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Micronutrients and athletic performance: A review

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ABSTRACT

Optimising nutrition intake is a key component for supporting athletic performance and supporting adaption to training. Athletes often use micronutrient supplements in order to correct vitamin and mineral deficiencies, improve immune function, enhance recovery and or to optimise their performance. The aim of this review was to investigate the recent literature regarding micronutrients (specifically iron, vitamin C, vitamin E, vitamin D, calcium) and their effects on physical performance. Over the past ten years, several studies have investigated the impacts of these micronutrients on aspects of athletic performance, and several reviews have aimed to provide an overview of current use and effectiveness. Currently the balance of the literature suggests that micronutrient supplementation in well-nourished athletes does not enhance physical performance. Excessive intake of dietary supplements may impair the body's physiological responses to exercise that supports adaptation to training stress. In some cases, micronutrient supplementation is warranted, for example, with a diagnosed deficiency, when energy intake is compromised, or when training and competing at altitude, however these micronutrients should be prescribed by a medical professional. Athletes are encouraged to obtain adequate micronutrients from a wellbalanced and varied dietary intake.

1. Introduction

The foundations of sport performance are training and nutrition/diet of the athlete, with nutritional strategies providing a supportive role in enhancing training adaption (Stellingwerff et al., 2019). In line with the evolution of methods to optimise training through various training prescriptions, there has been a concurrent evolution in nutrition strategies for athletes. Current nutritional consensus statements promote the periodisation of nutritional intake to optimise the adaptation from the training programme prescribed to the athlete (Stellingwerff et al., 2019). The theory of nutritional periodisation is planned energetic and macronutrient strategies to target the individual exercise sessions and overall training programme to aid in obtaining long term performance gains in athletes (Jeukendrup, 2017). Strategies of nutritional periodisation include manipulating carbohydrate and fat intake to upregulate key signalling pathways in the skeletal muscle and promote mitochondrial biogenesis, angiogenesis and increased lipid oxidation (Hansen et al., 2005; Hulston et al., 2010; Morton et al., 2009) or optimising protein intake to support hypertrophic responses in skeletal muscle (Stellingwerff et al., 2019). However, numerous metabolic processes and reactions involved in energy extraction from macronutrients, oxygen delivery and transfer, tissue repair, and growth and development are dependent on essential vitamins and minerals (Volpe, 2007).

Micronutrients are essential for life and include; vitamins which are organic compounds that support health, growth and reproduction and are needed in small amounts to prevent clinical deficiencies and declines in health (Fogelholm, 2015). A key feature of most vitamins is that the human body is unable to synthesise them (Fogelholm, 2015), therefore they must be obtained from dietary intake. Vitamins are classified based on their *in vivo* solubility, with A, D, E and K classified as fat-soluble and vitamins B and C classified as water soluble (Fogelholm, 2015). Minerals are inorganic substances that support physiological functioning (Fogelholm, 2015). The daily physiological requirements determine the mineral classifications, hence ~100 mg·day^{-1} of macrominerals (sodium, potassium, calcium, phosphorus and magnesium) and ~20 mg·day^{-1} of trace elements (iron, zinc, copper, chromium and selenium) are required by healthy individuals (Fogelholm, 2015).

Typically, a well-balanced diet in a healthy individual is adequate to obtain all of the micronutrients in recommended doses for normal bodily functions (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006), but the adequacy of these recommendations for athletes is a topic of discussion amongst researchers and clinical practitioners.

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Abbreviations

aOR	adjusted Odds Ratio
AST	aspartate transaminase
BF%	body fat percentage
BMD	bone mineral density
CI	confidence interval
CK	creatine kinase
C-RP	C-Reactive protein
DBPRCT	double-blind placebo randomised controlled trial
DOMS	delayed onset muscle soreness
EAR	estimated average requirement
F	female
FR	free radical
Hb	haemoglobin
HIIT	high-intensity interval training
IDA	iron deficiency anaemia
M	male
MAPK	mitogen-activated protein kinase
Mb	myoglobin
MFP	meat fish poultry
MVC	maximum voluntary contraction
ns	non-significant
PBM	peak bone mass
RDI	recommended dietary intake
ROS	reactive oxygen species
SDT	suggested dietary target
TT	time trial
TTE	time to exhaustion
UL	upper limit
URTI	upper respiratory tract infection
UVβ	ultraviolet-β
VO_2max	maximum volume oxygen consumed

Micronutrient supplementation is common among athletes, with a meta-analysis finding approximately 50% of athletes were using vitamin or mineral supplements (Knapik et al., 2016). Inference from research suggests that while intensity, duration and mode of exercise may dictate the macronutrient requirements of the athlete's diet, so will it determine the micronutrient needs of the athlete. Athletes competing in sports with higher energy requirements are likely to have increased micronutrient requirements, however there is insufficient data to quantify the levels of micronutrients required (Fogelholm, 2015). If the overall energy requirement of the athlete is high due to their training load, then this should be reflected in both the macro- and micro-nutrient intake of that individual. Most athletes will have high energy intakes to support their training, and if obtaining their increased energy intake from a well-balanced diet, then the dietary reference intakes for vitamins and minerals may be suitable (Volpe, 2007). However, regularly occurring circumstances exist where requirements for vitamins and minerals are increased. These include, but are not limited to, athletes with high sweat and urine losses (e.g. electrolytes, zinc) (Fogelholm, 2015), and athletes with low energy intakes (e.g. iron) or specific dietary preferences (e.g. vegetarian or vegan dietary pattern and haem iron) (Deakin and Peeling, 2015). Such circumstances may require exogenous supplementation with vitamins and minerals to support health and performance. Alternatively, supplementation may be considered by athletes in order to support adaptation to training, for example, iron supplementation may be required to support adaptation to altitude training (Garvican-Lewis et al., 2016). Some micronutrients are more likely to be of concern in particular sports. For example, vitamin D concentrations may be low in athletes training and competing in Winter sports or predominantly

Table 1
Roles, food sources and dietary recommendations for micronutrients in the general population.[a]

Nutrient	Roles in body and performance	Food sources	Sex	EAR	RDI	UL	SDT[b]
Iron (mg·day^{-1})[c]	Part of haemoglobin (carries oxygen) and myoglobin (in muscles), energy metabolism	Haem iron – meat, fish, chicken, seafood	F	8	18	45	–
		Non-haem iron – meat, fish, chicken, seafood, legumes, eggs, vegetables	M	6	8	45	–
Vitamin C (ascorbic acid) (mg·day^{-1})	Antioxidant, collagen synthesis, amino acid metabolism, immune function, thyroxine synthesis, enhances iron absorption	Citrus fruit, kiwifruit, strawberries, papayas, mangoes, tomatoes, broccoli, cauliflower, brussels sprouts, lettuce, capsicum, potatoes	F	30	45	NA	220[d]
			M	30	45	NA	190[d]
Vitamin E (alpha-tocopherol) (μg·day^{-1})	Antioxidant	Polyunsaturated plant oils (margarine, salad dressings, mayonnaise), nuts, seeds, leafy green vegetables, wheat germ, wholegrains, liver, egg yolks, fatty meats	F	–	7	300	15[e]
			M	–	10	300	19[e]
Vitamin D (μg·day^{-1})[f]	Bone mineralisation, immune function	Fortified milk, margarine and butter, beef, veal, egg yolks, liver, fatty fish (salmon, sardines, herring)		–	5	80	–
Calcium (mg·day^{-1})	Bone mineralisation, role in muscle contraction and relaxation, nerve function, blood clotting, maintaining blood pressure	Dairy products (milk, yoghurt, cheese), calcium fortified milks and juices, canned fish with bones, broccoli, silverbeet, bok choy, kale, legumes, almonds, sesame seeds		840	1000	2500	–

Abbreviations: EAR, estimated average requirement; F, female; M, male; RDI, recommended dietary intake; SDT, suggested dietary target; UL, upper limit.

