Molecular adaptations of lipolysis to physical activity

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Abstract

The purpose of the present study was to investigate the context of lipid metabolism research in physical activity, lipolysis, lipolysis hormone regulation and the fate of lipolysis products in exercise, fatty acid transporters, some genes involved in lipid metabolism, effect of resistance activity on lipolysis, adaptations of adipose tissue due to physical activity, lipoproteins and apoproteins and the effect of physical activity and to achieve a desirable conclusion and provide more relevant information from previous research. In this study, articles were searched in specialized databases and 40 related articles were selected based on inclusion and exclusion criteria, and the molecular adaptations of lipolysis to physical activity were investigated.
The amount of free fatty acid occurrence can be measured by estimating the amount of lipolysis. Fatty acids released from adipose tissue lipolysis form a major part of the active fuel, especially when the duration of training is long and the intensity is low to moderate. Physical activity through the release of more epinephrine from the adrenal glands and norepinephrine from the sympathetic nerve endings increases the rate of lipolysis of adipose tissue. Also, aerobic activity increases the genes involved in lipid metabolism and lipolysis.
The present study showed that the intensity and duration of training, diet and training positions influence lipolysis and lipid metabolism. The amount of adipose tissue lipolysis is controlled by triacylglycerol hormone-sensitive lipase. Resistance activity stimulates stimulating growth hormone, catecholamines, and enzymes involved in the lipolysis process. Endurance training also reduces the number of fat cells and reduces plasma triacylglycerol concentrations.

Keywords: Physical activity, Lipolysis, Lipoprotein

Introduction

Fat fuel reserves are the most important energy substrates for skeletal muscle metabolism during endurance exercise. The share of fats in total oxidative metabolism depends on several factors, including the intensity and duration of exercise, as well as diet and exercise situations. Oxidizable fats include triacylglycerol, free plasma fatty acids and intramuscular triglycerides. However, free fatty acids bonded with albumin in the blood move from adipose tissue and account for a major share of fat metabolism within the skeletal muscle during exercise. In humans, adipose tissue comprises between 10 to 25 percent of the body weight. Most of the adipose tissue is located subcutaneously and around the abdominal organs, but fewer reservoirs of this tissue are located between the skeletal muscles. The rate of movement of free fatty

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acids from adipose tissue depends not only on the amount of lipolysis but also on the plasma carrying capacity of free fatty acids and on the rate of re-esterification of free fatty acids by adipose tissue (1).

When exercising at a sub-maximal intensity, the skeletal muscle relies on the chemical energy of fat oxidation and carbohydrates to meet its needs. At rest, fatty acid oxidation is significantly involved in providing total energy. During physical activity a number of neural, metabolic and hormonal stimuli lead to increased lipolysis and release of fatty acids and fatty acids are increasingly oxidized by muscle cell mitochondria. As a result, the concentration of released fatty acids inside the muscle cells will decrease. This phenomenon stimulates the discharge of free blood fatty acids. The integrated process of release, transport, uptake and ultimately oxidation of fatty acids is increased by the joint action of epinephrine, norepinephrine which increases during exercise and also regulated by the decrease of circulating insulin (2). It should be noted that the steps of increasing fatty acid oxidation are very numerous and complex. This provides answer to the question why reaching a sustainable state of increased fat oxidation and reduced carbohydrate oxidation takes about 20 minutes and the reason is that carbohydrate-induced energy production is faster than fat. Thus, depending on the type and intensity of the activity of biochemical processes, the use of carbohydrate and fat also varies (1, 2).

Lipid metabolism in physical activity

Lipids in general are a large family of biological compounds that have little solubility in water. The three groups most commonly used in physical activity are fatty acids, triacylglycerols, and steroids. Fatty acids and triacylglycerols are important sources of energy during physical activity; the amount of free fatty acids found in the body is much lower than that of triacylglycerol (3).

Most of the triglycerides are inside the adipocytes; the cytoplasm of these cells is full of triacylglycerol, the particles that are surrounded by a single layer of phospholipid. In addition, these particles are coated with perilipins (a family of proteins that regulate access to these particles), the main metabolic activity of adipocytes is the synthesis and decomposition of triacylglycerol; synthesis of triacylglycerol is predominant in the satiety condition after eating, while its decomposition occurs in times of energy crisis, including hunger or long-term physical activity. If these two conditions are balanced throughout the day, the fat mass will remain constant, otherwise the amount of fat will increase or decrease. In the case of a positive energy balance that means increased calorie intake and decreased consumption during weeks, months or years, the fat mass increases and the consequent obesity will be serious threat to health. Since physical activity by negating energy balance increases the need to consume fatty acids as a fuel, lipolysis is increased in adipocytes, so physical activity is a beneficial way to control weight and obesity (3-6).

