Energy Metabolism during Endurance Exercise
耐力運動的能量代謝

Stephen H. S. Wong
Department of Sports Science & Physical Education,
The Chinese University of Hong Kong, HONG KONG

王香生
香港中文大學體育運動科學系

Abstract

Carbohydrates and fats become the major energy sources during prolonged exercise. The relationship between these energy metabolism processes depends upon the pre-game diet, exercise intensity as well as its duration. The present paper attempts to review such energy metabolism relating to endurance exercise.

摘要

進行耐力運動時，身體的能量供應主要由碳水化合物和脂肪提供，兩者的供能關係取決於運動前的飲食和運動時的強度和時間，本文旨在闡釋兩者在運動時的互動關係。

During prolonged, exhaustive exercise, the depletion of muscle glycogen and dehydration are the two main causes of fatigue (Ahlborg et al., 1967a; Armstrong et al., 1985). Exercise-induced dehydration adversely affects cardiovascular function and temperature regulation (Costill & Fink, 1974; Montain & Coyle, 1992a, 1992b). Besides the body hydration level, CHO availability is also of importance to exercise performance. Prolonged exercise at 60 to 80% O_2 max is known to reduce the CHO reserves of the body, and contributes to the onset of fatigue (Ahlborg et al., 1967b; Bergstrom & Hultman, 1966; Hermansen et al., 1967; Hultman, 1967).

The energy used to sustain steady-state aerobic exercise in humans is derived predominately from the oxidation of CHO and fat (Krogh & Lindhard, 1920). Normally, bodily protein oxidation does not contribute significantly to energy production (Romijn & Wolfe, 1992, Hood & Terjung, 1990). Therefore, the four major sources of energy for exercise are muscle glycogen, blood glucose (from liver glycogen), plasma free fatty acids (FFA), and intramuscular triglyceride (Coyle et al., 1986; Romijn et al., 1993).

During prolonged, strenuous exercise, muscle glycogen and blood glucose are important substrates for contracting muscle and fatigue often coincides with depletion of these CHO sources (Coggan & Coyle, 1987; Constantin-Teodosiu et al., 1992; Sahlin et al., 1990). One cannot oxidise fat at high enough rates to provide all the energy required by moderate to high intensity exercise (Davies & Thompson, 1979). At such high intensities, CHO oxidation must provide the major source of energy not available from fat (Coggan & Coyle, 1991). Consequently, fatigue often occurs when muscle glycogen becomes depleted (Coggan & Coyle, 1991). This is the rationale for the implementation of dietary CHO supplementation before, during, and after exercise.

The amount of energy stored in the form of triglycerides within adipocytes in the body is large, totalling 200 - 625 MJ (~50,000 - 150,000 kcal) in men and women with a normal body composition of between 10 - 30% body fat. Triglycerides stored in adipocytes can be hydrolysed into glycerol and FFA; the latter must bind to the protein carrier, albumin, for transport via the circulation to the exercising muscles (Bulow & Madsen, 1981). Additional triglyceride is stored in droplets within the muscle fibres and is available for oxidation following intramuscular lipolysis.

Despite the large amount of potential energy in the body's fat stores, the rate at which they can be oxidised is limited. Thus, CHO metabolism is needed to provide the additional substrate for oxidation as the intensity of exercise is increased. It has been suggested that increases in the level of lactate in blood may retard FFA release (Boyd et al., 1974; Fredholm, 1969; Issekutz et al., 1975) and thus, reduce the availability of FFA in parallel with increasing work intensity. However, it was found

that during moderate intensity exercise in endurance-trained athletes, plasma FFA and intramuscular triglyceride contribute equally to total fat oxidation (Hurley et al., 1986; Martin et al., 1993).