[a] Nutrient Reference Values for Australia and New Zealand adults (19–30 years) (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006).

[b] Definitions: EAR – daily nutrient level estimated to meet requirements of half the healthy individuals; RDI - average daily dietary intake level sufficient to meet nutrient requirements of nearly all (97–98 per cent) healthy individuals; UL - highest average daily nutrient intake level likely to pose no adverse health effects to almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse effects increases; SDT – daily average intake from food and beverages for certain nutrients that may help in the prevention of chronic disease (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006).

[c] Absorption lower in vegetarian diets, so intakes need to be up to 80% higher.

[d] Based on 90th centile of the current population intake; attained by replacing nutrient poor energy dense foods and drinks with plenty of vegetables, legumes and fruit.

[e] Based on 90th centile of the current population intake; Attained by including some polyunsaturated or monounsaturated fat and replacing nutrient poor energy dense foods and beverages with plenty of vegetables and moderate amounts of lean meats, poultry, fish, reduced-fat dairy foods and wholegrain cereals.

[f] With regular sun exposure there would be no need for dietary vitamin D. For people with little exposure to sunlight, a supplement of 10 μg·day^{-1} would not be excessive.

indoors (von Hurst and Beck, 2014), while iron stores may be more compromised in athletes/or physically active individuals undertaking high intensity and endurance based exercise (Martin et al., 2019). While sufficient intake of vitamins and minerals is considered paramount for individual health, the role of supplementation on exercise performance, either directly or indirectly, requires an updated review.

Therefore, this review aims to investigate the recent literature with regards to the effect of key micronutrients, iron, antioxidants (vitamin C and E), vitamin D and calcium and exercise performance. We focussed our search on literature published in the past 10 years using the Pubmed database. Several review papers were located which were used and citations from relevant papers were searched. Search terms included iron, antioxidant, oxidative stress, vitamin C, vitamin E, vitamin D, calcium, exercise, athlete, sport and performance. Only full manuscripts published in English were included (abstracts, theses and conference proceedings were not included).

Table 1 provides an overview of the micronutrients that will be discussed in the review including their roles in exercise, food sources and recommendations for dietary intake.

2. Iron

Maintaining optimal iron status is considered fundamental for sport performance, athletic training and health. Iron forms the functional component for oxygen delivery (haemoglobin) and storage (myoglobin), and is necessary for numerous biological processes including electron transfer reactions, gene regulation, and cell growth and differentiation (Beard, 2001). The majority of the body's iron is stored in the liver (~60%), with the remaining 40% found in muscle tissue, cells and the reticuloendothelial system (Beard, 2001). Of the stored iron, ~95% is sequestered in the liver bound to the storage protein ferritin, a 24 subunit protein capable of containing ~4500 atoms of iron, and the remaining 5% is bound to hemosiderin (Knovich et al., 2009). The total concentration of iron stored in the body is ~30–50 mg kg^{-1} body mass, however this will vary depending on age, sex and the specific tissue being examined (Beard, 2001).

In biological systems, iron is found in ferrous (Fe^{2+}) and ferric (Fe^{3+}) states. In extracellular fluids, Fe^{2+} is readily oxidised by oxygen to Fe^{3+} and will form ferric hydroxide ($Fe(OH)_3$) (Oliveira et al., 2014). The solubility of iron in the presence of oxygen in extracellular fluid is limited by the concentration of $Fe(OH)_3$ and the pH of extracellular fluid. The human body has no inherent mechanism of generating its own iron supply (Beard and Han, 2009), hence the iron status of an individual is largely dependent on dietary iron intake and recycling of iron within the body. The low solubility of iron at physiological pH levels (7.35–7.45) precludes urinary excretion as a major mechanism for eliminating iron in order to maintain iron status (Oliveira et al., 2014). Therefore, the primary mechanism for maintaining iron homeostasis is a tightly regulated feedback system coordinated by hepcidin, a 25 amino-acid peptide hormone encoded by the *HAMP* gene. Hepcidin regulates iron absorption so that it approximates to the daily iron loss (Nemeth and Ganz, 2009). Transcription of the *HAMP* gene is primarily influenced by bone morphogeneic proteins pathway that acts to directly regulate iron metabolism, with a separate pathway regulated by inflammation (Hare, 2017). Hepcidin exerts its effects on ferroportin, the primary iron export channel expressed on the basolateral surface of enterocytes, hepatocytes and macrophages, regulating the release of ferric iron into systemic circulation (Nemeth et al., 2004). Ferric iron released into circulation is bound to the vital protein transferrin, which is able to maintain iron in a stable form while allowing it to be available for biological processes (Gkouvatsos et al., 2012). The rate and production of transferrin proteins is regulated by the individual's iron status. Therefore, in iron depletion, transferrin production and plasma levels may increase by up to 100%. In iron sufficient individuals, the iron saturation of transferrin is typically in the range of 25–50%, a level that maintains adequate delivery of iron throughout the body, and in athletes may serve as a marker of tissue iron use (Beard and Han, 2009). Previous research has suggested that transferrin saturation of <15% be considered a marker of stage 2 iron deficiency, since this level of iron saturation is considered a physiologically insufficient level to maintain iron delivery to the bone marrow for erythropoiesis (Clenin et al., 2015). The bone marrow demands ~80% of the plasma iron pool daily, equating to ~20–30 mg·day^{-1} required to support effective erythrocyte production. The mean functional life of an erythrocyte is ~120 days, after which they are considered senescent and undergo phagocytosis by splenic macrophages followed by denaturing of haemoglobin to release haem-bound iron from globin chains (Beard and Han, 2009). The unbound haem iron is then released back into systemic circulation. However, the rate of influx from the macrophages is regulated by hepcidin's interaction with ferroportin on the cell surface. Once released back into circulation, iron will either be bound to transferrin proteins or sequestered in ferritin proteins (Fig. 1). Approximately 0.66% of iron is recycled each day in this manner. It should be noted that absorbed iron not bound to these transferrin transport proteins is capable of catalysing reactions resulting in the production of free radicals (FRs) which are detrimental to optimal functioning of the biological system (Oliveira et al., 2014). Recently, this process has been hypothesised as a likely mechanism of overtraining syndrome in skeletal muscle of athletes (Cheng et al., 2020).

