfeeding[20,21] and decreasing 24-hour RER with high-fat diets.[22-24] RER is the ratio of the volume of $CO_2$ produced to $O_2$ consumed and is an indication of energy substrate use. A higher RER is indicative of carbohydrate oxidation and lower RER is characteristic of fat oxidation. This metabolic response to carbohydrate, however, has limits and can be overwhelmed leading to nutrient storage. Carbohydrate overfeeding leads to storing carbohydrate and as these glycogen stores are filled, fat synthesis becomes increasingly stimulated. Therefore, not only excess fat intake can lead to increases in adipose tissue, but excess carbohydrate intake can also be stored as adipose. Thus, both surplus dietary carbohydrate and fat can negatively impact fat balance.

Generally, however, *de novo* lipogenesis appreciably contributes to adipose accretion only under massive carbohydrate overfeeding.[21,25] Acheson et al.,[21] while looking at glycogen storage and *de novo* lipogenesis, progressively overfed high-carbohydrate meals (86% carbohydrate and 3% fat) to healthy male subjects for 6 days (days 4–10) beginning at 3600 kcals on day 4 of the study and finishing at 5000 kcals on day 10 of the study. Net lipogenesis only began to occur on study day 5 (~30 g/day) and culminated in ~150 g/day of new fat production on day 10.[21] It is also important to note that even before day 10, when *de novo* lipogenesis reached a peak, fat balance was positive because the body oxidised the dietary carbohydrates in preference to the dietary fats.

Thus, positive fat balance and weight gain can result from either excess fat or carbohydrate intake. Still, as discussed, most weight-gain scenarios implicate dietary fat and subsequent positive fat balances. Exercise is considered one of the important tools for preventing the accumulation of body fat.[11-15,26] Exercise increases energy demands and macronutrient oxidation to meet those demands. Inherently, this means increased fat (endogenous or dietary) oxidation both during and post-exercise. The exact increase in fat oxidation, however, is dependent on intensity and duration of the exercise, as well as the individual's fitness level. In almost all forms, however, exercise increases utilisation of fat as energy compared with being sedentary. This increased oxidation helps decrease fat balance where the ultimate goal is zero for weight maintenance and negative for weight loss.

## 2. Fat Oxidation During Exercise

### 2.1 Intensity and Duration of Exercise

The intensity of physical activity is typically described by the percentage of the peak volume of oxygen an individual can consume ($\dot{V}O_{2peak}$). Upon the onset of exercise, increased energy demands are met primarily through the oxidation of two main fuel substrates, carbohydrates and fats. The nature of the exercise bout (i.e. duration, intensity, aerobic or

anaerobic) as well as the trained status of the individual, greatly influences the relative utilisation of these fuels. It has been demonstrated that higher aerobic and anaerobic energy needs are compensated for primarily by the metabolism of carbohydrates, whereas low to moderate power outputs (<65% $\dot{V}O_{2peak}$) elicit the oxidation of fats as the primary substrate. In fact, Stefanick[27] revealed that fat oxidation accounts for up to 90% of the total substrate utilisation during exercise of low to moderate intensity (<50% $\dot{V}O_{2peak}$). Lipid substrates are found in circulation as fatty acids liberated from adipose and from the diet as fatty acids, triglycerides, or within lipoproteins (e.g. chylomicrons, very low-density lipoprotein). Additionally, smaller, local sources within muscle beds (intra- and extracellular) are available as substrate when needed and can contribute substantially to total energy metabolism.[28]

Romijn et al.[29,30] exercised trained men and women to examine the effect of exercise intensity (25%, 65% and 85% $\dot{V}O_{2peak}$) and duration (120, 120 and 30 minutes, respectively) on substrate utilisation using infused deuterated palmitate and indirect calorimetry. These investigators reported that upon the onset of exercise at 25% of $\dot{V}O_{2peak}$, plasma fatty acid oxidation accounted for >80% of the total energy expenditure and 100% of lipid metabolism. While total fat oxidised did not change significantly over the exercise period of 2 hours, intramuscular lipid oxidation slowly increased to approximately 10% at 45 minutes, plateaued for another 45 minutes and then slowly decreased as a substrate for the duration of exercise. These data also indicate that moderate-intensity exercise (65% $\dot{V}O_{2peak}$) elicits the highest rates of fat oxidation (0.7 g/min), followed distantly by 25% and 85% $\dot{V}O_{2peak}$ (0.4 and 0.5 g/min, respectively). Using indirect calorimetry, a separate study identified Fatmax (workload at which the greatest fat oxidation occurs) to be at an intensity of 64% $\dot{V}O_{2peak}$ corresponding to 74% maximal heart rate.[31]

