REVIEW

Vitamin D deficiency in British South Asians, a persistent but avoidable problem associated with many health risks (including rickets, T2DM, CVD, COVID-19 and pregnancy complications): the case for correcting this deficiency

Barbara J Boucher

The Blizard Institute, Queen Mary University of London, London, UK

Correspondence should be addressed to B J Boucher: bboucher@doctors.org.uk

Abstract

High vitamin D deficiency rates, with rickets and osteomalacia, have been common in South Asians (SAs) arriving in Britain since the 1950s with preventable infant deaths from hypocalcaemic status-epilepticus and cardiomyopathy. Vitamin D deficiency increases common SA disorders (type 2 diabetes and cardiovascular disease), recent trials and non-linear Mendelian randomisation studies having shown deficiency to be causal for both disorders. Ethnic minority, obesity, diabetes and social deprivation are recognised COVID-19 risk factors, but vitamin D deficiency is not, despite convincing mechanistic evidence of it. Adjusting analyses for obesity/ethnicity abolishes vitamin D deficiency in COVID-19 risk prediction, but both factors lower serum 25(OH)D specifically. Social deprivation inadequately explains increased ethnic minority COVID-19 risks. SA vitamin D deficiency remains uncorrected after 70 years, official bodies using 'education', 'assimilation' and 'diet' as 'proxies' for ethnic differences and increasing pressures to assimilate. Meanwhile, English rickets was abolished from ~1940 by free 'welfare foods' (meat, milk, eggs, cod liver oil), for all pregnant/nursing mothers and young children (<5 years old). Cod liver oil was withdrawn from antenatal clinics in 1994 (for excessive vitamin A teratogenicity), without alternative provision. The take-up of the 2006 'Healthy-Start' scheme of food-vouchers for low-income families with young children (<3 years old) has been poor, being inaccessible and poorly publicised. COVID-19 pandemic advice for UK adults in 'lockdown' was '400 IU vitamin D/day', inadequate for correcting the deficiency seen winter/summer at 17.5%/5.9% in White, 38.5%/30% in Black and 57.2%/50.8% in SA people in representative UK Biobank subjects when recruited ~14 years ago and remaining similar in 2018. Vitamin D inadequacy worsens many non-skeletal health risks. Not providing vitamin D for preventing SA rickets and osteomalacia continues to be unacceptable, as deficiency-related health risks increase ethnic health disparities, while abolishing vitamin D deficiency would be easier and more cost-effective than correcting any other factor worsening ethnic minority health in Britain.

Key Words

- vitamin D deficiency
- UK South Asians
- long-standing
- correction
- justification

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A narrative review of vitamin D deficiency in South Asians in the UK

