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ORIGINAL ARTICLE

Influence of exercise on nutritional requirements

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Abstract There is no consensus on the best diet for exercise, as many variables influence it. We propose an approach that is based on the total energy expenditure of exercise and the specific macro- and micronutrients used. di Prampero quantified the impact of intensity and duration on the energy cost of exercise. This can be used to determine the total energy needs and the balance of fats and carbohydrates (CHO). There are metabolic differences between sedentary and trained persons, thus the total energy intake to prevent overfeeding of sedentary persons and underfeeding athletes is important. During submaximal sustained exercise, fat oxidation (FO) plays an important role. This role is diminished and CHO's role increases as exercise intensity increases. At super-maximal exercise intensities, anaerobic glycolysis dominates. In the case of protein and micronutrients, specific recommendations are required. We propose that for submaximal exercise, the balance of CHO and fat favors fat for longer exercise and CHO for shorter exercise, while always maintaining the minimal requirements of each (CHO: 40% and fat: 30%). A case for higher protein (above 15%) as well as creatine supplementation for resistance exercise has been proposed. One may also consider increasing bicarbonate intake for exercise that relies on anaerobic glycolysis, whereas there appears to be little support for antioxidant supplementation. Insuring minimal levels of substrate will prevent exercise intolerance, while increasing some components may increase exercise tolerance.

Keywords Carbohydrate oxidation · Fat oxidation · Supplementation · Immune · Risk factors

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Introduction

Although many studies have been conducted regarding the nutritional requirements of exercise, at present there does not seem to be a consensus on the recommended diet for exercise. This is not unreasonable based on the number of variables that could influence an individual's diet, including the type of exercise and person's capabilities and training status. One unifying approach could

be to evaluate the total energy expenditure of acute and chronic exercise, and recovery from exercise, and, in addition, analyze what specific substrates are used during the exercise to make recommendations for an athlete's nutritional intake. Many studies have emphasized the need to assess the total energy expenditure, and then insure that the individual takes in sufficient, but not excessive, energy. In fact, total energy intake has been reported not to match expenditure in many athletes during their competitive season (Horvath et al. 2000b), while in sedentary persons, the opposite may be true on a day-do-day basis (Meksawan et al. 2004, 2005). However, underreporting is common with self-report dietary surveys, therefore these data need to be interpreted carefully (Horvath et al. 2000b). The balance between carbohydrate, fat and protein intake has been, and remains, controversial; however, it is methodologically possible to determine the use of these substrates during exercise. If this is accomplished, then a diet that replenishes the stores and provides substrate during exercise can be recommended. This approach would allow the determination of energetic demands while also recommending micronutrients and supplements. As protein is generally not used as a fuel for exercise, its intake would have to be evaluated based on protein needs for other functions.

This review will evaluate the energy requirements of resistance, anaerobic and aerobically dominated exercise, and consider some important modulators of these, including gender, training, and age. No attempt will be made to evaluate nutritional requirements of specific types of exercise or sports. In addition, although hydration status is of critical importance for exercise tolerance, it is a broad topic unto itself, thus this review concentrates on substrate use and availability.

Estimating exercise energy expenditure

As both exercise intensity and duration influence the energy during exercise, and thus the substrates used, both of these factors have to be considered required to recommend diets. di Prampero (1981, 1986) considered the impact of intensity and duration for exercise bouts lasting from a few seconds to several minutes in a model that is expressed quantitatively in Eq. 1. The energy requirement of an exercise can be described as the relationship between maximal metabolic power (E'_{tot}, kW) and exhaustion times (t, s):

$$E'_{\mathrm{tot}} = \mathrm{AnS}t^{-1} + \alpha V'\mathrm{O}_{2\,\mathrm{max}} - \alpha V'\mathrm{O}_{2\,\mathrm{max}}\tau\left(1 - \mathrm{e}^{-(t/t)}\right), \quad (1)$$

where α is the energy equivalent of O_2, τ is time constant of the kinetics of $V'O_2$ at the onset of exercise, AnS is the amount of energy (kJ) derived from anaerobic energy utilization and $V'O_{2max}$ (kW) is the net maximal oxygen uptake.

Utilizing Eq. 1, it is possible to calculate the aerobic contribution (Aer, kW) to maximal metabolic power (E'_{tot}, kW) as the sum of the second and third term, and the anaerobic contribution is represented by the term $\mathrm{AnS}t^{-1}$. In turn, AnS is calculated as the sum of the energy derived from lactic acid production (Anl, kJ) plus that derived from maximal phosphocreatine (PCr) splitting in the contracting muscles (AnAl, kJ). The energy derived from complete utilization of PCr stores (AnAl) can be estimated assuming that, in the transition from rest to exhaustion, the PCr concentration decreases by 18.5 mmol/kg muscle (wet weight) in a maximally active muscle mass (e.g., assuming it corresponds to 30% of body mass). Assuming further, a P/O_2 ratio of 6.25 and an energy equivalent of 20.9 kJ/lO_2, AnAl can be calculated as: $(18.5/6.25)0.3 \times 468.2 =$ 416 J/kg body mass. Two studies have confirmed that PCr splitting and O_2 debt increase linearly with power output during aerobic exercise. In fact, they have demonstrated that PCr concentration sets the time course of oxidative metabolism at the start of exercise in humans (Francescato et al. 2003, 2008). The quantitative analysis described in Eq. 1 has been used by di Prampero and co-workers for many years to evaluate the energy requirements of exercise. Some examples include running (di Prampero et al. 1993), walking (Minetti et al. 1995), karate (Francescato et al. 1995), swimming (Capelli et al. 1995), bicycling (Capelli et al. 1993), speed skating (di Prampero et al. 1976), rowing (Celentano et al. 1974), gondola sculling (Capelli et al. 1990), multiple sclerosis patients (Olgiati et al. 1986) and microgravity in space (Antonutto et al. 1991).

