ABSTRACT
Ultraviolet radiation is a carcinogen that also compromises skin appearance and function. Because the ultraviolet action spectra for DNA damage, skin cancer, and vitamin D₃ photosynthesis are identical and vitamin D is readily available from oral supplements, why has sun protection become controversial? First, the media and, apparently, some researchers are hungry for a new message. Second, the controversy is fueled by a powerful special interest group: the tanning industry. This industry does not target the frail elderly or inner-city ethnic minorities, groups for whom evidence of vitamin D₃ insufficiency is strongest, but rather fair-skinned teenagers and young adults, who are at highest risk of ultraviolet photodamage. Third, evolution does not keep pace with civilization. When nature gave humans the appealing capacity for cutaneous vitamin D₃ photosynthesis, life expectancy was <40 y; long-term photodamage was not a concern; and vitamin D₃ deficiency, with its resulting skeletal abnormalities (rickets), was likely to be fatal in early life. In the 21st century, life expectancy approaches 80 y in developed countries, vitamin D₃ is available at the corner store, and the lifetime risk of skin cancer is 1 in 3 among white Americans. Medical and regulatory groups should avoid poorly reasoned, sensationalistic recommendations regarding unprotected ultraviolet exposure. Instead, they should rigorously explore possible cause-and-effect relations between vitamin D₃ status and specific diseases while advocating the safest possible means of ensuring vitamin D₃ sufficiency. Am J Clin Nutr 2008;88(suppl):570S–7S.

INTRODUCTION
The media and certain elements within the biomedical research community have created a controversy regarding the allegedly conflicting goals of sun protection and skin cancer prevention on the one hand and achieving optimal vitamin D concentration on the other hand. I will attempt to distinguish this pseudo-controversy from the true controversy surrounding the rather poorly documented health benefits of very high vitamin D concentrations, however achieved.

The curious and somewhat elusive basis of the pseudo-controversy lies in the often unstated assumption that vitamin D concentrations, specifically those of the inactive pre-hormone 25-hydroxyvitamin D [25(OH)D] that is measured in serum, are best achieved from increased sun exposure, which enhances the cutaneous photosynthesis of vitamin D within the irradiated epidermis. This assumption has framed discussions in the popular press and on the Internet, even though virtually all intervention studies suggesting a benefit for increasing the conventional “normal” or “sufficient” 25(OH)D concentration in specific population groups have examined the effect of oral vitamin D supplements, not increased exposure to sun or other ultraviolet (UV) sources (1), and have shown that vitamin D obtained from diet or supplements can fully substitute for vitamin D synthesized in the skin. This formulation of the debate also fails to acknowledge that the major motivation for sun exposure in the population at large is tanning, not improved general health. Thus, reports continue on the “debate” between professional groups with primary interests in skin health versus endocrinologic health, often where no such debate exists, creating confusion among the general public regarding recommended health behaviors. I briefly review these complex areas, but principally seek to explain the controversy that continues to dominate media coverage and, more recently, health policy deliberations.

ULTRAVIOLET RADIATION CAUSES MELANOMA AND NONMELANOMA SKIN CANCER
UV radiation is a proven carcinogen (2) that is responsible for most of the estimated 1.3 million skin cancer cases in the United States each year (3), which account for more than one-half of all human malignancies. Experienced clinicians and epidemiologists have long suspected that UV irradiation has a causal role in both nonmelanoma skin cancer and melanoma. Studies of hairless mice and other animal models have demonstrated this relation repeatedly since the 1920s (4, 5). Particularly for squamous cell carcinomas, the relation is direct, with more UV irradiation resulting in earlier onset and higher prevalence of cancers in both mice and humans (4, 6). A direct cause-and-effect relation for UV irradiation and basal cell carcinomas has also been documented in a mouse model (7). Although the dose-response relation between UV irradiation and melanoma is less obvious than for nonmelanoma skin cancer, at least in humans, studies have documented a cause-effect relation in multiple animal models (8, 9). In addition, patients with the rare disorder xeroderma pigmentosum, caused by a mutation in 1 of 8 DNA repair enzymes

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required to correct UV-induced DNA damage, develop nonmelanoma skin cancers and melanomas at least 1000 times as frequently as the general population beginning early in life, even when they attempt to avoid all sun exposure (10).

PUBLIC ATTITUDES TOWARD SUN EXPOSURE

In 1903, Niels Ryberg Finsen received the Nobel Prize for observing that sun exposure was therapeutic for cutaneous tuberculosis (11), and the idea that UV radiation exposure was healthful rapidly took hold among the public (12). In the 1920s, Coco Chanel championed the idea that sun tanning was glamorous (13). Like many of her pronouncements, the concept of a tan’s attractiveness became embedded in the public psyche and remains there to this day, nearly a century later, despite the revised medical and scientific perception of a tan as a DNA damage response (14) and widespread appreciation that UV radiation often leads to skin cancer (15).

The public perception that sunbathing is pleasant and that a suntan is attractive continues to motivate many people, especially teenagers and young adults, to attempt to tan their skin (16). These young people have a well-documented inability to imagine themselves being affected by photoaging and skin cancer when they become middle-aged or elderly.

