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Food-based strategies for prevention of vitamin D deficiency as informed by vitamin D dietary guidelines, and consideration of minimal-risk UVB radiation exposure in future guidelines

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There is widespread acknowledgement of the presence of vitamin D deficiency in the community and the pressing need to address this. From a public health perspective, emphasis has been placed on addressing vitamin D deficiency through dietary means. However, naturally rich food sources of vitamin D are few and infrequently consumed, and nutrition survey data from various countries have indicated that habitual vitamin D intakes in the community are much lower than the current vitamin D dietary guidelines. This review will briefly overview the extent of vitamin D deficiency within the community, its causes, and how our food chain, once its embraces the evidence-based practise of food fortification and potentially bio-fortification, can cater for meeting the dietary vitamin D needs of the community. Finally, international authorities, briefed with establishing vitamin D dietary guidelines over the past decade, have struggled with uncertainties and gaps in our understanding of the relative contribution of sunshine and diet to vitamin D status and vitamin D requirements for health maintenance. The review will also consider how emerging evidence of a possible minimal-risk UVB radiation exposure relative to skin cancer that also enables vitamin D production could greatly inform future vitamin D dietary guidelines.

1. Introduction

While there are many areas of controversy and debate within the vitamin D field, some points are undisputed. For example, vitamin D is undoubtedly an essential nutrient for the human body[1] and, worryingly, there is widespread acknowledgement of the presence of vitamin D deficiency in the community.[2] There is a pressing need to tackle this deficiency and it requires implementation of corrective and preventive public policy.[3] Estimates of dietary requirements for vitamin D are crucial from a public health perspective in providing a framework for prevention of vitamin D deficiency and optimizing vitamin D status of individuals.[4] In recognition of these points, over the last 10 years there has been re-evaluations of vitamin D recommendations by at least six expert bodies within the Northern hemisphere and these have established intake requirements between 10 and 20 μg d^{-1}, reviewed in detail elsewhere.[5–7] Habitual vitamin D intakes by many within the community are below these estimates of intake requirement,[1,8,9] which contribute to increased risk of vitamin D deficiency when ultraviolet B (UVB)-induced synthesis in the skin is limited. This clearly points towards the necessity for creative, and sustainable, food-based solutions to bridge the gap between current dietary intakes and recommendations. This review will briefly overview the extent of vitamin D deficiency within the community, its causes, and how our food chain (current and future) can cater for meeting the dietary vitamin D needs of the community, especially if components of it are modernised in relation to fortification with vitamin D. Lastly, as most of this decade's vitamin D recommendations were established in the context of presumed absence or markedly diminished dermal production of vitamin D, the review will consider how emerging evidence of a possible minimal-risk UVB radiation exposure relative to skin cancer that also enables vitamin D production could greatly inform future vitamin D dietary guidelines.

2. Extent of vitamin D deficiency within the community

While universal agreement on the definition of vitamin D deficiency is yet to be reached, the majority of expert bodies have suggested serum 25(OH)D concentrations below 25 or 30 nmol L^{-1} are indicative of increased risk of clinical vitamin D deficiency as it relates to nutritional rickets and

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osteomalacia.[1,10–15] Estimates of the prevalence of vitamin D deficiency using serum 25(OH)D < 30 nmol L^{-1} in representative population samples in the US (n = 16 180),[16] Canada (n = 11 336)[17] and Europe (n = 55 844)[18] have been reported as 5.0%, 7.4% and 13%, respectively. It should be stressed that these prevalence data, based on average yearly population-wide estimates, do not capture the differences by ethnicity in these regions, which can be significant. For example, across ethnic groups in the *National Health and Nutrition Examination Survey* (NHANES) 2011–2014 in the US, the prevalence of serum 25(OH)D < 30 nmol L^{-1} in non-Hispanic white, Hispanic, non-Hispanic Asian and non-Hispanic blacks have been recently reported as 2.1%, 5.9%, 7.6% and 17.5%, respectively.[16] A recent analysis of ethnic differences within the *Canadian Health Measures Survey* Cycles 1–3 showed that the prevalence of serum 25(OH)D < 30 nmol L^{-1} was 6% in white *versus* 20% in non-white Canadians.[19] Dark-skinned ethnic groups within Europe are also worryingly at much increased risk of vitamin D deficiency compared to their white counterparts (prevalence < 30 nmol L^{-1} in the range 28–65%, depending on the country and the ethnic group).[18,20]

3. Why is vitamin D deficiency prevalent within the community?

There is no one single underlying reason for vitamin D deficiency, but the combination of low UVB availability and/or exposure coupled with a low dietary vitamin D supply are of key importance.[21] The major source of vitamin D in humans is *via* the action of UVB radiation (290–315 nm) in sunshine on skin,[1] with estimates of synthesis in the skin providing 80–100% of the vitamin D requirements of the body.[22] However, there are several environmental factors that impede year-round synthesis, such as season, latitude and prevailing weather conditions, which determine the availability of UVB to stimulate the conversion of 7-dehydrocholesterol in the skin to pre-vitamin D_3.[21] This vitamin D-effective UVB availability was recently modelled for nine European countries/regions using a validated UV irradiance model.[23] The results showed that UVB availability decreased with increasing latitude (from 35°N to 69°N), while all locations exhibited significant seasonal variation in UVB. The number of months in which UVB availability was too low to allow for synthesis of vitamin D in the skin, referred to as the "vitamin D winter", was estimated to range from being largely absent in the very south of Europe to lasting for as long as 7 or 8 months in Northern Europe.[23] Beyond vitamin D-effective UVB availability, personal characteristics, such as skin pigmentation, age, attire, sunscreen usage, working environment, outdoor physical activity and sun exposure behaviour, can also prevent or impede vitamin D synthesis.[1,24] For example, melanin in skin reduces the penetration of UVB,[25] and thus contributes, at least in part, to lower vitamin D status in dark-skinned individuals, as mentioned above.[16,18–20] Dermal synthesis of vitamin D is less efficient in older than in younger adults.[26]

In the absence of sufficient UVB availability/exposure to enable synthesis in the skin, dietary supply of vitamin D is critical to meeting population requirements and prevention of vitamin D deficiency.[21] However, national nutrition surveys in Europe and the US highlight how mean population intake of vitamin D is in the range 3–8 μg d^{-1}, depending on the country.[8] There are also ethnic-related differences in vitamin D intake within some of these populations. As an example, while data from the National Diet and Nutrition Survey [NDNS] rolling programme in the UK suggest median vitamin D intakes of 3 to 4 μg d^{-1} for adults and older adults (86.3% white),[27] of note, data from the UK Biobank cohort (with 8024 South Asians, aged 40–69 years) suggest a median vitamin D intake of only 1.0, 1.5, 3.0 μg d^{-1} for Indian, Pakistani and Bangladeshi, respectively.[28] This lower intake coupled with skin pigmentation contributes to the higher prevalence of serum 25(OH)D < 30 nmol L^{-1} among Asian participants (59.6%; n = 52) compared with 19.6% in white participants (n = 1359) within the NDNS.[18]