Whereas the exercise intensity sets the rates of utilization, it is the duration that determines the total energy requirement for all forms of locomotion, as pointed out by di Prampero (2003). The greatest rate of potential energy is derived from PCr and can be influenced by its concentration in muscle; it has a high rate, but very low capacity (short duration). The substrate with the next highest rate of energy supply is anaerobic glycolysis and is dependent on intramuscular glycogen, which is highly concentrated around the fast twitch fibers that are recruited for high intensity, short duration exercise. Finally, the pathway with the greatest capacity is oxidative metabolism (VO_2) for which the contribution of substrates can easily be derived from the relationship between the respiratory quotient (RQ) and VO_2.

Estimating substrate requirements for exercise

The balance of substrates used [carbohydrates (CHO), fat] during exercises below the intensity where lactate increases significantly above resting levels in the venous blood (aerobic) can be estimated from the respiratory exchange ratio (RER) under conditions when arterial pressure of carbon dioxide (P_aCO_2) is constant and normal (~40 mmHg) (Meksawan et al. 2005), and if utilization of protein is assumed to be minimal. The relationship between RER and VO_2 (in absolute values) can be influenced by gender, training, age, etc.; however, if VO_2 is normalized for VO_{2max}, its relationship can be used to determine substrate use across all of these conditions. This relationship is shown in Fig. 1. As can be seen, the contribution of fat to total energy decreases, and CHO increases, as the intensity of the exercise increases, so that at VO_{2max}, primarily only CHO is used. The data in Fig. 1 are a combination of data from sedentary men and women, as there were no gender differences (Meksawan et al. 2005). It should be noted however, that even at intensities approaching and above the intensity where lactate begins to appear in blood, fat is supplying significant amounts of energy, thereby sparing glycogen.

A previous study has shown that sedentary males have higher VO_{2max} values than females; however, dietary intake of fat versus CHO does not appear to affect VO_{2max} for either gender (Meksawan et al. 2004). In both males and females, RER increases linearly and similarly with VO_2, reaching 1.0 (CHO use) at a VO_2 that is above the intensity where lactate begins to appear in blood above the resting level. When the fat and CHO oxidation (CHOO) are expressed as a function of the exercise VO_2, fat oxidation (FO) accounts for about 80% and CHO 20% of total energy expenditure. Although there is a tendency for FO to be higher on a high fat diet and lower on a low fat diet, these differences do not appear to be significant (Meksawan et al. 2005). From these data it is possible to calculate the energy derived from FO and CHOO as a function of time, and then multiply these by the exercise time, thus calculating the total energy spent from fat and from CHO. Assuming normal pre-exercise stores of fat and CHO, the dietary intake requirement of these two substrates to replenish the stores can be calculated. As these calculations assume that protein is not a primary source of energy during exercise, protein intake has to be judged by other means, as do micronutrients.

Substrate utilization in trained individuals and athletes is different from that of sedentary persons. It is well established that aerobic exercise training significantly improves VO_{2max}, as well as FO (Jeukendrup et al. 1998; Kiens et al. 1993, 1997; Calles-Escandon et al. 1996). In addition, endurance athletes have a significantly greater innate (genetic) FO as shown by the analysis of white blood cell fat oxidation (Pendergast et al. 2004). The latter appears to be a good estimate of the genetic predisposition for FO (Vladutiu et al. 2002; Pendergast et al. 2004). This can best be seen by comparing the percentage of energy from fats and carbohydrates from the sedentary subjects in Fig. 1 with that of the elite athletes in Fig. 2. As can be seen in Fig. 2, the RER as a function of time in an endurance run is

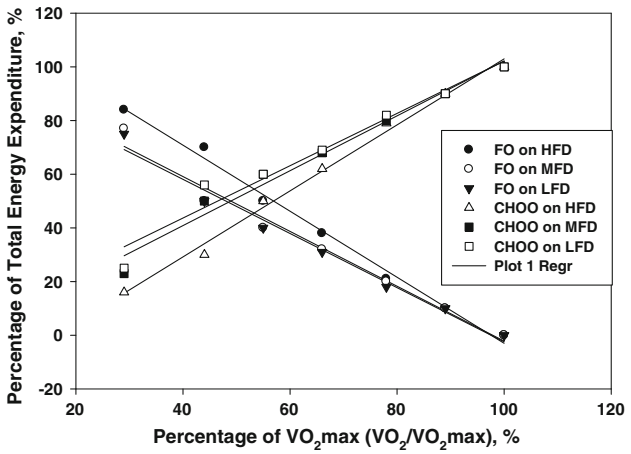

Fig. 1 The relationship between substrate utilization, expressed as % of total energy calculated from RER, and the VO_2 during a treadmill test is plotted for normalized maximal oxygen consumption (VO_2/VO_{2max}, %). The data are average values for a group of sedentary men and women on diet comprised of high fat (HFD, 55%), medium fat (MFD, 30%), and low fat (LFD 15%). Fat oxidation (FO) (*filled circle*, *open circle*, *filled inverted triangle*) and CHO oxidation (CHOO) (*unfilled upright triangle*, *filled square*, *unfilled square*) are shown for the three diets, respectively

