Environmental factors that influence the cutaneous production of vitamin D[1–3]

Michael F Holick

ABSTRACT All vertebrates, including humans, obtain most of their daily vitamin D requirement from casual exposure to sunlight. During exposure to sunlight, the solar ultraviolet B photons (290–315 nm) penetrate into the skin where they cause the photolysis of 7-dehydrocholesterol to precholecalciferol. Once formed, precholecalciferol undergoes a thermally induced rearrangement of its double bonds to form cholecalciferol. An increase in skin pigmentation, aging, and the topical application of a sunscreen diminishes the cutaneous production of cholecalciferol. Latitude, season, and time of day as well as ozone pollution in the atmosphere influence the number of solar ultraviolet B photons that reach the earth's surface, and thereby, alter the cutaneous production of cholecalciferol. In Boston, exposure to sunlight during the months of November through February will not produce any significant amounts of cholecalciferol in the skin. Because windowpane glass absorbs ultraviolet B radiation, exposure of sunlight through glass windows will not result in any production of cholecalciferol. It is now recognized that vitamin D insufficiency and vitamin D deficiency are common in elderly people, especially in those who are infirm and not exposed to sunlight or who live at latitudes that do not provide them with sunlight-mediated cholecalciferol during the winter months. Vitamin D insufficiency and deficiency exacerbate osteoporosis, cause osteomalacia, and increase the risk of skeletal fractures. Vitamin D insufficiency and deficiency can be prevented by encouraging responsible exposure to sunlight and/or consumption of a multivitamin tablet that contains 10 μg (400 IU) vitamin D. *Am J Clin Nutr* 1995;61(suppl):638S–45S

KEY WORDS Vitamin D, sunlight, osteoporosis, aging, recommended dietary allowance, RDA, osteomalacia, skin

Evolutionary perspective

The photosynthesis of vitamin D has been occurring for > 750 million y (1). The phytoplankton coccolithophor *Emeliani huxleii* that has existed in the Sargasso Sea for ≥ 750 million y not only synthesized carbohydrates when exposed to sunlight but also produced vitamin D. Although the exact role of vitamin D in early plant and animal forms is unknown, vitamin D played a critical role in the maintenance of a calcified skeleton in vertebrates as they left their calcium-rich ocean environment for land > 350 million y ago. It is likely that these early vertebrate species depended on vitamin D for the efficient use of scarce dietary calcium to preserve a rigid calcified skeleton. Because vitamin D can only be synthesized via a photochemical process, early vertebrates that ventured onto land either had to ingest foods that contained vitamin D or had to be exposed to sunlight to photosynthesize vitamin D in their skin to satisfy their body's vitamin D requirement.

Human historical perspective

Archeological records show that humans have worshipped the sun for its life-giving powers from almost the beginning of time. Although the relationship of sunshine to health began to be appreciated at the turn of the 20th century, its roots began > 300 y ago (2). As people began to migrate into city-centers in northern Europe and the industrial revolution began to take hold, the pall of pollution in the atmosphere in combination with the construction of multistoried structures in close proximity provided an environment for children that was devoid of direct exposure to sunlight. In 1650, Glisson, DeBoot, and Whistler wrote treatises on a bone-deforming disease in children that was endemic in Great Britain and northern Europe. The incidence of the disease commonly known as rickets or English disease continued to increase during the industrial revolution and by the turn of the 20th century, this crippling bone disease was epidemic in industrialized cities of northern Europe and the northeastern United States. A study conducted in Leiden, Netherlands, reported that >80% of young children dying of various causes had clinical manifestations of rickets. The consequences of this disease were quite profound especially for children, who often had deformities of their legs that made it difficult for them to walk. For women, a flat, deformed pelvis often caused difficulty in childbirth and resulted in a high incidence of infant and maternal morbidity and mortality (2).

The first insight into the potential cause of rickets was made by Sniadecki in 1822 (3). He reported that children who lived in Warsaw had a relatively high incidence of rickets compared with children who lived in nearby rural areas and who were

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essentially free of this bone disease. He advocated that children be carried into the open air, especially into the sun, the direct action of which on their bodies must be regarded as one of the most efficient methods for the prevention and cure of this disease. He concluded "thus, strong and obvious is the influence of the sun on the cure of the English disease (i.e., rickets) and frequent occurrence of the disease in densely populated towns where the streets are narrow and the dwellings of the working class are poorly lit." It was inconceivable to the medical community at this time that exposure to sunlight could have any benefit to either preventing or curing this bone disease and, as a result, the epidemic of rickets in the 19th century continued its unrelenting course. In 1889, an investigative committee of the British Medical Association reported that rickets was unknown in rural districts of the British Islands but that it was prevalent in large industrialized towns (2). A year later, Palm (4) reported the results of an epidemiologic survey regarding the potential factors that cause rickets. He found that children who had poor nutrition and who lived in squalor in cities in China, Japan, and India were not afflicted with this dreaded disease whereas children living in industrialized centers throughout Europe were afflicted by rickets. He concluded that the only common denominator for rickets in children was the lack of sunlight exposure. He encouraged systematic sunbathing as a means for preventing and curing rickets and encouraged the scientific community to appreciate the healthful effects of sunlight. However, Palm's insightful observations and suggestions went unheeded by the medical and scientific communities, just as Sniadecki's had been.

