

# Food fortification to tackle vitamin D deficiency: to address classic or non-classic effects?

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Vitamin D is a hormone synthesised in the skin from 7-dehydrocholesterol following ultraviolet B (UVB) radiation exposure from sunlight. Limited dietary sources of vitamin D make it difficult for certain groups to maintain optimum serum 25-hydroxyvitamin D (25OHD) levels. These high-risk groups include individuals residing at high latitude, dark-skinned populations and those who avoid the sun for medical or cosmetic reasons or who wear full body clothing for religious reasons.<sup>1</sup>

The classic role of vitamin D in optimising bone health through mineral homeostasis is undisputable. Deficiency causes rickets (impaired mineralisation of the growth plates) in children and osteomalacia (impaired mineralisation of pre-formed bone) in children and adults, which can manifest as muscle pain, weakness, delayed development and bony deformities.<sup>2</sup> However, over the last three decades or so understanding of the non-classic role of vitamin D against inflammation and infection has evolved. A growing body of evidence suggests a role for vitamin D in immune modulation through 1,25-dihydroxyvitamin D [1,25(OH)<sub>2</sub>D], the active form of 25OHD, regulating the expression of vitamin D-responsive genes which influence immune cell signalling pathways.<sup>3</sup>

Vitamin D deficiency has been linked to autoimmune conditions such as type 1 diabetes mellitus (T1DM), multiple sclerosis, Crohn's disease and infections such as tuberculosis. The article by Harvey JN<sup>4</sup> proposes food fortification with vitamin D to reduce the incidence of T1DM. In autoimmune conditions, it is challenging to conclude causality given the observational nature of the majority of studies and also the widespread prevalence of vitamin D deficiency globally. Due to the practical difficulties in excluding the influence of confounding environmental factors on disease incidence, the results of most studies can only be speculative at best. Through monozygotic twin studies we understand that environmental factors play a key role in the pathogenesis of T1DM. Childhood obesity, seasonal infections, enterovirus exposure, gut microbiome and vaccination programmes are some of the factors that have been considered to

influence the incidence of T1DM. Studies evaluating vitamin D receptor polymorphisms in T1DM have been small and heterogeneous, thereby providing conflicting results.<sup>5</sup> Moreover, ethnic minority groups, who are disproportionately affected by vitamin D deficiency, are often under-represented in these studies.

Whether optimising 25OHD levels beyond those essential for bone health through vitamin D supplementation and fortification protects against autoimmune disease onset or supports its treatment is yet to be elucidated. Clarifying the specific role of vitamin D in prevention or treatment of autoimmune diseases would require prospective randomised clinical trials which poses several logistic challenges.

Despite increased numbers of cases of nutritional rickets and growing evidence that vitamin D deficiency is a major public health problem in the UK, we continue to have a conservative threshold for 25OHD adequacy in comparison to other countries.<sup>6</sup> Given the general reluctance to adopt mandatory or systematic food fortification of vitamin D in the UK to achieve optimum bone health,<sup>6</sup> proposing the adoption of fortification to achieve a higher threshold for non-bone health benefits is currently improbable. Nonetheless, food fortification is the most economically feasible way forward to tackle the vitamin D deficiency crisis in the UK whilst concurrently reducing health inequalities.<sup>7</sup>

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