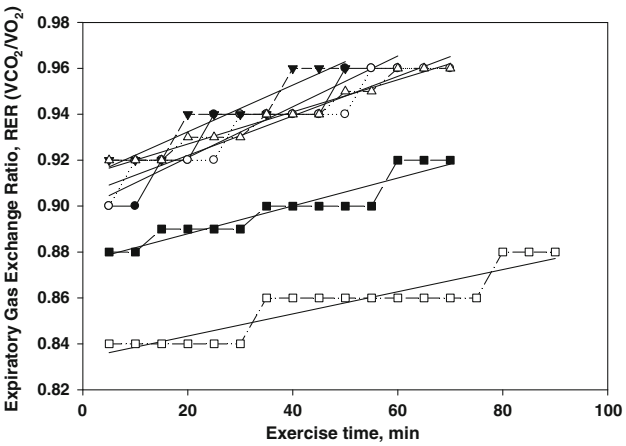

Fig. 2 Respiratory exchange ratio (RER) is plotted as a function of walking/running time for sedentary subjects (*filled circle*), trained subjects (*filled inverted triangle*), and elite (*filled square*) men exercising at 70, 80 and 85% of maximal oxygen consumption on a diet with 20–25% fat (61% CHO, 19% protein) (*closed symbols*) versus a diet of 40–45% fat (45% CHO, 15% protein) (*open symbols*)

lower, i.e., greater fat oxidation, and exercise time longer on both low and high fat diets in athletes, and more so in elite athletes. Thus, at a given percentage of VO_{2max}, FO in athletes contributes more to the total metabolism than in sedentary. This relationship is born out practically, as less gifted or trained athletes can run a marathon at about 60% of VO_{2max}, while elite athletes can run at 80–89% of VO_{2max}, thus accounting for, in addition to VO_{2max}, the differences in time to complete the race. The main concept of energy utilization during exercise is to consider at what rate substrates are used and the capacity of the substrate stores, in order to determine how long intensity can be sustained (di Prampero 1986). For fat, the higher its rate of oxidation, the more glycogen that is spared, and thus the rate of depletion of glycogen is slower, and exercise duration is prolonged (glycogen sparing).

Contribution of CHO to exercise metabolism

CHO metabolism (glucose or glycogen) is a readily available substrate with high metabolic power (Costill et al. 1981; Costill and Hargreaves 1992; Romijn et al. 1993). In fact, about 75% of the mitochondrial membrane is available for CHO metabolism (Hoppeler et al. 1999). High availability of CHO and low availability of free fatty acids (FFAs) results in increased CHO oxidation and reduced FO via reduced adipose tissue lipolysis by insulin (Campbell et al. 1992) or by inhibiting long chain fatty acid uptake into the mitochondria by inhibition of carnitine palmitoyltransferase I (CPT I) activity (Coyle et al. 1997). In addition, the decrease in pH that can accompany glycolysis also inhibits FO. Many studies have shown the importance of glucose at low exercise levels, and intramuscular glycogen at higher submaximal VO_2 levels, with glycogen used exclusively at higher exercise intensities (above VO_{2max}), i.e., anaerobic glycolysis. In fact, it is well established that the consequences of glycogen depletion in sustained submaximal exercise or the secondary consequence of lactate build up in high intensity exercise results in termination of exercise and fatigue (Costill and Hargreaves 1992).

Contribution of fat to exercise metabolism

Fat is an essential macronutrient that provides energy via β-oxidation (37.7 J/g, about twice that of CHO). It requires greater oxygen for oxidation. Fat also protects vital organs, provides insulation, transports fat-soluble vitamins, and serves as a precursor of steroid hormones, bile acids and vitamin E. FFAs are transported with short chains freely diffusing and longer chains transported via transporters carnitine palmitoyltransferase II (CPTII) (MIM # 600649) and CPTII (MIM # 255110) through the mitochondrial membrane (outer and inner, respectively) (Vladutiu et al. 2002). Studies have demonstrated that a fat droplet is adjacent to about 26% of the mitochondria where fat is transported through the mitochondrial membranes (Hoppeler et al. 1999). Genetic defects in CPTI or CPTII result in a significant reduction in fat oxidation, increased glycogen use, exercise intolerance and in some cases, myopathy (Vladutiu et al. 2002).

The availability of fat and CHO has the potential to alter the patterns of substrate oxidation as the metabolism of these substrates is interrelated. Randle et al. (1963) suggested that increased FFA availability leads to increased fat oxidation (Schrauwen et al. 1997), and concomitantly decreased CHO oxidation through inactivation of pyruvate dehydrogenase. In addition, high fat diets increase the activity of CPTII, one of the rate limiting steps in fat oxidation (Chen et al. 1992).

Effects of exercise on substrate utilization

Fat provides the majority of substrate used during exercise at 25% of maximal VO_2, and comes mainly from the blood (Romijn et al. 1993). However, during moderate exercise (65%VO_{2max}), the contribution by FO is less, but still important, with intramuscular stores being the primary source of both fat and CHO (glycogen). During high intensity exercise (85%VO_{2max}), intramuscular glycogen is the major source of substrate and CHO oxidation is the major energetic pathway (Romijn et al. 1993). At low exercise levels where FO dominates, exercise can be sustained for up to 2 h. Alternately, a progressive increase in plasma FFA and glucose availability over time was observed at 65%VO_{2max}. However, at higher exercise levels (85%VO_{2max}), exercise time became limited, presumably due to glycogen depletion (Costill et al. 1981; Costill and Hargreaves 1992; Romijn et al. 1993).