From a public health perspective, the percentage of the population with a habitual daily nutrient intake lower than the Estimated Average Requirement (EAR) is taken as an estimate of the percentage of the population with probable inadequate intakes.[29] Thus, there is a significant gap between the aforementioned typical intakes and the EAR for vitamin D of 10 μg d^{-1} and, on this basis, the vast majority of individuals in North America and Europe, with the exception of some Nordic countries, have inadequate intakes of vitamin D.[9,30–34] While often referred to as a possible means of closing this gap, relying on supplements as an appropriate public health strategy to increase intakes across the population has intrinsic limitations because supplements are only effective in those who consume them, and their uptake across the population is usually lower than ~40%, on average.[34–36] There is also the potential for overdosing with vitamin D supplements. For example, the NHANES assessment of trends in supplementation shows that daily supplemental vitamin D intake of 100 μg (the current Tolerable Upper Intake Level [UL] for vitamin[1]), or more, prior to 2005–6 was less than 0.1%, but thereafter climbed to 3.2% in 2013–14.[37] Instead, emphasis has been placed on food-based approaches for addressing inadequacy of dietary vitamin D within the population.[2,5,8,38]

4. The current food supply of vitamin D and how its needs to change to cater for meeting the dietary vitamin D needs of the community

There are very few food sources rich in vitamin D. For example, out of a total of approximately 2000 food codes in the UK's food composition database,[39] expressed per 100 g of food, only 3% of foods have vitamin D contents in the range 1.5–5 μg per 100 g and a mere 1% each have vitamin D contents in the range 5–10 and >10 μg per 100 g, respectively (see

Table 1 Vitamin D content of top 5 percent of food codes with richest content within the UK food composition database[39]

Category	Food(s)[a]	Vitamin D content (μg per 100 g)
≥10 μg vitamin D per 100 g (23 food codes)	Cod liver oil	210
	Herring (salted and smoked, grilled, pickled)	10.9–25
	Cod roe	18
	Salmon (pink or red – canned in brine, smoked, wild – baked or grilled)	10.1–13.6
	Sprats	13
	Jackfish	13
	Chicken egg yolk	12.6–12.8
	Kippers (cooked, grilled)	10.1–11.1
	Chicken whole egg, dried	12.2
5–10 μg vitamin D per 100 g (27 food codes)	Salmon (wild or farmed – steamed, rock salmon, smoked, grilled)	6.8–9.8
	Kippers (flesh only grilled)	9.0
	Baking fat/margarine	5.8–8.8
	Mackerel	8.2–8.5
	Trout	7.1–8.2
	Sardines	5.1
	Breakfast cereal	8.3
	Duck eggs	5
1.5–5 μg vitamin D per 100 g (69 food codes)	Fish and fish products, various (24 codes, including fish fingers, herring roe, sushi, salmon, eel, sardines, tuna, red snapper, pangasius)	1.5–4.9
	Powdered drinks	2.1–4.5
	Meat and meat products (pork ribs, weiner schnitzel, chicken skin, beef burger, minced veal, pork fat)	1.5–2.1
	Egg and egg dishes (omelette, fu yung, scrambled eggs, fried eggs, boiled eggs, poached eggs, fried eggs)	1.8–4
	Breakfast cereal, various (9 codes)	3.9–4.7
	Fat spreads	3–4.5
	Milk (evaporated, dried)	1.5–3.1

[a] May contain the same food but cooked/processed in different ways, in which case content range is provided.

Table 1). In the case of the latter two higher content categories, fish and fish products made up 70% and 78% of the foods codes, respectively,[39] but fish are not frequently consumed by many in the population.[27,34] Even within the 1.5–5 μg per 100 g category, 35% of food codes were again fish, with eggs and egg products as well as meat and meat products making up 17% and 10%, respectively.[39] While collectively accounting for between approximately a third to two-thirds of the mean daily vitamin D intake, in quantitative terms, these three food groups still only each contributed ≤1 μg d^{-1} towards the mean vitamin D intakes of adult participants in the Irish and UK national nutrition surveys.[27,34]

While encouraging greater consumption of these relatively vitamin D-rich foods within the community would undoubtedly enhance vitamin D intakes, of note, there have been recent calls for a radical transformation of the global food systems, with emphasis on increased consumption of plant-based foods and reductions in animal-derived foods for many as part of a more sustainable flexitarian type diet.[40] It is now feasible, however, to further increase the vitamin D content of some of these animal-derived foods by use of a vitamin D-biofortification process, which at minimum could mitigate a potential decrease in population vitamin D intakes in Westernised countries which adopt such food systems transformations, if not facilitating an increase in vitamin D intake in those countries that maintain the more typical western diet. In the biofortification process, the animal-derived food produce has increased vitamin D and/or 25-hydroxyvitamin D contents by virtue of addition of vitamin D and/or 25-hydroxyvitamin D (where permissible) to the respective farmed fish, livestock, or poultry feeds.[5] Encouragingly, data from recent animal feeding trials highlight how this approach can increase the total vitamin D content of eggs, beef, pork and farmed Atlantic salmon by 1.7- to 3.6-fold, depending on the food as well as the form and dose of vitamin D added to the feedstuffs.[41–45] Furthermore, and of importance, evidence from randomized controlled trials (RCTs) with these eggs and salmon highlight their effectiveness in positively influencing serum 25(OH)D status in adults and older adults;[41,45] however, such studies with vitamin D-biofortified beef or pork have yet to be undertaken.[21]

