Vitamin D supplementation for athletes: Too much of a good thing?

Vitamin D is currently a hot topic with many athletes routinely advised dietary supplementation. In this article Dr Graeme L Close and Prof William D Fraser examine the potential advantages and disadvantages of vitamin D supplementation for athletic populations.

Vitamin D synthesis and function
Vitamin D is a lipophilic pro-hormone that can be obtained from dietary sources but is predominately synthesised in skin actioned by solar ultraviolet-B (UVB) radiation. Under optimum conditions, physiological sufficiency can be met through endogenous synthesis. Active vitamin D (1,25(OH)2D3) has important functions in many target tissues acting via the vitamin D receptor (VDR). Few foods contain high vitamin D and endogenous synthesis is the predominant source of vitamin D. One full body minimal erythema dose of UVB produces ~20,000 IU of vitamin D, the equivalent of 8 fresh farmed salmon or 50 standard oral multivitamins. Despite 1,25(OH)2D being the most active biological metabolite in man, total 25(OH)D is measured as the clinical marker of vitamin D deficiency.

Functions of vitamin D metabolites are the regulation of calcium and bone metabolism. Indeed, skeletal pathologies (e.g., rickets, osteomalacia) ensue when total 25 (OH)D concentrations are low. All tissues in the human body are thought to possess the VDR and thus, any deficiency in 25 [OH]D may have wider consequences than just compromised bone health. In relation to skeletal muscle, low total 25(OH)D concentrations in the elderly increase the risk of falls and we observed that vitamin D supplementation in highly trained football players improves vertical jump height and 10-m sprint times (Close et al., 2012).

Problems with defining vitamin D deficiencies
Defining vitamin D deficiency is complicated as to what concentrations of total 25(OH)D constitute vitamin D deficiency, insufficiency, sufficiency and toxicity (Table 1). Zittermann (2003) defines “optimum vitamin D status” between 100-250 nmol.L⁻¹ whereas the US Institute of Medicine (IoM) define inadequate vitamin D status as <50 nmol.L⁻¹ suggesting potential adverse events when levels are >125 nmol.L⁻¹. The Scientific Advisory Committee on Nutrition (SACN) and the Food Standards Agency (FSA) of the UK define vitamin D deficiency as <25nmol.L⁻¹ although these definitions have been subject to recent critique. These recommendations are in relation to adequate bone function and there are currently no guidelines to maximise neuromuscular performance. It has been argued that some diseases may be preventable or modified by increasing total 25(OH)D >120 nmol.L⁻¹ and therefore deficiency should be defined as <120 nmol.L⁻¹ (Heaney, 2011). Finally, it is argued that there are no recorded cases of “true” vitamin D intoxication <350 nmol.L⁻¹ and therefore safe but sufficient concentrations may lie between 120 – 225 nmol.L⁻¹ (Heaney, 2011).

Table 1. Classification of vitamin D status based on total 25(OH)D concentrations. Definitions from US Institute of Medicine except the definition of ‘Optimal’, which is taken from Heaney (2011).

<table>
<thead>
<tr>
<th>Serum Total 25(OH)D</th>
<th>Status</th>
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</thead>
<tbody>
<tr>
<td>&lt;12 nmol.L⁻¹</td>
<td>Severely deficient</td>
</tr>
<tr>
<td>12.5 – 30 nmol.L⁻¹</td>
<td>Deficient</td>
</tr>
<tr>
<td>30 – 50 nmol.L⁻¹</td>
<td>Inadequate</td>
</tr>
<tr>
<td>&gt;50 nmol.L⁻¹</td>
<td>Adequate</td>
</tr>
<tr>
<td>&gt;120 nmol.L⁻¹</td>
<td>Suggested Optimal</td>
</tr>
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25 [OH]D in society and athletes
Total 25(OH)D deficiency is widespread in several populations owing to lack of adequate sun exposure, indoor lifestyle, use of sun-screen and poor dietary habits. In the UK between October and March, synthesis of vitamin D3 is almost impossible and the majority of the UK population may become vitamin D deficient during the winter. Athletes are also susceptible to total 25(OH)D deficiency, even those that train outdoors. In 61 UK based athletes (Rugby League, Soccer and Jockeys) using the classifications from Table 1 we reported that 7%, 28% and 26% were severely deficient, deficient or inadequate, respectively, with only 38% adequate and 2% demonstrating optimal concentrations (Close et al., 2012). However, the physiological consequence of this deficiency is not yet fully defined.