By the turn of the 20th century, rickets continued to be a major health problem in the industrialized cities of Europe and North America. By this time numerous theories for the cause of rickets had surfaced, including the following: *1*) an inherited disorder, *2*) a lack of activity, *3*) a nutritional deficiency, *4*) a lack of exposure to sunlight, and *5*) an infectious disease. In 1911, Koch (5) inoculated dogs with *Streptococcus longus* and found that 10% of the dogs developed rickets. He therefore concluded that rickets was an infectious disease. In 1919, Huldschinsky (6) exposed four rachitic children to radiation from a mercury arc lamp and demonstrated by x-ray analysis that rickets was cured after 4 mo of therapy. He also showed that the phototherapy was not a local effect on the bone by demonstrating that exposure of one arm to this radiation had an equal and dramatic curative effect on both arms. Two years later, Hess and Unger (7) exposed seven rachitic children in New York City to sunshine and reported by x-ray examination marked improvement in the severity of rickets in each child. Thus, one century after Sniadecki first suggested the importance of sunlight exposure for the prevention of rickets it was unequivocally shown that exposure to sunlight alone could prevent and cure this crippling bone disease. These observations led Hess and Weinstock (8) and Steenbock and Black (9) to expose a variety of foods and other substances including lettuce, vegetable oils, and rat feed to ultraviolet radiation. They found that ultraviolet irradiation imparted antirachitic activity to most of the substances. These observations led Steenbock (10) to conclude that there may be a great utility in irradiating food substances to prevent and cure rickets in children. This concept led to the addition of provitamin D to milk and its subsequent irradiation to impart antirachitic activity. As a result, rickets was eradicated as a significant health problem in the United States and other countries. Once the structure of vitamin D was characterized, it was chemically synthesized and directly added to milk, making the irradiation of milk obsolete.

Photosynthesis of precholecalciferol in human skin

During exposure to sunlight, the solar ultraviolet B photons with energies between 290 and 315 nm penetrate the skin where they cause the photolysis of 7-dehydrocholesterol (provitamin D_3) to precholecalciferol (previtamin D_3) (**Figure 1**). Precholecalciferol is thermodynamically unstable and undergoes an internal [1–7] sigmatropic shift of a proton from C-9 to C-10 and subsequent isomerization to form cholecalciferol. Once formed, cholecalciferol exits the skin into the circulation bound to the vitamin D–binding protein (2, 12, 13).

In warm-blooded animals such as humans, it was thought that, at 37 °C, precholecalciferol slowly converted over a period of 2–3 d to cholecalciferol (12). This was based on observations that in an isotropic organic solvent such as hexane at 37 °C, ≈80% of precholecalciferol had thermally equilibrated to cholecalciferol by 3 d. However, it is now recognized that in the skin this isomerization reaction is ≈10 times faster than in an organic solvent (14) (**Figure 2**). Thus, within 8 h, 80% of precholecalciferol is converted to cholecalciferol. For cold-blooded vertebrates, the rapid cutaneous isomerization of

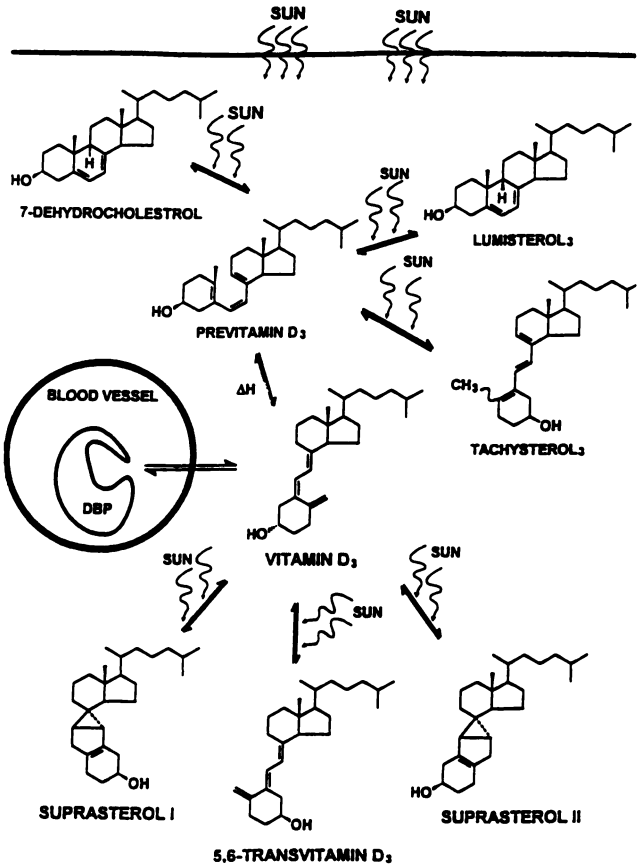

FIGURE 1. Photochemical events that lead to the production and regulation of cholecalciferol (vitamin D_3) in the skin. Reproduced with permission (11).