Long-standing racial inequalities in the UK are common and recognised as reducing both access to health care and the quality of health, a recent report describing them as affecting ethnic minorities in the UK (Feb 2022) 'throughout the life course from birth to death' and as being 'rooted in experiences of structural, institutional and interpersonal racism' (1; https://www.nhsrho.org/wp-content/uploads/2022/02/RHO-Rapid-Review-Final-Report_v.7.pdf). These problems have been well-known for over 50 years (2). The increased risks of severe illness and death in ethnic minority groups during the COVID-19 pandemic have highlighted these problems and greatly increased public awareness of this inequitable situation which has also been discussed in both houses of parliament. The resultant UK report on these problems, examining five strategic areas, identified significant barriers to treatment arising from multiple problems including poorer education, deficiencies in health literacy and communication, greater poverty, and discriminatory interactions deterring people from seeking healthcare (1; https://www.nhsrho.org/wp-content/uploads/2022/02/RHO-Rapid-Review-Final-Report_v.7.pdf). These appear to be the same problems that, for vitamin D deficiency, it was suggested should be overcome by 'education' and 'assimilation' almost 70 years ago (2). A specific factor unavoidably aggravating South Asian (SA) vitamin D deficiency is increased skin pigmentation (3), aggravated by the use of facial coverings for socio-religious reasons by many SA women. The failure to correct vitamin D deficiency in children early on was particularly regrettable since rickets had been eradicated after its return in England between the two world wars by the provision of five extra welfare foods, including cod liver oil, from early in World War II (https://discovery.nationalarchives.gov.uk/details/r/C10953) (4). These foods were provided for all expectant and nursing mothers and their children up to the age of 5 (extra milk, eggs, meat, orange juice (later rose- hip syrup) and cod liver oil), together providing extra protein, vitamin C and vitamin D. That programme was well publicised, widely available and paralleled by increased awareness of the benefits of outdoor fresh air and sunshine for babies and young children. Thus, nutritional rickets virtually disappeared in Britain during and after World War II as it had elsewhere with the use of cod liver oil (4). Pandemic lockdown led to advice to all adults in the UK to take the 'recommended daily amounts' of vitamin D, 400 IU/day, all year round (https://www.nutritionsociety.org/policy/public-health-england-publishes-new-advice-vitamin-d) in 2020, though this intake is too low to correct all deficiency (5). Deficiency in the UK has run at significant levels for decades, for example, summer/winter at 17.5%/5.9% in White Britons, 38.5%/30.8% in Black Britons and 57.2%/50.8% in SAs, respectively, in representative UK biobank subjects at recruitment over 14 years ago and at similar levels more recently (6, 7). Furthermore, only 5% of the UK Biobank SA volunteers had baseline 25(OH)D values ≥ 50 nmol/L, a level used to define repletion in the USA and across Europe, at recruitment (6, 7).

As rickets and adult osteomalacia in women emerged in SAs resident in post-war Britain, many studies were made of the problem in specialist centres developing research technology for assessment of the adequacy of an individual's vitamin D 'status', first by bioassays of circulating 'vitamin D' and later through measurement of serum concentrations of serum 25(OH)D by immunoassay and then by mass spectroscopic methodology (8, 9).

25(OH)-vitamin D is produced predominantly by the liver by hydroxylation of cholecalciferol in the carbon 25 position. 25(OH)D remains in the circulation bound to various D binding proteins and to albumin with a clearance half-time of ~2 weeks (10). Early studies provided information able to inform official bodies about vitamin D deficiency in rickets and osteomalacia and its increased prevalence in SA communities, but this information was not followed by effective measures to reduce the problem. Prof O'Riordan and colleagues working in cities such as London and Glasgow did, however, advise fortification of chapatti flour, widely used by SA communities, for correction of their D deficiency since this had been shown to be more effective than the provision of daily supplements (11, 12), but this was never acted upon. Successive official bodies and their advisors on public health and on nutrition must have known the scale of the problem over those years but usually continued to advise correction of the problem by 'education' and 'assimilation' of immigrant communities into the 'English way of life' (2). Those bodies did advise issuing pamphlets in several SA languages, but the lack of literacy in many immigrants, most notably amongst women from Bangladesh who usually prepared meals, reduced their value. Infant feeding in east London was still reported as inadequately nutritious in 2020 (13). The hesitancy of official bodies to provide supplemental vitamin D over those early decades was probably aggravated by the fear of precipitating infantile hypercalcaemia, which had been a health scare in the mid-1950s when many hospital admissions were noted of babies with symptomatic hypercalcaemia, corrected quite easily by extra fluids

