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A review of Vitamin D: Its potential for use in athletes who live and train above 33 degrees latitude during winter months.

The purpose of this review is to evaluate the current research into the effect of vitamin D on athletic performance and make recommendations to those who live above 33 degrees latitude. Firstly, an over view of what vitamin D is, the range of roles it plays within the human body and an over view of the current research will be provided.

Like vitamins A, E and K, vitamin D is also a fat-soluble vitamin. It can be found naturally in foods such as herring, salmon and mackerel whilst it is also added to others and available as a dietary supplement often with calcium or vitamin K2. It is important to highlight that low vitamin D status is not just a problem for athletes. A meta-analysis carried out by Autier & Gandini (2007) showed a significant reduced mortality risk with supplementation of vitamin D. Vitamin D insufficiency affects almost 50% of the population worldwide (Holick, 2007) and although often described as a vitamin, it is actually a secosteroid hormone. Many vitamins, like vitamin C for example, act as antioxidants or cofactors in enzymatic reactions; however, vitamin D has many other functions that are significantly different.

Vitamin D aids the absorption of calcium, which in turn will help bone development, immune function, blood pressure, nerve signals along with strength and mass of muscle. Mitochondria are the aerobic power houses of our cells and a combination of glucose and oxygen help to produce Adenosine Triphosphate (ATP), which is a high-energy molecule, used frequently in muscle contractions. Sinha et al (2013) suggested improved mitochondrial efficiency through training would help improve phosphocreatine recovery times. In this study it was found that those who supplemented with oral vitamin D over a period of 10-12 weeks reduced phosphocreatine recovery from 34.4 seconds to 27.8 seconds. In a parallel study, those who had low vitamin D levels had reduced mitochondrial function. This type of evidence provides support to the argument that vitamin D has a multitude of actions within the body. Adding further support to this is the work of Mach & Fuster-Botella (2016) who acknowledged the importance of vitamin D3 in regulating the gut microbiota and that gut flora affects endurance performance. Their systematic review of literature concluded that microbiota provided a key role in controlling the oxidative stress and inflammatory responses as well as improving metabolism and energy expenditure during intense exercise. Bashir et al (2016) made the link between improved gut health, vitamin D status and immunity. These authors concluded that Vitamin D3 modulates the gut microbiome of the upper GI tract, which might explain its positive influence on gastrointestinal diseases, such as inflammatory bowel disease or bacterial infections. Previously work from He et al (2013) established and investigated the association between vitamin D status and the incidence, severity and duration of upper respiratory tract infections (URTI) during a 16-week winter training period in 225 male and female athletes. These authors concluded that those athletes exhibiting a higher vitamin D status displayed significantly fewer URTI episodes. Furthermore, it was noted that when a URTI was present, athletes reduced their training load by an average of 24%, this is likely to have a negative impact on performance over time. Although this study provides an apparent positive relationship between performance and vitamin D status, this study followed a cohort and looked for associations between biomarkers. This approach gives an insight in to potential mechanisms, but a true cause-and-effect relationship cannot be drawn. Providing further support for vitamin D as a modulator of inflammation is a study from Timms et al (2002) that demonstrated a dramatic reduction in the inflammatory proteins CRP and matrix metal-loproteinase (MMP), after supplementation in 171 healthy adults, with a 68% reduction in MMP.

Vitamin D has wide ranging effects throughout the body and subsequently there are multiple vitamin D receptors (VDR) in the body's tissues. The major source of vitamin D for humans is casual exposure of the skin to sunlight. Older people often find themselves inside and at a reduced level of sun exposure, which leaves them at risk of osteomalacia, a disease involving muscle weakness and pain. With the aforementioned in mind, many studies (Houston, 2011; Harris & Dawson-Hughes, 2002; Bischoff-Ferrari, 2004) on vitamin D have focused on older adults. However Cannell et al (2009) reviewed the effect of Vitamin D on athletic performance. These authors suggested that even as early as the 1940's German studies highlighted low vitamin D as being a causal factor for reduced performance. Cannell et al (2009) mentioned that early studies were not looking at serum 25 (OH)D concentrations, which made it difficult to identify an ideal level of vitamin D for peak performance.