Current dietary recommendations

Current dietary recommendations for the general adult population are 45–65% (5–12 g/kg) for CHO, 20–35% (limited saturated and trans fats) for fats and 0.8 g/kg/day (1.2–1.8 g/kg) for proteins (Manore 2005; Phillips 2006). Recommendations for linoleic acid in this group are 14–17 and 11–12 g/day for men and women, respectively, and 1.6 and 1.1 g/day for men and women, respectively, for linolenic acid (Manore 2005). Recommendations for fat intake in the general population have been suggested to be less than 30% (10% from saturated and 330 mg/day from cholesterol) of total energy intake to promote health (American Heart Association, LaRosa et al. 1990), with the balance CHO and protein. Dietary expenditures, and thus

intakes, of athletes need specific consideration due to the athletes' physiological make up and the requirements of the specific types of exercise they are engaged in.

In pioneering work by di Prampero et al. (1969, 1970, 1978, 1981) in dog muscle, the time course and energy balance between high-energy phosphates, anaerobic glycolysis (lactate production) and VO_2 were extensively studied. Using this model, the energy requirement, and thus nutritional needs, can be evaluated for specific exercise types, i.e., resistance exercise, types dominated by anaerobic pathways and finally aerobic pathways. These data support the use of Eq. 1 to estimate the contribution of the various energy sources (di Prampero 1981). In addition, di Prampero et al. (1969, 1993) proposed and validated a method to estimate the anaerobic contribution to exercise from blood lactate measurements. It is important to note that glycogen is used for both. It is further worth noting that during the recovery from this type of exercise, lactate is converted to pyruvic acid and reduced. As the half-time of the VO_2-off response is fast (about 26 s) (di Prampero et al. 1969), and lactate is available, little glycogen is used during recovery from exercise. The implication of these data for dietary intake is that it is essential to provide sufficient CHO in the diet to reestablish the glycogen stores in the muscle and liver (Costill and Hargreaves 1992). This replenishment takes 24–48 h. It is time course dependent, and depends on the level of depletion, i.e., the greater the depletion, the longer the time to reestablish the stores (Costill et al. 1981; Costill and Hargreaves 1992). As fat oxidation in this type of exercise is minimal, the fat intake should be kept at the minimal recommended dietary level (about 30%). As high intensity exercise may result in protein degradation, some have argued that protein intake should be increased, especially for athletes performing at higher exercise intensities (Evans 2001, 2004). However, this is not agreed upon in all studies (Koopman et al. 2007; Kumar et al. 2009). Perhaps a recommendation to keep protein intake at 15–19% would be a reasonable compromise based on the sum of the results of these studies.

Role of nutrition in resistance exercise

Energy requirement of resistance exercise

Resistance exercise is characterized by short bursts of near maximal power output, and as such requires primarily high-energy phosphates and some contribution of anaerobic glycolysis (di Prampero 1986). Resistance exercise has been shown to reduce the excretion of nitrogen, thus minimizing the need for increased amino acid availability and protein intake (Evans 2001, 2004). This confirms that dietary protein intake of up to 1.6 g/kg/day may increase the hypertrophic response to resistance training (Evans 2001). In these cases, the energy to resynthesize ATP would be anaerobic, and mostly dependent upon glycogen during muscle contractions and recovery. This being the case, a diet high in CHO would be advisable, while sustaining the minimal fat intake requirements. During this type of exercise it may be the case that the total energy and protein intake may not be determined by the apparent energy required to "lift the weights". Resistance exercise depresses muscle protein synthesis, whereas protein breakdown does not appear to be accelerated (Kumar et al. 2009). Resistance exercise, when matched with appropriate nutrition, is a significant anabolic stimulus for muscle hypertrophy due to increased muscle protein synthesis from amino acids and attenuation of muscle protein breakdown by CHO (Tipton and Ferrando 2008).

Exercise training appears to increase basal muscle protein turnover (Kumar et al. 2009). Some studies have suggested that athletes, particularly those involved in resistance exercise, require more protein; however, this is not the general recommendation (Phillips 2006). Studies have shown that high protein intake (30–35%) may be effective in weight loss, particularly fat mass loss. If weight loss is important, this may be useful for sedentary people as well as athletes (Phillips 2006). Increased protein synthesis is a transient storage phenomenon; however, when it is coupled with the upregulation of anabolic signaling pathways initiated by exercise, it provides the amino acids and energy for protein synthesis (Miller 2007). There are suggestions that post-exercise ingestion of protein is required to achieve a protein balance and maximize adaptive responses of muscle (Koopman et al. 2007); however, this is not universally found. Post-exercise amino acid availability does not appear to be critical for protein synthesis (Kumar et al. 2009).