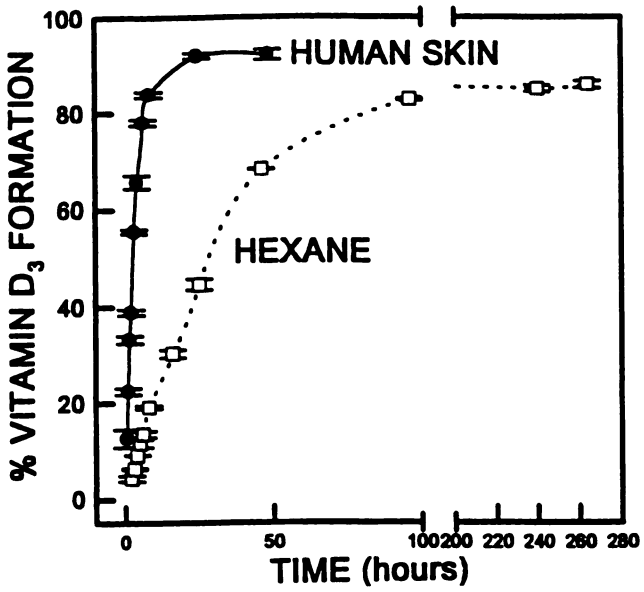

FIGURE 2. Thermal isomerization of precholecalciferol (previtamin D_3) at 37 °C in *n*-hexane (□) and in human skin (●). Adapted from reference 14.

precholecalciferol to cholecalciferol probably played an important role in supplying these animals with their vitamin D requirement. Although the exact mechanism for this rapid effect is not known, it has been speculated that it is due to the fact that 7-dehydrocholesterol is in the membrane fraction of skin cells and that upon irradiation, the confirmation of precholecalciferol is maintained in a *s-cis, s-cis* rather than in the more thermodynamically favored *s-trans, s-cis* confirmation, the former of which is more efficiently converted to cholecalciferol (XQ Tian and MF Holick, unpublished results).

Endogenous factors that regulate the cutaneous production of cholecalciferol

Melanin is an excellent natural sunscreen that efficiently absorbs solar ultraviolet radiation, including ultraviolet B radiation (290–320 nm). Thus, melanin pigmentation in the skin competes for ultraviolet B photons and thereby decreases the efficiency for the photosynthesis of precholecalciferol (15–17).

Aging has a dramatic effect on the skin. Skin thickness decreases linearly with age in humans after the age of 20 y. An analysis of the concentrations of 7-dehydrocholesterol in the epidermis of people of varying ages revealed a marked age-dependent decrease in epidermal concentrations of this vitamin D precursor (18). When healthy young and elderly adults were exposed to a whole-body dose of the same amount of simulated solar radiation that was comparable with being at Cape Cod on a sunny afternoon in the summer for ≈15 min, the circulating concentrations of vitamin D increased to a maximum of 78.1 nmol/L (30 ng/mL) within 24 h in the young volunteers whereas the older subjects (aged 62–80 y) were only able to achieve a maximum concentration of ≈20.8 nmol/L (8 ng/mL) (19) (**Figure 3**).

FIGURE 3. Circulating concentrations of vitamin D in response to a whole-body exposure to one minimal erythemal dose in healthy young and elderly subjects. Reproduced with permission (11).

Exogenous factors that affect the cutaneous production of cholecalciferol

Sunlight and ozone

During exposure to sunlight, solar ultraviolet B photons photolyze 7-dehydrocholesterol to precholecalciferol. However, precholecalciferol can also absorb solar ultraviolet B photons, and therefore, during prolonged exposure to sunlight, precholecalciferol is photolyzed principally to a biologically inert isomer lumisterol (16) (**Figure 4**). Thus, prolonged exposure to sunlight cannot cause the overproduction of precholecalciferol because, once formed, the amount of precholecalciferol is maintained in a quasi-photostationary state, ie, no more than ≈10–15% of the initial cutaneous concentrations of 7-dehydrocholesterol will be converted to precholecalciferol (Figures 1 and 4).

Similarly, when cholecalciferol is produced in the skin, it is sensitive to both ultraviolet B and ultraviolet A radiation up to ≈345 nm. Thus, once cholecalciferol is formed, if it does not escape into the circulation, when exposed to sunlight it is photolyzed to at least three other products, including 5,6-*trans*-cholecalciferol, supersterol I, and supersterol II (Figure 1) (20).