High-intensity exercise, 85% $\dot{V}O_{2peak}$, results in more carbohydrate oxidation, specifically muscle glycogen. Interestingly, relative lipid oxidation doesn't drop significantly compared with moderate-intensity exercise, still totalling approximately 30% of energy expended. The lipids oxidised at 85% $\dot{V}O_{2peak}$, however, are composed of plasma and intramuscular sources in equal quantities.[29,30] It should be made clear that the percentage of energy coming from fat does not unequivocally equate to absolute grams of fat being used as substrate. Total fat oxidation depends on the percentage of energy from fat oxidation and the rate of energy expenditure (intensity). A point worth noting is that while the percentage of fat being oxidised is lower in high-intensity (85% $\dot{V}O_{2peak}$) exercise, it is at a similar rate as at low-intensity (25% $\dot{V}O_{2peak}$) exercise (0.4 and 0.5 g/min, respectively). Advantages at these individual intensities vary and should be scrutinised for maximal health benefit, both cardiovascular and weight.

The data indicate that both the intramuscular triglyceride (IMTG) stores as well as the circulating fatty acids contribute equally to the overall fat oxidation (~60%) during the first 60 minutes of the moderate-intensity exercise bout. After 60 minutes there was a progressive increase in reliance on plasma free fatty acids as the contribution of IMTG slowly decreased with time.[29,30] While IMTG are mentioned, it should be noted that oxidation of IMTG during exercise is controversial because of contradictory study results.[32-35] This controversy appears to stem mainly from high between-biopsy variability when using the muscle triglyceride extraction technique in estimating net IMTG oxidation.[35,36]

### 2.2 Aerobic and Anaerobic Exercise

While distinctly different, aerobic and anaerobic/intermittent exercises share the ambiguous trait of using all energy-producing pathways (e.g. creatine, glycolysis, tricarboxylic acid cycle) at the whole-body level. However, on average, different types of exercise rely more heavily on one or the other energy-producing pathways and on different substrates. Anaerobic/intermittent exercise relies predominantly on the anaerobic glycolysis pathway, which uses carbohydrate as substrate and aerobic exercise relies

on the tricarboxylic acid cycle and fat, or a mixture of fat and carbohydrate for energy production. For example, Christmass et al.[37] compared intermittent (treadmill speed set at 120% $\dot{V}O_{2peak}$; 12 **:** 18 second work **:** rest intervals) continuous bouts of less intense exercise that were matched for average oxygen uptake ($\dot{V}O_2$) over the entire period, resulting in isoenergetic bouts of exercise (0.81 and 0.85 kJ/min/kg, respectively). Their findings suggest a 3-fold lower fat oxidation rate in intermittent (0.1 g/min) versus continuous (0.3 g/min) bouts of exercise.

### 2.3 Dietary Effects

It should be noted that all of the studies cited in sections 2.1 and 2.2 were performed in the post-absorptive state and that recent meals will alter substrate utilisation. Ainslie et al.[38] investigated the metabolic response to high carbohydrate, protein or fat meals/snacks during 7.5 hours of walking. The high-fat diet was found to increase fat oxidation over the exercise period of 450 minutes. A limitation of this study is that the enduring nature of the exercise resulted in a negative energy balance of >1000 kcals in all treatments altering oxidation patterns. Hawley et al.[39] fed highly trained cyclists a high-fat meal 90 minutes prior to intense exercise. The investigators found an increased rate of the fat oxidation and fatty acid concentration during the 20-minute exercise period.

In an investigation of performance and substrate utilisation during an exercise bout, Hawley et al.[39] manipulated the diet to attain either: (i) high-plasma free fatty acids (HIFAT), glucose and insulin (CHO); or (ii) glucose while inhibiting free fatty acids (NA). During 20 minutes of cycling at 80% $\dot{V}O_{2peak}$, HIFAT treatment resulted in 2- and 3-fold higher fat oxidation compared with CHO and NA, respectively. In a similar pre-exercise feeding study, Whitley et al.[40] examined cyclists for 90 minutes at 70% $\dot{V}O_{2peak}$ followed by a 10km time trial, 4 hours after consuming isoenergetic high-carbohydrate or high-fat diets. They also reported higher fat oxidation rates, but only during the first 15 minutes of the 90-minute exercise period despite there being treatment effects on plasma free fatty acid and RER. These two studies, while not in total agreement, do indicate that the diet influences substrate utilisation during exercise. This effect is also probably modulated by pre-exercise meal timing. When the timing of the high-fat meal was delayed (90 vs 240 minutes), there was a 2-fold decrease in plasma free fatty acids.