CHO is stored as glycogen, both within the muscle fibres and liver (Bergstrom & Hultman, 1966; Nilsson & Hultman, 1973). Consumption of a high CHO diet is associated with an increased rate of CHO oxidation during exercise (Bergstrom et al., 1967; Galbo et al., 1979; Martin et al., 1978), and increased muscle glycogenolysis (Gollnick et al., 1972). In view of the importance of CHO for exercise performance, the goal of CHO nutritional strategies during exercise is to optimise the availability of muscle and liver glycogen and blood glucose, so as to enhance and maintain CHO oxidation (Costill & Hargreaves, 1992).

Thus, it is clear, through the literature, that during exercise of low to moderate intensity, most of the energy is derived from the oxidative phosphorylation of CHO and the lipolysis of fat. The activation threshold is low in Type I motor neurons and it increases in neurons activating Type IIa to Type IIb. Therefore, during prolonged exercise at intensities of 60 - 75% O_2 max, muscle glycogenolysis occurs primarily in the Type I muscle fibres (Gollnick et al., 1973, 1974; Tsintzas et al., 1995, 1996; Vollestad et al., 1984), although there may be some glycogen degradation in Type IIa fibres (Vollestad et al., 1984).

In the study by Ball-Burnett et al. (1990), a 2 h period of dynamic exercise was performed at an intensity of 61% O_2 max. The glycogen degradation, as estimated in pooled samples of fibres, was most pronounced in Type I fibres. Histochemical analyses showed that the glycogen loss during the first 15 min of exercise was detectable in 75% of Type I fibres, compared with 28% of Type II fibres. These results confirmed earlier studies with histochemical analyses of glycogen degradation during exercise which show that glycogen depletion initially occurs in Type I fibres, and then gradually increases, first in Type IIa fibres and then in Type IIb fibres (Essen, 1978; Gollnick et al., 1973; Thomson et al., 1979; Vollestad & Blom, 1985).

More recently, samples of single muscle fibres from the m. vastus lateralis were analysed before and after exhaustive running at 70% O_2 max (Tsintzas et al., 1996). Glycogen concentrations in Type I fibres decreased from 317.0 ± 34.2 to 31.6 ± 10.3 mmol•kg^{-1} dry weight, and in Type II fibres from 443.4 ± 44.9 to 103.9 ± 29.2 mmol•kg^{-1} dry weight. Tsintzas et al. (1996) concluded that compromised CHO availability specifically in Type I fibres is associated with fatigue during prolonged, constant pace running.

As mentioned previously, muscle glycogen is the most important substrate during prolonged exercise. Its rate of utilisation is most rapid during the early part of exercise and is related to exercise intensity (Gollnick et al., 1974; Saltin & Karlsson, 1971). As muscle glycogen declines with continued exercise, blood glucose becomes more important as a CHO fuel source. Muscle glucose uptake can increase up to 20 - 40 times the resting level, depending upon the exercise intensity and duration (Hargreaves, 1990, 1995). During the latter stages of prolonged exercise, glucose delivery may become a rate-limiting factor as arterial blood glucose levels decline (Ahlborg et al., 1974; Ahlborg & Felig, 1982; Katz et al., 1991). Accompanying the increased muscle glucose uptake is an increase in liver glucose output, so that blood glucose levels usually remain at, or slightly above, resting levels. Liver glycogenolysis supplies the majority of liver glucose output; however, during the latter stages of prolonged exercise, when liver glycogen levels are low, gluconeogenesis is an important source of glucose (Felig & Wahren, 1975). Under such circumstances, liver glucose output may fall behind muscle glucose uptake, resulting in hypoglycaemia.

There is increasing evidence that lactate, derived from contracting and inactive muscle, is an important oxidative substrate for contracting skeletal (Stanley et al., 1986) and cardiac (Gertz et al., 1988) muscle, and gluconeogenic precursor for the liver (Wasserman et al., 1991). Lactate is also a valuable metabolic intermediate, rather than simply being a waste product of anaerobic glycolysis (Brooks, 1986).

To summarise, the relative contribution of CHO and fat will be influenced by a number of factors. These include mainly exercise intensity and duration, preceding diet and substrate availability. Besides, training status, environment, age and gender can also influence the process of energy metabolism (Hargreaves, 1995).

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