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Aerobic Exercise: Fat Cremation and Heart Rejuvenation

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ABSTRACT:

A large amount of body fat is stored in the form of triglyceride within adipose tissue as well as within muscle fibers. These stores must be mobilized into free fatty acids (FFA) and transported to muscle mitochondria for oxidation during exercise. Fatty acids from adipose tissue are mobilized into plasma and carried by albumin to muscle for oxidation. As exercise intensity increases from low (25% VO2max) to moderate (65% VO2max) to high (85% VO2max), plasma FFA mobilization declines. However, total fat oxidation increases when intensity increases from 25% to 65% VO2max, due to oxidation of intramuscular triglycerides, which provide about one-half of the fat for oxidation. Endurance training characteristically increases fat oxidation during moderate intensity exercise by accelerating the oxidation of intramuscular triglyceride without increasing the mobilization or oxidation of plasma FFA. Carbohydrate ingestion during the hours before exercise, even in relatively small amounts, reduces fat oxidation during exercise largely through the action of insulin. Fat supplementation and special diets have limited ability to increase fat oxidation in people, especially during sport competitions. Fat from body stores and/or dietary supplementation cannot adequately replace muscle glycogen and blood glucose as fuels for intense exercise. Although a greater proportion of energy is derived from fat within the fat burning zone (FBZ), total energy expenditure is greater with high-intensity exercise. It is total energy expenditure, regardless of the source, that is paramount for achieving sensible weight loss via negative energy balance.

INTRODUCTION:

Adipose tissue triglycerides are a very important source of fuel to meet energy demands during exercise. Increases in lipolytic rate and availability of fatty acids that occur during exercise require the coordination of neural, hormonal and circulatory events, which facilitate

delivery of fatty acids from adipose tissue to the working muscle for oxidation. The two main sources of energy during muscular exercise that fuel aerobic ATP synthesis in human skeletal muscle are fat (triglyceride) and carbohydrate (glycogen and glucose) stored within the body. It is well known that diets containing predominantly carbohydrate are necessary to maintain glycogen stores at high levels during bouts of intense exercise and those diets are apparently optimal for promoting training-induced improvements in performance[16]. The primary reason that glycogen reserves are essential for the body is that it can only slowly convert their body fat stores into energy during exercise. Therefore, when muscle glycogen and blood glucose concentrations are low, the intensity of exercise must be reduced to a level that can be supported by the body's limited ability to convert body fat into energy. With endurance training, athletes can markedly increase the rate at which body fat can be oxidized, thus allowing them to exercise longer before becoming exhausted due to glycogen depletion.

Whether high-intensity exercise is the best bet for training and weight loss? The concept of the fat burning zone is highly attractive to the exercise enthusiasts of today, many of whom are more interested in weight loss than the pursuit of fitness. Training zone charts adorn the walls of Gyms and fitness centers across the world and body-conscious exercisers religiously adhere to the recommended limits for exercising heart rates without a proper knowledge about it. In the this article we have discussed fat metabolism during exercise and fat burning zone to impart better understanding about the selection of training intensity for fitness enthusiastic peoples .

BODY FAT STORES

Fat is stored in the body in the form of triglyceride in adipose tissues under the skin. These fatty acids consist of chains of carbon atoms with hydrogen atoms attached. There is more stored energy (9 kcal/g) in a gram of fat than in an equal weight of carbohydrate (4 kcal/g). Typically, about 50,000 to 60,000 kcal of energy are stored as triglycerides in the entire mass of all of adipose tissues throughout the body. In contrast, if all of this energy were stored as carbohydrate in the form of glycogen, glycogen is stored with a bound with water molecules, which are very heavy, resulting in a total energy store weight of more than 100 pounds. Thus

large amount of energy is stored in a relatively small mass of triglycerides, which provide a marvellous way for people to carry fuel as they move from place to place.