Nutrient intake in resistance exercise

In addition to PCr supplying energy for resynthesis of ATP, creatine and phosphocreatine are intracellular buffers during the transition from rest to exercise and in exercises requiring very high energy turnover like weight lifting. Creatine loss is replaced by dietary intake and endogenous synthesis. Although it has long been known that creatine supplementation is beneficial in patients with reduced endogenous synthesis and those with low dietary intake (Brosnan and Brosnan 2007), supplementation in normal individuals has been controversial. Creatine levels are highly variable, and are influenced by muscle fiber type, age, and disease, but apparently not by training and gender (Balsom et al. 1994). Testosterone and creatine supplementation appear to facilitate muscle protein synthesis, while growth hormone, androstenedione and dehyroepiandrostenedione do not

(Tipton and Ferrando 2008). Creatine supplementation increases lean body mass in men and women and may also reduce protein breakdown (Tarnopolsky 2000). Although some studies have shown that oral administration of creatine increases its stores in the muscle (Feldman 1999), this is not universally found. Its role in exercise metabolism remains controversial. These results may be due to the creatine levels of the subjects prior to supplementation and the dose. It has been shown that a dose of 20 g/day for 5–6 days increases creatine concentration (30% as phosphocreatine) and performance. However, increasing the dose above 20 g/day does not further improve storage of PCr, or performance (Casey et al. 2000). Similarly, in persons with high initial creatine levels (150 mmol/kg dry mass), there was no benefit of creatine supplementation (Casey et al. 2000). In addition, the benefit of creatine supplementation is improved when CHO intake, and presumably glycogen, is increased. There is concern, however, regarding the potential risk of long-term creatine supplementation, particularly in its pure form, as it may cause down regulation of creatine synthesis (Feldman 1999; Silber 1999).

Role of nutrition in exercise requiring "anaerobic" energy

Energy requirement for anaerobic exercise

As described above, Eq. 1 can be used to estimate the contribution of anaerobic energy to exercise from lactate measurements in blood (di Prampero et al. 1969, 1981, 1993). It can be seen that for exercises more that 15 s and less than 4 min, anaerobic glycolysis dominates the balance of energy supplied, with $\dot{V}O_2$ playing a role as time increases. However, it is important to note that glycogen is used for both.

Nutrient intake for anaerobic exercise

During heavy exercise and training, CHO intake by athletes may need to be as much as 8–10 g/kg/day (60–70% of total energy intake) to maintain muscle and liver glycogen stores (Costill and Hargreaves 1992). This diet may result in reduced essential fatty acids, fat-soluble vitamins and micronutrients. For example, a low fat diet, particularly in those who restrict their lean meat use, has been shown to result in inadequate intake of zinc and vitamin E (Retzlaff et al. 1998). In athletes where the energy expenditure is higher, most athletes consume sufficient amounts of protein (Horvath et al. 2000b); however, they may have insufficient energy intake, and micronutrients (Horvath et al. 2000b; Ziegler et al. 2002) such as calcium, zinc and iron. This is particularly true for female athletes. It has also been suggested that athletes in high intensity training on a low fat diet may have reduced vitamins A, E, C and B_{12} (Horvath et al. 2000b).

During prolonged exercise or intensities above where lactate appears in the blood, muscle glycogen is an important substrate. It has been well documented that depletion of muscle glycogen and hypoglycemia are associated with reduced exercise performance and fatigue (Costill and Hargreaves 1992). In addition to fasting or over exercising, very high fat diets can decrease muscle glycogen content, impair exercise performance and lead to fatigue (Jeukendrup et al. 1998). High CHO intake 3–4 h prior to exercise has been shown to ensure adequate CHO during exercise (Costill and Hargreaves 1992). Post-exercise nutrition to reestablish stores of glycogen in the liver and muscle is critical to maintain chronic exercise capabilities. It has been suggested that immediate intake of CHO post-exercise (1 g/kg every 2 h, 7–10 g/kg/day), particularly with foods that have a high glycemic index, greatly improves recovery from exercise (Burke 1997).

Another potential nutritional contribution to anaerobic metabolism during exercise is the role of bicarbonate (HCO_3) supplementation. There is convincing evidence in animal models that HCO_3 plays an important role in acid–base balance in muscle during exercise, particularly in fast twitch muscle (Grossie et al. 1988). Studies have shown that 0.3 g/kg bicarbonate and 0.3–0.5 g/kg citrate taken orally with 1–2 L of water 60–90 min pre-exercise increases the pH in the extracellular space (McNaughton 2000). Higher doses spread over a day or so may have prolonged effects, including improved training duration (McNaughton et al. 1999). These changes have shown performance enhancement in high intensity exercise (either 1–7 min continuous or 30–60 min intermittently) (Burke 2007). HCO_3 may also assist in high intensity exercise training regimens (Edge et al. 2006).

Role of nutrition in exercise requiring "aerobic" energy

Energy requirement for aerobic exercise

During the transition to a "steady state" in reasonably fit persons, the kinetics of the $\dot{V}O_2$ on-response is rapid enough [$t_{1/2}$ = 17 s (animal) −26 s (human)] that anaerobic glycolysis (lactate formation) is not required. Hydrolysis of phosphocreatine (PCr) provides the energy deficit between demand and supply by $\dot{V}O_2$ (Francescato et al. 2008). This relationship is such that PCr concentration sets the rate kinetics of the adjustment of $\dot{V}O_2$ to "square wave" exercise; thus, it is one of the main controllers of $\dot{V}O_2$ (Francescato et al. 2008). Based on this relationship, the depletion of PCr at the start of exercise would slow $\dot{V}O_2$,

and require an obligatory contribution from anaerobic glycolysis, leading to lactate production (di Prampero et al. 1969; Francescato et al. 2008). It has been shown that low intramuscular PCr interferes with metabolism during exercise. In addition, in unfit subjects the slow adjustment of VO_2 results in significant anaerobic glycolysis during the transition from rest to exercise. This can be reversed by exercise training (Pendergast et al. 1979; Cerretelli et al. 1977, 1979). However, there is not convincing evidence that PCr supplementation in subjects with normal levels get additional benefits (Balsom et al. 1994; Casey et al. 2000).