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and restricted vitamin D intakes. This problem probably resulted from the use of the many vitamin D-fortified infant milk and pre-prepared infant feeds available, almost all of which were also fortified with vitamin D and whose contents have since been modified (14). There was no suggestion of any manufacturing error that could have caused unduly high vitamin D intakes at that time. Many workers felt this 'outbreak' was due to unrecognised cases of Williams syndrome, and genetic causes do present this way. However, for the several admissions every week to the children's wards seen during a personal 3-month attachment to the paediatric department of an east London teaching hospital as a medical student in the late 1950s, this condition (incidence up to 20/10,000 births) (15, 16) could not have been the whole explanation. This unfortunate outbreak of infantile hypercalcaemia in Britain was largely abolished by the reduction of an excessive fortification of infant foodstuffs, but official bodies advising on public health and nutrition have still not provided effective measures for correcting vitamin D deficiency in the UKs SA communities. The welfare food scheme did continue to offer supplemental cod liver oil from 1941 to 1961 when it was discontinued (17) (https://discovery.nationalarchives.gov.uk/details/r/C10953). Cod liver oil was still offered in antenatal clinics until late 1994 when it was discontinued (due to the teratogenicity of its excessive vitamin A content (18)), without alternative provision of vitamin D.

A new scheme, the 'Healthy-Start' began in 2006 and aimed to provide vouchers for buying extra food to low-income families with children under 3 years. This was not, therefore, available to all as it had to be applied for and signed up by a health professional. Overall uptake was poor and tended to fall over time as it was difficult to access and not well publicised. Uptake was much higher when these vouchers could be offered locally to all eligible women (19, 20). Take up of such benefits was also improved by improved literacy in American Hispanics (21), but no such schemes were provided in the UK. A review of the Healthy-Start scheme in 2015 showed poor uptake, suggesting that supplementation, or the scheme, should be available to all families with children up 5 years (22, 23). 'Universal' rather than targeted provision of vitamin D from the Healthy-Start scheme increased uptake rates by 17% in one local scheme, leading to reductions in the incidence of rickets in children under 5 years in the North of England by 59% (incidence rates falling from 120/100,000 to 49/100,000) (21). Currently, information on the benefits of vitamin D in pregnancy and childhood in the UK is provided by the NHS online (https://www.nhs.uk/conditions/vitamins-and-minerals/vitamin-d/) There is also sporadic reporting on the need for, and benefits of, taking vitamin D in many 'women's' magazine articles and in newspapers (e.g. https://www.thetimes.co.uk/article/vitamin-d-are-you-sure-youre-getting-enough-0hmqjxdjq).

Such guidance, however, relies on people seeing it, being able to read and being able to afford the various supplements suggested. Furthermore, many Sylheti-speaking Bangladeshi immigrants remained unable to read Bengali or English for some time so the NHS pamphlets produced in various languages were not always useful (https://www.nhs.uk/about-us/health-information-in-other-languages/).

Maternal vitamin D deficiency increases the risk of gestational diabetes, toxaemia of pregnancy and of small-for-dates births, all of which pose added risks to both mother and baby (24, 25). It also increases the risk of stillbirth which remains more common in SA women than other British women (26). Furthermore, the deficiency increases offspring obesity risks which persist into later life through recognised epigenetic effects *in utero* (27, 28). Thus, ensuring repletion in all women of childbearing age would help to reduce later health risks, and by reducing later health care costs, should prove to be a cost-effective measure. This proposition is supported by the finding of increased cardiovascular disease (CVD) risk markers and increased insulin resistance in offspring that relate directly to the severity of maternal deficiency in Indian children in the Mysore Parthenon study aged 9.5 years while increases in HDL-cholesterol and reductions in fasting insulin in adolescence were found with increased childhood 25(OH)D in the British ALSPAC study (29, 30).