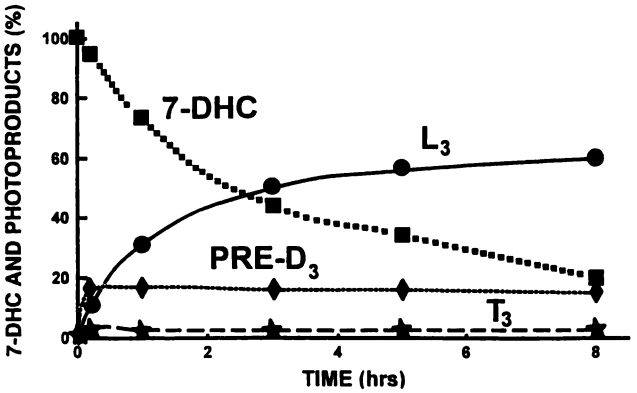

FIGURE 4. Exposure of 7-dehydrocholesterol (7-DHC) to simulated equatorial sunlight resulting in the photoproduction of precholecalciferol (PRE-D_3) and its photoisomers lumisterol (L_3) and tachysterol (T_3). Reproduced with permission (11).

The stratospheric ozone layer efficiently absorbs solar radiation <290 nm. There is increased concern about the effect of chlorofluorohydrocarbons on depleting the stratospheric ozone layer, thereby resulting in transmission of a larger number of higher-energy solar ultraviolet photons. Although the exact impact of the depletion of the ozone layer on the cutaneous production of cholecalciferol is not well understood, it is recognized that when human skin is exposed to narrow band radiation (295–300 nm), that ≈65% of the original 7-dehydrocholesterol content is converted to precholecalciferol (13) (**Figure 5**). This is in comparison with when skin was exposed to simulated solar radiation where the maximum formation of precholecalciferol was <20%. Thus, it is interesting to speculate that if the ozone layer was substantially depleted not only would this markedly increase the incidence of skin cancer and skin damage but it could potentially cause an increase in the cutaneous production of cholecalciferol that could lead to vitamin D intoxication.

In many industrialized cities, air pollution that contains ozone can be quite substantial. If the ozone concentration in the atmosphere is increased, the ozone would efficiently absorb ultraviolet B photons thereby reducing the cutaneous photosynthesis of precholecalciferol. This could ultimately result in an increased risk of vitamin D deficiency in young children and in elderly people who live in the polluted cities in the United States and northern Europe.

Sunscreens and clothing

There is great concern today about the damaging effects of chronic exposure to sunlight. Long-term exposure to sunlight can cause significant damage to the structural integrity of the skin and can increase the risk of skin cancer. As a result of this heightened awareness of the damaging effects of sunlight, there has been a major effort to encourage the use of a topical sunscreen with a sun protection factor of ≥15 before going outdoors. Although there is no question of the benefit of sunscreen use for the prevention of sunburn, skin cancer, and skin damage, it has been shown that sunscreens can prevent the beneficial effect of sunlight, the production of precholecalciferol (21). This is not surprising because the radiation that is responsible for causing sunburn and skin damage is the same radiation that is responsible for producing precholecalciferol. The topical application of a sunscreen with a sun protection factor of 8 prevented any increase in circulating concentrations of vitamin D after a whole-body exposure to simulated sunlight that would have caused a mild sunburn (21). Chronic sunscreen use by elderly people can cause vitamin D insufficiency and vitamin D deficiency (22).

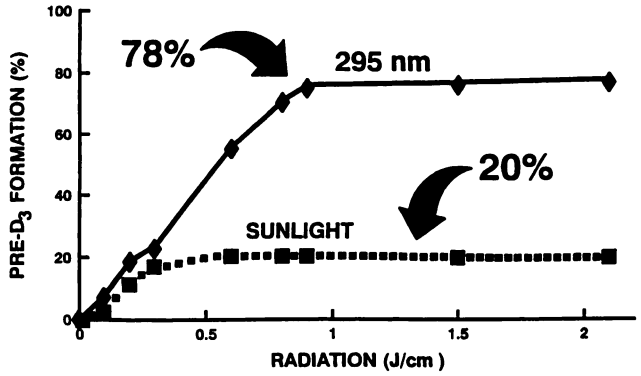

FIGURE 5. Photosynthesis of precholecalciferol (PRE-D_3) from 7-dehydrocholesterol after exposure to simulated sunlight (■) or 295 ± 5 nm radiation (◆). Adapted from reference 13.

Most clothing absorbs solar ultraviolet B radiation (23). When human volunteers wore fabrics made out of white or black cotton, wool, or polyester and were then exposed to simulated sunlight for up to 40 min, there was no elevation in circulating concentrations of vitamin D (23). In some societies in which clothing is worn over most sun-exposed areas, there is an increased risk of vitamin D deficiency rickets in children, and osteomalacia and osteoporosis in adults (24). In an epidemiologic study performed in Saudi Arabia, where extensive coverage by garments is promoted, circulating concentrations of 25-hydroxyvitamin D were measured in 100 consecutively studied Saudi Arabian mothers and their neonates. A high prevalence of subjects with subnormal values (59% and 70% of the mothers and neonates, respectively) was found. In addition, plasma calcium concentrations were below the normal range in 61% of the mothers and 59% of the newborns (24).