The relationship between exercise and dietary lipids was also investigated at a lower exercise intensity. Ainslie et al.[38] studied low-intensity walking for 450 minutes while the subjects consumed isocaloric high fat, high carbohydrate and mixed meals/snacks during rest intervals. Fat oxidation (g/min) differences were observed only between the high-fat and high-carbohydrate diets. Lower RER values resulted from the high-fat diet beginning during the first minute of walking and throughout the 450 minutes compared with the high-carbohydrate condition.[38]

Studies have also been performed using longer dietary treatments. Schrauwen et al.[22] have demonstrated that chronic high-fat (60% of kcals) diets lead to a non-exercise-related increased fat oxidation calculated from within a lower 24-hour non-protein respiratory exchange ratio (24h-NPRER). Furthermore, unlike dietary carbohydrates that cause a rapid and substantial rise in 24h-NPRER shortly after starting the high-carbohydrate intake,[21] the resultant shift to a lower 24h-NPRER from a diet high in lipids is much slower.[41] Additional research[23,24,41] confirmed this transition and established that exercise (1.8 × resting metabolic rate = 24-hour energy expenditure) speeds this adaptation. Examining the robustness of this adaptation, Burke et al.[42] fed well trained subjects a high-fat (70% of kcals from fat) diet for 5 days followed by 1 day of a high-carbohydrate (18% of kcals from fat) diet. Steady-state cycling for 120 minutes at 70% $\dot{V}O_{2peak}$ resulted in higher rates of lipid oxidation and lower RERs during exercise on day 7.

These studies demonstrate that while exercise intensity has primary influences on substrate utilisation during exercise, diet will alter the substrate utilisation. The amount of fat, timing of the meal and

duration of the dietary treatment all influence the degree of the dietary effect.

### 2.4 Sex Differences

Animal studies have demonstrated that estradiol (E2) greatly influences substrate selection during aerobic exercise in rats.[43-47] Effects include an increase in lipid oxidation during treadmill exercise[44] and the sparing of glycogen depots in E2-supplemented male and ovariectomised rats. Additional effects include increased muscle lipoprotein lipase activity[43,48-50] and plasma free fatty acid concentrations.[43] Cumulatively, these results suggest possible secondary effects of increased fat oxidation during aerobic exercise.

In human studies, however, the evidence that women oxidise fat preferentially during exercise[51-54] is not clear due to some contradicting studies.[55-58] However, as noted in two excellent reviews on sex differences in metabolism,[59,60] interpretation of the data is difficult because of differences in control of the menstrual cycle and some differences between the characteristics of the subjects between sexes. Collectively, however, the evidence suggests that women oxidise proportionately more lipids compared with males.[51-54,61-65] In addition, Horton et al.[53] demonstrated in females at rest, increased uptake of infused lipid tracer via vastus lateralis biopsy in both fasted and fed states, which suggests that females would be more sensitive to recent diet treatments than men.

At least two exercise studies[63-65] have elaborated on the implication of E2 from animal studies showing increasing lipid oxidation in women. Ruby et al.[64] showed increased lipid metabolism during 90 minutes of treadmill running at 65% $\dot{V}O_{2peak}$ by measuring glycerol appearance in amenorrheic females during E2 supplementation using transdermal E2 administration. Additionally, in two publications, Zderic et al.[63,65] demonstrated increased fat oxidation at the 90% lactate threshold (LT), but not the 70% LT in the luteal phase, when circulating E2 levels are greater. Thus, there seems to be growing evidence of a sex difference in lipid metabolism and presumably fat oxidation during exercise in eumenorrheic females using tracer infusions. We could not find research directly measuring dietary fat oxidation as part of a sex comparison.