British SA communities have higher age-adjusted mortality rates than indigenous Britons, aggravated by their increased rates of obesity, metabolic syndrome, type 2 diabetes and cardiovascular disease (31, 32). Obesity increases the rates and severity of vitamin D deficiency through well-recognised mechanisms. Diffusion of vitamin D and 25(OH)D through the enlarged fat mass 'dilutes' serum 25(OH)D, but, in addition, hepatic 25-hydoxylation is reduced in obesity (33, 34). Hepatic 25-hydroxylation of vitamin D is also suppressed in diabetes by the PPARγ-coactivator-1α (PGC1α) enzyme which becomes up-regulated in diabetes (33), no doubt being aggravated by the fatty infiltration of the liver commonly seen in T2DM (34). These phenomena have usually been dismissed as 'reverse confounding', but, in fact, they must increase deficiency-related health risks since lowering serum 25(OH)D levels reduces the availability of 25(OH)D to target tissues which are known to depend on circulating 25(OH)D as a substrate for activation to form calcitriol *in*

situ (35). Additional support for the importance of this mechanistic evidence comes from the fact that increased intakes are needed in overweight/obese subjects (by ×1.5 to ×3 or ×4-fold) as compared to the intakes needed by slim people to achieve comparable serum 25(OH)D levels (36). Emerging data also suggest the need for increased vitamin D intakes in disorders of glucose homoeostasis. For example, normoglycaemic SA women with increased insulin resistance needed to achieve serum 25(OH)D values of at least 80 nmol/L to normalise their insulin resistance (37). Also, in prediabetes with early hyperglycaemia, it took achieved 25(OH)D values of 100 nmol/L in the D2d trial for insulin resistance to be reduced (38), though 25(OH)D values of 25 nmol/L are thought to be adequate to prevent rickets by the UK's Scientific Advisory Committee on Nutrition SACN (39). 25(OH)D levels ≥ 100 nmol/L in the D2d trial also reduced the risks of later T2DM by up to 70% though it required daily doses of 4000 IU/day to achieve these effects, not seen on 3200 IU/day (38). If confirmed in further population groups, these findings could lead to the transformation of T2DM risks in the SA population groups that suffer high rates of obesity, prediabetes and vitamin D inadequacy. The suggestion of causality from the aforementioned data is supported by the fact that vitamin D is necessary for normal islet beta cell function (40, 41, 42). Also, American adults with diabetes in the NHANES 111 and NHANES 2001–2014 cohorts showed significant reductions in HOMA-IR, HbA1c, blood lipids and C-reactive protein (CRP) with baseline 25(OH)D values > 75 nmol/L vs <25 nmol/L (Ptrend < 0.05) (43). That study also reported significant reductions, prospectively, in age-adjusted mortality rates overall (by −34%), in cardiovascular mortality rates (by −31%) and in cancer mortality rates (by −51%) in subjects whose baseline 25(OH)D values had been >75 nmol/L vs <25 nmol/L (43).

The inverse relationships between vitamin D status, metabolic syndrome and its components are well-known (44, 45, 46). A report of seven studies meeting specific inclusion criteria for one recent meta-analysis found that low vitamin status was significantly associated with metabolic syndrome as a whole. Four of the five studies that allowed examination of individual components of MetS with vitamin D status showed inverse associations of obesity, BMI, dyslipidaemia, blood pressure, insulin resistance and glycaemia with vitamin D status, and review of seven recent randomised controlled trials of vitamin D supplementation in metabolic syndrome found significant benefits on blood pressure, abdominal adiposity and all aspects of insulin and glucose metabolism, but not on other features (47). The severity of non-alcoholic fatty liver disease, common in diabetes and obesity, relates inversely to vitamin D status, no doubt because both obesity and liver damage will reduce 25-hydroxylation of vitamin D (48, 49, 50). As in any disorder reducing 25(OH)D production, this increases the risks of adverse effects on target tissues in general by reducing substrate availability for *in situ* calcitriol production. Recently, extra-hepatic health benefits were reported following vitamin D supplementation in non alcoholic fatty disease of the liver (NAFDL) (improvements in HDL-cholesterol, body weight, BMI, alanine aminotransferase (ALT), insulin resistance and fasting glycaemia), the authors suggesting that vitamin D supplementation should be included in the management of patients with NAFDL (51).