A traditional fortification approach (*i.e.*, exogenous nutrient is added to the food in controlled amounts; whether on a mandatory or voluntary/optional basis) to increasing intakes of vitamin D within the population has been implemented by many countries.[46] The WHO–FAO have suggested that of the three strategies that can be considered for addressing micronutrient inadequacies, food fortification has potentially the widest and more sustained impact, and is generally more cost-effective than other interventions.[38] Such traditional fortification of breakfast cereals with vitamin D explains how they can contribute 5% and 13% to the mean daily intake in UK and Irish adults, respectively,[27,34] when most plant-based foods, including cereals, naturally have no or very low vitamin D content.[39] Approximately 26% of participants in the Irish nutrition survey consumed vitamin D-fortified breakfast

cereals.[34] It has been suggested that optimal food vehicles selected to deliver adequate vitamin D to the majority of the population are those that are frequently consumed population wide, affordable, and accessible.[6] Data from NHANES 2011–2014 in the US and the 2015 *Canadian Community Health Survey—Nutrition* both show that ~40% of the mean daily vitamin D intake of adults is from milk and dairy alone.[30,47] The US and Canada have a long experience of traditional fortification of food, especially of milk/dairy foods and margarine, a practice which has its origins in these countries' response to childhood rickets.[8] From a European perspective, Finland is the country with the most progressive vitamin D fortification of milks and spreads, and the beneficial impact of this on population vitamin D status has been illustrated recently.[48,49] In other European countries where fortification of milk is voluntary and the uptake is far less than in the US or Finland, the impact of vitamin D-fortified milk and dairy on adequacy of intake of the vitamin is understandably low. For example, data from national nutrition surveys in the UK and Ireland show that in general the percentage contribution that milk and dairy makes to the mean daily intake of vitamin D are low (≤7%) for those aged 11 years upwards in both populations.[27,34] It has been suggested that compared to mandatory, voluntary fortification is less likely to produce an impact on micronutrient status, as it does not create a level playing field for food producers, thereby increasing the risk that industry may focus on higher cost or niche products rather than ones aimed at higher risk consumers who have limited vitamin D intake from other sources.[50]

The problem of fortifying a single food staple, *e.g.*, milk, or focusing on a commodity sector such as dairy, is that it does not increase the vitamin D supply in non- or low consumers.[51] Thus, while acknowledging the valuable contribution fortified milk makes to vitamin D intakes among consumers, particularly in children, and the continued need for fortification of milk and other dairy products, it has been suggested that additional strategic approaches to fortification, including potentially biofortification, of a wider range of foods, have the potential to increase vitamin D intakes in the population.[8,51] In terms of diversification of food fortification, there is good evidence from both food-based RCTs and dietary data modelling exercises that fortification of flour for bread baking with vitamin D can have significant impact in terms of improving vitamin D intakes and status.[52–55] It should also be said that the safety profile of vitamin D-fortified bread and/or milk in terms of risk of intakes exceeding the UL for vitamin D in population intake data modelling exercises has been shown to be very good.[32,53] Also, as mentioned above, in light of recent calls for a more sustainable flexitarian type diet,[40] the fortification of non-animal derived foods, such as cereals, bread, edible oils, and possibly biofortification of edible mushrooms using UV irradiation, with vitamin D may be of increasing importance for the World's population.[21]

The WHO has suggested that one of the greatest challenges facing the global health community is how to take proven interventions and implement them in the real world.[56] Food fortification with vitamin D is one such example where the evidence of its effectiveness is of a very high order, and progress towards its implementation becomes key for those countries where the prevalence of serum 25(OH)D < 30 nmol L^{-1} warrant it. While there is no one-size fits all, there are general principles and guidance in terms of food fortification, particularly in the form of the FAO–WHO guidelines,[38] as well as the experience of countries like the US, Canada and Finland. Not only implementation, but subsequent evaluation of any vitamin D fortification policies considered relevant in those countries where is its deemed necessary, are of critical importance in terms of addressing vitamin D inadequacy in the community.[3]

5. Vitamin D dietary guidelines as target intakes for food-based strategies, and the potential impact of minimal-risk UVB radiation exposure on their future guidelines

Uncertainty and gaps in the available data about the relative contribution of sunshine and diet to vitamin D status and vitamin D requirements for health maintenance have presented considerable difficulty for each of the international authorities who have set new dietary recommendations for vitamin D over the last decade.[1,10–12] Consequently, each have followed an approach that prioritizes the identification of vitamin D intake values that will maintain serum 25(OH)D concentrations above chosen cut-offs when dermal production of vitamin D is absent or markedly diminished. While not focussed on establishing dietary recommendations for vitamin D, the Public Health England *Advisory Group on Non-Ionising Radiation* did comprehensively consider and report on sunlight exposure of the skin's contribution to vitamin D production and status.[57] On completion of their respective exercises, the various international authorities have highlighted vitamin D research recommendations and needs, several of which relate to sun and UVB (see Table 2).

While the need to further clarify how physiological factors (*e.g.*, skin pigmentation, genetics, age, body weight and body composition) and environmental factors (*e.g.*, sunscreen use) affect vitamin D synthesis has been stressed,[1,10,11] one key recommendation, set out by the US Institute of Medicine (IOM) in 2011, was the need to investigate whether a minimal-risk UVB radiation exposure relative to skin cancer exists that also enables vitamin D production.[1] It has been shown that high-level ultraviolet radiation (UVR) exposure conditions (such as those attained during sunbathing or ski holidays) increase not only serum 25(OH)D concentrations in white individuals but also biomarkers of epidermal DNA damage, such as urinary levels of cyclobutane pyrimidine dimers (CPD).[58,59] Of importance, since publication of the IOM report in 2011, there have also been a limited number of studies which have tried to address the balance between minimal-risk UVB exposure and

Table 2 Research recommendations related to areas of vitamin D photobiology arising from vitamin D dietary reference value revision exercises

International expert group responsible for vitamin D dietary guidelines (year of their report)	Research recommendations related to sun/ultraviolet B (UVB) and vitamin D
The US Institute of Medicine (2011)[1]	Clarify the influence of age, body weight, and body composition on serum 25(OH)D levels in response to intake/exposure
The US Institute of Medicine (2011)[1]	Investigate whether a minimal-risk UVB radiation exposure relative to skin cancer exists that also enables vitamin D production
The US Institute of Medicine (2011)[1]	Clarify how physiological factors such as skin pigmentation, genetics, age, body weight, and body composition influence vitamin D synthesis
The US Institute of Medicine (2011)[1]	Clarify how environmental factors such as sunscreen use affect vitamin D synthesis
The UK Scientific Advisory Committee on Nutrition (2016)[10]	Further research is required on the effect of aging on cutaneous vitamin D synthesis
The European Food Safety Authority (2016)[11]	There is a need for further research to study the respective impact of vitamin D dietary intake and sunlight exposure on serum 25(OH)D concentrations
The European Food Safety Authority (2016)[11]	Future studies should investigate food-based strategies to ensure adequate vitamin D intakes accounting for latitude, sunlight exposure and diet