Physiological processes of recycling iron within the body are highly efficient, such that only 1.0–2.0 mg of iron is lost daily (~1 mg·day^{-1} in males and up to 2 mg·day^{-1} in females) (Clenin et al., 2015); this amount is then required to be absorbed from dietary intake (Carpenter and Mahoney, 1992). Based on the total obligatory iron losses that occur daily, and daily iron bioavailability and absorption rates of ~10%, the World Health Organisation and other national organisations have calculated daily iron requirements for various populations, sexes and age groups (Monsen et al., 1978; Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006) (Table 1). The bioavailability of iron from ingested food is used to determine the active amount of iron absorbed from the total nutrient content of the diet. Numerous physiological factors will influence the bioavailability of iron from the diet, with the individual's iron status being the primary influence of iron absorption, and secondary factors such as dietary constitutes and nutrients, exercise, maturity and sex all reported to alter iron bioavailability in order to maintain iron homeostasis (Carpenter and Mahoney, 1992) (see Table 2).

Within an exercise setting, obligatory iron losses are exaggerated, while regulation of iron absorption and recycling appears to be influenced by alterations in hepcidin. Following an acute bout of exercise, an inflammatory-derived increase in hepcidin may occur approximately 3 h post-exercise and may be sustained for up to 6 h post-exercise. Exercise associated factors known to influence this hormones activity are training/exercise frequency, duration, intensity, modality, and nutrition practices prior to exercise (Sim et al., 2013; Badenhorst et al., 2015; Peeling et al., 2009a, 2014). Without a subsequent exercise session, hepcidin will likely return to baseline levels by 12–24 h post-exercise (Peeling et al., 2009b). Regular exposure to periods of altered iron homeostasis post-exercise have been shown to reduce iron stores by 25–40% over a 6-week training period (McKay et al., 2019). This situation would support the higher prevalence rates of iron deficiency in male (3–11%) and female (20–40%) athletes (Fallon, 2004; Malczewska-Lenczowska et al., 2009; Parks et al., 2017) when compared to the general population. A recent study of female military personnel found that iron stores (serum ferritin) decreased from 57 to 38 μg L^{-1} over 16 weeks of basic combat training (Martin et al., 2019). As such, the recommended daily intake (RDI; average daily dietary intake level sufficient to meet the nutrient requirements of 97–98 per cent the population (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006)) of iron for athletes may be higher when compared to age and

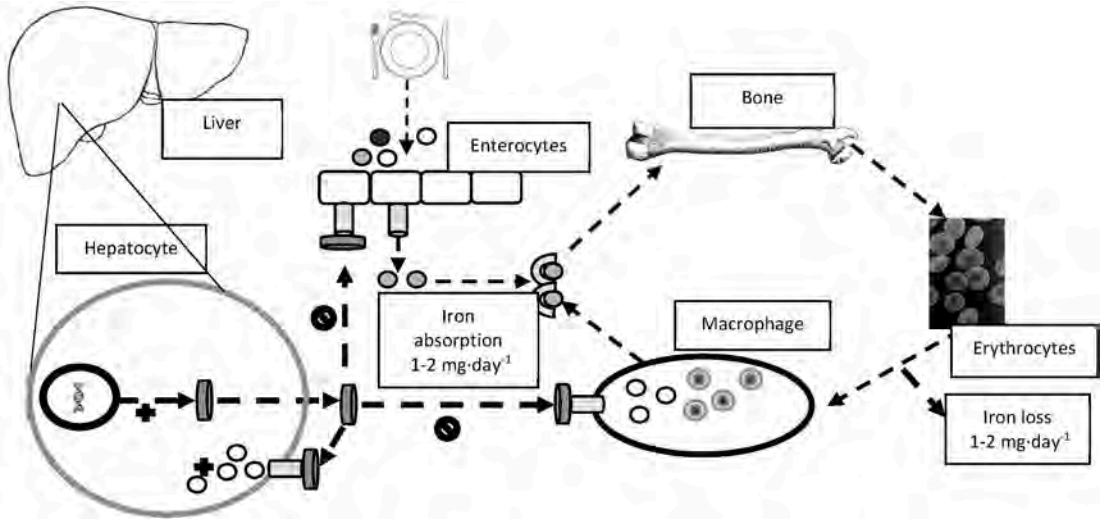

Fig. 1. Diagrammatic representation of iron intake and recycling within the body and the role of hepcidin in iron homeostasis.

sex-matched populations, however this is still an area of investigation in athletes. Additional considerations for altered iron homeostasis and risk of iron deficiency is low energy intake associated with Relative Energy Deficiency in Sport (RED-S). RED-S is 'a syndrome referring to impaired physiological function including, but not limited to, metabolic rate, menstrual function, bone health, immunity, protein synthesis, cardiovascular health caused by relative energy deficiency. The cause of RED-S is energy deficiency relative to the balance between dietary energy intake and energy expenditure required for health and activities of daily living, growth and sporting activities' (Mountjoy et al., 2014). Research has demonstrated that both overall lower iron intake caused by low food intake, and exacerbated hepcidin levels and consequent disruption to effective iron homeostasis caused by the physiological stress of low energy availability, are possible contributors to iron deficiency in athletes with RED-S (Badenhorst et al., 2019). For an update on iron consideration for athletes, the authors refer the reader to the review by Sim et al. (2019). This narrative review provides a comprehensive overview of iron deficiency in athletes over the last decade and highlights regular screening for athletes (quarterly or annually depending on gender and previous history of iron deficiency), the progression of iron depletion to iron deficiency anaemia in athletes and serum markers for detection, the impact of iron depletion and anaemia on athletic performance, the effectiveness of supplementation for athletic performance, and suggests a specific focus on iron in athletes who have chronic high training loads and who are training or competing at altitude.