## 3. Post-Exercise Oxidation of Lipids

As previously discussed in section 2.1, the mix of macronutrient oxidised during exercise is dependent on many factors including intensity and duration of the exercise. Exercise also alters post-exercise nutrient oxidation and metabolism. Although there is a tendency to think of the effects of exercise as limited to the exercise period, the recovery phase and post-exercise phase constitute a much longer period of time. Even though the effects of previous exercise may be small to modest, the longer time period can in some instances result in major cumulative effects. Studies have demonstrated an increased post-exercise lipid oxidation and postprandial lipaemia attenuation after endurance[53,66-71] and resistance exercise.[72-76]

### 3.1 Intensity and Duration

Among other influences, intensity and duration define the energy expended during an acute bout of exercise, but evidence indicates that it is the total energy expenditure of the bout of exercise that provides the platform for the understanding of post-exercise lipid metabolism.[69,77,78] Isocaloric exercise at 66% $\dot{V}O_{2peak}$ for 45 minutes and 33% $\dot{V}O_{2peak}$ for 90 minutes resulted in similar 6-hour recovery non-protein RER ($\dot{V}CO_2/\dot{V}O_2$), $\dot{V}O_2$ and fat oxidised.[79] Thus, while the exercise bouts differed in intensity and had a very different acute effect on fat oxidation, the total energy expenditures were similar and post-exercise periods did not differ in fat oxidation.

Focusing on the effects from acute periods of exercise, data clearly indicate a decreased RER and increased lipid oxidation during the post-exercise period.[53,70,72,74,75,80] This post-exercise phenomenon continues despite the feeding of carbohydrate (1.5mL of fluid and 0.09g of carbohydrate per net kcal expended) during or after (in a pasta meal) the exercise session seemingly favouring lipids regardless of macronutrient intake. The effect of exercise

dampens a possible diet-induced reliance on carbohydrate in the post-exercise period.[81,82] One theory for this effect is that metabolic pathways are directed to fill glycogen first.[83] This means that all gluconeogenic precursors (including lactate, available amino acids, glycolytic intermediates) will be spared from further oxidation and get converted to glucose for storage as carbohydrate. Lipids become the preferred substrate for energy conversion as the body recovers in the post-exercise period.

### 3.2 Aerobic versus Anaerobic

Anaerobic exercise also increases post-exercise fat oxidation. Resistance exercise data demonstrate increased lipid oxidation lasting up to 3 hours post-exercise.[75] Binzen et al.[72] exercised resistance-trained women on nine exercises for three sets of ten repetitions each at 70% one repetition maximum and completed within 45 minutes. The collection of metabolic gases via indirect calorimetry indicated lowered RER and increased fat oxidised throughout the 20-minute post-exercise period. These data agree with previously stated results because the RER was still significantly lower at 120 minutes post-exercise, the end of data collection, and appeared to be able to last for some time, based on the slope of the line.

Comparisons between aerobic and resistance exercise, however, have revealed a difference in post-exercise fat metabolism.[84,85] These studies discovered that resistance exercise lends itself to higher fat oxidation and lower RERs compared with aerobic exercise. When energy expended during exercise was matched by an isocaloric intake, resistance exercise oxidised more fat.[84] Melanson et al.[85] showed similar post-exercise fat oxidation and RER between types of exercise with less energy expended during the resistance (322 kcals) than the aerobic (464 kcals) periods of exercise. This difference may be coupled to the source of substrate used during either type of exercise, with resistance exercise utilising more carbohydrate. Following the aforementioned theory, more available glucose (or gluconeogenic precursors) will be shunted towards glycogen replenishment and fat used for current energy needs.

A weakness of post-anaerobic exercise RER data is that the whole body bicarbonate pool tends to increase during recovery due to changes in acid-base balance. Therefore, RER or the $CO_2 : O_2$ ratio would be lowered after high-intensity, anaerobic activity because there is less $CO_2$ being expelled due to bicarbonate sequestration. The distinction between lowered RERs because of this phenomenon or because of increased fat oxidation needs further examination.

While most exercise results in health benefits, a fine methodological point needs to be discussed regarding exercise studies. Post-exercise studies require that we distinguish between chronic adaptations of training and metabolic modifications caused by an acute bout of exercise, as nicely reviewed by Gill and Hardman.[86] The transient nature of metabolic messenger RNA (mRNA) exists at the heart of this distinction. Evidence suggests that mRNA responds to exercise within 4 hours of cycling (~60% $\dot{V}O_{2peak}$)[87] and returning to baseline as soon as 24 hours in rats.[88] Thus, allowing for a minimum of 48 hours[89,90] and up to 60+ hours of no exercise[91-93] will decrease the likelihood of acute exercise bias on studies examining the effects of exercise training on postprandial lipid metabolism. In summary, researchers need to differentiate the two and control for acute effects of exercise when looking at exercise training effects on lipid metabolism.