Latitude and season

It is well known that vitamin D deficiency is more prevalent during the winter months. In 1897, Kassowitz (25) reported on the increased incidence of rickets during the winter months and its decline during the summer and autumn. In the winter, people are outdoors less frequently and wear more clothing, thereby decreasing the surface area exposed to sunlight. Furthermore, the zenith angle of the sunlight increases in the autumn and winter and the amount of solar ultraviolet radiation that reaches the earth's surface is substantially reduced.

The quantity of ultraviolet photons that penetrate to the earth's surface is dependent on many factors, including path length through which sunlight penetrates through the stratospheric ozone layer and the distance that solar radiation must travel through the atmosphere as a function of solar zenith angle, which depends on latitude, season, and time of day. An extensive evaluation of the effect of season and time of day was carried out in Boston (42 °N). In June and July, the cutaneous photolysis of 7-dehydrocholesterol to precholecalciferol was at a maximum (**Figure 6**). There was a gradual decline in the efficiency of conversion of 7-dehydrocholesterol to precholecalciferol after August, and by October <4% of 7-dehydrocholesterol was photolyzed to precholecalciferol between the hours of 1130 and 1430 Eastern Standard Time (EST). By November, however, there was little if any detectable production of precholecalciferol and this continued until March when precholecalciferol synthesis was detected. Thus, between the months of November and February, little if any cutaneous cholecalciferol synthesis occurred in Boston (26). A similar study was conducted in Edmonton, Canada (52 °N). The photosynthesis of precholecalciferol ceased after the middle of October and did not recur until the middle of April. In Los Angeles (34 °N) and San Juan, Puerto Rico (18 °N), precholecalciferol production occurred throughout the year (26) (Figure 6).

The effect of time of day on the cutaneous production of precholecalciferol can be quite substantial. During July, the photolysis of cutaneous 7-dehydrocholesterol to precholecalciferol occurred as early as 0700 EST and as late as 1700 EST. However, in the spring and autumn, precholecalciferol synthe-

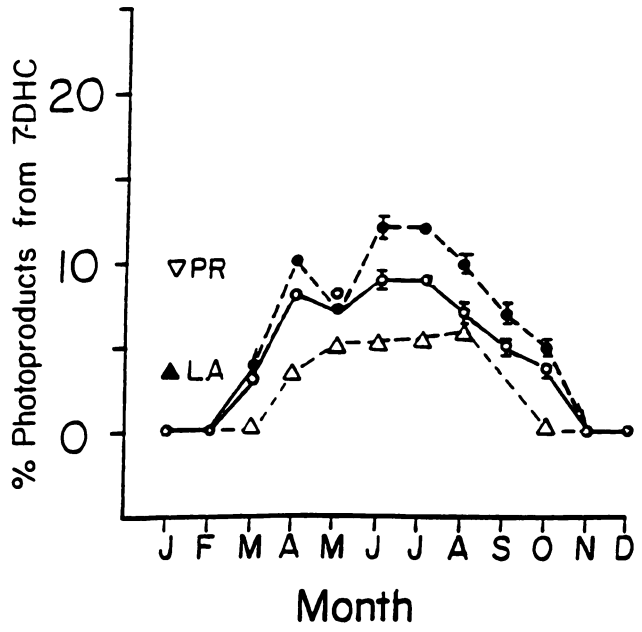

FIGURE 6. Photosynthesis of precholecalciferol after exposure of 7-dehydrocholesterol (7-DHC) to sunlight in Boston (42 °N) for 1 h (○) and 3 h (●); in Edmonton, Canada (52 °N) for 1 h each month for 1 y (△); and in Los Angeles (34 °N) (▲) and Puerto Rico (18 °N) (▽) in January. $\bar{x}$ ± SEM. Adapted from reference 26.

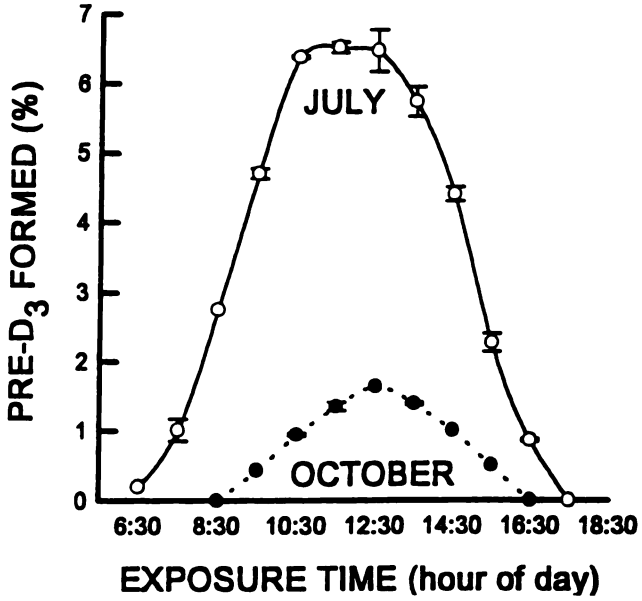

FIGURE 7. Photosynthesis of precholecalciferol (PRE-D_3) at various times on cloudless days in Boston in October (●) and July (○). $\bar{x}$ ± SEM. Reproduced with permission (11).

sis began around 1000 EST and ceased around 1500 EST (27) (**Figure 7**).