ULTRAVIOLET ACTION SPECTRA AND BIOLOGICAL RESPONSES

In the 1980s, studies of healthy human volunteers and multiple narrow-band UV light sources determined the relative efficacy of different wavelengths of light in producing sunburn and suntan (17) as well as epidermal DNA damage (18). The action spectra for all these responses are strikingly similar, with peak efficacy in the UVB portion of the spectrum (17–290–300 nm) and efficacy reduced by approximately an order of magnitude at 313 nm (still in the UVB range) and by 4–5 orders of magnitude by 400 nm, the beginning of the visible spectrum (17, 18). An overlapping group of researchers determined the action spectrum for vitamin D photosynthesis in skin in the same manner and found it to be extremely similar, also peaking at ~300 nm and falling off exponentially with longer wavelengths of light (19; Figure 1). The virtual identity of these multiple action spectra implies that DNA damage is responsible for tanning [experiments have confirmed this relation (14)] and that vitamin D photosynthesis cannot occur in the absence of DNA damage, even though vitamin D production is a consequence of UV effects on cell membranes rather than on DNA itself (20).

Formation of DNA photoproducts is linear with UVB dose over a very wide range (18). In contrast, pre-vitamin D conversion to the inactive photoproducts lumisterol and tachysterol balances vitamin D photosynthesis (conversion of 7-dehydrocholesterol to pre-vitamin D) (20). Hence, the concentration of pre-vitamin D remains there to this day, nearly a century later, despite the revised medical and scientific perception of a tan as a DNA damage response (14) and widespread appreciation that UV radiation often leads to skin cancer (15).

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SKIN PHOTOTYPE INFLUENCES ACUTE AND CHRONIC ULTRAVIOLET RADIATION RESPONSES

The content of epidermal melanin, a large polymer that efficiently absorbs photons across the entire UV and visible light range, and a related but less well understood set of determinants (termed phototype) substantially determine the effects of UV radiation on human skin (23). An individual’s phototype reflects the extent of sunburning versus subsequent tanning after an initial moderate sun exposure after a long period of little or no exposure (27; Table 1). Phototypes strongly affect the acute and chronic risks of UV exposure and the rate of vitamin D photosynthesis (Figure 3).

Phototype I or II skin burns readily with a first moderate UV exposure and then tans minimally, if at all (23, 27). Persons with this type of skin achieve maximal vitamin D photosynthesis rapidly after, for example, ~2–8 min of midday spring or summer sun exposure in New York or Boston and only slightly longer in Alaska or Scandinavia (28). With longer and repeated sun exposures, such persons suffer very substantial DNA damage that is eventually manifested as photoaging and skin cancer. Persons with phototype III skin, who commonly have a reasonably light baseline complexion, experience DNA damage and produce vitamin D at similar rates to persons with phototype I or II skin after a first UV exposure, but burn less and tan more readily (23, 27). With multiple exposures, the tanning response dominates, reducing the rate of vitamin D photosynthesis. After comparable UV exposure, persons with phototype III skin have considerably less cumulative DNA damage and hence less severe photoaging and less skin cancer risk than do those with phototype I or II skin (23). Constitutively high epidermal melanin content protects persons with phototype VI skin, who often have African
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The pseudo-controversy

In recent years, numerous newspaper reporters, freelance journalists, and television news anchors have reported on the “medical controversy” that pits the unwanted effects of acute sunburn, photaging, and skin cancer against both well-established and postulated benefits of vitamin D photosynthesis. These reports rarely note that sun exposure also produces tanning, at least in persons genetically capable of tanning, which is a cosmetic and lifestyle goal of many viewers or readers. Simplistically stated, the question posed by these articles and reports is: should the public maximize vitamin D levels through intentional UV exposure to reduce their risk of internal cancers, hypertension, diabetes, multiple sclerosis, and a litany of other disorders that some believe are due in part to “insufficient” vitamin D levels (28)? By framing the issue in this way, the media reports ignore the fact that people can obtain ample vitamin D from a combination of diet, supplements, and incidental protected sun exposure (30).

Reports often cite studies that measured low or low-normal concentrations of vitamin D in darkly pigmented individuals, such as inner-city minority groups, or among the frail elderly to justify promoting unprotected sun exposure. However, such coverage rarely notes that these at-risk groups have inefficient cutaneous vitamin D photosynthesis. In darkly pigmented people, melanin absorbs the UV photons that generate vitamin D (29), and in the elderly, their thinned epidermis may contain less 7-dehydrocholesterol, the cell membrane constituent that UVB converts to pre-vitamin D (29), and in the elderly, their thinned epidermis may contain less 7-dehydrocholesterol, the cell membrane constituent that UVB converts to pre-vitamin D (29).