Nutrient intake for aerobic exercise

Previous studies clearly indicate that an optimal amount of CHO and fat from blood and muscle are needed for exercise performance, as depletion of either or both results in termination of exercise, fatigue, and perhaps even muscle myopathies. Maximizing glycogen stores with high CHO diets is widely recommended (Costill et al. 1981); however, this is not universally accepted (Hawley et al. 1997). High dietary fat intake may cause an increase in fat oxidation and glycogen sparing, which when combined with greater intramuscular fat stores (Hoppeler et al. 1999) may prolong exercise performance in athletes (Horvath et al. 2000a; Pendergast et al. 1996a) and in sedentary subjects (Meksawan et al. 2005). In other studies, a high fat diet resulted in an increase in endurance running time (Muoio et al. 1994; Horvath et al. 2000b). A very low fat diet was shown to reduce intramuscular fat stores (Coyle et al. 2001; Hoppeler et al. 1999), and it would be expected that this would reduce exercise endurance. Intramuscular lipid droplets are significantly reduced after prolonged exercise (Decombaz et al. 2001) when on a low-fat diet (Hoppeler et al. 1999) and in untrained subjects (Decombaz et al. 2001). These low stores increase the dependence on glycogen and lead to reduced endurance exercise performance (Horvath et al. 2000a; Pendergast et al. 1996a, b; Meksawan et al. 2005).

Exercise performance on diets with about 15% protein, 15% fat, and 70% CHO have been compared to diets with 15% protein, 35% fat and 45% CHO, as well as a diet with 15% protein, 40% fat and 45% CHO in both athletes and sedentary subjects (Horvath et al. 2000a; Pendergast et al. 1996a, b; Meksawan et al. 2005). In these studies, it was demonstrated that endurance exercise time (running/walking at 70% of VO_{2max}) was shortest on the lowest fat diet, and increased on both the medium and higher fat diets (see Fig. 2). It should also be noted that the RER was lower for athletes (higher fat oxidation) than sedentary individuals; however, in both groups the RER did not increase significantly as a function of time. In fact, the ratio of substrate use (CHO and fat) was relatively constant as a function of time, emphasizing the glycogen sparing role of fat oxidation. It can also be concluded that subjects will stop exercising when either glycogen (Costill and Hargreaves 1992) or fats (Hoppeler et al. 1999) are depleted.

As can be seen in Fig. 2, athletes and elite athletes have lower RERs, respectively, than sedentary subjects during exercise at all intensities. In Fig. 2, it can also be seen that the endurance exercise time is higher on a high fat (40–45%) than on a lower fat (15–20%) diet in all three groups. However, the higher the fitness level (sedentary to athletic), the greater the effect that diets have. The greatest effect of the diet was on the elite athletes who were more highly trained and had a greater genetic disposition for fat oxidation (Vladutiu et al. 2002; Pendergast et al. 2004).

The potential benefit to exercise performance of a higher fat intake, while maintaining sufficient CHO intake, appears to require long-term intake (Helge 2002). Short-term intake (<7 days) increases fat stores and fat oxidation; however, it has minimal effect on performance in athletes (Starling et al. 1997; Burke et al. 2002). Longer term intake has significant effects on performance of athletes (Lambert et al.1994; Muoio et al. 1994; Horvath et al. 2000a, b; Pendergast et al. 1996a, b) and sedentary subjects (Meksawan et al. 2005). High fat intake before exercise or immediately post-exercise cannot be supported by research evidence (Jeukendrup et al. 1998). Post-exercise nutrition to reestablish stores of fat in the muscle is critical for maintaining chronic exercise capabilities.

It can be concluded that for short duration exercise at a higher percentage of VO_{2max}, CHO intake and glycogen stores are critical. However, if exercise is sustained for longer periods of time, stores of both glycogen and fat are important, as depletion of either would result in exercise intolerance. For example, in ultra distance runs like the Iditarod, the sled dog diets have extremely high fat content.

Micronutrient intake and exercise

Acute and chronic exercise could increase the micronutrient requirements thereby playing an important role in the immune and antioxidant systems of both sedentary individuals and athletes. High doses of antioxidants can minimize the effects of radical oxygen species or, alternatively, generate pro-oxidant effects. However, these benefits appear to occur only when there are nutritional deficiencies, and not in well trained athletes (Margaritis and Rousseau 2008). In addition, heavy use of antioxidants has unknown long-term risks.

Vitamin supplementation has also been widely speculated. For example, phytoestrogens that bind to estrogen receptors lead to an upregulation of the expression of antioxidant genes (Vina et al. 2007). However, evidence

has been reported that does not support this postulate (Vina et al. 2007). Subjects on a higher fat diet had improved intake of vitamin E and zinc and corrected the deficiencies seen on a very low fat diet (Meksawan et al. 2004).

