EDITORIAL

Vitamin D—Time for Reassessment

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It is a fact of life in the current scientific world that publicity, hype, and spin are more important in spreading information—and disinformation—about nutritional medicine than peer-reviewed scientific studies. The recent publicity over vitamin C is an obvious instance of this; a paper from the University of Pennsylvania Center for Cancer Pharmacology was the subject of a press release that presented it as new data that may have shown a damaging effect of vitamin C, even though any number of other studies have shown the opposite. When the principal author, Ian A. Blair, was finally contacted by telephone (the press release was issued while he was out of the USA on holiday), he apparently said ‘Absolutely for God’s sake don’t say vitamin C causes cancer’. This did not stop the media from reporting exactly that, with the result that many patients world-wide have been stopping or reducing their vitamin C intake, with possible detrimental effects on health. As has been pointed out repeatedly, if the study had shown a positive effect from vitamin C, or any other nutrient, experience tells us that it would not have been ‘hyped up’ in this manner.

Which is also exactly what did not happen with a crucial review article on vitamin D in the American Journal of Clinical Nutrition in 1999 [1]. This paper, by Dr Reinhold Vieth in Toronto, laid out strong arguments that we are all deficient in vitamin D, because we have grossly misjudged the necessary intake for adults and the amount needed to cause toxicity. Since then Dr Veith has continued to accumulate evidence of this, his central thesis, and for the further ramifications of it. In this issue he brings the argument up to date [2] and discusses the implications, not just for bone health, but for a number of other diseases as well.

Ironically, this comes at a time when the US Environmental Protection Agency is advising that ultraviolet light, and therefore sunlight, is so dangerous that we should ‘protect ourselves against ultraviolet light whenever we can see our shadow’. Following this advice is likely to lead to an increase in vitamin D deficiency diseases, and is effectively discriminatory against the many individuals with darker skin types who now live in higher latitudes. As a physician who has seen two cases of rickets this year, I find this of great concern. Whether sunlight is the direct and principal cause of skin cancers and other skin damage is a separate debate, which we cannot cover here, but the evidence is mounting that following current guidelines on sun protection will have detrimental effects on other areas of health.

There are several important, only recently understood, and not yet widely appreciated, points to be made about vitamin D:

- Healthy/desirable levels are much higher than was thought. Laboratory reference ranges for 25-hydroxyvitamin D (25(OH)D) are still stated at around 40–100 nmol l\(^{-1}\). But people who live in equatorial regions, and spend much of their days in the sunlight (farmers and lifeguards, for instance), consistently show levels over 100 nmol l\(^{-1}\), and even above 200 nmol l\(^{-1}\). Because humanity evolved in such an environment, it is clear that the vitamin D exposure that parallels the ‘Stone Age diet’, as the environment in
which humanity evolved, was much higher than the levels we have now come to regard as normal. Yet vitamin D deficiency is widespread in developed countries, and food fortification is disappointing as a solution to this.

- Sunlight gives much more than we can get in (even fortified) food. Our understanding of the chemistry of vitamin D now makes it clear that it is not really a vitamin; the reason we need dietary intakes is only because we are so deprived of ultraviolet light, which triggers its synthesis in the skin. A whole-body sunlight exposure barely sufficient to trigger tanning (the minimum melanogenesis dose, which may be as little as 15 min for pale-skinned individuals) has been established to be equivalent to the consumption of as much as 10,000 IU of vitamin D. Compare this with the US recommended dietary allowance (RDA) for adults under 50 years of age, which now stands at 200 IU. Studies of everyday sunlight exposure conducted in San Diego, California, found that normal urban inhabitants spend very little of their time in full sunlight [3], which accounts for the generally lower vitamin D levels, most of which will have been obtained from food, rather than from the effect of sunlight.

- Toxicity requires much more than was thought. Clearly the high levels of vitamin D synthesis, and of serum 25(OH)D, mentioned above are not toxic. A number of studies have in fact shown that toxicity requires doses in the milligram range (1 mg = 40,000 IU). Vieth suggests that the widely held view that vitamin D is the most toxic of all the vitamins derives from the fact that physiological doses are in the microgram range not the milligram range, as with most other vitamins.

- Vitamin D2 is a poor substitute, other analogues probably too. Textbooks still assume that ergocalciferol, vitamin D2, manufactured by yeast organisms, is equivalent to cholecalciferol, D3, the human physiological form. But the evidence currently available indicates that it has about 25% of the potency of D3 in humans. Nonetheless it is still used widely. In Australia, where sunlight is close to becoming illegal, for instance, D3 is not licensed for use in food, only D2.

- Older people need more than young adults, coloured people more than white people. The US RDA for people over 70 years has recently been trebled to 600 IU day\(^{-1}\), an impressive increase. Laboratory evidence shows that, with increasing age, the skin becomes progressively less efficient at synthesizing 25(OH)D. Yet older people typically spend less time in the sunlight, are more prone to vitamin D deficiency, and to the consequent diseases—osteomalacia, osteoporosis, bone fractures. Recent evidence indicates that vitamin D strengthens muscles, thus presumably improving balance and movement and preventing falls, thus reducing fractures independently of its effect on bone density.

- The RDA was derived from what is in a spoonful of cod liver oil, not from what we actually need. A spoonful of cod liver oil, such as so many mothers used to give to their children daily (in some countries such as Iceland this is still a breakfast custom), contains a little less than 400 IU. As has been observed with other nutrients, the original RDA appears to have been set on a pragmatic basis, rather than in relation to human needs; 400 IU has proved ineffective at preventing deficiency levels of vitamin D, and estimates of requirements continue to rise over the years.

- The vitamin appears to be 25(OH)D, whereas 1,25-dihydroxyvitamin D (1,25(OH)2D) is the hormone. 25(OH)D has always been thought to have no physiological role, needing to be converted to 1,25(OH)2D to achieve its effects. But it now appears to be in fact better correlated than 1,25(OH)2D with the deficiency diseases, rickets and osteomalacia. Only a surprisingly small proportion of 25(OH)D is converted into 1,25(OH)2D, which has a clear hormonal effect on the active uptake of calcium from the gut. The physiological role of 25(OH)D appears to lie elsewhere.

- Our current sun phobia is contributing to many other diseases. Recent work has demonstrated the presence of receptors for vitamin D in an enormous range of tissues, and physiological roles for the nutrient in a number of different contexts (preventing and
treat seasonal affective disorder (SAD), enhancing activation of thyroid hormone in
the periphery, and modulating immune function, for instance). Epidemiological data point
to an effect of sunlight exposure and vitamin D in reducing the prevalence of a number
of the major degenerative diseases that have been increasing in recent decades—cancer,
hypertension, hypercholesterolaemia (see previous volumes of this journal), diabetes and
heart disease in a wider sense—probably all the components of Syndrome X in
fact—multiple sclerosis, and vulnerability to infections.

In the light of all this new evidence, it is clear that we must revise our policies and our
advice, not only to sufferers from the bone diseases of old age, but to those suffering from
or at risk from a range of other degenerative diseases, and indeed to all those wishing to
prevent such diseases and to maintain good health. Our current phobic recommendations for
the avoidance of sunlight exposure start to look like the worst possible public health
measure.

REFERENCES