With iron being a mineral that, in optimal physiological levels, will support athletic performance and adaptation, early detection of iron depletion is important to prevent the declines in aerobic performance that are observed in athletes with iron deficiency anaemia (Rubeor et al., 2018). Inference from previous research has suggested that for every 1 g L^{-1} decrease in Hb, there is a 1.04 kcal increase in energy expenditure (Hinton et al., 2000) decreasing the aerobic exercise efficiency and increasing the work capacity in iron deficiency anaemic athletes (Hinton et al., 2000; Garvican et al., 2011). The severity of iron deficiency, individual responses to treatment, time course of treatment and the legality of anti-doping agencies, should all be considered when prescribing appropriate supplementation for iron deficiency correction (Clenin et al., 2015). Due to the exacerbated rates of iron loss with regular training, all athletes, regardless of iron status, should consider increasing dietary iron intake by reviewing meal composition and bioavailability of iron in their diet. Exogenous sources of iron are primarily provided as oral supplements in the ferrous form to reduce gastrointestinal distress (Santiago, 2012) and have been shown to increase serum ferritin concentrations by 40–80% over a 12-week supplementation period (Garvican et al., 2014). Inference from research suggests that to maximise iron absorption from oral iron supplements, especially when ferritin levels exceed 30 $\mu g \cdot L^{-1}$, alternate morning and daytime supplementation should be considered to improve fractional iron absorption in the presence of an activated hepcidin response as iron stores are improved (McCormick et al., 2019; Stoffel et al., 2017). This treatment may be recommended for athletes who are iron deficient but not anaemic, since meta-analyses on this supplementation protocol in this cohort has shown to improve iron stores and reduce athletes' rating of fatigue during training and competition (Burden et al., 2015; Houston et al., 2018). The final treatment option is parenteral iron (injection or infusion). This treatment has been shown to increase iron stores by 200–400% in 1–42 days, with limited gastrointestinal effects. This option may be recommended for severe iron deficiency anaemia when aerobic performance and health of the athlete is compromised (Woods et al., 2014; Garvican et al., 2014). However, as noted previously, elite athletes will require support from sport physicians and medical clearance from anti-doping agencies prior to treatment.

3. Antioxidants

Exercise is associated with the production of reactive oxygen species (ROS) including free radicals (FRs). A FR is an unstable molecule with one or more unpaired electrons in its outer shell (Clarkson and Thompson, 2000). The collective name of ROS refers to oxygen centred radicals, but also non-radicals which are reactive derivatives of oxygen such as hydrogen peroxide (Powers and Jackson, 2008). Incomplete reduction of oxygen during oxidative metabolism within the electron transport system within mitochondria, specifically the inhibition of ubiquinone cytochrome *c* reductase (complex III) by antimycin has been shown to increase ROS (Raha et al., 2000; Bailey et al., 1999). It is noted that exercise may generate ROS and FRs through alternative pathways, such as through ischaemic perfusion (Finaud et al., 2006), lactic acid production, increases in adrenaline and catecholamine production, and through the inflammatory response to muscle damage (Clarkson and Thompson, 2000).

Exercise is proposed to create an optimal exercise redox balance that stimulates skeletal muscle adaptation (Cheng et al., 2020). With optimal rest and recovery, structural and metabolic adaptations will occur within the skeletal muscle that will support exercise performance (Cheng et al., 2020). Extreme exercise whether that be prolonged in duration and intensity can increase oxidative stress, creating an imbalance between the production of ROS and the body's ability to counteract ROS through an adequate antioxidant defense system (Pingitore et al.,

Table 2
Factors affecting the absorption of haem and non-haem iron.

Iron type	Sources	Absorption at apical surface of small intestine	Absorption efficiency	Dietary factors affecting iron absorption
Haem	Animal sources bound in porphyrin ring present in Hb and Mb. Released in lumen and stomach by proteolytic enzymes. Does not require binding proteins.	Specific haem transporters on apical surface of enterocytes, haem carrier protein 1. Haem oxygenase 1 releases ferrous iron in cytoplasm of enterocyte iron pool.	Efficient absorption ~40%, ~10% of all dietary iron is haem iron	Inhibitors: • Calcium
Non-haem	All other forms of iron, and typically bound to other food components in ferric form. Reduced to ferrous form by brush border enzymes (duodenal cytochrome *b*) or dietary reducing agents (ascorbic acid).	Soluble ferrous form is transported across the apical surface of gastrointestinal tract by divalent metal transporter 1 channels.	Absorption depends on levels of inhibitors and enhancers ~2–20%	Enhancers: • Ascorbic acid • MFP factor • Organic acids Inhibitors: • Phytates • Polyphenols • Calcium

Abbreviations: Hb, haemoglobin; Mb, myoglobin; MFP, meat fish poultry.

2015; Cheng et al., 2020). This same stimulus however, also ensures the upregulation in the endogenous antioxidant defense system (Pingitore et al., 2015; Criswell et al., 1993). It should be noted that the relationship between exercise and oxidative stress is complex, and depends on the type, intensity and duration of exercise, as well as the genetics, sex, age, fitness and nutritional status of the individual athlete (Margonis et al., 2007; Braakhuis and Hopkins, 2015; Pingitore et al., 2015). Biomarkers of oxidative stress typically include the increases in formation of free radicals, antioxidant enzymatic activity, oxidative damage to cell components such as lipids, protein and DNA that contribute to immune dysfunction, muscle damage and fatigue (Finaud et al., 2006; Powers and Jackson, 2008). However, direct measurement of oxidative stress, inclusive of FR production and ROS cell component damage is difficult due to the short half-life of reactive molecules and the relatively rapid influence of diet and cell metabolism on cell antioxidant states (Powers and Jackson, 2008). An absence of effects may be due to a lack of sensitivity of measures.

Antioxidants may aid in counteracting FR damage by pairing with and stabilising the unpaired electrons of FRs, lessening their damaging effects. Antioxidants can be produced endogenously, and include glutathione, uric acid, lipoic acid, bilirubin, and coenzyme Q10; enzymatic antioxidants include superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase. Alternatively, antioxidants can be obtained through dietary intake or supplements. Dietary antioxidants include ascorbic acid (vitamin C), tocopherol (vitamin E), carotenoids (β-carotene) and polyphenolic extracts (Peternelj and Coombes, 2011). The roles, food sources and recommended intakes for Vitamin C and E are shown in Table 1.

Many athletes consume antioxidant supplements in the hope of preventing or reducing oxidative stress and improving their performance. An extensive 2011 review of over 150 mainly small and low quality studies found antioxidant supplementation reduced exercise induced oxidative stress (Peternelj and Coombes, 2011). Most studies however, also found no beneficial effects on exercise-induced muscle damage and performance, and several studies found negative effects of antioxidant supplements on health and performance (Peternelj and Coombes, 2011). There is concern that high dose antioxidant supplementation might attenuate the beneficial effects of exercise, and interfere with critical ROS-mediated physiological processes (Peternelj and Coombes, 2011).

Since 2011, further studies (Askari et al., 2012; Braakhuis et al., 2014; de Oliveira et al., 2019; Paulsen et al., 2014a, 2014b) have been undertaken and several reviews (Braakhuis, 2012; Braakhuis and Hopkins, 2015; Clifford et al., 2019; Mason et al., 2020; Morrison et al., 2015; Pingitore et al., 2015; Righi et al., 2020; Stepanyan et al., 2014) have systematically investigated the effects of vitamin C, vitamin E and other antioxidants on oxidative stress and performance in athletes. Studies are difficult to compare due to variations in study design and participants (recreational to elite), different training and exercise regimes, and variations in the combinations and doses of antioxidants provided (Peternelj and Coombes, 2011), as well as variation in the duration of interventions and outcome measures. For the remainder of this section, we have largely focused on research undertaken since 2011 investigating the effect of vitamin C and E on performance outcomes.