Modulators of energy and nutritional requirements

Effects of gender on exercise nutrition

Protein metabolism is lower in females (Tarnopolsky 2000); however, protein turnover in young men and women appears to be similar during exercise (Kumar et al. 2009). Females have higher fat oxidation and lower CHO oxidation than men during exercise (Tarnopolsky 2000). These gender differences are primarily associated with estrogen levels in females (Tarnopolsky 2000). The data shown in Fig. 1 and for sedentary individuals and athletes in Fig. 2 represent data for men and women combined, indicating that there was no gender differences in fat oxidation or the effect of training. Some recent studies have suggested, however, that exercise training in females does not improve fat oxidation, particularly in older women.

Studies have suggested that supplementation of CHO (>8 g/kg/day), protein and creatine in females improves performance. Females show less of an increase in lean body mass than men after creatine supplementation (Tarnopolsky 2000).

Effects of training on exercise nutrition

Exercise training induces adaptations in skeletal muscle that alter substrate utilization. Fat oxidation at rest is greater in trained individuals, and this is independent of energy intake, body composition or changes in lipolysis (Calles-Escandon et al. 1996). It has been well documented that exercise training increases fat oxidation and reduces CHO oxidation during exercise at absolute VO_2 levels (Holloszy and Coyle 1984; Coggan et al. 1990). This increase in fat oxidation could occur in as few as several days of training, and is due to increased intramuscular triglyceride oxidation and reduced glucose oxidation (Phillips et al. 1996). Fatty acid profiles (phospholipids and triglycerides) in muscle in trained and untrained individuals are different even though the composition of their dietary fatty acids is similar (Andersson et al. 2000). The increase in fat oxidation is associated with the increase in VO_{2max} with training, secondary to increased size and number of mitochondria, capillary density and related enzymes (Jeukendrup et al. 1998; Spina et al. 1996; Kiens et al. 1993). Thus, expressing fat oxidation as a function of VO_{2max} minimizes the differences in fat oxidation as a result of training and inter-individual differences.

Exercise training appears to increase basal muscle protein turnover (Kumar et al. 2009), affecting both degradation and resynthesis similarly. These two changes lead to a protein balance, so the decision about protein intake should be determined primarily by acute needs.

Effects of aging on exercise nutrition

It is well known that VO_{2max} decreases with age (Evans and Cyr-Campbell 1997; Hawkins and Wiswell 2003). There is also an increased fat mass and deterioration in muscle mass, muscle function and metabolism (Manetta et al. 2005) with aging, all of which could influence the exercise energy expenditure and nutritional intake in the elderly. It has been reported that the ability to use fat as a fuel during exercise is reduced in older individuals (Blaak 2000). Muscle fat oxidation is the main determinant of total body fat oxidation at rest and during exercise. Fat oxidation has been shown to be lower in the elderly (25–35%), even after correction for VO_{2max} (Sial et al. 1996). Reduced muscle FO is influenced by hormonal and pharmacological stimulation of lipolysis which has been shown to be diminished with age (Blaak 2000). In addition, mitochondrial function also decreases with age (Blaak 2000). A critically important change with aging that affects nutrition is the reduction of VO_{2max}, as substrate utilization at absolute VO_2s is strongly related to a person's VO_{2max}.

Aerobic exercise training in younger and elderly individuals has been shown to increase physical activity and fat oxidation (Solomon et al. 2008). Part of the reduced fat oxidation may be related to increased visceral and subcutaneous fat and decreased muscle mass with aging, which may be a secondary effect of inactivity.

Aging appears to reduce the responses of myofibrillar protein and anabolic signaling in response to resistance exercise (Kumar et al. 2009). Higher than recommended protein intake (1.0–1.6 g/kg/day) in older individuals has been shown to be associated with increased bone-mineral density (when adequate calcium is taken) and increased muscle mass in people performing resistance exercise; there is no apparent health risk (Lucas and Heiss 2005; Evans 2004).

Effect of nutrition on oxidative stress, blood lipids and immune function

Exercise-induced oxidative stress has been linked to muscle fatigue and damage, as well as reduced exercise performance, and is a key element in overtraining (Konig et al. 2001). It is well recognized that exercise training is beneficial for blood lipids and lowers the risk of cardiovascular disease (CVD). This has led to the recommendation of low

fat diets, particularly low in saturated fats. However, the link between CVD and fat intake is eliminated when factors concomitant with high fat diets are considered (Hu et al. 1997). It is true that elevated levels of cholesterol and low density lipoproteins (LDL) (Schaefer 1993) are related to high mortality from CVD (LaRosa et al. 1990). However, cholesterol is synthesized by the body, and can be obtained from food derived from other nutrients. Thus, dietary fat intake may not be the cause of high cholesterol is individuals. Similarly, high triglycerides are associated with CVD (Austin et al. 1998); however, they are also a source of FFAs for oxidation. Low fat diets are often recommended as they lower high-density lipoprotein cholesterol (HDL-C); however, they also elevate triglycerides (Sacks and Katan 2002). Although these data suggest that a low fat diet may be beneficial, the studies described above did not control total energy intake. Higher fat diets, if unregulated, typically result in higher total energy intake as well, which would influence cholesterol and triglycerides. Studies where total energy intake was matched to expenditure on a high fat diet (42%) in athletes failed to show weight gains or negative effects on blood lipoprotein levels (Leddy et al. 1997; Brown and Cox 1998). In fact, HDL-C levels were improved. In the same studies, however, lowering fat intake to 16% compromised total cholesterol, HDL-C ratios and levels of ApoA1 and HDL-Cg.