Importance of sun-mediated photoproduction of cholecalciferol for human bone health

Osteoporosis is a disease of the bone whereby there is a loss of both matrix and mineral that ultimately causes a compromise of the skeleton's architectural integrity resulting in fracture. There are a variety of factors that are implicated in the etiology of osteoporosis, including low lifetime dietary intake of calcium, endocrinopathies including hyperparathyroidism and hyperthyroidism, as well as aging (28). Recently, it has been suggested that a polymorphism for the vitamin D receptor may result in a predisposition for developing osteoporosis (29).

It is not well appreciated that vitamin D deficiency can exacerbate osteoporosis and increase a person's risk of developing skeletal fractures. Vitamin D deficiency results in transient hypocalcemia, which leads to secondary hyperparathyroidism, and a return of serum calcium concentrations to normal. Secondary hyperparathyroidism causes hypophosphatemia, which lowers the calcium-phosphorus product below normal, causing a mineralization defect that leads to osteomalacia. Secondary hyperparathyroidism also increases the number of bone resorption cells (osteoclasts), which, in turn, erode and worsen osteoporotic bone.

It has been assumed in the United States that vitamin D deficiency is no longer a health problem because many foods are fortified with vitamin D. However, there is only one major food that is fortified with vitamin D: milk. It has been generally believed that because milk is such an important food source for both children and adults that the vitamin D content in milk is carefully monitored. It is now recognized that >70% of milk samples from all sections of the United States and in British Columbia, Canada, do not contain between 10.6 and 13.2 μg (423 and 528 IU) vitamin D/L (400 and 500 IU/quart). Most samples in the United States were found to be under-fortified with vitamin D. Most disturbing was the finding that between 14% and 21% of skim milk samples contained no detectable vitamin D (30, 31) (**Table 1**).

There is strong evidence that vitamin D intake from the diet is low in elderly people in the United States (32). A recent analysis of vitamin D status of elderly residents at a local nursing home revealed that at the end of the summer, 56% of the residents tested were borderline to overtly vitamin D deficient and that by the middle of winter 83% were (33) (**Figure 8**).

The major causes of vitamin D deficiency in elderly people include *1*) decreased milk consumption because of lactase deficiency and concern about milk's fat and energy content, *2*) decreased outdoor activities, *3*) decreased capacity of their skin

TABLE 1
Vitamin D content of milk samples, as a percentage of the amount stated on the package label[1]

Source [no. of samples]	Percentage of stated amount of vitamin D			
	<5[2]	5–80	81–120	>120
			%	
Eastern states[3] [41]	5 [2]	44 [18]	27 [11]	24 [10]
Illinois [6]	0	17 [1]	17 [1]	67 [4]
Louisiana [8]	25 [2]	25 [2]	12 [1]	38 [3]
Utah [24]	29 [7]	33 [8]	12 [3]	25 [6]
All United States [79]	14 [11]	37 [29]	20 [16]	29 [23]
British Columbia [15]	7 [1]	20 [3]	27 [4]	47 [7]

[1] Reproduced with permission (31). Number of samples in brackets.
[2] The samples in this group had undetectable amounts of vitamin D.
[3] New Hampshire, Vermont, Massachusetts, New Jersey, Pennsylvania, Maryland, and Virginia.

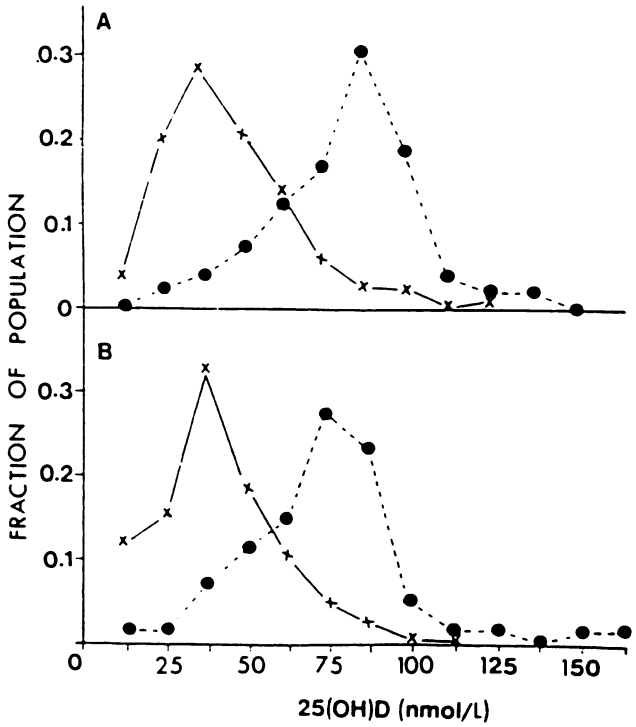

FIGURE 8. Circulating concentrations of 25-hydroxyvitamin D [25(OH)D] in elderly nursing home residents with (●) and without (x) vitamin D supplements in September (A) and February (B). Each data point represents $n - 10$ to n, eg, 75 represents all samples between 65 and 75.0 nmol/L. Reproduced with permission (33).

to produce vitamin D because of age-related changes, and *4*) use of sunscreens and clothing that prevent or diminish the cutaneous production of vitamin D.