3.1. Vitamin C

A review by Mason et al. found both acute (Yimcharoen et al., 2019; Thompson et al., 2001a) and chronic supplementation (Thompson et al., 2001b, 2003; Nieman et al., 2002) with vitamin C decreased (Thompson et al., 2001b; Yimcharoen et al., 2019) or had no effect (Thompson et al., 2001a, 2003; Nieman et al., 2002) on markers of oxidative stress associated with exercise (Mason et al., 2020). For example, Yimcharoen et al. (2019) found plasma malondialdehyde was significantly decreased post exercise when 1000 mg ascorbic acid was given prior to 30 min of moderate intensity cycling compared with a placebo. Braakhuis (2012)

in a review found that vitamin C in doses of 200–1000 mg·day^{-1} reduced oxidative stress in exercising participants; while higher doses (>1000 mg·day^{-1}) reduced training-induced adaptations. A recent meta-analysis of randomised controlled trials found vitamin C attenuated oxidative stress (measured by lipid peroxidation) and inflammatory response (interleukin-6) to a single bout of exercise, but had no effect on levels of creatine kinase, C-Reactive protein, cortisol, or muscle soreness or strength (Righi et al., 2020). Overall, there appears to be no positive effect of vitamin C supplementation on aerobic performance in athletes (Mason et al., 2020). Braakhuis (2012) found that vitamin C in doses of 1000 mg or more per day significantly impaired sport performance in 4 out of 12 studies, possibly by reducing mitochondrial biogenesis, while 4 studies found non-significant impairments in performance, however these findings were not significant. Where performance improved (by a small amount in four studies), three studies provided vitamin C acutely or for one week (Braakhuis, 2012). Two recent studies have found vitamin C to have no beneficial effects on performance (Roberts et al., 2011; Askari et al., 2012) (Table 3). A study in trained female runners found 1000 mg·day^{-1} vitamin C for three weeks likely decreased running training speed and increased markers of oxidative stress (Braakhuis et al., 2014). Findings are mixed regarding the beneficial effects of vitamin C on post exercise muscle recovery (Mason et al., 2020). Braakhuis (2012) concluded that doses of vitamin C (up to 250 mg·day^{-1} (which can be obtained from 5 plus servings of fruit and vegetables)) may be adequate to reduce oxidative stress without impairing training adaptations. This is similar to the suggested dietary target for vitamin C of 220 mg·day^{-1} for the general population (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006). Intakes of >200 mg·day^{-1} for one to two weeks may be advantageous in times of increased exercise stress (Braakhuis, 2012).

Table 3
Interventions investigating the effect of Vitamin C supplements on performance in athletic populations.

Author (year), country	Population group	Study design	Supplement	Results
Askari et al. (2012), Iran	60 male student athletes, 21 y	DBPRCT for 8 weeks	Intervention – Vitamin C 200 mg·day^{-1}; Control – placebo	No change between or within groups for TTE or BF%; no changes in AST or CK between or within groups
Roberts et al. (2011), United Kingdom	15 recreationally active males, 23 y	DBPRCT for 4 weeks	HIIT training plus: Intervention – Vitamin C 1000 mg·day^{-1}; Control – placebo (electrolyte sports drink)	No differences between groups in any performance measures (VO_2max, running economy, 10-km TT, distance run)
Braakhuis et al. (2014), New Zealand	23 trained female runners, 31 y	DBPRCT for 3 × 3.3 weeks	HIIT endurance training plus: Intervention – Vitamin C 1000 mg·day^{-1}; Control – placebo	Vitamin C likely slower intervals (1.3% all runners; 2.1% faster runners); unclear 5-km TT and Incremental$_{peak}$; Vitamin C increased oxidative stress (catalase 23%, protein carbonyls 27%, superoxide dismutase 8.3%)

Abbreviations: AST, aspartate transaminase; BF%, body fat percentage; CK, creatine kinase; DBPRCT, double-blind placebo randomised controlled trial; HIIT, high-intensity interval training; TT, time trial; TTE, time to exhaustion; VO_2max, maximum volume oxygen consumed.

3.2. Vitamin E

Vitamin E supplementation has been shown in several studies to reduce oxidative stress markers (Mason et al., 2020; Meydani et al., 1993; Rokitzki et al., 1994). However, one study found increased plasma lipid hydroperoxides post-exercise in triathletes (McAnulty et al., 2005), and a meta-analysis found vitamin E did not result in significant protection against exercise-induced lipid peroxidation or muscle damage (Stepanyan et al., 2014). A review paper suggested that acute intake of vitamin E around competition time may be beneficial (Braakhuis and Hopkins, 2015), however the majority of studies found chronic vitamin E supplementation did not improve performance and, in some cases, inhibited performance (Braakhuis and Hopkins, 2015). Simon-Schnass and Pabst (1988) found vitamin E supplementation over four weeks maintained aerobic working capacity at high altitude (>5000 m), with the supplemented group showing no increase in pentane (a marker of lipid peroxidation) compared with the control group. Altitude increases oxidative stress, and vitamin E may offset impairment by maintaining red blood cell structure. One study has found a beneficial effect of vitamin E on post-exercise muscle recovery (Rokitzki et al., 1994), while others have found no impact (Mason et al., 2020; Beaton et al., 2002). Overall, there appears to be no clear scientific rationale for recommending vitamin E to athletes.

3.3. Vitamin C and vitamin E (combined)

Earlier studies combining 500 mg vitamin C and 400 IU vitamin E per day have not demonstrated negative effects on skeletal muscle adaptive responses to endurance training (Mason et al., 2020; Yfanti et al., 2010). However other more recent studies suggest 1000 mg·day^{-1} of vitamin C with vitamin E may impair protein signalling or physiological adaptations following resistance and endurance exercise (Morrison et al., 2015; Paulsen et al., 2014a, 2014b; Mason et al., 2020). Recent studies in athletic populations (Table 4) have shown no differences in performance outcomes with vitamin C and E supplementation (de Oliveira et al., 2019; Paulsen et al., 2014a). In one study, greater increases in strength were observed in the placebo group compared with the group supplemented with vitamin C and E (Paulsen et al., 2014b). Increased markers of oxidative stress were observed in a recent study where male football players received 500 mg·day^{-1} vitamin C and 400 IU·day^{-1} vitamin E for 15 days (de Oliveira et al., 2019). In a recent systematic review and meta-analysis of randomised controlled trials where vitamin C, E or both were consumed alongside a supervised exercise training programme of at least four weeks duration, it was found that supplementation did not attenuate aerobic exercise-induced performance as measured by maximum aerobic capacity or endurance performance (Clifford et al., 2019). With resistance training, there were no effects on lean mass or muscle strength. It was concluded that vitamin C and/or E supplementation did not blunt exercise induced adaptations or physiological function, however, this did not transfer to benefits in adaptations or performance. It should also be noted that the 18 studies included in the review had relatively small sample sizes and none of the studies were conducted in elite athletes (Clifford et al., 2019). Few studies have investigated the effects of combined vitamin C and vitamin E supplementation on muscle recovery post-exercise, and the results have been mixed (Mason et al., 2020). Overall, there is a lack of evidence supporting combined supplementation of vitamins C and E on performance outcomes.