Similar to immune function, blood lipids were negatively affected by a diet of 15% fat in both athletes (Leddy et al. 1997) and sedentary subjects (Meksawan et al. 2005), while a diet with 55% fat did not negatively affect them. These changes included lower HDL cholesterol and apolipoprotein A1, and a negative effect on the ratios of HDL/LDL and A1/A2.

Although the aspects described above relate to the effect of nutrition on exercise performance, a secondary consequence is the effect of nutrition on the stress caused by exercise. It is well documented that relatively intense (high % of VO_{2max}) or prolonged exercise results in an increase in inflammatory, as well as anti-inflammatory, mediators, thereby increasing the risk of inflections (Gleeson et al. 2004). These changes can occur in the absence or presence of exercise-induced muscle damage, and are exacerbated by nutritional deficiencies (Gleeson et al. 2004). It is clear that high levels of exercise, particularly overtraining, can suppress the immune system (Nieman 1997). Acute exercise induces increased levels of leukocytes (T cells, CD4+, CD8+ T cells) and neutrophils (Yamada et al. 2000); however, this is reversed with exercise training. Natural killer cells are also released post-exercise and rapidly react to viruses and bacteria (Mackinnon 2000). Pro-inflammatory cytokines (IL-1ß, IL-6, and TNF-α) are also increased post-exercise. Recent evidence suggests that elevated IL-6 post-exercise may actually have an anti-inflammatory role.

Deficiencies in fat, CHO and protein, as well as micronutrients (i.e., iron, zinc and vitamins A, E, B6 and B12) can lead to immunocompetence. Ironically, excesses of fat, CHO, protein or micronutrients can also lead to immunocompetence. CHO depletion, due to inadequate dietary intake or acute or chronic use, can lead to increased hormones in the circulation, as well as inflammatory immune responses (Gleeson et al. 2004). Also ironically, both low (Venkatraman et al. 1997, 2000a, b, 2001; Venkatraman and Pendergast 1998, 2001, 2002) and very high levels (Gleeson et al. 2004) of fat may also compromise the immune system. Although ascorbic acid (vitamin C) is increased transiently post-exercise, there is no evidence that supplementation above daily requirements is required in either athletes or non-athletes (Peake 2003). There is currently a lack of support for taking high doses of antioxidant vitamins, glutamine, zinc, probiotics and Echinacea to prevent or minimize exercise-induced immune stress (Gleeson et al. 2004).

Although immune function is influenced by exercise intensity and duration, it has been suggested that higher fat diets are more inflammatory, and less anti-inflammatory. In other studies, where total energy intake was matched to energy expenditure, it was shown that for subjects on a low fat diet (15%), exercise resulted in a greater increase in inflammatory cytokines, chemokines and plasma soluble intercellular adhesion molecules (Venkatraman et al. 1997, 2000a, b, 2001; Venkatraman and Pendergast 1998, 2001, 2002) than when on a medium fat diet (30%). In these studies, it was also shown that a diet with 55% fat did not negatively affect immune function.

Summary

The approach of this review was to discuss the nutritional needs of sedentary individuals and athletes based on their total energy expenditure and the balance of substrates used during exercise. di Prampero (1986) expressed the impact of intensity and duration for exercise bouts lasting from a few seconds to several minutes (Eq. 1) and this relationship can be used to estimate energy expenditure. When data from Eq. 1 are coupled with data of RER as a function of VO_{2max} (Fig. 1), it allows the balance of CHO and fat required for exercise to be calculated for the general population, as well as athletes. Adding an analysis of protein and micronutrient requirements to the total, fat and CHO requirements for exercise allows a dietary plan to be formulated for both the general population and athletes that is based on the intensity and duration that a person exercises.

As described above, nutrition for exercise follows the same "U" shaped pattern that is observed for many physiological functions. Specifically, too high energy

intake results in obesity, whereas too low an intake results in exercise intolerance. High CHO and low fat intake, or the reverse, high fat and low CHO, result in exercise intolerance and fatigue during submaximal sustained exercise. Thus, the recommendation for nutrition should be at the nadir of the "U" for optimal results. Total energy intake can be estimated sufficiently from standard data and Eq. 1. The balance of CHO and fat can be estimated from Fig. 1, and a diet prescribed. For example, for very high intensity exercise (VO_{2max} or above), CHO (55%) intake is essential; however, minimal levels of fat (30%) and protein (15%) are required. In this case, it may be helpful to ingest bicarbonate to assist with acid–base balance. For lower exercise intensities (60–80% VO_{2max}), where fat oxidation spares glycogen, the percentage of fat and CHO can be adjusted (40 and 45%, respectively), with protein constant (15%). At still lower percentages of VO_{2max}, the ratio may be shifted more toward fat, levels (20 and blood borne substrates become more important. For very high intensity resistance exercise, higher protein, with CHO at 55% and fat at 25%, plus creatine supplementation are important.

In addition to the energy provided for exercise from a proper nutritional plan, one has to be mindful of the health consequences that can occur from inappropriate nutritional intake. It is clear that very high levels of fat and low CHO, or very low levels of fat and high CHO, have negative effects on immune function and can lead to cardiovascular risk factors. In addition, there are essential fatty acids needed for cellular and other functions, thus requiring a minimal level of fat and micronutrient intake. Individual food choices may influence the capacity of energy delivery during exercise, and may influence cardiovascular risk factors such as blood lipoproteins and immune factors.

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