

Symposium: Limits to fat oxidation by skeletal muscle during exercise – introduction

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ABSTRACT

HAWLEY, J. A. Symposium: Limits to fat oxidation by skeletal muscle during exercise—introduction. *Med. Sci. Sports Exerc.*, Vol. 34, No. 9, pp. 1475–1476, 2002. Lipids, in the form of adipose tissue triacylglycerol (TG), intramuscular triglyceride (IMTG), and dietary-derived fatty acids (FA) from plasma TG (chylomicrons), and very low-density lipoproteins (VLDL), represent the largest store of nutrient energy in humans. Yet despite the abundance of endogenous TG, there is limited capacity for FA oxidation during exercise: there are no mechanisms that match the availability and metabolism of FA to the rate of energy expenditure. Because of the body's limited carbohydrate (CHO) stores, and because depletion of muscle and liver glycogen reserves often coincide with exhaustion, there is interest in several nutritional interventions that increase FA availability and rates of fat oxidation during exercise: such strategies have the potential to slow the rate of glycogen utilization and delay the onset of fatigue. The five papers comprising this symposium provide a synopsis of 1) the regulation of fat oxidation in human skeletal muscle during aerobic exercise; 2) selected nutritional techniques that increase fat oxidation, spare endogenous CHO stores, and modify exercise capacity; and 3) dietary manipulations that alter macro-nutrient availability and muscle gene expression. **Key Words:** ADIPOSE TISSUE LIPOLYSIS, EXERCISE CAPACITY, GENE EXPRESSION, LIPIDS, METABOLIC REGULATION, MUSCLE TRIACYLGLYCEROL, PERFORMANCE

TRIACYLGLYCEROL AS AN ENERGY SOURCE FOR SKELETAL MUSCLE DURING EXERCISE

Lipids provide the largest nutrient store of chemical energy used to power biological processes. Compared with the finite reserves of carbohydrate (CHO), endogenous fat depots in humans represent a potentially unlimited source of fuel for oxidation by skeletal muscle. Lipid is principally stored as triacylglycerol (TG) in adipose tissue. A further physiological important store of TG can be found within skeletal muscle (IMTG), mostly adjacent to the mitochondria (14). IMTG content is highly variable and depends on fiber type (4), training status (7,8), and diet (3,12). Finally, FA for oxidation by skeletal muscle during exercise can be derived from plasma TG (chylomicrons) and very low-density lipoproteins (VLDL) formed from dietary fat in the postabsorptive state (6).

FACTORS AFFECTING FAT OXIDATION BY SKELETAL MUSCLE DURING EXERCISE

Despite the vast stores of endogenous TG, there is limited capacity for FA oxidation, especially during exercise

>65% of maximal O₂ uptake ($\dot{V}O_{2max}$). Under physiological conditions, the rate of FA oxidation depends on the interaction of a variety of factors that include the relative exercise intensity (9), training status (7,8), plasma FFA concentration (5), and endogenous (3) and exogenous (2) CHO availability.

In certain situations, FA oxidation is impaired because of a failure of the rate of lipolysis to meet the energy demands of the muscle (i.e., exercise at $\geq 85\%$ of $\dot{V}O_{2max}$). However, even when lipid is infused during intense exercise in concentrations far in excess of muscle requirements, less than 50% of total energy is derived from fat (10). This is because the muscle is also a major site of control of the rate of FA oxidation. During intense exercise, accelerated glycolytic flux results in high rates of pyruvate and acetyl-CoA formation that inhibit carnitine palmitoyltransferase 1 activity and, in turn, FA entry into the mitochondria (11). Increased glycolytic flux resulting from the ingestion of CHO before exercise also directly inhibits LCFA oxidation (2).

FAT OXIDATION AND EXERCISE CAPACITY

Because of the body's limited CHO stores, and because depletion of muscle and liver glycogen reserves often coincide with exhaustion (1), there is interest in a variety of nutritional interventions that increase FA availability and rates of fat oxidation during exercise: such strategies have the potential to attenuate the rate of glycogen utilization, delay the onset of fatigue, and enhance the performance of

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selected athletic events. Increasing FA availability by fat ingestion or i.v. infusion of intralipid, or feeding individuals high-fat, low-CHO diets, increases FA oxidation during exercise. Indeed, some of the highest rates of whole-body fat oxidation reported in the literature have been observed after short-term (<5 d) high-fat diets (13). The alterations in macronutrient availability that accompany these diets have also been shown to modify muscle gene expression. Yet, despite marked effects on metabolism underpinned by cellular changes that favor fat oxidation, the impact of dietary manipulations on exercise capacity is equivocal.

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The five papers comprising this symposium provide a state-of-the-art synopsis of 1) the regulation of fat oxidation in human skeletal muscle during exercise; 2) selected nutritional strategies that increase fat oxidation, spare endogenous CHO stores, and modify exercise capacity; and 3) dietary manipulations that alter macronutrient availability and muscle gene expression.

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