Triglyceride is also stored in droplets directly within the muscle fibers (intramuscular triglyceride), placing this fuel in close proximity to the site of oxidation in the muscle mitochondria. Intramuscular triglyceride accounts for 2,000-3,000 kcal of stored energy, making it a larger source of potential energy than muscle glycogen, which can contribute only about 1,500 kcal. Unfortunately, because it is technically difficult to measure intramuscular triglyceride from muscle biopsy samples, relatively little is known about the rate at which intramuscular triglyceride can be oxidized during exercise or how this energy store changes in response to acute and chronic training. It is clear, however, that intramuscular triglyceride can provide energy for intense exercise at less than one-third the rate attributed to muscle glycogen. Therefore, during strenuous training or competition energy from intramuscular triglyceride should be considered as supplementary to that supplied by muscle glycogen. In addition to energy supplied by intramuscular triglycerides, it should be noted that plasma triglycerides are another source of energy for muscle. In the fasted state, there is a small amount of triglyceride produced by the liver that is bound to very-low-density lipoproteins in plasma. Although muscle can break down this plasma triglyceride to some extent during exercise, its contribution to energy is very small[10].

MOBILIZATION AND OXIDATION OF FAT DURING EXERCISE

The large stores of triglyceride within adipose tissue are mobilized at relatively slow rates during exercise. Exercise stimulates an enzyme, hormone sensitive lipase, that dissolve the triglyceride molecule into free fatty acids (FFA) and one glycerol molecule ; this process of breaking down triglycerides is known as lipolysis. The primary factor thought to be responsible for the stimulation of adipose tissue lipolysis during exercise is the increasing plasma concentration of epinephrine, which activates betareceptors in adipocytes additional hormonal factors probably also play a role[1].

The glycerol released from this reaction is water soluble and diffuses freely into the blood. Its rate of appearance in the blood provides a direct measure of the amount of triglyceride

hydrolyzed in the body. Where as fatty acids are not water soluble and thus require a protein carrier to allow them to be transported through cells and within the blood stream. At rest, about 70% of the FFA released during lipolysis are reattached to glycerol molecules to form new triglycerides within the adipocytes. However, during low-intensity exercise, this process is attenuated at the same time as the overall rate of lipolysis increases; as a result, the rate of appearance of FFA in the plasma increases by up to five fold [20]. Once they enter the plasma, the FFA molecules are loosely bound to albumin, a plasma protein, and transported in the circulation. Some of the fatty acids are eventually released from albumin and bound to intramuscular proteins, which in turn transport the FFA to the mitochondria for oxidation[19].

EXERCISE INTENSITY AND FUEL USE

The contributions of carbohydrate, i.e. muscle glycogen and blood glucose, and of fat, i.e., plasma FFA from adipose tissue plus intramuscular triglyceride, to total energy expenditure during exercise at these various intensities are shown in Table - 1. It should be noted that although the contribution of plasma FFA to the fuel supply declines as exercise intensity increases from 40% to 75% VO2max, total fat oxidation increases.

Table – 1

Energy expenditure and fuel selection Values[12]