In the United States, the major source of vitamin D for elderly people is milk (when it is fortified with an adequate amount of vitamin D), vitamin D supplements, and exposure to sunlight. Multivitamin pills that contain 10 μg (400 IU) ergocalciferol are an excellent source of vitamin D for elderly people. However, it is often difficult to convince elderly people to take a multivitamin pill each day, especially because many are already taking several other medications for their health.

It is casual exposure to sunlight that provides most humans with their vitamin D requirement (34). An evaluation of monthly frequency of fractures of white females demonstrated a significant increase in the incidence of hip fracture during the winter months (**Figure 9**) (35). Although some of this increase can be directly related to an increased risk of falling in icy conditions, this is not the full explanation for these observations. There is mounting evidence that elderly people develop vitamin D insufficiency and deficiency during the winter that can exacerbate osteoporosis, cause osteomalacia, and increase their risk of fracture (36, 37). A study of 18 rural Maine women (mean age 77 y) revealed that most of the loss of bone mineral density in the lumbar spine occurred during the fall and winter when the sun was unable to produce cholecalciferol in the skin. The marked 3.6% reduction in bone mass correlated with a 13 ± 6% decline in blood concentrations of 25-hydroxyvitamin D and a 27 ± 11% increase in parathyroid hormone (PTH) concentrations. The decrease in the bone mass during the spring and summer was negligible and this correlated with a

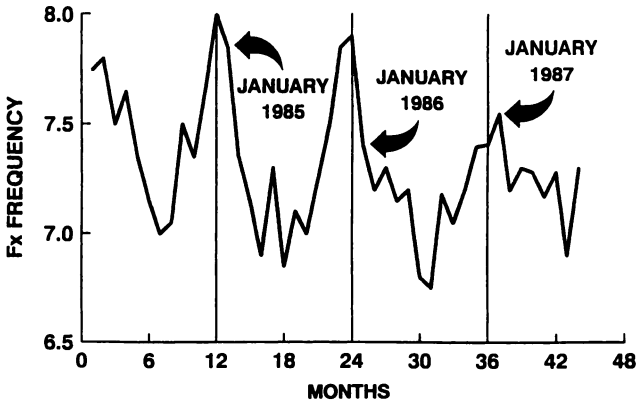

FIGURE 9. Fracture (Fx) incidence as a function of season in women. Reproduced with permission (35).

44% increase in blood 25-hydroxyvitamin D and a 14.5% decline in PTH concentrations toward baseline (**Figure 10**). Several studies have now demonstrated that increases in calcium and vitamin D intake increase bone mineral density and decrease fracture risk (11, 36–39).

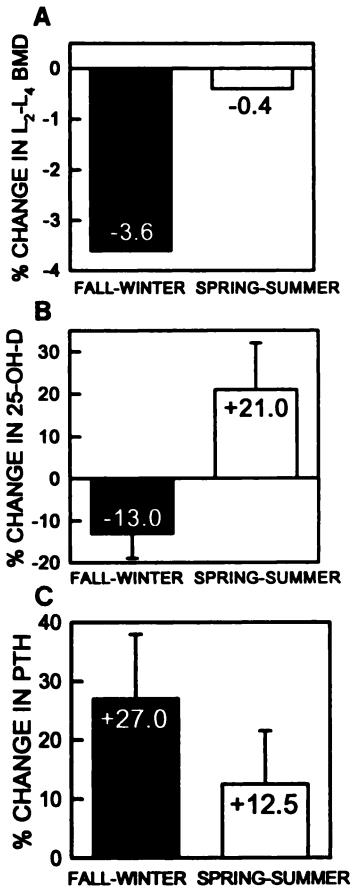

FIGURE 10. Percent change in the L_2–L_4 bone mineral density (BMD) (A); 25-hydroxyvitamin D (25-OH-D) concentrations (B); and circulating immunoreactive intact parathyroid hormone (PTH) (C) during the winter and summer in women in rural Maine. Adapted from reference 37.

