Dietary Nitrates in Sports Nutrition

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Higher intake of nitrates from the diet can increase the bioavailability of nitric oxide (NO) via the nitrate–nitrite–NO pathway. Increased production of NO (e.g., in mitochondria, blood vessel cells, muscle cells) may improve physical performance. Nevertheless, the increased availability of NO via daily diet or supplementation does not always lead to improved performance in some individuals. Research observations suggest there might be fibre-type specific effects of dietary nitrates (DN) intake. It seems that ergogenicity is somehow related to the fibre-type ratio in muscles, augmenting the exercise economy and performance more likely via type II muscle fibres than type I. Therefore, more consistent and positive improvements in physical performance are usually observed in less-trained athletes (VO_{2max} <65 mL/min/kg) or untrained. Statistically non-significant effects on performance are less likely observed in well-trained and elite endurance-trained athletes (VO_{2max} >65 mL/min/kg). It is also essential to follow the correct supplementation plan (acute/chronic use) to enhance exercise economy or performance, whereas the chronic use of DN brings more consistent results. Nevertheless, DN offer easily available, safe and efficient ergogenic aid for some athletes who seek to improve their performance.

Keywords: ergogenic aid ; nitrate-nitrite-nitric oxide pathway ; nitric oxide ; physical performance

1. Introduction

Nitric oxide plays a crucial role in signalling and physiological regulatory functions of the human body which are crucial to exercise economy and performance (i.e., vasodilatation, mitochondrial respiration, glucose and calcium (Ca^{2+}) homeostasis, skeletal muscle contractility and fatigue development). Because the NO molecule is highly unstable, there is a constant need for its regeneration [1][2][3][4].

Interestingly the substrates for NO syntheses (L-arginine and nitrates) come from our daily diet and therefore can be manipulated [5]. Increased availability of NO via diet has been linked to ergogenic effects [6][7]. However, not all athletes can benefit from these nutritional strategies [8][9]. It seems the effectiveness of NO inducing foods and supplements is limited by an athlete's training status (aerobic fitness) and muscle fibre type ratio [10].

2. NO Metabolism and Physiological Importance

Nitric oxide synthesis in the human body is carried out via two pathways: the NO synthase (NOS)-dependent pathway and nitrate–nitrite–NO (NO_3^-–NO_2^-–NO) pathway [11]. Formation of NO via the NOS-dependent pathway is carried out through the utilisation of L-arginine and oxygen (O_2). Thus, this reaction relies on the delivery of O_2 [12]. Insufficient O_2 delivery during high-intensity exercise may cause this pathway to become dysfunctional [13]. Therefore, the O_2-independent pathway can substitute NO production [14] via the reduction of NO_3^- from the diet (e.g., green leafy vegetables, beetroot, radish) [15]. Nitrates are reduced by oral anaerobic bacteria to NO_2^- [16]. Subsequently, part of the total NO_2^- is reduced to NO in the acidic environment of the stomach where it protects the organism from some pathogens [17]. The rest of the NO_2^- is then transported via the upper gastrointestinal tract into the blood reaching its plasma peak level 2–3 h postprandially [18]. The reduction of NO_2^- to NO substitutes the O_2-dependent NOS pathway in various tissues under hypoxic or acidic conditions [19]. These are conditions that typically occur in working muscles during vigorous exercise [20].

Increased NO bioavailability via food or supplements may increase exercise performance, as it is the critical factor in three physiological mechanisms related to physical exercise. Firstly, NO increases skeletal muscle O_2 delivery via vasodilatation [21]. Secondly, it reduces the O_2 cost of mitochondrial ATP resynthesis via an increased number of ATP molecules (P) formed per O_2 molecules consumed (O) in the electron transport chain (P/O ratio) [22], possibly via the interaction with five-coordinated cytochrome C oxidase [23][24]. Lastly, it improves the efficiency of muscle contractility via reduced creatine phosphate (PCr) cost of force production [25] and changes in Ca^{2+} metabolism within the muscle cells [26]. Enhancing these physiological mechanisms leads to more efficient energy metabolism, lower O_2 demands of the working muscles and higher muscle contractility [26] and therefore an increase in exercise economy and performance [1][22][25].

