# Vitamin D status in Africa is worse than in other continents

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Serum concentration of 25-hydroxyvitamin D (25[OH]D) is the best marker for nutritional vitamin D status. Although no consensus has been reached on the optimal 25(OH)D concentration, all guidelines agree that serum 25(OH)D concentrations of less than 30 nmol/L (12 ng/mL) should be avoided in all age groups.[1] Several reviews of vitamin D status have concluded that vitamin D deficiency is highly prevalent around the world, yet Africa is considered to be largely spared from severe vitamin D deficiency.[2,3] A global review of serum 25(OH)D concentrations found that concentrations of less than 30 nmol/L are present in around 7% of the global population.[2] Severe vitamin D deficiency affects around 5% of the US population (based on a representative sample from the National Health and Nutrition Examination Survey 2011–14), 14% of individuals in Europe (based on 11 randomised controlled trials), and a higher percentage of people living in the Middle East or Gulf states.[4] Severe vitamin D deficiency has also been observed in Northern India and China.[5] In *The Lancet Global Health*, Reagan M Mogire and colleagues[6] describe the vitamin D status of 21 474 individuals living in 23 African countries. Severe vitamin D deficiency (<30 nmol/L 25[OH]D) was found in about 18% of these individuals, suggesting that Africa could be the continent with the highest frequency of severe vitamin D deficiency (table). This finding is in contrast with the widely held view that the extensive sunshine in Africa, the cradle of mankind, was indicative for the optimal vitamin D status of early humans.[10] The study by Mogire and colleagues[6] has some major limitations, in that it does not evaluate a representative sample of the African population, does not use accuracy-validated assays, and overrepresents North African countries and South Africa, with no data on many African countries. Nevertheless, the study does improve our global view of vitamin D deficiency.

Most guidelines consider a serum 25(OH)D concentration of less than 50 nmol/L (20 ng/mL) to represent vitamin D deficiency, because at this concentration, a compensatory mechanism is activated to maintain normal calcium homoeostasis, with implications for optimal bone health and muscle function. Several reviews have reported that 30–40% of the global population has this mild deficiency. Therefore, Africa should no longer be considered an exception.

Vitamin D and iodine are inactive but essential substrates for generating the active hormones calcitriol and triiodothyronine, which activate specific nuclear receptors and thereby regulate a large number of genes.[11] Iodine deficiency can lead to a wide range of disorders, including congenital iodine deficiency syndrome. Similarly, vitamin D deficiency can lead to rickets and many other disorders, with implications for bone and general health. The discovery of the dual origin of vitamin D (dietary intake or synthesis in the skin during exposure to sunlight), about a century ago, soon resulted in health-care strategies that aimed to eliminate endemic nutritional rickets in the so-called western world. In 1929, Alfred F Hess wrote that "within the next decade or even sooner, [rickets] will be almost completely eradicated, so that it will become as rare as infantile scurvy since the widespread use of orange juice".[12] Nutritional rickets is affecting more than one third of infants and children in Mongolia, northern China, and Northern India, and it is still prevalent in other countries.[13] Extensive surveys of rickets in Canada, UK, and New Zealand concluded that rickets was not found in infants or children who were receiving vitamin D supplements. However, children not receiving such supplements, and especially those with mothers who had dark skin and a poor vitamin D status, and those receiving prolonged breastfeeding, were at the highest risk.[11] Nutritional rickets is also increasingly recognised as a health issue in African countries, as a result of calcium deficiency, vitamin D deficiency, or both.[13]

Similar to that for iodine deficiency, we need a worldwide strategy with regional adjustments to cope

| | Geographical area | Prevalence, % | |
|---|---|---|---|
| | | Cutoff of <25 nmol/L or <30 nmol/L | Cutoff of <50 nmol/L |
| Hilger et al (2014)[2] | Global | 7% | 37% |
| Herrick et al (2019)[7] | USA | 5% | 18% |
| Cashman et al (2016)[8] | EU countries (adults) | 13% | 40% |
| Arabi et al (2010)[9] | Iran and Jordan | 50% | 90% |
| Durazo-Arvizu et al (2014)[3] | Ghana and Seychelles | <1% | <7% |
| Mogire et al (2019)[6] | African continent | 18% | 34% |
| Zhang et al (2013)[5] | China | Around 37% | Around 72% |

Cutoffs refer to serum concentrations of 25(OH)D used to define vitamin D deficiency. 25(OH)D=25-hydroxyvitamin D.

***Table:*** **Vitamin D deficiency by geographical area and serum 25(OH)D cutoff**

with poor vitamin D status. Vitamin D deficiency can be corrected by adequate vitamin D intake, but most natural foods (even breast milk from mothers living in tropical climates) have a low vitamin D content, and there is not enough oily, vitamin D-rich fish in the oceans to provide sufficient vitamin D to the world population. Greater exposure to sunlight improves vitamin D status, but the ultraviolet B (UVB) light that is needed for the photochemical synthesis of vitamin D is also carcinogenic. During the evolution of humans and other vertebrates, the carcinogenic properties of UVB light might not have been as much of a problem, because the mean life expectancy was 50 years or less, and UVB-induced DNA damage tends to have latent effects. Nowadays, UVB irradiation increases the risk of skin cancer in older adults and the elderly population. Therefore, vitamin D supplementation is the only practical and safe solution to improving vitamin D status. The practical implementation vitamin D supplementation might vary between countries and for specific risk groups. Potential strategies include vitamin D supplementation of food (as successfully implemented in Finland), or vitamin D supplementation of infants and small children (a cheap option that has been in place in many high-income countries [eg, the USA, Canada, Australia, New Zealand, and western European countries] for almost a century and was recently successfully implemented in Turkey). In other situations, an ad hoc strategy will be needed. Of course, the first and major efforts should be directed towards eradication of severe vitamin D deficiency in the individuals who are most at risk, such as infants, small children, and pregnant women. The other major risk groups are individuals with dark skin living in moderate climates and all people with poor exposure to sunlight due to personal, cultural, or religious reasons. WHO and UNICEF should lead a strategy to eradicate the most severe consequences of worldwide vitamin D deficiency. The lessons learned from the successful battle against iodine deficiency, such as the requirement for effective strategies tailored by country and regular monitoring of deficiency status, can guide WHO, governmental organisations, and clinicians.

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