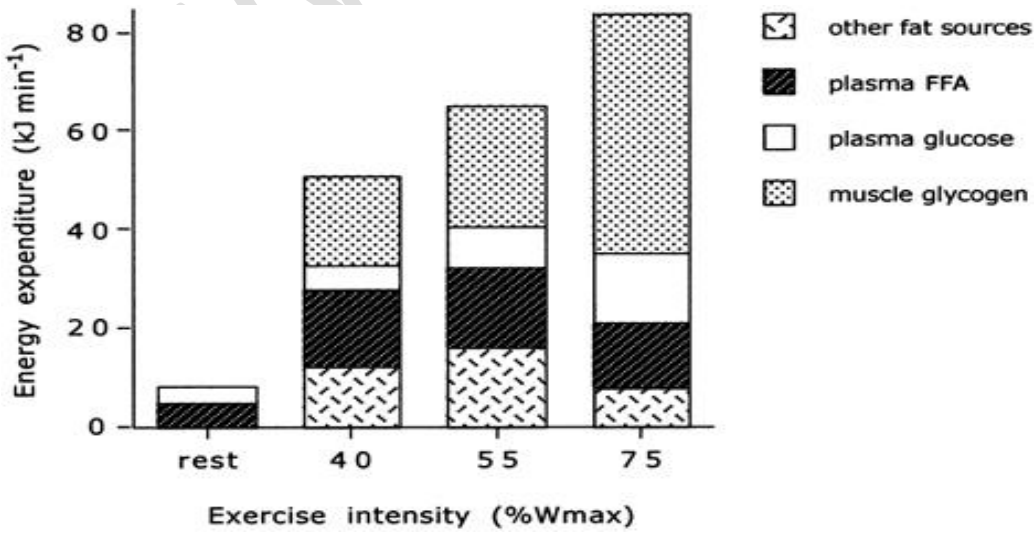

Table - 2

Contribution of substrates to total energy expenditure during cycling exercise at 40, 55 and 75 % W_{max} (Work Maximum) [12]

Source of Energy	40% W_{max}	55% W_{max}	75% W_{max}
Free fatty acids	31 %	25 %	15 %
Other fat sources	24 %	24 %	9 %
Plasma glucose	10 %	13%	18%
Muscle glycogen	35 %	38%	58 %

It shows that both carbohydrate and fat oxidation rates increased proportionally as the exercise intensity was increased up to a workload of 55 % W_{max}. However, as exercise intensity was increased to 75 % W_{max}, both muscle glycogen and plasma glucose oxidation rate markedly increased and fat oxidation rate markedly decreased. This decrease in fat oxidation rate involved a significant decline in the oxidation rate of both plasma FFAs and TG fat sources (sum of intramuscular and lipoprotein-derived TG).

At the start of exercise (regardless of the intensity), a series of biochemical events is initiated by neurological stimulation. Glycolysis (an anaerobic means of ATP/energy provision, fuelled by carbohydrate) is primed by hormones and neurotransmitters to take over from phosphagen-mediated energy sources[3]. Thereafter, if the exercise is of a low- to moderate-intensity, energy demands are met increasingly by fat in the form of muscle triglycerides and plasma free fatty acids. If the exercise is of a high intensity, energy from carbohydrate-derived fuels predominates[4].

This shift from fat to carbohydrate oxidation during high-intensity exercise is essential to increase both the magnitude and the rate of energy release. Muscle can extract more energy per liter of oxygen consumed from carbohydrate than from fat. As carbohydrate oxidation

yields more than 5 kcal of energy with the consumption of one liter oxygen, whereas on the other hand, the mixture of carbohydrate and fat oxidised during moderate intensity exercise releases only 4.86 kcal per liter of oxygen. The energy expenditure increases in line with exercise intensity: The energy expenditure in 20 minutes of very high-intensity exercise is 404 kcals as compared with only 244 kcals in moderate-intensity exercise performed for the same duration. Thus, although oxygen uptake may be a limiting factor, the greater energy demands of high-intensity exercise can be met. Unfortunately, though, the 'fast-twitch' muscle fibers recruited in high-intensity work are relatively inefficient and bring about the decline in power associated with intensive exercise [6].

Highly trained male endurance runners use more fat as a fuel during low-intensity exercise than do untrained healthy men despite similar rates of lipolysis and FFA uptake from plasma. This increase in fat oxidation must be related to an increased percentage of FFA uptake oxidized, a greater contribution from intramuscular triglyceride stores, or both. Additionally, lipid kinetics return to baseline more rapidly in trained than in untrained subjects after completing an exercise bout of the same absolute intensity [11].

FAT BURNING ZONE

There are two components involved in the total energy cost of exercise: first, the energy cost of the activity itself, which accounts for most of the caloric expenditure; secondly, the energy expended in recovery while the metabolic rate remains elevated above resting levels. This 'excess post exercise oxygen consumption' (*EPOC*) is fuelled by fat. Intriguingly, not all exercise is sufficient to bring about a meaningful EPOC: it is generally agreed that such exercise must be carried out at more than 70% of VO2 max [4]. Although this mechanism is not entirely understood, it seems that the metabolic disturbance of exercise determines the magnitude and duration of EPOC.