Vitamin D status and COVID-19 illness

Increased severity and mortality of COVID-19 infections have been reported in Black, Asian and Ethnic minority (BAME) communities, including SAs, throughout the current pandemic (52). Correcting vitamin D deficiency by supplementation with vitamin D3 has been found to produce modest reductions in upper respiratory tract infections, the number of exacerbations of asthma in children and chronic pulmonary disease in adults (53). The possibility that current rates of vitamin D deficiency generally and the increased rates of deficiency in BAME, and in SA communities in particular, might be worsening COVID-19 infection rates and the severity of illness has been considered from the start of this epidemic (54). Infection rates, the need for intensive care and mortality rates due to COVID-19 infections have been used to judge COVID-19 severity clinically, and the increased severity and higher death rates amongst ethnic minorities have been painfully obvious globally throughout the pandemic (55, 56). The high death rates in BAME communities have been blamed on social deprivation with overcrowding and inability to self-isolate, plus discriminatory practices against such communities, together with the increased rates of obesity and T2DM that these groups suffer (57). However, even with the inclusion of measures of social deprivation as a factor increasing COVID-19 risks, it has not been possible to account for all of the increased COVID-19 risks in ethnic minorities while vitamin D status has either been excluded from analysis or eliminated in multiple regression analyses by adjustment for ethnicity, obesity and diabetes, as would be expected since those factors cause D deficiency (58). Furthermore, that observation is supported by the fact that increased COVID-19 risks are not only common amongst ethnic minority healthcare workers but have been common

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among senior medical staff who are unlikely to have been in poverty; indeed 28 of the 33 deaths from COVID-19 amongst senior medical staff being remembered in the *BMJ* recently were of ethnic minority origin (https://www.bmj.com/COVID-memorial). Thus, there are likely to be some unaccounted-for factors causing increased COVID-19 disease severity in Britons of BAME origin. The suggestion that vitamin D deficiency is one such factor is becoming increasingly credible on mechanistic grounds. In brief, it is well established that lack of vitamin D reduces the ability of the immune system to modulate the over-production of pro-inflammatory cytokines and underproduction of anti-inflammatory cytokines commonly seen in severe infections, including COVID-19 illness, that increase the risks of cytokine storms and the risk of developing the acute respiratory distress syndrome (ARDS). Abolishing vitamin D deficiency should reduce these risks since vitamin D specifically reduces the secretion of pro-inflammatory cytokines and increases the secretion of anti-inflammatory cytokines (59, 60). Additionally, vitamin D promotes secretion of the defensins, and of cathelicidin, which are microbiocidal agents protective against infection (60). Reduced pulmonary production of the angiotensin ll receptor (ANG2R) in ARDS is associated with poor outcomes, but its secretion is increased specifically in the lungs by vitamin D while increased pulmonary ANG2R secretion is associated with the reduction in the severity and risks of ARDS experimentally (61).

The use of vitamin D_3 in COVID-19 illness has had little success in treatment, but it has usually been given in ever-increasing bolus doses (62, 63), and large bolus doses are known to induce self-regulatory effects that reduce calcitriol formation and that persist for at least 3 months (64). These effects include the reduction of hepatic 25 hydroxylation of vitamin D and the stimulation of the secretion of fibroblast growth factor-23 which is known to inhibit the action of the 1-alpha-hydroxylase enzyme that activates 25(OH)D to form calcitriol in both renal and non-renal target tissues (3, 35). That the self-regulatory effects of large bolus dosing can have biological consequences is demonstrated by their failure to prevent rickets in deficient children (64).

In contrast, treatment with 25(OH)D [calcifediol) does not induce adverse self-regulatory effects (64), and there are now several observational reports of its use in COVID-19 showing reductions in the need for intensive care and in mortality (65). In line with what is known about the protective effects of vitamin D against infection, reduced rates of COVID-19 illness are reported with higher pre-pandemic serum 25(OH)D levels in almost 200,000 people across all 50 states of the USA (66), the risk reductions plateauing at 25(OH)D values of ~100 nmol/L and similar risk reductions are reported from elsewhere (67, 68). Furthermore, UK-Biobank Data report reductions in COVID-19 illness rates in those reporting taking vitamin D supplements at recruitment but not in those taking any other types of vitamin supplements (69).