Season, time of day, length of day, cloud cover, air pollution, skin melanin content, and sunscreen are among the factors that affect UV radiation exposure and subsequent vitamin D synthesis. Matsuoka et al (1987) stated that when wearing sunscreen of factor 30 or above there was a 95% reduction in Vitamin D production. Vitamin D production from sunlight operates on a negative feedback loop; therefore vitamin D toxicity from UVB radiation is not typically a concern (Vieth, 1999). It has been recommended that a fair skinned person spends 20-30 minutes with direct sunlight exposure on the face and forearms, 2-3 times per week to achieve sufficient vitamin D levels in the summer (Pearce & Cheetham, 2010). Vitamin D absorbed through sunlight due to direct contact with the skin is not useful in its current form. The liver is the site for the first hydroxylation where it is converted from Vitamin D 25-hydroxylase (25-OHase) to 25(OH)D. Then 25(OH)D requires another hydroxylation in order for it to become the biologically active form of Vitamin D, known as 25(OH)2D. This is a very important substance as it helps to stimulate the absorption of calcium (Lips et al, 2006). Holick (2008) suggested that between the hours of 1000 and 1500 hours is the optimal time of day for exposure during spring, summer and autumn. However, Holick and Chen (2008) explained that due to a change in the zenith angle of the sun unfortunately for residents of the UK, solar radiation is negligible throughout October to April due to the geographic latitude, thus the potential need for supplementation.

Public Health England (PHE) currently recommend that serum 25 (OH)D concentrations should not fall below 25 nmol/litre at any time of year to protect musculoskeletal health. A Reference Nutrient Intake (RNI) for vitamin D, of 10 µg/d (400 IU/d) is recommended for everyone aged 4 years and above. The importance of daily vitamin D intake is supported by data from the United States National Health and Nutrition Examination Survey (NHANES 2005-2006) in which the recommended dietary allowance (RDA) for all adults aged 18-70 years old is 600 IU/day. Importantly vitamin D intakes are reported to be well below the RDA when supplements were not included. Average intake levels for males and females from foods alone ranged from 204-288 IU/day and 144-276 IU/day respectively. The study by Holick et al (2011) on behalf of the Endocrine Society proposed that 1500-2000 IU/d might be required to consistently maintain adequate vitamin D status throughout winter. Tripkovic et al (2012) conducted a systematic review and meta-analysis and found vitamin D3 to be more effective at raising serum concentrations than vitamin D2 and should therefore be considered the superior form of supplementation. Toxicity from vitamin D3 supplementation is rare and data from human studies suggests that prolonged intakes up to 10,000 IU/day have not been associated with adverse effects (Yetley et al, 2008). However, the current tolerable upper intake level (UL) for vitamin D as set by the Institute of Medicine (2011) is 4000 IU/day. Although the UL is not a recommended level of intake, it is an estimate of the highest level of intake, which carries no appreciable risk of adverse health effects. Unfortunately a lack of good experimental design amongst studies leaves no clear conclusion as to the effectiveness of Vitamin D for improved athletic performance. For example Wyon et al (2014) studied elite ballet dancers who took 2000 IU of Vitamin D per day over four months. They were found to suffer fewer injuries and performed better, but dancers serum 25 (OH) levels were not analysed and thus the researchers were unable to state if it was the vitamin D that was responsible.