Vitamin D supplementation
The RDA for vitamin D in the UK is 400 IU. Many authors suggest this is too low and have advocated supplementation with considerably higher doses. Studies on vitamin D consumption and the effects on total 25(OH)D during the...
summer report variable results, however those performed during the winter are more consistent. Aloia et al. (2008) investigated the supplemental doses required to increase serum 25\([\text{OH}]\text{D}\) to >75 mmol.L\(^{-1}\) and observed that 5000 IU daily for individuals <55 mmol.L\(^{-1}\) was sufficient. As well as dosage, length of supplementation is important. A study in Scottish women reported it takes 6-8 months to get into a new balance state when receiving 400IU or 1000IU of vitamin D3 daily.

We recently investigated the effects of supplementing athletes with either 20,000IU or 40,000IU per week (single bolus weekly) for 6 and 12 weeks on total 25\([\text{OH}]\text{D}\). Six weeks of 20,000 IU vitamin D3 per week increased total 25\([\text{OH}]\text{D}\) from a mean of 50 to 80 mmol.L\(^{-1}\) and by 12 weeks this increased to 95 mmol.L\(^{-1}\). Using 40,000IU per week, serum total 25\([\text{OH}]\text{D}\) increased at 6 weeks to 95 mmol.L\(^{-1}\) with no further change at 12 weeks (data under review). Data suggest both doses are effective at increasing serum total 25\([\text{OH}]\text{D}\) in a short period of time although neither dose resulted in serum total 25\([\text{OH}]\text{D}\) concentrations suggested as optimal by Heaney (2011). This protocol of supplementing once per week as opposed to daily may be advantageous to the busy athlete and reduce the chances of forgetting to take supplements.

**Dangers of toxicity from vitamin D supplementation**

Concern with toxicity is being raised regarding detrimental effects of both short and long-term exposure to high circulating concentrations of 25\([\text{OH}]\text{D}\). Hypercalcaemia occurs when serum adjusted calcium concentrations are >2.6 mmol.L\(^{-1}\) (10.5 mg.dL\(^{-1}\)). Clinical signs and symptoms of hypercalcaemia depend on the magnitude and the rapidity of onset and include anorexia, weight loss, polyuria, heart arrhythmias, fatigue, and soft tissue calcification. When serum calcium is >3.0 mmol.L\(^{-1}\) (12 mg.dL\(^{-1}\)) the kidney’s ability to reabsorb calcium is limited, hypercalciuria will occur (10.0 mmol.L\(^{-1}\), 25mg.dL\(^{-1}\)) and diabetes insipidus can result. This may happen with increased calcium and/or vitamin D intake.

There is uncertainty about the progressive health effects for regular ingestion of even moderately high amounts of vitamin D over several decades. Most evidence is based on short-term exposure (<6 months) from adult populations with few data specific to children or groups such as athletes. A number of variables may affect the onset of toxic symptoms in the face of excess vitamin D intake but there has been a paucity of longer term studies investigating the effects of doses >10,000 IU weekly or the maintenance of serum total 25\([\text{OH}]\text{D}\) >250 nmol.L\(^{-1}\).

Studies in the elderly have highlighted the dangers of high dose vitamin D. In community dwelling women (>70y) given placebo or 500,000IU each autumn or winter for 3-5 years, high-dose vitamin D3 resulted in a significant increase in falls and fractures with the event occurrence highest in the 3 months following commencement of supplementation. Animal studies suggest that high dose vitamin D triggers a feedback loop where 1,25\([\text{OH}]\text{D}\) \(_2\)D metabolism increases to decrease the possibility of hypercalcaemia and this may reduce calcium availability negatively affecting muscle contraction, relaxation and overall function. Recent data from Copenhagen (Durup et al., 2012) has described a reverse J shaped curve association between serum total 25\([\text{OH}]\text{D}\) and mortality with both low (10 nmol.L\(^{-1}\) and high 140 nmol.L\(^{-1}\) serum total 25\([\text{OH}]\text{D}\) concentrations increasing all-cause mortality with the lowest mortality in the mid-range (50nmol.L\(^{-1}\)). Clearly, a blanket high dose supplementation for all athletes may therefore be detrimental.

**Practical recommendations**

- Athletes with serum total 25\([\text{OH}]\text{D}\) <50 nmol.L\(^{-1}\) should consider appropriate vitamin D supplementation.
- Athletes are encouraged to eat a diet rich in vitamin D and to obtain sensible sun exposure during the summer months although not at the risk of sun burn.
- Mega doses of vitamin D supplementation should be discouraged until comprehensive evidence shows it is safe and there are physiological benefits to such doses.
- Excessive vitamin D supplementation may lead to toxicity and be detrimental to athletes’ performance and health.

**References**