The decomposition of triacylglycerol is called lipolysis. Lipolysis occurs in the cytosol with the help of two lipases, triacylglycerol lipase and monoacylglycerol lipase. The former catalyzes the ester bond hydrolysis of one- and three-unit positions of glycerol into 2-monoacyl glycerol and two fatty acids. The monoacyl glycerol lipase then completes the decomposition of triacylglycerol by the ester bond hydrolysis of the 2-position glycerol unit (3).

The amount of lipolysis is better estimated by measuring glycerol release. Glycerol in the blood seems to be the only product of lipolysis and once released, it cannot be reused by adipose tissue, since adipose tissue lacks glycerol kinase. The amount of free fatty acid (FFA) occurrence can also be measured by estimating lipolysis (1). Fatty acid released from lipolysis of adipose

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tissue is a major part of the active fuel, especially when the training period is long and its intensity is low to moderate. Metabolism of FFA (long chain linked to albumin) is a complex process and involves several steps: moving FFA from adipose tissue, transporting into plasma, penetrating through the cell membrane and between pores, transporting into the cytoplasm, and intracellular metabolism (1, 4).

Hormonal regulation of lipolysis

Physical activity through the release of more epinephrine from the adrenal glands and norepinephrine from the end of the sympathetic nerve increases the rate of lipolysis of adipose tissue. Epinephrine and norepinephrine bind to beta-adrenergic receptors located in the plasma membrane of adipocytes and cause cAMP cascade and consequently PKA activation. PKA phosphorylates two proteins that play an important role in accelerating lipolysis. One of these proteins is perilipin A. Its phosphorylation results in the uptake of triacylglycerol lipase by the fat particles. Another protein is triacylglycerol lipase, which phosphorylation activates it. As a result, the rate of hydrolysis of triacylglycerol to 2-monoacylglycerol and two fatty acids is greatly increased. 2-monoacyl glycerol is then rapidly hydrolyzed to glycerol and fatty acid. Unlike catecholamines, insulin decreases the lipolysis rate by stimulating phosphorylation and activating phosphodiesterase. This enzyme hydrolyzes cAMP to AMP thus disabling the cAMP cascade (1). In isolated rat adipocytes, it has been shown that catecholamines, glucagon, growth hormone, adrenocorticotropic and various pituitary and intestinal hormones increase lipolytic activity. But in isolated human adipocytes only catecholamines, thyroid-stimulating hormone, and parathyroid hormone have been reported to be good stimulants for lipolysis. The amount of adipose tissue lipolysis is controlled by the hormone-sensitive triacylglycerol lipase (HSL) that breaks down the triacylglycerol ester bonds to FFA and glycerol (1, 4).

The fate of lipolysis-derived products during physical activity

Triacylglycerols in adipose tissue are decomposed into fatty acids and glycerol that enter the bloodstream and are absorbed by other tissues. Fatty acids by bonding to albumin are transported in the bloodstream, and glycerol enters the liver, which is used for glucose synthesis. Intramuscular glycerol derived from the hydrolysis of triacylglycerols of muscle cells has a similar fate. Fatty acids that are transported by blood enter the liver, which are used to produce triacylglycerols or enter the muscles, where they bind to fatty acids from the breakdown of muscle cell triacylglycerols and are oxidized to CO_2 (1).

Detection of fatty acid transmitters

It appears that a number of fatty acid-binding proteins take long-chain fatty acids (LCFAs) across the plasma membrane. These include fatty acid binding protein peripheral membrane (FABPPM) and the transducer protein family of fatty acids (FATP1-5) and glycosylated FAT (fatty acid translocase) (FAT / CD36). Evidence suggests that when either of these transducers is expressed in mismatched cells, they can independently enhance FA fatty acid transport. In recent years, much of the information is gained from the transfer of long-chain fatty acids (LCFAs) to FAT / CD36 and FABPM, the proteins with appropriate antibodies. The maximal amount of FA in the muscle tissue has been substantially varying, and exists more in the heart, red muscles and white muscles, respectively. In rodent heart and muscle, both FAT / CD36 and FABPPM appear to be crucial and physiological transmitters of FA across the plasma membrane. When it was determined that LCFA translocation through sarcolemma was carried out by the protein, it was important to determine

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whether the rate of LCFA translocation could change severely (within minutes) or chronically (Exercise-adjusted responses and in metabolic diseases).