Few studies have focused on the effects of whole foods and overall dietary patterns on oxidative stress in athletes. Dietary patterns which emphasise plant-based foods e.g. vegetarian (Satija and Hu, 2018) and Mediterranean (Visioli and Galli, 2001) dietary patterns tend to be high in antioxidants. Whole foods contain a range of antioxidants in natural

Table 4
Interventions investigating the effect of Vitamin C and Vitamin E supplements on performance in athletic populations.

Author (year), country	Population group	Study design	Supplement	Results
Paulsen et al. (2014a), Norway	54 recreationally endurance trained adults, 20–45 y	DBPRCT for 11 weeks	Endurance training plus: Intervention - Vitamin C 1000 mg·day^{-1} + vitamin E 235 mg·day^{-1}; Control - placebo	No differences between groups in any performance measures (VO_2max, 20m shuttle run, submaximal running); Vit C & E attenuated increases in mitochondrial biogenesis
de Oliveira et al. (2019), Brazil	21 trained male footballers, 19.9 y	DBPRCT for 15 days	Intervention – Vitamin C 500 mg·day^{-1} + vitamin E 400 IU·day^{-1}; Control – placebo	No differences between groups in any performance measures (power, agility, anaerobic power) or muscle recovery; No differences between groups in CK or DOMS; increased markers of oxidative stress with Vit C & E
Paulsen et al. (2014b), Norway	32 recreationally strength trained adults, 24 y (intervention), 27 y (control)	DBPRCT for 10 weeks	Strength training plus: Intervention - Vitamin C 1000 mg·day^{-1} + vitamin E 235 mg day^{1}; Control - placebo	Greater increases in strength (biceps curl ($p = 0.04$), MVC knee extensors (ns)) with placebo; Vit C & E interfered with cellular signalling (MAPKs and ubiquitination levels) post-exercise

Abbreviations: CK, creatine kinase; DBPRCT, double-blind placebo randomised controlled trial; DOMS, delayed onset muscle soreness; MAPK, mitogen-activated protein kinase; MVC, maximum voluntary contraction; ns, non-significant; VO_2max, maximum volume oxygen consumed.

amounts which may act synergistically for optimal antioxidant effects (Pingitore et al., 2015). A recent randomised controlled trial in elite endurance athletes compared the effect of consuming antioxidant rich foods (more than double the usual intake) with eucaloric control foods during an altitude training camp (2320 m). Antioxidant foods increased the antioxidant capacity (uric-acid free (ferric reducing ability of plasma (FRAP)) and reduced some inflammatory markers (plasma interleukin (IL) −13, micro CRP), but had no effect on oxidative stress (urine 8-epi-$PGF_{2\alpha}$) or changes in acute cytokine (plasma Interferon (INF)-γ, tumour necrosis factor (TNF)-α, IL1α, IL1β, IL1RA, IL2, IL5, IL6, IL7, IL8, IL10, IL12p70, IL13, IL17, monocyte chemoattractant protein (MCP)-1) response to exercise tests (Koivisto et al., 2019). A study by Watson et al. (2005) found that oxidative stress increased when male athletes followed a restricted fruit and vegetable diet for two weeks, compared with the same athletes consuming a habitually high antioxidant diet. There were no measurable changes in performance, but there was an increased F2-isoprostane concentration (oxidative stress marker) and increased perception of effort on the fruit and vegetable restricted diet (Watson et al., 2005). More high quality, well-designed and well controlled research studies regarding the effects of dietary patterns high in fruits and vegetables are needed in athletes to investigate the effects of these dietary patterns on oxidative stress and performance.

While there is some evidence that athletes consuming antioxidants have reduced oxidative stress, overall antioxidant supplementation is not associated with improved exercise performance. In some situations, antioxidant supplementation may be justified, for example, when athletes are exposed to high levels of oxidative stress (e.g. altitude training) or fail to meet dietary antioxidant requirements (e.g. restrictive eating patterns or low energy intake associated with disordered eating). There is a concern that high/supramaximal doses of antioxidants may negatively affect important ROS-mediated physiological processes required for adaption and performance, though doses that would have this effect are not typically obtained through a healthy diet. Currently, there is insufficient evidence to recommend antioxidant supplements for athletes. Obtaining a range of antioxidants from a varied and balanced diet including fruit and vegetables is advised.

4. Vitamin D

Although there are small amounts of vitamin D in food, endogenously produced vitamin D_3 is the most significant source, accounting for more than 95% of supply (Touvier et al., 2015). Calcitriol, the active

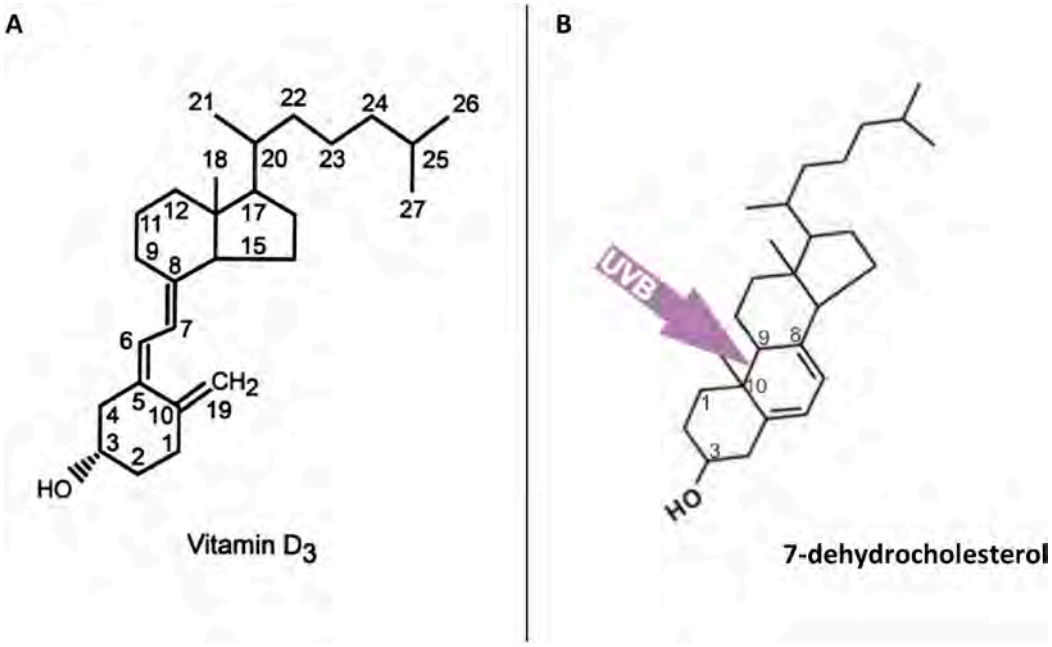

Fig. 2. A: The structure of vitamin D_3 and its numbering system. Fig. 2B: Ultra violet-β radiation cleaves 7-dehydrocholesterol between carbon 9 and 10, forming previtamin D_3. Heat-induced isomerisation occurs almost immediately, and vitamin D3 is formed (DeLuca, 2004). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

form, is generated from sequential hydroxylation of vitamin D_3 (Fig. 2A), a prohormone which is synthesised when unprotected skin is exposed to sunlight (Holick et al., 1980). The ultraviolet-β (UVβ) radiation in sunlight penetrates the skin and causes the conversion of 7-dehydrocholesterol to pre-vitamin D (Fig. 2B).