Vaccination against various infections has been suggested to induce higher antibody titres in subjects with higher vitamin D status, a suggestion supported by a trial showing greater antibody responses with correction of deficiency by supplementation, with immunisation using a standard antigen (70). A recent study has also reported higher antibody titres in response to one of the newer COVID-19 vaccines in those with better initial vitamin D status (71).

As already mentioned, CVD rates, risks and mortality are higher in SAs than in other Britons. Atherosclerosis progression is significantly driven by inflammation, including the remote inflammatory effects of obesity (72, 73). Arterial plaque instability leads to acute events and results largely from the release by plaque infiltrating macrophages of the matrix metalloproteinase enzymes, MMP 2/9, that destroy the plaque matrix (74). Secretion of MMP 2/9 is also a feature of severe lung damage in COVID-19 illness (73, 74, 75). It is, therefore, of relevance that inflammation is reduced by vitamin D, that macrophage secretion of MMP 2/9 is reduced by vitamin D and that circulating MMP 2/9 levels relate inversely to serum 25(OH)D in deficiency and have been noted to fall dramatically with supplementation in an observational study (76, 77). One recent trial reports reductions in CVD risk with correction of deficiency as already mentioned, and a recent non-linear Mendelian randomisation (MR) analysis has shown dose-wise reductions in CVD risk with genetic variants that increase 25(OH)D values in subjects whose measured 25(OH)D values were below 50 nmol/L but not in those with higher 25(OH)D values (78). CVD risks are increased by hypertension. It is also of interest, therefore, that renin secretion is suppressed by vitamin D (79). Reductions in blood pressure and risks of hypertension are reported with higher genetic predictions of 25(OH)D by MR analysis (80). Overall, therefore, it could be expected that avoidance of vitamin D deficiency and ensuring 25(OH)D concentrations over time of at least 50 nmol/L should gradually reduce the risks of CVD, including acute events. This would be of especial benefit to minority groups in the UK, as it would in any vitamin D-deficient population group with a high mortality rate from CVD events.

Another risk factor specific to SA Communities that increases health risks such as T2DM, CVD, obesity and metabolic syndrome dose-wise over time is betel-chewing (81, 82, 83). Dose-wise increases in intracellular peripheral blood mononuclear cell production of the 24-hydroxylase catabolic for calcitriol are seen along with dose-wise reductions in both intracellular and serum calcitriol in relation to increasing betel-quid usage, a phenomenon that may aggravate the adverse effects of inadequate vitamin D provision (84). Furthermore, metabolic syndrome, T2DM and CVD risks are transmitted by betel-chewing fathers to their children, though whether this applies to women chewers and whether it is reversible across generations is unknown (82, 83).

Discussion

From available data, it can be expected that abolishing the common UK problem of vitamin D deficiency would improve both skeletal and non-skeletal health, and do so most markedly in those with the highest deficiency rates, namely SA and other ethnic minority groups, and improve their COVID-19 survival as it does in older people supplemented pre-infection (85, 86).

Furthermore, if the tobacco commonly added to betel quid chews in the UK and elsewhere adds to the risks of the non-cancerous disorders mentioned earlier in the same way that smoking does, independently, in Taiwan (where tobacco is not added to betel chews (83)), this will add to the importance of reducing betel-chewing, already targeted for reducing the high oro-pharyngeal cancer risks seen in betel-chewers, though this is difficult as the habit is highly addictive (87). The risks of metabolic syndrome, T2DM and CVD fall over time after betel-cessation in the same way that lung cancer rates fall after smoking-cessation (88). Community-based programmes in schools, religious centres and workplaces have reduced betel-chewing rates prospectively in other countries and could prove valuable in reducing ethnic health disparities in the UK (89, 90).