FA translocation may be regulated by transducer proteins in several ways: 1. Change the expression of FA transmitters. 2. Alteration of their cellular subunit distribution. 3. Possible altering of their activity in the plasma membrane.

Since the levels of protein expression do not change over a period of less than 30 minutes, acute regulation of FA transmission is accomplished by two other mechanisms. On the other hand, chronic mechanism responses that are observed after a few hours or days occur through the first or second mechanisms, or both. The results show that FAT / CD36 facilitates the translocation of LCFA from sarcolemma; and in coordination with FABPPM and the carotene palmitoyl transferase (CPT1) crosses the mitochondrial membrane. The fatty acids entered in cells may be oxidized by beta-oxidation, or converted to reserves such as IMGT, which are altered into MGAT (monoacylglycerol acyl transferase) and diacyl glycerol transferase (DGAT) by esterification (6-8). At rest, LCFA metabolism (Esterification and oxidation) is controlled by various hormones including epinephrine, insulin, leptin and adiponectin. Recent studies in human subjects have shown that FA metabolism is caused by insulin resistance conditions, and in these individuals, changes in FA metabolism occur at the plasma membrane level and within intracellular FA intramuscular transfers (6-9).

Some genes involved in lipid metabolism

Research on LXR gene expression has shown increased expression of this gene through endurance training (10). It has been reported that low-intensity physical activity (thousand steps of walking) 3 sessions per week increases the expression of LXRA, PPARY (Nuclear Oxysterol Receptor) gene in human leukocytes. In another study, it was reported that high intensity interval endurance training for 12 weeks increased PPARY gene expression (Transcription factors of genes involved in fat metabolism) in the skeletal muscle (7, 11, 12).

In another study, the researchers concluded that two sessions of combined training per week (aerobic training at an intensity of 55-70% of Vo_{2max} and resistance training at an intensity of 60-80% of 1RM) for 6 and 12 months increased PPARY and PPAR gene expression .The effect of LXR activity on glucose deficiency-induced fatigue in rats was investigated. The results showed that activation of LXR increased the consumption of fatty acids during exercise and prevented glucose deficiency-induced fatigue (13).

Various studies have reported the possible cause of increased HDL following physical activity due to adaptations to exercise. These adaptations include: increased activity of lipoprotein lipase (LPL), lecithin cholesterol acyltransferase (LCAT), hepatic lipase (HL), phospholipid transfer protein (PLTP), cholesteryl ester transfer protein (CETP) and ABCA1, ABCG1 cholesterol transporter genes, whose activities are regulated by LXR. These adaptations contribute to the formation and alteration of HDL and accelerate the reverse cholesterol transfer (RCT) process. C.a.MEF2 gene increases percentage of slow-twitch fibers and is one of the markers of oxidative metabolism (Such as myoglobin, PGC-1a, doubles EXC). According to the research conducted, the importance of LXR gene in cholesterol homeostasis indicates that moderate-intensity (endurance) physical activity increases LXR gene, while HDL-C levels are significantly increased, and TC, LDL-C, and TG levels are significantly reduced; thus, the mechanism of an increase in HDL-C by physical activity is likely to increase LXR gene expression (7,13 ,14).

The effect of resistance activity on lipolysis

In one study, moderate-intensity resistance training (10 Repetitions) and short rest

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periods between sets (1 min) induced a severe anabolic hormone response to catecholamines, and the lipolytic response time to hormones appears to be different (Catecholamines increase lipolysis rate) (15). Physical activity improves lipid oxidation by decreasing the concentration of malonyl coenzyme A. Circular resistance activity by stimulating growth hormone and catecholamines, and the enzymes involved in the lipolysis process, increase lipolysis in obese individuals, which are the major triggers of lipolysis. Today both resistance and endurance training are widely recommended for maintaining health and weight control (14, 15).

Gutto et al showed a significant increase in glycerol and NEFA concentrations in healthy individuals during endurance activity after resistance exercise, with concomitant increases in growth hormone concentrations and catecholamines, and decreased insulin concentration (lipolysis inhibitor) (6). Circular resistance exercise with a short rest interval and moderate intensity (50% of one repetition maximum (1RM)) in obese individuals increases triacyl glycerol lipase activity and is a potent stimulator of growth hormone and catecholamines, both of which are major lipolysis stimulants. It is likely that the main reason for the lower increase in lipolysis in this study compared to the study by Ormesbi et al is due to the differences in subjects, as in the present study overweight men were used.Researchers also reported that obese individuals had higher insulin concentrations and lower plasma levels of growth hormone than normal subjects. This low increase in lipolysis is likely due to the inhibition of growth hormone in these individuals and is not related to the activation of alpha-adrenergic receptors by catecholamines (8).