Close et al (2013) conducted two studies to investigate the effect of high dosage vitamin D supplementation (20000 to 40000 IU) over a 12-week period. In the first study, thirty club-level athletes received a placebo, 40000 IU/week or 20000 IU/week of vitamin D3 for 12 weeks during winter months. Supplementation was able to correct the existing vitamin D deficiencies and increased the group average vitamin D status to ≥30 ng/ml. Despite this, supplementation had no significant effects on vertical jump, 1 repetition maximum (RM) testing or 20-meter sprints when compared to placebo. Interestingly, although the higher dose (40000 IU/week) caused a sharper increase in vitamin D status, after 12-weeks there was no difference between either dose. This suggests that to maintain adequate vitamin D status throughout winter, more is not necessarily better. In a subsequent study, an 8-week dose of 5000 IU/day vitamin D3 was found to increase 10-meter sprint times and vertical jump height compared to placebo. However, the experimental sample consisted of 5 youth team football players (after drop outs). The most consistent increases in performance were seen in 1 RM bench press and 1RM back squat; however external training and diet were not recorded or controlled for across the 8-week study. Given the small sample size, there is possibility that any performance improvements were due to chance or differences in diet and activity levels, which were not controlled. Interestingly, 3 of the 5 athletes were not able to achieve ‘optimal’ vitamin D status of ≥40 ng/ml following supplementation. At present there is a lack of empirical evidence that supports the role of an optimal vitamin D status. As previously highlighted Cannell et al (2009) stated that most research is largely descriptive and adequate randomised control trials are lacking.

Similar to Close et al (2013) another study that investigated vitamin D supplementation during winter months was that of Barker et al (2013). This study aimed to establish whether or not, higher vitamin D concentrations were associated with faster recovery of skeletal muscle after injury. Subjects were not assigned randomly in this quasi-experimental design, therefore cause and effect cannot be established. Due to strict inclusion criteria, only 14 non-smoking subjects were included, with a minimum exercise time of 3 bouts of 30 minutes continuous exercise per week in the year prior to the study commencing. Data collection took place in December through to March in Salt Lake City, at this time sunlight levels would be lowest in this region during winter months. Subjects were asked to keep their diet consistent with that of the rest of the year. Researchers used measured markers of inflammation and vitamin D levels before, during and after an intense exercise protocol to analyse muscle repair rate. Subjects performed 10 sets of 10 repetitions on single leg jumps, with one of their legs randomly assigned as the test leg and the other as the control. The researchers aims included trying to find out if pre exercise serum 25(OH)D concentrations could predict muscular weakness immediately, 48 hours and 72 hours post intense exercise. They also wanted to identify if markers of inflammation in the blood might predict alterations in serum 25 (OH)D concentrations. They did find that in this small population pre exercise serum concentrations of 25 (OH)D provided a significant influence ($p < 0.05$) on the rate of recovery of skeletal muscle strength after an acute bout of intense exercise. With the aforementioned in mind, UK based athletes and other athletes living and training above 33 degrees latitude in the winter, that do not supplement or expose themselves to artificial UVB radiation during the winter months, have to rely on diet and summer vitamin D stores throughout this period. The lack of quality dietary sources results in ongoing catabolism of internal stores and vitamin D status can fall significantly in the winter (Morton et al, 2012).

The concern within team sports continues and Morton et al (2012) found that within a group of professional soccer players, their winter vitamin D status was found to fall to <50% of their previously recorded summer values. This is of particular concern because individuals engaged in outdoor training would be expected to have a higher vitamin D status due to increased exposure to sunlight. Interestingly however, a study carried out by Jastrzębska, Kaczmarczyk, & Jastrzębski (2016) showed no significant improvement in peak power performance between a control group and a supplemented group (SG) within a cohort of football players. In the 8-week trial the SG and control group were subjected to the same high intensity interval-training programme. The control group had no supplemented vitamin D whereas the SG was given

5000IU per day for the 8 weeks. The authors did acknowledge however that seasonal serum 25 (OH)D concentrations may need to be allowed for in a supplement plan for the soccer players. Heaney et al (2003) suggested that the athlete who is below 30ng/ml of serum 25 (OH)D should take 3000-5000 IU per day of vitamin D3. These authors also recommended that once an athlete reaches the sports health optimal level of 50ng/ml, that taking more than 10000 IU per day is not necessary.