A recent secondary analysis from VITAL trial data reports no benefit of supplementation for incident fractures in those over 60 years. An accompanying comment states that the VITAL study showed 'no important health benefits from supplementation in the general population of older adults', even 'when initially deficient' (91, 92). However, valid sub-group analyses of VITAL data for supplementing at 2000 IU/day have shown significant reductions, by ~20%, in several disorders. Those reductions included incident cancer in those with healthy weights (BMIs <25 kg/m^2), probably because increasing BMI increases the vitamin D intakes needed to correct deficiency (×1–4 fold) (36, 93). Significant falls in autoimmune disease were found overall, and a significant reduction in cancer mortality, by up to ~40%, from the 3rd year of the study onwards (94, 95). Thus, a general condemnation of vitamin D supplementation from VITAL results is not justified. Indeed, priority was called for in following up on the effect on cancer mortality in a report on findings from VITAL data (93).

Furthermore, the possibility of being able to detect health benefits in the VITAL trial was reduced, even in deficient subjects, since volunteers were told at recruitment that they could take personal vitamin D supplements at up to 800 IU/day. This feature makes the lack of assessment of vitamin D status at the end of the study especially regrettable since achieved vitamin D status has emerged as an important determinant of many health outcomes (96). For example, serum 25(OH)D values of ≤100 nmol/L reduced T2DM risk in subjects with pre-diabetes significantly (by up to 70%) in the D2d study, where taking 4000 IU/day achieved such levels but not in those taking 3200 IU/day, higher intakes being needed in dysglycaemia, as in obesity (33, 38).

As discussed already, many non-skeletal health benefits have now been reported from randomised controlled trial (RCT) data analyses with varying 25(OH)D threshold values (97). Many health benefits of better D status have been confirmed by MR analyses, especially non-linear MR analyses where benefits shown include reductions in CRP and in the risks of CVD, hypertension, T2DM and dementia. Such benefits were, however, limited to subjects with deficient 25(OH)D values, supporting the desirability of avoiding deficiency at the population level (78, 80, 98, 99, 100). Recent trial data haves reported reduced COVID-19 risks following supplementation (101). To suggest, therefore, as the recent NEJM reports do (91, 92) that the general use of vitamin D supplements in adult populations should stop on the basis of the negative findings from the VITAL study with its design problems is likely to prove irresponsible; especially since deficiency remains common in virtually all populations. This view is supported by the many significant findings of significant health benefits from better vitamin D status now emerging from trials and from MR analyses as already mentioned.

Overall, since higher vitamin D status from supplementation or through genetic variation reduces many non-skeletal health risks and since deficiency remains common globally, it is clear that avoiding deficiency has the potential to improve public health. Modern lifestyles

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continue to reduce exposure to sunlight. Thus, long-term avoidance of vitamin D deficiency and ensuring 25(OH)D values of 50 nmol/L or above is increasingly likely to improve UK health and to reduce health disparities currently of concern in British ethnic minorities as they would in any other highly deficient populations. Ensuring vitamin D repletion through food fortification, possibly of flour(s), plus targeted supplementation of those with high risks of deficiency) (well-known factors linked to high risks of vitamin D deficiency in adults: obesity, minority ethnicity, dark skin, vegetarianism, veganism, diabetes, older age, indoor or night shift work, pregnancy, breast-feeding, infancy, wearing covered up clothing, being house-bound or in residential care) has been suggested as a cost-effective measure for the UK (102). However, higher than average supplemental provision will be necessary where higher status or intakes are required for achieving health benefits, (as in obesity and diabetes) (36, 38). Furthermore, reducing vitamin D deficiency rates would be easier and more cost-effective than correcting any of the socio-economic factors recently identified as increasing UK ethnic minority health disparities (1) or reducing the rates of betel-chewing, though these measures are also highly desirable.

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