Adaptations to adipose tissue resulting from physical activity

Endurance training reduces white adipose tissue, while, Sympathetic systems reduce fat breakdown and reduce fat mass due to exercise. Exercise, probably in adolescent female rats may increase adipose tissue (4, 15). Endurance training reduces the number of fatty cells, but in general most exercises cannot reduce the number of fatty cells in humans (16, 17).

Enzymes

In white adipose tissue, exercise enhances mitochondrial enzymes such as cytochrome oxidase (CCO) and malate dihydrogenase (MDH), but mitochondrial enzymes in brown adipose tissue did not show any significant change or decrease in rats. Endurance training increases the supply of adipose tissue glucocorticoid, which in turn produces adaptations that facilitate lipolysis (4, 15-18).

Lipoproteins

Because blood lipids are insoluble in organic solutions such as blood plasma, they are transported by carrier molecules with a combination of protein and lipid. Free plasma fatty acids are transferred by binding to albumin. Triglycerides, phospholipids, and cholesterol along with proteins form a spherical pathway structure called lipoprotein. In the structure of lipoproteins, cholesterol esters and hydrophobic glycerides are located in center, and the proteins of polar phospholipids (Apolipoproteins) and free cholesterol surround the hydrophobic lipids as a surface polar layer. Surface polar components make lipoproteins soluble and allow insoluble lipids to flow into the bloodstream. Four main groups of lipoproteins have been identified in the blood that are differentiated depending on the amount of protein and fat in them, namely, LDL, HDL, VLDL, chyl. The higher the protein to fat ratio in these particles, the higher their density, and the higher the density lipoprotein, the smaller in size it is. LDL and HDL carry cholesterol, and VLDL and chylomicrons mainly contain triglycerides (3, 6, 13).

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Classification of lipoproteins based on ultracentrifuge in size: Chylomicrons > VLDL> LDL> HDL. HDL is an alpha protein because it usually has an electrophoretic motion similar to that of alpha 1 globulin. LDL lies in the beta-globulin region, so it is called beta-lipoprotein. VLDL lies between the alpha and beta globulins and is called pre-beta lipoprotein. Chylomicrons reside at or near the source of sampling, so their electrophoretic motion is similar to that of gamma globulins (3, 6, 19). Proteins in lipoproteins are called apoproteins or apolipoproteins (APOs) (3, 19, 20).

Chylomicrons are the largest lipoprotein particles and contain about 90% triglycerides and 1-2% of protein. The most abundant apolipoproteins in chylomicrons are APOE, APOC2 and APOB48. Chylomicrons are responsible for the transmission of dietary lipids (triglycerides, cholesterol and fat-soluble vitamins) from the intestinal microvilli to the liver and other organs of the body. VLDL, like chylomicrons are rich in triglycerides. High intake of carbohydrates increases the hepatic synthesis of triglycerides, which in turn increases VLDL production. The dominant fuel for muscle and skeletal cells is triglycerides, and in the absence of chylomicrons and in between meals, the liver packs TG and cholesterol into VLDL to provide triglycerides for these cells, sending it to the cells. Thus, forming TG in the liver is an immediate stimulus for the formation and secretion of VLDL. LDL, or Beta-Lipoprotein, is called the body fat. 50% of its weight is cholesterol and has the highest percentage of cholesterol in lipoproteins. It is produced by the metabolism of VLDL in the bloodstream and is responsible for the transfer of cholesterol made in the liver to the peripheral tissues. Its major apoprotein is APOB100. LDL is lipoprotein atherogenetic and its increase in serum is the most important risk factor for coronary heart disease (CHD). Unlike VLDL (CHY), which is only a few hours into the bloodstream, LDL particles stay stable for 3 days in the bloodstream, and with the oxygen of fats inside are swallowed by macrophages to become foam cells and deposited on the arteries, causing atherosclerotic plaque (3, 6, 13, 21).

