



Your Monthly Update

Dear Colleague

Welcome to the October newsletter from Pure Bio Ltd.

In an attempt to summarise much of the recent research being published and disputed between various peer review publications, our featured topic for this month is vitamin D

We always welcome feedback and suggestions.

Vitamin D

The Facts

Vitamin D has a well-established role in calcium homeostasis and the maintenance of healthy bone.

Recent articles indicate that the prevalence of vitamin D deficiency is much higher than previously recognized (more than 90% in patients with chronic pain, according to a recent study published by the Mayo Clinic), and that vitamin D supplementation is much safer than previously recognized. The recent studies by Al Faraj (2003), Vieth, Chan, and MacFarlane (2001), and Heaney et al (2003) all used daily doses of 4,000 IU per day or more with no evidence of adverse effects.

Recent articles have also suggested that vitamin D may have a role in the prevention and treatment of many chronic diseases.

Indications for Vitamin D (*and sunlight*)

- *Osteoporosis and other metabolic bone diseases*
- *Prevention of various cancers including prostate, breast, colon, lung*
- *Decreases risk of autoimmune diseases e.g. type I diabetes, RA, MS*
- *Mental health problems – SAD, PMS, depression, general mood*
- *Elevated BP and heart disease*
- *Obesity*
- *Important for the foetal skull and brain, therefore important in pregnancy and early childhood*

Physiology of Vitamin D

Sunlight converts 7-dehydrocholesterol in the skin into vitamin D3 (cholecalciferol). Cholecalciferol is transported to the liver & converted to 25-hydroxycholecalciferol (25-OHD₃). 25-OHD₃ is then transported to the kidneys & converted to

1,25-dihydroxycholecalciferol (1,25(OH)₂D₃), which is the highly active form of vitamin D and is 10 x more potent than cholecalciferol. 1,25(OH)₂D₃ increases calcium and phosphorus absorption in the intestine, induces osteoclast maturation for bone remodelling, and promotes calcium deposition in bone and a reduction in parathyroid hormone (PTH).

Recent research indicates that virtually all cells have the capacity to convert 25-OHD₃ into 1,25(OH)₂D₃, and that this conversion mechanism is an intracellular anti-cancer mechanism.

The production of 1,25(OH)₂D₃ should occur everywhere in the body, thus requiring much more 25-OHD₃ than previously thought. When there is insufficient 25-OHD₃, the production of 1,25(OH)₂D₃ in the kidneys is a compensatory mechanism to increase serum calcium, even if that means mobilising it from the bone. A further response to the high 1,25(OH)₂D₃ and the low 25-OHD₃ is increased production of PTH. Suppression of PTH by vitamin D is also clinically important since relatively lower levels of PTH appear to promote and protect health, and higher levels of PTH correlate with increased risk for myocardial infarction, stroke, and hypertension.

The objective in blood testing is therefore a high range of 25-OHD₃ (75 – 100 nmol/l) with a lower range of normal for 1,25(OH)₂D₃ (20 – 50 pg/ml). Research shows that PTH begins to elevate once 25 drops below 110 nmol/l.

Impact of Vitamin D on disease

Vitamin D deficiency and musculoskeletal pain

Vitamin D deficiency causes dull, achy musculoskeletal pain that is incompletely responsive to manual treatments. The pain may be widespread or confined to a particular area, most commonly the low back and lumbar spine. The mechanism by which this pain is produced has been clearly elucidated: 1) vitamin D deficiency causes a reduction in calcium absorption, 2) production of parathyroid (PTH) hormone is increased to maintain blood calcium levels, 3) PTH results in increased urinary excretion of phosphorus, which leads to hypophosphataemia, 4) insufficient calcium phosphate results in deposition of unmineralized collagen matrix on the endosteum and periosteum of bone, 5) when the collagen matrix hydrates and swells, it causes pressure on the sensory-innervated periosteum resulting in pain. (3) Indeed, several clinical investigations have recently shown that vitamin D deficiency is particularly common among people with musculoskeletal pain.

Non-musculoskeletal manifestations of hypovitaminosis D

Both the peripheral and central nervous systems have multiple sites of action for vitamin D, and it appears likely that vitamin D modulates serotonin and melatonin synthesis and metabolism. Alterations in vitamin D levels appear to explain, at least in part, the adverse psychological effects of sunlight deprivation, such as which occurs in winter. Preliminary evidence suggests that vitamin D deficiency may also be particularly common among patients with inflammatory and autoimmune disorders, and that vitamin D may modulate inflammatory responses.

Nutritional Supplement Treatment Options to balance vitamin D deficiency and decreased bone calcium

Vitamin D3 400i.u. PE — *use a maximum of 2 x daily, unless blood monitoring is regularly undertaken*

Calcium – various compounds – citrate, aspartate, MCHA

Text by Tracy S Gates, for and on behalf of Pure Bio Ltd.

Tel:01403 730342

Email: info@purebio.co.uk

www.purebio.co.uk

Calcium combination products e.g. +CAL+ PE

Zinc – picolinate, citrate, orotate

Potassium – aspartate, citrate

Magnesium – aspartate, citrate, glycinate, orotate

Selenium – selenomethionine, citrate

EPA/DHA

Vitamin A – oil based

*** * * All of the above are available from Pure Bio Ltd. * * ***

References:

The Clinical Importance of Vitamin D (Cholecalciferol): A paradigm shift with implications for all healthcare providers – Alex Vasquez, DC, ND, Gilbert Manso, MD, John Cannell MD

Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers and cardiovascular diseases – M F Holick

The UV Advantage – M F Holick, M Jenkins. Ibooks, New York 2004

Vitamin D – New Developments and AK – W Gerz MD

For further information, contact:

Tracy S Gates
Director
PURE BIO LTD.
01403 730342
info@purebio.co.uk