Once formed, pre-vitamin D immediately begins to isomerise into vitamin D_3 in a temperature-dependent manner which can continue for up to three days following one exposure of UVβ (Holick et al., 1980). The newly formed vitamin D_3 diffuses into the circulatory system, bound to vitamin-D binding protein and undergoes two further hydroxylations to reach the bioactive metabolite 1,25-dihydroxyvitamin D_3 or calcitriol. The first hydroxylation takes place in the liver (Fig. 3), catalysed by vitamin D-25-hydroxylase, producing the major circulating form, 25-hydroxyvitaminD_3 (25(OH)D_3) (DeLuca, 2004).

Calcitriol is so tightly regulated that circulating calcitriol is not a good measure of vitamin D status (Jones, 2008), whereas 25(OH)D has a much longer half-life with a mean of 89 days (Datta et al., 2017). Total 25(OH)D (includes 25(OH)D_3 and 25(OH)D_2 as most assays do not differentiate between the two) is the major circulating form and is a better indicator of vitamin D status. The concentration of 25(OH)D is measured either in nmol·L^{-1} or ng·ml^{-1} and the level considered to infer adequacy is controversial and varies between 50 and 80 nmol L^{-1} (20 and 32 ng ml^{-1}) (Ross et al., 2011; Holick et al., 2011).

A number of variables are involved in determining the amount of vitamin D_3 that will be generated in human skin due to sun exposure. Skin pigmentation, age, adiposity, season, latitude, protection from clothes or sun screen, and baseline level all play a role (Mazahery et al., 2015) making a determination of the level of required exposure very difficult. Additionally, there are very few sources of dietary vitamin D (Table 5), so in the absence of sun exposure, vitamin D supplements are often the only realistic option for ensuring adequacy.

Intake of vitamin D is measured either in micrograms (μg) or international units (IU), the latter now being the most commonly used. RDIs vary, reflecting the controversy over adequacy of 25(OH)D concentrations, and ranges between 200 and 1000 IU·day^{-1} for adults, with safe upper limits varying between 4000 and 10,000 IU·day^{-1} (Ross et al., 2011; Holick et al., 2011).

Some of the key risk factors for vitamin D deficiency in athletes and sports people are recognisable and should be addressed. Athletes with very dark skin will absorb less UVβ radiation than their pale skinned colleagues and therefore be at greater risk of poor status (Jablonski and Chaplin, 2000; Krzywanski et al., 2020). Gymnasts, ballet dancers and other athletes who train predominantly indoors or during the Winter months also demonstrate high rates of vitamin D deficiency (von Hurst and Beck, 2014). It is important that these risk factors for deficiency be recognised, and athletes tested for vitamin D status and supplemented, if necessary. Because vitamin D is fat soluble, and is stored in adipose tissue, it has been common practice in both research and public health settings to give bolus doses (50,000–150,000 IU) at wide timepoints. Although this seems practical for compliance and convenience purposes, there is increasing evidence that smaller doses taken daily or even weekly, are more physiologically appropriate and more effective (Ketha et al., 2018; Martineau et al., 2019).

Table 5
Vitamin D content of the main food sources in the New Zealand diet (New Zealand Food composition database Version 10, Foodworks, 2020; Xyris Software).

Food	Vitamin D_3 μg·100 g^{-1}	Vitamin D_3 IU·100 g^{-1}
Eggs	1.75	70
Butter	5.19	207
Fresh king salmon	25.52	1028
Canned salmon	5.52	220
Sardines	8.30	332
Canned tuna	2.26	90
Kahawai	10.90	436

4.1. Vitamin D in health and performance

The role of vitamin D in bone health is very well understood, with vitamin D deficiency being a recognised risk factor for fractures in children (Delshad et al., 2020) and stress fractures in athletes and military trainees (McClung and Karl, 2010). Of relevance to athletes, vitamin D is also associated with muscle strength and respiratory infection, with recent research also investigating relationships with testosterone secretion (Krzywanski et al., 2020) and antioxidant action (Ferrari et al., 2020).

In 2014, we published a review on vitamin D and skeletal muscle function in athletes (von Hurst and Beck, 2014). Subsequently a number of reviews have assessed more recent research on this topic (Han et al., 2019; Zhang et al., 2019; Chiang et al., 2017; Ksiazek et al., 2019) and the conclusions have been very similar to those found in our review. There is a wide variability in the dose, duration and methods of the various trials, making any conclusions hard to draw. Many of the trials were for short periods and had low participant numbers. Baseline status was often not measured, or not used as a criteria for inclusion, and the outcome measures were very different. However, there is benefit of vitamin D supplementation on muscle strength and/or power when athletes have low baseline status (25(OH)D ≤ 25 nmol L^{-1}), and the

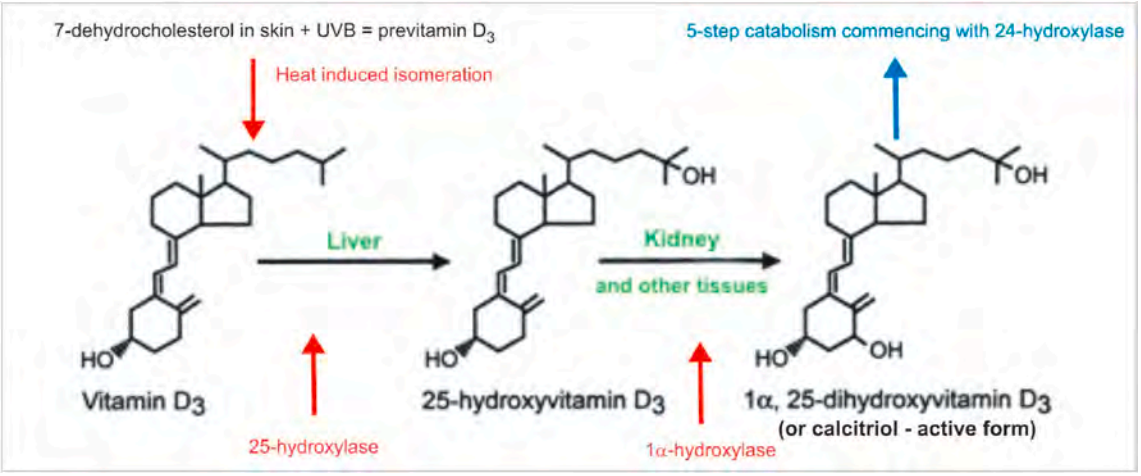

Fig. 3. The metabolic activation of vitamin D_3 to the bioactive metabolite 1,25-dihydroxyvitamin D_3 or calcitriol involves the hydroxylation of carbon 25, then carbon 1 (see Fig. 2A for numbering) (adapted from DeLuca (2004)).

optimal 25(OH)D concentration for overall performance appears to be around 80–100 nmol L^{-1}. Recently published work suggests that vitamin D may play an important role in muscle recovery. Mechanistic studies have demonstrated an increase in muscle regeneration after injury with vitamin D supplementation combined with resistance exercise (Latham et al., 2021), potentially through the regulation of mitochondrial health and oxidative capacity (Ashcroft et al., 2020).