In order to recover from exercise, the body undertakes several active (energy-consuming) processes for up to an hour afterwards: phosphate is reunited with creatine and ADP; *hemoglobin* and myoglobin (oxygen-carrying pigment within the muscle) are resaturated with oxygen; *lactate* is oxidised or resynthesised to glycogen; circulation and breathing increase.

In addition, the return to homeostasis following high-intensity exercise is further delayed by the demands of glycogen resynthesis and increased hormonal activity. Interestingly, in the glycogen-depleted state, this prolonged EPOC period is fuelled by lipid as blood glucose is used to replace muscle glycogen[14].

Clearly, the calorific value of EPOC has implications for those seeking to reduce body weight. Indeed, after 20 minutes of high intensity exercise (70% VO2max) EPOC is approximately 30 kcal and if such exercise were performed five times a week for 52 weeks, the EPOC period alone would amount to 7,800 kcal or the energy equivalent of approximately 1 kg fat[16].

DIETARY CARBOHYDRATE AND FAT OXIDATION DURING EXERCISE

Fat oxidation during exercise is very sensitive to the interval between eating carbohydrate and the onset of exercise and to the duration of the exercise. This is due in part to the elevation in plasma insulin in response to the carbohydrate meal and the resultant inhibition of lipolysis in adipose tissues, thus reducing the mobilization of FFA into the plasma. This effect is evident for at least 4 h after eating 140 g of carbohydrate that has a high glycemic index [13]. Under these conditions, the carbohydrate meal reduces both total fat oxidation and plasma FFA concentration during the first 50 min of moderate-intensity exercise. However, this suppression of fat oxidation is reversed as the duration of exercise is increased; after 100 min of exercise, the rate of fat oxidation is similar, whether or not carbohydrate was eaten before exercise. It appears that the body relies heavily on carbohydrate and less on fat when people have eaten carbohydrate during the previous few hours, and therefore carbohydrate is preferred when it is available. It is likely that insulin plays a role in regulating the mixture of carbohydrate and fat oxidized during exercise[18].

This reduction in fat oxidation and increase in carbohydrate oxidation is not usually detrimental if all of the increase in carbohydrate oxidation is derived from glucose in the blood from the meal, thus having little influence on muscle glycogen use. Therefore, at present, there is little basis for recommending that people refrain from eating carbohydrate before exercise because such a meal will simply shift energy metabolism to less of a reliance

on oxidation of plasma FFA and more on blood glucose oxidation, with lesser effects on muscle glycogen and intramuscular triglyceride utilization.

SUMMARY

Fat provides the highest concentration of energy of all the nutrients. One gram of fat equals nine calories. This calorie density, along with our seemingly unlimited storage capacity for fat, makes fat our largest reserve of energy. One pound of stored fat provides approximately 3,600 calories of energy. While these calories are less accessible to athletes performing quick, intense efforts like sprinting or weight lifting, fat is essential for longer, slower lower intensity and endurance exercise such as easy cycling and walking. As exercise intensity increases from low (25% VO2max) to moderate (65% VO2max) to high (85% VO2max), plasma FFA mobilization declines. However, total fat oxidation increases when intensity increases from 25% to 65% VO2max, due to oxidation of intramuscular triglycerides, which provide about one-half of the fat for oxidation. Endurance training characteristically increases fat oxidation during moderate intensity exercise by accelerating the oxidation of intramuscular triglyceride without increasing the mobilization or oxidation of plasma FFA. Similarly, during low-intensity exercise with little intramuscular triglyceride oxidation, the increased fat oxidation of trained people does not appear to be caused by increased mobilization of FFA into plasma, but rather by a greater rate of oxidation of the FFA removed from the blood during exercise. Exciting new research is shedding light on the complex mechanisms that facilitate the delivery and transport of these fatty acids in skeletal muscle. Understanding the factors involved in the regulation of lipid availability and oxidation is an important step towards the clarification of how exercise can best be implemented as a first line for prevention and/or treatment of obesity-related disorders.

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