Recommendations

How much sunlight exposure is required to provide the body with its vitamin D requirement? There is no simple answer to this question. It depends on the person's age, the person's sensitivity to sunlight, the latitude, the season, the time of day, and how much skin is directly exposed to sunlight. However, when we gave young adults a whole-body exposure of one minimal erythemal dose of simulated solar ultraviolet B radiation, their circulating concentrations of vitamin D increased to a peak of ≈52 nmol/L (20 ng/mL). After a single oral dose of either 10 000 or 25 000 IU ergocalciferol, circulating concentrations of vitamin D increased to a maximum after 24 h of ≈31.2 and 125 nmol/L (12 and 48 ng/mL), respectively (34) (**Figure 11**). These data clearly demonstrate that the skin has a large capacity to produce cholecalciferol and that whole-body exposure to one minimal erythemal dose of simulated solar ultraviolet radiation is comparable with taking an oral dose of between 250 and 625 μg (10 000 and 25 000 IU) vitamin D.

It is recommended for white, elderly people in Boston to expose their hands, face, and arms two to three times a week to suberythemal amounts of sunlight. For example, for an elderly person who goes outside in the afternoon in July and experiences a sunburn after 30 min exposure, exposure of 5–10 min is adequate. To stay outdoors for a longer period of time, an elderly person should apply a sunscreen with a sun protection factor of ≥15 on all sun-exposed areas (after an initial 5–10 min without a sunscreen) to prevent the damaging effects due to excessive chronic exposure to sunlight. For active children and adults, it is casual everyday exposure to sunlight that provides them with their vitamin D requirement. Therefore, there is no need for concern about sunscreen use in decreasing their ability to produce vitamin D. It is recommended that children and young and middle-aged active adults who have sensitivity to sunlight should always wear a sunscreen when outdoors.

The recommended dietary allowance (RDA) for vitamin D is 5 μg (200 IU) (40). It is likely, however, that without casual exposure to sunlight, the RDA for vitamin D is at least two to three times more, or ≈10–15 μg (400–600 IU/d) (39). This is also supported by studies that have demonstrated that increasing the vitamin D intake by 10 μg (400 IU) can be protective of bone mass especially during the winter (36–38).

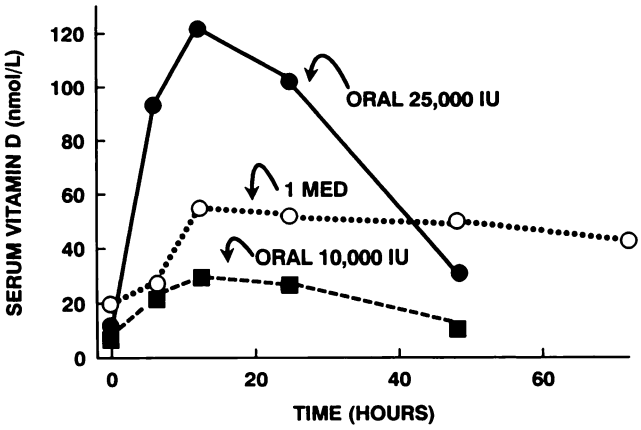

FIGURE 11. Circulating concentrations of vitamin D after either a single oral dose of 10 000 or 25 000 IU ergocalciferol or exposure of the whole body of an adult to one minimal erythemal dose of simulated sunlight. Reproduced with permission (34).

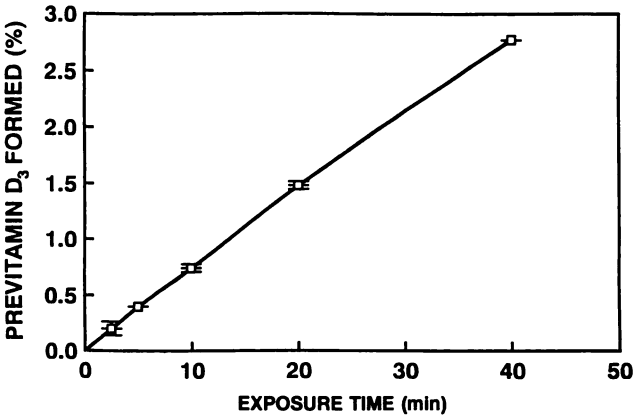

FIGURE 12. Production of precholecalciferol (previtamin D_3) from 7-dehydrocholesterol after exposure at a distance of 0.3 m from a Eurosun light source produced by Wolff System Technology (Atlanta, GA). $\bar{x} \pm$ SEM. Adapted from reference 34.

Unfortunately, glass and most plastics absorb most if not all solar ultraviolet B photons. As a result, exposure to sunlight through glass or plastic will not produce vitamin D in the skin. An alternative is to consider designing indoor lighting that would provide a small amount of ultraviolet B radiation to promote the cutaneous synthesis of cholecalciferol in a passive manner. An analysis of exposure to simulated radiation at distances of 0.3 and 1.5 m from the source revealed that this kind of exposure is capable of promoting precholecalciferol synthesis in skin (34) (**Figure 12**). Thus, it is likely that in the future indoor lighting that contains ultraviolet B radiation in selected areas (such as activity rooms, eating areas, etc) will be an important source of vitamin D for elderly people who cannot depend on environmental exposure to sunlight to provide them with their vitamin D requirement.

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