synthesis of vitamin D in skin. These highlight how UVR exposure pattern and dose as well as skin phototype may colour conclusions drawn on the concurrent beneficial and hazardous impact of sunlight exposure.[60–62] For example, Felton *et al.* performed an experimental study to determine the dual impact of repeated simulated low-level summer sunlight exposures on vitamin D status and DNA damage/re-pair in light-skinned (phototype II, n = 10) and brown-skinned adults (phototype V, n = 6).[60] The UV exposure (1.3 standard erythema dose; 35% skin surface area exposed, 3 times a week *via* an irradiation cabinet) over 6 weeks was provided in January and February when ambient UVB is negligible at UK latitudes,[23] and was intended to approximate 13–17 minutes exposure to June midday sunlight exposure for most days of the week. Mean serum 25(OH)D concentration increased significantly from 36.5 to 54.3 nmol L^{-1} in phototype II subjects, and from 17.2 to 25.5 nmol L^{-1} in phototype V subjects, over the 6 weeks of UV exposure. In addition, the low-level UVR exposures induced CPD (as assessed using immunostaining of keratinocyte nuclei in sections of skin biopsies) in the white phototype II skin, and, to a much less extent, in the brown phototype V skin.[60] Comparison of CPD following a single UVR exposure with that after 6 weeks of repeated exposures provided no evidence for regular low-level exposures leading to DNA damage accumulation, pointing towards effective repair between exposures. Nevertheless, the CPD-positive nuclei biopsy data also showed that a substantial proportion of damaged cells were still present 24 hours post-UVR in the white skin, which led the authors to caution that potential remained for mutagenesis after each DNA-damaging event.[60]

In a dose–response study, healthy subjects of all skin types (I–VI, n = 6–20) were exposed to low sub-sunburn doses of UVR (20–80% their individual sunburn threshold dose or minimal erythema dose [MED]).[61] For context, UVR levels at 20% MED are profoundly lower than those causing visibly detectable erythema. These doses were achieved using an irradiation cabinet (35% skin surface area exposed), with a month allowed between exposures. Blood was sampled immediately before and 1 week after UVR exposure and analysed for 25(OH)D. Skin biopsies, taken unexposed as well as 15 minutes and 48 hours after UVR, were used for estimation of total CPD levels. The study showed that there was no influence of skin type on response of serum 25(OH)D or on CPD in skin biopsies, with mean UVR effects over the dose range of 1.6 nmol L^{-1} increase in serum 25(OH)D and 0.13 arbitrary unit increase in CPD for every 20% MED increment.[61] Of note, no dose could be identified at which 25(OH)D was produced without detectable DNA damage, irrespective of skin type. This is in contrast to a report of lower DNA damage in individuals with skin type II than IV given an equivalent 65% MED.[62] Assessment of CPD levels at 48 hours after UVR showed that most of the CPD evident immediately after UVR (15 min) had been repaired.[61] Also of note, while all skin types gained equivalent serum 25(OH)D and total epidermal CPD levels, people with the lightest skin (types I–III grouped) showed little CPD gradient across the epidermis, whereas those with the darkest skin (types IV–VI grouped) showed a steep gradient, with highest measured CDP levels in the superficial epidermis and virtually no detectable damage in the germinative basal layer,[61] where UVR is most likely to initiate skin cancers. This is suggestive of an increasingly favourable balance of vitamin D and DNA damage responses toward dark skin types, and in lighter skin types, even low sub-sunburn UVR levels produce DNA damage in basal cells, where carcinogenic risk is greatest, despite concurrent vitamin D synthesis. This is consistent with the much lower skin cancer incidence seen in people with darker skin types.[63]

Overall, the findings of these controlled low-dose summer sunlight equivalent exposure studies are encouraging in that they provide evidence of benefits in terms of enhancement of vitamin D status in light-skinned people which are concurrent with low-level, non-accumulating DNA damage. However, they also raise a caution in that unrepaired cutaneous DNA damage, especially at a basal cell level, were seen at 24–48 hours following even these low, sub-erythema, UVR doses.[60,61] Agreement on whether such findings support or challenge guidance on gaining vitamin D "safely" through brief sun exposures below their visible sunburn level,[64,65] is of high priority in terms of informing future vitamin D dietary guideline evaluations. For example, should it be the latter

case, then the various international authorities, at least for light-skinned people, are likely to retain their current approach that prioritizes the identification of vitamin D intake values that will maintain serum 25(OH)D concentrations above chosen cut-offs when dermal production of vitamin D is absent or markedly diminished. There is an increasing evidence-base which supports the tailoring of recommendations for different skin types both with regards to skin cancer/DNA damage and vitamin D production,[60,61,63,66] and these could be taken account of. If on the other hand, there is agreement there is a threshold UVR level, below which risk of DNA damage in epidermis is minimal in white skin, and yet is sufficient to allow meaningful synthesis, it could have very important impact on future vitamin D dietary guideline evaluations.

For example, it would allow more emphasis be placed on an approach which quantifies the sun exposure required to meet vitamin D status targets year-round and whether this can be achieved in a simply defined manner as an alternative to increasing the oral intake of vitamin D to meet dietary recommendations. Webb *et al.* recently performed calculations and modelling of the time necessary to be spent outdoors in the UK to ensure adequacy of vitamin D status (*i.e.*, serum 25(OH)D $\geq$ 25 nmol L^{-1}) year-round, without being sunburnt under the differing exposure scenarios evaluated.[67] The modelling showed that, in specified conditions, white individuals across the UK need 9 minutes of daily sunlight at lunchtime from March through to September so as to maintain serum 25(OH)D concentration $\geq$25 nmol L^{-1} during winter, when status would naturally be at its nadir.[67] This estimate assumes that forearms and lower legs (representing 35% skin surface area) are exposed unprotected from June to August, while in the remaining, cooler months only unprotected hands and face need to be exposed (representing 10% skin surface area). The modelling also found that exposing only unprotected hands and face throughout summer did not maintain serum 25(OH)D concentration $\geq$25 nmol L^{-1} during winter.[67] It should be stressed that a 25 nmol L^{-1} threshold is the minimum used by an international authority with many agencies selecting 40 or 50 nmol L^{-1} as the basis for their dietary recommendations.[1,11–13] Thus, to achieve these higher serum 25(OH)D thresholds *via* the sun exposure route only will dramatically increase the required minutes of summer sun exposure needed. It is also acknowledged by both research groups and agencies that there is a need for further research based on natural sunlight exposure to complement existing data which is based on extrapolation of UVR from artificial sources to solar UVR.[10,60]