### 3.3 Effects of Dietary Fat Consumed Post-Exercise

Gill et al.[80] investigated the effects of exercise on dietary fat metabolism approximately 12 hours after a 60-minute period of walking. The researchers fed previously exercised subjects a tracer-containing emulsion of 1,1,1-$^{13}$C tripalmitin in a high-fat meal. The exercise consisted of men walking at 60% $\dot{V}O_{2peak}$, after which, continuous data collection occurred for 8 hours and intermittently thereafter. They demonstrated significantly increased exogenous fat oxidation through approximately 20 hours post-exercise.

In a similar study looking at fat oxidation using whole-room calorimetry, Votruba et al.[70] fed an average Western diet (30% fat, 55% carbohydrate, balance protein) to female subjects after performing cycle ergometry during two different treatments in addition to a control treatment. Exercise was performed at light intensity (25% $\dot{V}O_{2peak}$) and heavy intensity (85% $\dot{V}O_{2peak}$) calculated to expend ~300 kcals above rest. Light- and heavy-intensity exercise treatments similarly increased fat oxidation approximately 12g in the 11.5 hours post-exercise compared with rest.[70] This study also determined that heavy-intensity exercise results in less fat oxidised during exercise compared with light-intensity exercise. This results in less total fat oxidation in the heavy intensity exercise treatment due to decreased fat oxidation during exercise.

## 4. Conclusion

Exercise increases the demand for energy and thus, macronutrient oxidation. Inherently, this means an increase in fat (endogenous or exogenous) oxidation both during and post-exercise. Considerations of intensity, duration, post-exercise effects, fitness, etc., are necessary in an effort to maximise this effect. However, regardless of the gradation of 24-hour fat oxidation, exercise increases utilisation of fat as energy in whatever time period is looked at. This increased oxidation helps decrease fat balance (fat intake – fat burned = fat stored; i.e. fat balance),[22-24,69,81,82,94] where the ultimate goal is zero for weight maintenance and negative for weight loss.

Perhaps this can be best illustrated with an example of how exercise can quantitatively affect energy and fat balance. Thirty minutes of brisk walking (6 km/hour) will yield approximately 154 kcals expended for a 70kg person. Approximately 80% of these calories expended come from fat,[29,30] yielding about 13.5g of fat oxidised above resting levels. Light-intensity exercise is suggested to also consume approximately 12g of fat for 12 hours post-exercise, above that consumed at rest.[70] In total, this increases energy expenditure and fat oxidation to ~25g above rest during a day. Also, as an additive effect, decreasing fat intake decreases fat balance. Thus, fat balance can be synergistically affected by increasing exercise and decreasing fat intake.

Increasing fat oxidation with exercise by 25g and decreasing dietary fat consumption by 25g will decrease fat balance (50g) and thus, energy balance (225 kcals). This also helps to partially explain why exercise alone doesn't often lead to weight loss. In comparison, one can attain greater results with diet modification compared with the 500–1500 kcal/day changes in energy balance that can be attained through energy restriction. We should add, however, that this is an oversimplification because there are multiple feedback loops leading to autoregulation of energy intake and, to a smaller degree, energy expenditure that helps regulate bodyweight. The milieu associated with bodyweight regulation and weight loss are not retold in this article; however, they are discussed in another thorough review.[95]

Effects of how inactivity affects energy and fat balance might best be illustrated with the results from a study by Shepard et al.[96] Subjects in a random, crossover design consumed either a eucaloric high-carbohydrate (25% fat) or high-fat (50% fat) diet for 14 days under free-living activity conditions.[96] On day 15, indirect calorimetry measured $\dot{V}O_2$ and $\dot{V}CO_2$ gases used to determine NPRER, energy expenditure and nitrogen-corrected macronutrient oxidation while the subjects were sedentary. Data indicate that this sedentary day, while consuming the high-fat diet, resulted in a positive fat balance compared with the high-carbohydrate diet (1790.8 ± 510.4kJ; −62.8 ± 510.4kJ, respectively). In summary, while eucaloric on an average day, a high-fat diet in combination with no exercise for 1 day would result in net body fat accumulation (1790kJ = 428 kcals ~50g) in 1 day. It should be noted that data do not exist that will allow us to fully extrapolate these findings; however, weight gain does appear to result from small additive effects that occur periodically throughout our daily lives.

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