When taking a close look at performance, testosterone and strength have been widely linked and some cross-sectional research suggests a positive association between vitamin D and total testosterone (Nimptsch et al, 2012). Interestingly however, there wasn’t a parallel relationship between vitamin D and testosterone changes in seasonal differences. A cause and effect relationship between vitamin D and testosterone was examined further by Pilz et al (2011). In the experimental group, 200 overweight, non-diabetic participants consuming 3332 IU/day of vitamin D3 for 1 year were observed. Those in the experimental group significantly increased their total testosterone levels by 25.2%. At first glance this seems very impressive, however it must be taken into consideration that the men in the study started with testosterone values at the lower end of the acceptable range (9.09–55.28 nmol/l for males aged 20–49 years) and supplementation caused an increase to take them to more respectable levels.

Considerations for further research into the effectiveness of Vitamin D3 supplementation and performance should be directly linked with the issues already highlighted and have an athlete centric approach. Issues within the research in this field were highlighted by a meta-analysis of cross sectional studies carried out by Hagenau et al (2009). These authors found that latitude didn't accurately predict vitamin D status and that other factors such as clothing, time spent indoors and outdoors, dietary vitamin D and other genetic factors may explain the lack of association between latitude and vitamin D status in the studies. Many authors including Cannell et al (2009) and Willis et al (2008) have identified that athletes with dark skin, who have greater levels of melanin in their skin are more susceptible to lower concentrations of serum 25 (OH)D than those with light and fair complexions. There have been very few studies into the influence of race and vitamin D levels in athletes. A comparison of mean vitamin D concentrations between 31 professional white and 58 black professional NFL players was carried out by Shindle et al (2011). These authors found that during the spring vitamin D levels were significantly lower in the black players than they were in the white players (20.4 ng/mL vs. 30.3 ng/mL; $P < 0.001$). More research into the direct relationship between speed, strength, power and vitamin D status should also be taken into account. Cannell et al (2009) highlighted that there have been very few studies investigating speed and vitamin D. In fact the only study that these authors highlighted in their review was one from 1938, that compared 100m sprint times between two groups which had the same training programme; one was exposed to irradiation with a sun lamp and the other wasn't. An improvement was recorded but because the methodology was in Russian and the sample (4) size was so small, no clear conclusions could be drawn. Much of the research into the relationship between the number and size of type 2 muscle fibre hypertrophy and vitamin D status has only been carried out in rat studies (Birge & Haddad, 1975; Wassner, Li, Sperduto & Norman, 1983). Whilst these studies show promising results in fibre growth following supplementation in rats, those studies evaluating the effects in human adults are less conclusive. A meta-analysis of 52 studies which examined the effects of vitamin D supplementation on grip strength showed no significant effect (SD -0.02, 95%CI -0.15,0.11) on proximal lower limb strength (SD 0.1, 95%CI -0.01,0.22) in adults with vitamin D concentrations >10 ng/mL (Stockton et al, 2011).

To conclude, more studies are needed with tighter controls of diet and training programmes. Measures of strength, speed and power need to be standardised and participant samples need to be clearly defined between elite and recreational athletes to help draw more accurate conclusions for supplement dosages. More studies with the aforementioned controls would be useful when investigating those in the same sport but living and training at different latitudes. It would also be useful to see the effect on a cohort of athletes, both team and individual, that train overseas during winter months for a period of 1 week or more and compare within the cohort on a following season when they don't train overseas below 33 degrees latitude. Despite some conflicting evidence it does appear that athletes who either live above 33 degrees latitude or have dark skin will tend to suffer from lower vitamin D levels. Therefore it would be advised that blood tests are carried out intermittently during seasonal changes to make sure that the serum 25OH(D) levels are maintained above 20ng/ml in all athletes. A supplementation of the active form of vitamin D3 would be recommended around the 4000IU per day, to be taken with dietary fats, as vitamin D3 is a fat-soluble vitamin.

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