Guzikowski *et al.*[65] in their data modelling showed how 10 minutes of near-noon sun exposure of 10% and 33% skin surface area by white young (<21 years) adults in the UK during each month of the year could be translated to vitamin D intake-equivalent estimates. These estimates allow us to see how UVR exposure under different constructs goes some, if not all, of the way towards meeting existing dietary vitamin D recommendations. Using the UK Scientific Advisory Committee on Nutrition's recommended vitamin D intake of 10 μg day^{-1} (needed to maintain 97.5% of the population with serum 25(OH)D $\geq$ 25 nmol L^{-1} (ref. 10)) as a benchmark shows that during the "vitamin D winter" in the UK (*i.e.*, October–March[23]) even exposing 33% of the whole body for that 10 minutes would only equate to an oral vitamin D intake of between 2–7 μg day^{-1} (0–2 μg day^{-1} with 10% of the whole body exposed),[65] dependent on the month. In contrast, the 10 min exposure during April to September (the sunnier part of the year) would equate to an intake in the range of 14–28 μg day^{-1} (4–8 μg day^{-1} with 10% of the whole body exposed), dependent on the month. The Guzikowski *et al.*[65] data would also predict that a 10 min exposure, but using the exposure pattern suggested by Webb *et al.*[67] above *i.e.* 35% from June to August and 10% for March–May plus September, would provide for vitamin D intake equivalent estimates in the range of 21–18 μg day^{-1} and 2–7 μg day^{-1}, respectively, dependent on the month. Thus, summer UVR exposure of unprotected skin, if agreed to be "safe", is highly effective in satisfying vitamin D needs, but is relatively ineffective during the "vitamin D winter" months at Northerly latitudes. This is why the various current dietary recommendations were established in the context of presumed absence or markedly diminished dermal production of vitamin D.

Finally, should there be agreement that it is possible to gain vitamin D "safely" through brief sub-erythemal summer sun exposure, this could still impact on dietary recommendations, even if authorities wish to continue establishing such vitamin D intake values to cover needs during the winter period when UV's contribution would be expected to be minimal. For example, similarly to the impact of brief summer sunshine exposure on winter prevalence of serum 25(OH)D concentration $\geq$25 nmol L^{-1}, as highlighted by Webb *et al.*,[67] we had shown previously that when self-reported data on summer sun exposure by older adults (>64 years) were incorporated into the modelling to establish the dietary requirements for vitamin D in winter-time; the vitamin D intakes that maintained serum 25(OH)D concentrations $\geq$25 nmol L^{-1} in 97.5% of individuals were 7.9 μg d^{-1} in those who were exposed to a minimum of 15 min d^{-1} of summer sunshine and 11.4 μg d^{-1} in those who were not.[68] We found a similar effect of sun preference in younger adults (20–40 years).[69]

The above example is just one where collaboration between photobiologists and nutritionists can make important contributions to public health guidelines and policy; there are many other examples but these are outside the scope of the present review.

6. Conclusions

There have been a number of re-evaluations of dietary guidelines for vitamin D by expert groups in the Northern hemisphere over the last 10 years. Despite this, however, vitamin D deficiency is still common. This is largely as a consequence of a vitamin D winter with limited UVB availability, limited per-

sonal UVB exposure for some individuals even in the presence of ample UVB, and inadequate dietary supply, amongst other factors. Accordingly, increasing vitamin D intakes across the population distribution is important from a public health perspective to reduce the high degree of inadequacy of vitamin D intake, and fortification, including biofortification, of a range of foods, which accommodate diversity, is key in this regard.

The current dietary guidelines for vitamin D were beset by uncertainty and gaps in the available data about the relative contribution of sunshine and diet to vitamin D status and vitamin D requirements for health maintenance.[1,10–12] Consequently, they were established using an approach that prioritizes the identification of vitamin D intake values that will maintain serum 25(OH)D concentrations above chosen cut-offs when dermal production of vitamin D is absent or markedly diminished. Should there be agreement on whether a minimal-risk UVB radiation exposure relative to skin cancer that also enables vitamin D production exists, it could have a major bearing on further revisions of dietary guidelines for vitamin D. The increasing evidence-base on differential impact of UVR in relation to skin cancer/DNA damage and vitamin D production in different skin types will also be of importance in further revisions of dietary guidelines for vitamin D. Such skin-type specific recommendations for safe sunlight exposure are evident in recent vitamin D advice around the Covid-19 pandemic.[70]

Conflicts of interest

There are no conflicts to declare.

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References

1 Institute of Medicine, *Dietary Reference Intakes for Calcium and Vitamin D*, The National Academies Press, Washington, D.C, 2011.

2 K. D. Cashman and M. Kiely, Towards prevention of vitamin D deficiency and beyond: knowledge gaps and research needs in vitamin D nutrition and public health, *Br. J. Nutr.*, 2011, **106**, 1617–1627.

3 K. D. Cashman and M. Kiely, Contribution of nutrition science to the vitamin D field-Clarity or confusion?, *J. Steroid Biochem. Mol. Biol.*, 2019, **187**, 34–41.

4 K. D. Cashman, Vitamin D requirements for the future-Lessons learned and charting a path forward, *Nutrients*, 2018, **10**, E533, DOI: 10.3390/nu10050533.

5 A. Hayes and K. D. Cashman, Food-based solutions for vitamin D deficiency: putting policy into practice and the key role for research, *Proc. Nutr. Soc.*, 2017, **76**, 54–63.

6 D. E. Roth, S. A. Abrams, J. Aloia, G. Bergeron, M. W. Bourassa, K. H. Brown, M. S. Calvo, K. D. Cashman, G. Combs, L. M. De-Regil, M. E. Jefferds, K. S. Jones, H. Kapner, A. R. Martineau, L. M. Neufeld, R. L. Schleicher, T. D. Thacher and S. J. Whiting, Global prevalence and disease burden of vitamin D deficiency: A roadmap for action in low- and middle-income countries, *Ann. N. Y. Acad. Sci.*, 2018, **1430**, 44–79.

7 K. D. Cashman and D. Vitamin, dietary requirements and food fortification as a means of helping achieve adequate vitamin D status, *J. Steroid Biochem. Mol. Biol.*, 2015, **148**, 19–26.

8 M. Kiely and L. J. Black, Dietary strategies to maintain adequacy of circulating 25-hydroxyvitamin D concentrations, *Scand. J. Clin. Lab. Invest., Suppl.*, 2012, **243**, 14–23.

9 B. Roman Viñas, L. Ribas Barba, J. Ngo, M. Gurinovic, R. Novakovic, A. Cavelaars, L. C. de Groot, P. van't Veer, C. Matthys and L. Serra Majem, Projected prevalence of inadequate nutrient intakes in Europe, *Ann. Nutr. Metab.*, 2011, **59**, 84–95.