An important consideration for athletes is the role of vitamin D in immune function, particularly in the prevention or amelioration of upper respiratory tract infections (URTIs). Trials with vitamin D supplementation and URTIs are afflicted by the same issues as the studies mentioned above, i.e. variability in methodology making it difficult to draw conclusions. However, an individual participant data meta-analysis which analysed the data from 10,933 participants from 25 randomised, controlled trials was able to overcome many of these difficulties (Martineau et al., 2019). The investigators concluded that vitamin D was protective against acute URTIs, and was especially effective in those who were very deficient (adjusted Odds Ratio (aOR) 0.30, 95% confidence interval (CI) 0.17 to 0.53). Participants who were receiving daily or weekly doses, in contrast to those receiving bolus doses, also saw a greater benefit (aOR 0.81, 95% CI 0.72 to 0.91).

New and emerging research interests in athletes include the roles of vitamin D as an antioxidant, and in the secretion of testosterone. Ferrari et al. (2020) found a correlation between 25(OH)D and ROS concentrations in male soccer players. However, recent research would indicate no relationship between vitamin D and testosterone concentrations in Polish track and field athletes (Krzywanski et al., 2020).

5. Calcium

The binding of calcium to troponin C is fundamental to muscle contraction, and therefore theoretically has an impact on performance. Certainly, calcium losses increase during exercise, largely through sweat, and such losses have been ameliorated by calcium supplementation of 800 mg day^{-1} albeit in the presence of a low calcium diet (Martin et al., 2007). To date, however, there is no evidence of calcium supplementation having a direct effect on athletic performance (Heffernan et al., 2019).

Calcium and vitamin D, along with physical activity, are the mainstays of bone mineralisation. However, in some cases, physical activity can put bone at risk, especially when nutrition is inadequate. Intense physical activity does increase the risk of stress fractures and therefore building and maintaining optimal bone health should be a consideration of every athlete. Optimum bone mineral density (BMD) is achieved in the first two decades of life, and referred to as Peak Bone Mass (PBM) (Weaver et al., 2016). Although many factors, modifiable and non-modifiable, influence the achievement of PBM, calcium has been identified as one of the most important (Nakayama et al., 2019; Golden et al., 2014). There is an inverse relationship between BMD and stress fracture history, and together with vitamin D, low dietary calcium is recognised as a key nutritional risk factor for exercise-induced stress fractures (Abbott et al., 2020). Absorption of adequate dietary calcium appears to be inhibited only when 25(OH)D concentrations fall to below 10–12 nmol·L^{-1} (Aloia et al., 2010). However, when dietary calcium intake is low, 1,25$(OH)_2$D acts to maintain serum calcium concentrations by increasing intestinal absorption. If calcium homeostatis is not achieved, then 1,25$(OH)_2$D together with parathyroid hormone (PTH) increases renal reabsorption of calcium and resorption of calcium from bone (Christakos et al., 2017). Although it is difficult to find a direct relationship between calcium intake and performance, there is a relationship between stress fractures and performance; stress fractures interrupt training and competition, and low BMD is related to a longer recovery and time to recommence training (Nattiv et al., 2013).

The best sources of dietary calcium are dairy products such as milk and yoghurt. These are also a good source of protein, and if fortified, will provide vitamin D. Consumption of low fat milk has been associated with reduced risk of stress fractures in female athletes (Nieves et al., 2010). There is no RDI of calcium specific to athletes, but achieving the RDI (Commonwealth Department of Health and Ageing Australia Ministry of Health New Zealand National Health and Medical Research Council, 2006) for adults (1000 mg day^{-1}) and adolescents (1300 mg·day^{-1}) would be highly advisable for athletes wishing to maintain BMD and reduce risk of stress fractures. Special attention should be given to those on diets that exclude dairy products to ensure suffient intake of calcium.

6. Discussion/conclusion

Athletes may self-prescribe micronutrient supplements in order to optimise recovery, boost immune function and improve performance. Inference from the literature reviewed would suggest that overall micronutrient supplementation does not tend to improve performance if a mineral deficiency is not present and that supramaximal doses may be associated with several negative consequences for athletes. These include the possibility of blunting the body's natural defence system (in the case of antioxidant supplements), high doses impacting the absorption of other nutrients, possible toxicity, and the expense and distraction from adequate energy intake and a quality diet. An additional concern for elite athletes is a lack of batch testing of formulated supplements that could increase the risk for some over the counter and readily available micronutrient supplements containing banned substances. Therefore, in elite athletes or athletes that undergo regular testing as part of competition schedules, professional prescription of supplements and adequate dietary intake are strongly recommended. In instances of deficiency, supplementation will be required, especially if the athlete is diagnosed with iron or vitamin D deficiency. In these circumstances, supplementation may aid performance through to the correction of the micronutrient deficiency, and as a result, support oxygen delivery, muscle and bone strength and immune system functioning. Supplementation with micronutrients may also be beneficial, when competing at altitude (vitamin E, iron), in conditions with low sunlight (winter or low latitudes), or when the diet is restricted, for example, athletes following low energy diets or dietary patterns which restricts certain food groups. Athletes who may present with micronutrient deficiencies are those who are in a low energy availability state and may have symptoms of Relative Energy Deficiency in Sport syndrome (including fatigue, low bone density and impaired immune function). A variety of foods from all food groups and an energy intake to meet training requirements is recommended to ensure adequate intake of all micronutrients. In the case of iron and vitamin D, a blood test is recommended to confirm micronutrient status prior to supplementation taking place. Working with a registered nutritionist or dietitian may be of benefit in order to maximise micronutrient intake from the diet. Future research incorporating well-designed randomised controlled trials are required to assess the effect of micronutrients on recovery and the functional components of health and performance. While supplemental trials have previously been completed and discussed in this review, research in dietary patterns and whole foods that would optimise micronutrient status are in need of investigation. This research may be highly beneficial to elite athletes who are cautious of supplements that are not batch tested, and as a result rely more on whole foods to prevent micronutrient deficiencies.

CRediT authorship contribution statement

Kathryn L. Beck: Writing – original draft, Writing – review & editing. **Pamela R. von Hurst:** Writing – original draft, Writing – review & editing. **Wendy J. O'Brien:** Writing – original draft, Writing – review & editing. **Claire E. Badenhorst:** Writing – original draft, Writing – review & editing, All authors contributed equally to the writing, reviewing and editing of this review and all authors approved the final version of the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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