Alpha lipoprotein is called good fat. It is the smallest lipoprotein in size and made by the intestine and the liver. Its major apolipoproteins are APOA1, APOA2. HDL transfers excess cholesterol to the liver for excretion from tissues); such as transport is called cholesterol reverse transport. HDL plays a scavenger role. The ability of HDL to take on cholesterol depends on an enzyme called Lysine cholesterol acyltransferase (LCAT), which first esterifies cholesterol and then places it in the center of the HDL. When HDL has accumulated enough lipid to become spherical, is removed and metabolized by the liver. Cholesterol removed from HDL in the liver is used for the synthesis of bile acids or secreted in the form of VLDL. HDL as a source of E, C and apoproteins also provides other lipoproteins with this apos (6, 13, 22). In a study, Yaktayar et al Investigated different exercise programs and lipid profiles of non-athlete males and found that the threshold for increasing APO A comprised performing exercise over 1 hour per session for over 12 weeks .They also found that LDL levels in the endurance and combination groups were significantly decreased, but LDL levels were not significantly elevated after 8 weeks of resistance training. Also in this study, HDL levels increased with resistance training, contradicting some previous studies (23).

Some researchers have concluded that the intensity of training can affect HDL levels, so that HDL levels can increase significantly after high-intensity training compared to low-intensity training (24). On the other hand, the subjects' weight and sex, as well as the protocol and duration of training can be important factors in HDL response to different exercise trainings. The precise mechanism underlying elevation of HDL levels remains unclear. However, it

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has been suggested that an increase in HDL is due to increased APO A, increased levels and activity of LPL and lysine cholesterol acetyltransferase enzymes, and decreased hepatic lipase activity (25, 26, 27). The levels of APO B in the combination group were significantly reduced, which may be due to the longer duration of the combined training sessions. APO B can be a major protein in LDL, therefore, with decreasing LDL levels, APO B levels decreased and LPL levels increased in all three training groups (endurance, strength, combination) but this increase was not significant. This result is consistent with most previous studies. Since lipoprotein lipase enzyme is involved in the lipolysis of VLDL, TG, and chylomicrons, increased LPL concentration increases lipolysis as well as HDL levels (28, 29).

Discussion

Obesity and overweight are nowadays one of the major health problems in the world. Despite the complexity of the obesity process, changes in lifestyle patterns including decreased physical activity and increased calorie intake, which are major contributors to a positive energy balance are regarded the main reasons for obesity and overweight. Regarding the risks of obesity and overweight and the role of physical activity in obesity control, this study aimed to investigate the effect of physical activity on the breakdown process of stored fat (triglyceride) in adipocytes. Most of the fat stored in the body is in the form of triglycerides, or acylglycerol, in adipocytes. However, a small fraction of triglycerides is also stored as fuel in the skeletal muscle cells. Both fatty acids stored in adipose tissue and circulating fatty acids can be a potential source of energy for muscle cells. In addition, small but physiologically significant amounts of fatty acids are stored as triglycerides in muscle cells. In activities with an intensity of above 70 to 80% of maximal oxygen consumption (VO_{2max}), the participation of fats in energy supply is reduced and the share of carbohydrates increases. This reflects the fact that there are limitations to increased fatty acid oxidation to release energy and regenerate ATP (3, 4, 5). The share of fats in total oxidative metabolism depends on several factors, including the intensity and duration of exercise, as well as diet and training situations. Oxidizable fat fuels include triacylglycerol, free plasma fatty acids and intramuscular triglycerides. Free fatty acids bonded to albumin in the blood move from adipose tissue and account for the major contribution of fat metabolism within skeletal muscle during exercise. During physical activity a number of neural, metabolic and hormonal stimuli lead to increased lipolysis and release of fatty acids, and fatty acids are increasingly oxidized by muscle cell mitochondria. Physical activity improves lipid oxidation by decreasing the concentration of malonyl coenzyme A. Circular resistance exercise elevates lipolysis in obese individuals by stimulating growth hormone and catecholamines, and the enzymes involved in the lipolysis process. These hormones are the main drivers of lipolysis (28, 29).

Conclusion

Physical activity through the release of more epinephrine from the adrenal glands and norepinephrine from the end of the sympathetic nerve increases the rate of lipolysis of adipose tissue. Unlike catecholamines, insulin slows down the rate of lipolysis by stimulating phosphorylation and phosphodiesterase activation. The amount of adipose tissue lipolysis is controlled by the hormone-sensitive lipase triacylglycerol (HSL), which breaks down the esterified bonds of glycerol triacyglycerol into FFA and glycerol. Since lipoprotein lipase is an enzyme involved in the lipolysis of VLDL, TG, and chylomicrons, increasing LPL concentration increases lipolysis as well as HDL levels. Regular aerobic exercise through high energy expenditure increases HDL levels and reduces plasma triacylglycerol concentrations.

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