10 Scientific Advisory Committee on Nutrition, *Report on vitamin D and health*, 2016; available online: http://www.sacn.gov.uk/pdfs/sacn_vitaminD_and_health_report_web.pdf (accessed 1 July 2016).

11 EFSA NDA Panel (EFSA Panel on Dietetic Products, Nutrition and Allergies), Scientific opinion on Dietary Reference Values for vitamin D, *EFSA J.*, 2016, **14**(10), e04547.

12 Nordic Council of Ministers, *NORDEN Nordic Nutrition Recommendations, 5th Edition (NNR5) – Vitamin D*, 2012. https://norden.diva-portal.org/smash/get/diva2:704251/FULLTEXT01.pdf (accessed August 2013).

13 German Nutrition Society, New reference values for vitamin D, *Ann. Nutr. Metab.*, 2012, **60**, 241–246.

14 Health Council of the Netherlands, *Evaluation of Dietary Reference Values for Vitamin D*, Health Council of the Netherlands, The Hague, 2012 (publication no. 2012/15E).

15 C. F. Munns, N. Shaw, M. Kiely, B. L. Specker, T. D. Thacher, K. Ozono, T. Michigami, D. Tiosano, M. Z. Mughal, O. Mäkitie, L. Ramos-Abad, L. Ward, L. A. DiMeglio, N. Atapattu, H. Cassinelli, C. Braegger, J. M. Pettifor, A. Seth, H. W. Idris, V. Bhatia, J. Fu, G. Goldberg, L. Sävendahl, R. Khadgawat, P. Pludowski, J. Maddock, E. Hyppönen, A. Oduwole, E. Frew, M. Aguiar, T. Tulchinsky, G. Butler and W. Högler, Global consensus recommendations on prevention and management of

nutritional rickets, *J. Clin. Endocrinol. Metab.*, 2016, **101**, 394–415.
16 K. A. Herrick, R. J. Storandt, J. Afful, C. M. Pfeiffer, R. L. Schleicher, J. J. Gahche and N. Potischman, Vitamin D status in the United States, 2011–2014, *Am. J. Clin. Nutr.*, 2019, **110**(1), 150–157.
17 K. Sarafin, R. Durazo-Arvizu, L. Tian, K. W. Phinney, S. Tai, J. E. Camara, J. Merkel, E. Green, C. T. Sempos and S. P. Brooks, Standardizing 25-hydroxyvitamin D values from the Canadian Health Measures Survey, *Am. J. Clin. Nutr.*, 2015, **102**, 1044–1050.
18 K. D. Cashman, K. G. Dowling, Z. Škrabáková, M. Gonzalez-Gross, J. Valtueña, S. De Henauw, L. Moreno, C. T. Damsgaard, K. F. Michaelsen, C. Mølgaard, R. Jorde, G. Grimnes, G. Moschonis, C. Mavrogianni, Y. Manios, M. Thamm, G. B. Mensink, M. Rabenberg, M. A. Busch, L. Cox, S. Meadows, G. Goldberg, A. Prentice, J. M. Dekker, G. Nijpels, S. Pilz, K. M. Swart, N. M. van Schoor, P. Lips, G. Eiriksdottir, V. Gudnason, M. F. Cotch, S. Koskinen, C. Lamberg-Allardt, R. A. Durazo-Arvizu, C. T. Sempos and M. Kiely, Vitamin D deficiency in Europe: pandemic?, *Am. J. Clin. Nutr.*, 2016, **103**, 1033–1044.
19 S. P. J. Brooks, L. Greene-Finestone, S. Whiting, V. E. Fioletov, P. Laffey and N. Petronella, An analysis of factors associated with 25-hydroxyvitamin D levels in white and non-white Canadians, *J. AOAC Int.*, 2017, **100**, 1345–1354.
20 K. D. Cashman, K. G. Dowling, Z. Škrabáková, M. Kiely, C. Lamberg-Allardt, R. A. Durazo-Arvizu, C. T. Sempos, S. Koskinen, A. Lundqvist, J. Sundvall, A. Linneberg, B. Thuesen, L. L. Husemoen, H. E. Meyer, K. Holvik, I. M. Grønborg, I. Tetens and R. Andersen, Standardizing serum 25-hydroxyvitamin D data from four Nordic population samples using the Vitamin D Standardization Program protocols: Shedding new light on vitamin D status in Nordic individuals, *Scand. J. Clin. Lab. Invest.*, 2015, **75**, 549–561.
21 K. D. Cashman, Vitamin D deficiency: Defining, prevalence, causes, and strategies of addressing, *Calcif. Tissue Int.*, 2020, **106**(1), 14–29.
22 M. G. Kimlin, Geographic location and vitamin D synthesis, *Mol. Aspects Med.*, 2008, **29**, 453–461.
23 C. M. O'Neill, A. Kazantzidis, M. J. Ryan, N. Barber, C. T. Sempos, R. A. Durazo-Arvizu, R. Jorde, G. Grimnes, G. Eiriksdottir, V. Gudnason V, *et al.*, Seasonal changes in vitamin D-effective UVB availability in Europe and associations with population serum 25-hydroxyvitamin D, *Nutrients*, 2016, **8**, E533.
24 A. R. Webb, Who, what, where and when-influences on cutaneous vitamin D synthesis, *Prog. Biophys. Mol. Biol.*, 2006, **92**, 17–25.
25 L. Y. Matsuoka, J. Wortsman, J. G. Haddad, P. Kolm and B. W. Hollis, Racial pigmentation and the cutaneous synthesis of vitamin D, *Arch. Dermatol.*, 1991, **127**, 536–538.
26 M. F. Holick, Deficiency of sunlight and vitamin D, *Br. Med. J.*, 2008, **336**, 1318–1319.
27 National Diet and Nutrition Survey (NDNS) rolling program, https://www.gov.uk/government/statistics/ndns-results-from-years-7-and-8-combined (accessed 14 December, 2018).
28 A. L. Darling, D. J. Blackbourn, K. R. Ahmadi and S. A. Lanham-New, Vitamin D supplement use and associated demographic, dietary and lifestyle factors in 8024 South Asians aged 40–69 years: analysis of the UK Biobank cohort, *Public Health Nutr.*, 2018, **21**, 2678–2688.
29 EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA), Scientific opinion on principles for deriving and applying dietary reference values, *EFSA J.*, 2010, **8**, 1458.
30 J. M. Hess, C. J. Cifelli, A. Agarwal and V. L. Fulgoni 3rd, Comparing the cost of essential nutrients from different food sources in the American diet using NHANES 2011–2014, *Nutr. J.*, 2019, **18**, 68.
31 J. C. Newman, A. M. Malek, K. J. Hunt and B. P. Marriott, Nutrients in the US Diet: Naturally occurring or enriched/fortified food and beverage sources, plus dietary supplements: NHANES 2009–2012, *J. Nutr.*, 2019, **149**, 1404–1412.
32 Y. A. Shakur, W. Lou and M. R. L'Abbe, Examining the effects of increased vitamin D fortification on dietary inadequacy in Canada, *Can. J. Public Health*, 2014, **105**, e127–e132.
33 H. Vatanparast, M. S. Calvo, T. J. Green and S. J. Whiting, Despite mandatory fortification of staple foods, vitamin D intakes of Canadian children and adults are inadequate, *J. Steroid Biochem. Mol. Biol.*, 2010, **121**, 301–303.
34 L. J. Black, J. Walton, A. Flynn, K. D. Cashman and M. Kiely, Small increments in vitamin D intake by Irish adults over a decade show that strategic initiatives to fortify the food supply are needed, *J. Nutr.*, 2015, **145**, 969–976.
35 C. Orces, The association between body mass index and vitamin D supplement use among adults in the United States, *Cureus*, 2019, **11**, e5721.
36 S. J. Whiting, K. A. Langlois, H. Vatanparast and L. S. Greene-Finestone, The vitamin D status of Canadians relative to the 2011 Dietary Reference Intakes: an examination in children and adults with and without supplement use, *Am. J. Clin. Nutr.*, 2011, **94**, 128–135.
37 M. R. Rooney, L. Harnack, E. D. Michos, R. P. Ogilvie, C. T. Sempos and P. L. Lutsey, Trends in Use of High-Dose Vitamin D Supplements exceeding 1000 or 4000 International Units daily, 1999–2014, *J. Am. Med. Assoc.*, 2017, **317**, 2448–2450.
38 L. Allen, B. de Benoist, O. Dary and R. Hurrell, *Guidelines on food fortification with micronutrients*, World Health Organization and Food and Agriculture Organization of the United Nations. Geneva, 2006. http://apps.who.int/iris/bitstream/10665/43412/1/9241594012_eng.pdf (accessed 28 June 2016).
39 Food Standards Agency, UK, *McCance and Widdowson's Composition of Foods integrated dataset*, 2008. http://www.food.gov.uk/science/dietarysurveys/dietsurveys/ (accessed 1 June 2011).