3. The Role of Dietary Nitrates in Exercise Physiology

Dietary nitrates have become a trendy topic in sports nutrition as even minor enhancement of human physiology may positively affect high-intensity exercise and, therefore, competition results. For example, there may be a decrease in the time of time-trial physical activities [27]. It is also important to note that these changes seem to be highly related to the dosage and supplementation protocol where exercise economy can be improved after a single dose of DN [28][29][30][31][32][33], but exercise performance is more likely augmented after chronic use of DN [34][35][36]. Interestingly, it seems ergogenicity is somehow related to the fibre-type ratio in muscles, augmenting the exercise economy and performance more likely via type II muscle fibres (MFs) than type I [37].

During high-intensity exercise, oxidative phosphorylation is diminished as the O_2 supply is inadequate, and reliance on the anaerobic metabolic pathway of ATP regeneration is favoured [38]. Long-duration high-intensity exercise and intermittent high-intensity exercise lead to exercise-induced hypoxemia causing the muscles to become hypoxic and acidic [39]. This impairment in homeostasis may also disrupt the functioning of the NOS-dependent pathway, and continuation of exercise is highly dependent on the activity of type II MFs [40][41]. Therefore, the reliance on the substitutional NO_3^-–NO_2^-–NO pathway independent of O_2 supply is increased [40][42]. Moreover, type II MFs have a lower blood supply which affects the partial pressure of O_2 within the microvasculature ($P_{mv}O_2$) causing a lower O_2 supply compared to type I MFs [43][44][45]. This phenomenon underlines the reliance on the NO_3^-–NO_2^-–NO pathway in type II MFs and even more under hypoxia [24][46]. Lastly, most recent studies suggest improvement in muscle force production and mitochondrial oxidative phosphorylation are more likely observed in type II MFs than in type I MFs after DN supplementation [47][48].

Furthermore, it has already been suggested in earlier studies that neither acute nor chronic DN supplementation can improve performance in highly trained cyclists [49][50][51], runners [52][53] or cross-country skiers [54]. These groups of endurance-trained athletes tend to have a higher type I MF ratio [55][56][57]. In contrast, high doses of DN (8.4–9.6 mmol) improved performance in highly trained kayakers and rowers [29][58] but not low doses (~4–5 mmol) [29][59]. In this context, the upper body muscles (e.g., biceps brachii, triceps brachii, deltoid, trapezius or latissimus dorsi) are well described as muscles with a higher type II MF ratio [60][61][62]. Moreover, as highly trained athletes develop specific adaptation to rowing [63] and kayaking [64], increased type II MF ratio or MF hypertrophy is more likely [65]. This exercise modality, which mostly involves muscle groups with a higher type II MF ratio [65], can be another example of the fibre-type specific effects of DN [37][66].

4. Training Status as a Limiting Factor

Nutritional strategies to increase the bioavailability of NO and possibly physical performance have been under the scope of research for many years, and there are some crucial variables (e.g., muscle fibre-type ratio, physiological limitations) which can influence their effectiveness [8][67]. Consumption of DN in the form of either nitrate-rich foods or supplements generally increases plasma levels of NO_2^- [28] which interestingly does not always lead to improvement in exercise performance especially in well-trained endurance and elite endurance athletes [68].

Fibre-type specific effects of DN supplementation have been demonstrated in animal experiments where muscle force development increased in type II (fast-twitch glycolytic fibres type IIx) but not in type I MFs (slow-twitch oxidative fibres) [34]. Reliance on the NO_3^-–NO_2^-–NO pathway is higher in type II MFs due to the lower O_2 tension (pO_2) than in type I [43]. Therefore, an increase in the bioavailability of NO mainly affects type II MFs [37]. Endurance-trained athletes are likely to have a higher ratio of slow oxidative type I MFs compared to non-trained and recreationally active athletes [69] or the inactive population and the elderly [70]. This phenomenon also relates to higher aerobic fitness in highly and elite trained athletes which cannot be augmented any further [68]. These seem to be possible explanations for the lower ergogenicity of DN supplementation in highly trained or elite athletes, as some studies failed to enhance the performance of the participants [52][71]. It has been suggested that the efficiency of DN supplementation is related to an athlete's training status, especially in high-intensity endurance exercises, e.g., time-trial performance, where O_2 delivery is impaired and reliance on type II MFs is higher [68].

Significant beneficial effects of DN intake in elite endurance athletes are less likely to be observed. This athletic group demonstrated a high proportion of type I MFs, elite exercise performance close to the athlete's physiological limits or NO_3^--mediated vasodilation in non-prioritized muscles which may lead to reduced O_2 delivery to the essential muscles working very close to their maximal cardiac output [69]. All these factors are now suggested as potential causes of the lower ergogenicity in highly trained athletes.

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