40 W. Willett, J. Rockström, B. Loken, M. Springmann, T. Lang, S. Vermeulen, T. Garnett, D. Tilman, F. DeClerck, A. Wood, *et al.*, Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems, *Lancet*, 2019, **393**(10170), 447–492.

41 A. Hayes, S. Duffy, M. O'Grady, J. Jakobsen, K. Galvin, J. Teahan-Dillon, J. Kerry, A. Kelly, J. O'Doherty, S. Higgins, K. M. Seamans and K. D. Cashman, Vitamin D-enhanced eggs are protective of wintertime serum 25-hydroxyvitamin D in a randomized controlled trial of adults, *Am. J. Clin. Nutr.*, 2016, **104**, 629–637.

42 S. K. Duffy, J. V. O'Doherty, G. Rajauria, L. C. Clarke, K. D. Cashman, A. Hayes, M. N. O'Grady, J. P. Kerry and A. K. Kelly, Cholecalciferol supplementation of heifer diets increases beef vitamin D concentration and improves beef tenderness, *Meat Sci.*, 2017, **134**, 103–110.

43 S. K. Duffy, A. K. Kelly, G. Rajauria, J. Jakobsen, L. C. Clarke, F. J. Monahan, K. G. Dowling, G. Hull, K. Galvin, K. D. Cashman, A. Hayes and J. V. O'Doherty, The use of synthetic and natural vitamin D sources in pig diets to improve meat quality and vitamin D content, *Meat Sci.*, 2018, **143**, 60–68.

44 S. K. Duffy, J. V. O'Doherty, G. Rajauria, L. C. Clarke, A. Hayes, K. G. Dowling, M. N. O'Grady, J. P. Kerry, J. Jakobsen, K. D. Cashman and A. K. Kelly, Vitamin D-biofortified beef: A comparison of cholecalciferol with synthetic versus UVB-mushroom-derived ergosterol as feed source, *Food Chem.*, 2018, **256**, 18–24.

45 I. E. Graff, J. Øyen, M. Kjellevold, L. Frøyland, C. G. Gjesdal, B. Almås, G. Rosenlund and Ø. Lie, Reduced bone resorption by intake of dietary vitamin D and K from tailor-made Atlantic salmon: A randomized intervention trial, *Oncotarget*, 2016, **7**, 69200–69215.

46 K. D. Cashman and M. Kiely, *Vitamin D and food fortification*, in *Vitamin D*, ed. D. Feldman J. Wesley Pike, R. Bouillon, E. Giovannucci, D. Goltzman and M. Hewison, Elsevier, 4th edn, 2017.

47 O. Auclair, Y. Han and S. A. Burgos, Consumption of milk and alternatives and their contribution to nutrient intakes among Canadian adults: Evidence from the 2015 Canadian Community Health Survey-Nutrition, *Nutrients*, 2019, **11**, E1948.

48 E. Vartiainen and S. M. Virtanen, Successful nutrition policy: improvement of vitamin D intake and status in Finnish adults over the last decade, *Eur. J. Public Health*, 2017, **27**, 268–273.

49 T. Jääskeläinen, S. T. Itkonen, A. Lundqvist, M. Erkkola, T. Koskela, K. Lakkala, K. G. Dowling, G. L. Hull, H. Kröger, J. Karppinen, E. Kyllönen, T. Härkänen, K. D. Cashman, S. Männistö and C. Lamberg-Allardt, The positive impact of general vitamin D food fortification policy on vitamin D status in a representative adult Finnish population: evidence from an 11-y follow-up based on standardized 25-hydroxyvitamin D data, *Am. J. Clin. Nutr.*, 2017, **105**, 1512–1520.

50 C. L. Luthringer, L. A. Rowe, M. Vossenaar, *et al.*, Regulatory monitoring of fortified foods: identifying barriers and good practices, *Glob. Health Sci. Pract.*, 2015, **3**, 446–461.

51 K. D. Cashman and M. Kiely, Tackling inadequate vitamin D intakes within the population: fortification of dairy products with vitamin D may not be enough, *Endocrine*, 2016, **51**, 38–46.

52 K. H. Madsen, L. B. Rasmussen, R. Andersen, C. Mølgaard, J. Jakobsen, P. J. Bjerrum, E. W. Andersen, H. Mejborn and I. Tetens, Randomized controlled trial of the effects of vitamin D–fortified milk and bread on serum 25-hydroxyvitamin D concentrations in families in Denmark during winter: the VitmaD study, *Am. J. Clin. Nutr.*, 2013, **98**, 374–382.

53 R. E. Allen, A. D. Dangour, A. E. Tedstone and Z. Chalabi, Does fortification of staple foods improve vitamin D intakes and status of groups at risk of deficiency? A United Kingdom modeling study, *Am. J. Clin. Nutr.*, 2015, **102**, 338–344.

54 A. M. Natri, P. Salo, T. Vikstedt, A. Palssa, M. Huttunen, M. U. Kärkkäinen, H. Salovaara, V. Piironen, J. Jakobsen and C. J. Lamberg-Allardt, Bread fortified with cholecalciferol increases the serum 25-hydroxyvitamin D concentration in women as effectively as a cholecalciferol supplement, *J. Nutr.*, 2006, **136**, 123–127.

55 K. D. Cashman and R. O'Dea, Exploration of strategic food vehicles for vitamin D fortification in low/lower-middle income countries, *J. Steroid Biochem. Mol. Biol.*, 2019, **195**, 105479, DOI: 10.1016/j.jsbmb.2019.105479.

56 D. Peters, T. Nhan and A. Taghreed, Alliance for Health Policy and Systems Research, World Health Organization, *Implementation Research in Health: A Practical Guide*, World Health Organization, 2013.

57 Public Health England's Advisory Group on Non-Ionising Radiation, *Ultraviolet radiation and vitamin D: the effects on health*, 2017. Ref: PHE publications gateway number: 2016617 (https://www.gov.uk/government/publications/ultraviolet-radiation-and-vitamin-d-the-effects-on-health).

58 B. Petersen, H. C. Wulf, M. Triguero-Mas, P. A. Philipsen, E. Thieden, P. Olsen, J. Heydenreich, P. Dadvand, X. Basagaña, T. S. Liljendahl, G. I. Harrison, D. Segerbäck, A. W. Schmalwieser, A. R. Young and M. J. Nieuwenhuijsen, Sun and ski holidays improve vitamin D status, but are associated with high levels of DNA damage, *J. Invest. Dermatol.*, 2014, **134**, 2806–2813.

59 T. S. Liljendahl, N. Kotova and D. Segerbäck, Quantification of ultraviolet radiation-induced DNA damage in the urine of Swedish adults and children following exposure to sunlight, *Biomarkers*, 2012, **17**, 634–641.

60 S. J. Felton, M. S. Cooke, R. Kift, J. L. Berry, A. R. Webb, P. M. Lam, F. R. de Gruijl, A. Vail and L. E. Rhodes, Concurrent beneficial (vitamin D production) and hazardous (cutaneous DNA damage) impact of repeated low-level summer sunlight exposures, *Br. J. Dermatol.*, 2016, **175**, 1320–1328.

61 B. B. Shih, M. D. Farrar, M. S. Cooke, J. Osman, A. K. Langton, R. Kift, A. R. Webb, J. L. Berry, R. E. B. Watson, A. Vail, F. R. de Gruijl and L. E. Rhodes, Fractional sunburn threshold UVR doses generate equivalent vitamin D and DNA damage in skin types I-VI but with epidermal DNA damage gradient correlated to skin darkness, *J. Invest. Dermatol.*, 2018, **138**, 2244–2252.

62 J. M. Sheehan, N. Cragg, C. A. Chadwick, C. S. Potten and A. R. Young, Repeated ultraviolet exposure affords the same protection against DNA photodamage and erythema in human skin types II and IV but is associated with faster DNA repair in skin type IV, *J. Invest. Dermatol.*, 2002, **118**, 825–829.

63 Cancer Research UK, http://www.cancerresearchuk.org, accessed 30th April, 2020.

64 A. R. Webb and O. Engelsen, Calculated ultraviolet exposure levels for a healthyvitamin D status, *Photochem. Photobiol.*, 2006, **82**, 1697–1703.

65 J. Guzikowski, J. Krzyścin, A. Czerwińska and W. Raszewska, Adequate vitamin D(3) skin synthesis versus erythema risk in the Northern Hemisphere midlatitudes, *J. Photochem. Photobiol., B*, 2018, **179**, 54–65.

66 M. D. Farrar, A. R. Webb, R. Kift, M. T. Durkin, D. Allan, A. Herbert, J. L. Berry and L. E. Rhodes, Efficacy of a dose range of simulated sunlight exposures in raising vitamin D status in South Asian adults: implications for targeted guidance on sun exposure, *Am. J. Clin. Nutr.*, 2013, **97**, 1210–1216.

67 A. R. Webb, A. Kazantzidis, R. C. Kift, M. D. Farrar, J. Wilkinson and L. E. Rhodes, Meeting vitamin D requirements in white Caucasians at UK latitudes: providing a choice, *Nutrients*, 2018, **10**, E497, DOI: 10.3390/nu10040497.

68 K. D. Cashman, J. M. Wallace, G. Horigan, T. R. Hill, M. S. Barnes, A. J. Lucey, M. P. Bonham, N. Taylor, E. M. Duffy, K. Seamans, S. Muldowney, A. P. Fitzgerald, A. Flynn, J. J. Strain and M. Kiely, Estimation of the dietary requirement for vitamin D in free-living adults ≥64 y of age, *Am. J. Clin. Nutr.*, 2009, **89**, 1366–1374.

69 K. D. Cashman, T. R. Hill, A. J. Lucey, N. Taylor, K. M. Seamans, S. Muldowney, A. P. Fitzgerald, A. Flynn, M. S. Barnes, G. Horigan, M. P. Bonham, E. M. Duffy, J. J. Strain, J. M. Wallace and M. Kiely, Estimation of the dietary requirement for vitamin D in healthy adults, *Am. J. Clin. Nutr.*, 2008, **88**, 1535–1542.

70 S. A. Lanham-New, A. R. Webb, K. D. Cashman, J. L. Buttriss, J. L. Fallowfield, T. Masud, M. Hewison, J. C. Mathers, M. Kiely, A. A. Welch, K. A. Ward, P. Magee, A. L. Darling, T. R. Hill, C. Greig, C. P. Smith, R. Murphy, S. Leyland, R. Bouillon, S. Ray and M. Kohlmeier, Vitamin D and SARS-CoV-2 Virus/COVID-19 disease, *BMJ Nutr. Prev. Health*, 2020, in press.