A recent study (32) of a convenience sample of 93 healthy young adults recruited from the University of Hawaii and a Honolulu skateboard shop questioned the frequently suggested serum 25(OH)D sufficiency cutoff of 75 nmol/L. The investigators recruited these prototypic “surfer dudes” [mean age, 24 y; mean body mass index (in kg/m²), 23.6] on the basis of a self-reported minimum outdoor sun exposure of 15 h (mean, 29 h) per week during the preceding 3 mo; 40% reported never using sunscreen, and the group overall reported an average of 22.4 h per week of unprotected sun exposure. All were clinically tanned. Nevertheless, the group’s mean 25(OH)D concentration, measured by 2 standard techniques (HPLC and radioimmunoassay), was 79 nmol/L, and 51% had a level below the suggested 75-nmol/L cutoff for sufficiency (32). The study group was multi-racial, but even among the 37 white subjects, the mean value was only 92.8 nmol/L and the highest value was 155 nmol/L (32). These data suggest that a public health goal of >75 nmol/L, not to mention >150 nmol/L, for the entire population might be unachievable by unprotected sun exposure.

That article also highlights a little-emphasized aspect of vitamin D insufficiency. Specifically, most persons with 25(OH)D concentrations <75 nmol/L have no detectable disease or health problems and probably never will. They appear perfectly healthy. Instead, the definition relies completely on statistical associations between a low vitamin D concentration and one or (sometimes) more diseases, a definition that is variably and imprecisely articulated in the literature (1), all of which affect only
a small minority of sufficient or insufficient groups. This suggests that the great majority of persons receive no detectable (at least no detected) benefit from a 25(OH)D concentration >75 nmol/L and, conversely, no harm from a lower level. Even more curious, in many instances, the statistical associations on which the insufficient status is based are not with measured 25(OH)D concentrations but instead with presumptive correlates such as insolation (amount and intensity of incident UV irradiation) in the general geographic region of residence. Latitude, altitude, season, cloud cover, smog, and other variables affect insolation, which is generally high near the equator and low near the poles; and lifestyle choices introduce enormous variation in sun exposure even among individuals in identical climates.

The safe sun position, as articulated by the American Academy of Dermatology (33) and other professional dermatologic organizations for many years, is based on the irrefutable facts that UV irradiation causes nonmelanoma skin cancer, melanoma, and photoaging; the only established health benefit of UV irradiation is vitamin D photosynthesis; and vitamin D can be obtained from the diet or from oral supplements. These organizations therefore recommend lifelong sun protection, especially for fair-skinned individuals at high risk of photodamage (1).

THE TRUE CONTROVERSY

The real controversy is whether increasing a person’s conventionally normal serum 25(OH)D concentration has health benefits, as some epidemiologic studies have suggested but prospective randomized studies have not confirmed (1, 33). A thorough discussion of the quality and consistency of the epidemiologic and observational data, which some have interpreted to support a health benefit of serum 25(OH)D concentrations far above those associated with normal skeletal maintenance, is available elsewhere (1) and is beyond the scope of this discussion.

A recent example is illustrative. Several much-referenced reports link colorectal cancer incidence (26, 34, 35) to low vitamin D concentrations within the conventional normal range or a presumptive proxy, little sun exposure, which is usually based on residence in a poorly insolated area, as noted above. Although other epidemiologic or observational studies of similar size and design (grade B, level 2 or 3 in the hierarchy of evidence-based medicine; 36) found no statistical relation or even an inverse relation between sun exposure and colorectal cancer or closely related diseases (37–41), the popular media coverage of the topic has selectively and prominently cited the positive reports at the suggestion of interviewed “experts.”

However, a prospective, randomized, placebo-controlled trial (grade A, level 1 for medical decision making; 36) of vitamin D supplementation (400 IU/d) for 7 y or longer involving >36,000 postmenopausal women found no relation between colorectal cancer risk (incidence or mortality; tumor grade, stage, or size) and supplement use, total vitamin D intake, or amount of sun exposure (crudely and indirectly calculated, as in the positive epidemiologic studies; 42). Although the investigators found an inverse correlation with baseline serum 25(OH)D concentrations, they found no indication that increasing initially low vitamin D concentrations by supplementation decreased cancer risk over the subsequent 7 y (42). An accompanying editorial (43) and the investigators themselves noted that 7 y of supplementation might be too short, the subjects might have received a dose of vitamin D that was too low, they might have had a lifestyle that was too healthy, or they might have been too young (62 y on average) to develop this cancer in large numbers. In brief, the authors concluded that no result is ever definitively negative. Nevertheless, it is most unlikely that even larger, longer-lasting randomized controlled trials than this multimillion dollar effort will ever be performed. Yet, <2 mo later, the media prominently covered a far less definitive multivariable model study inversely linking cancer, including colorectal cancer, risk statistically to 6 indirect historical measures of sun exposure and presumptively correlated vitamin D concentrations (44), with no reference to the gold-standard negative colorectal cancer study (42). Such selective reporting continues through the present.

IRRELEVANCE OF BOTH CONTROVERSIES TO SUN PROTECTION

A neglected but critical point is that the true, optimal level of 25(OH)D for musculoskeletal health, cancer prevention, or any of the other claimed benefits is irrelevant to the proven value of sun protection. Whatever this optimal level, ample vitamin D can be obtained from diet, supplements, and incidental sun exposure (45–48). Intentional unprotected sun exposure to increase vitamin D photosynthesis is not only unnecessary but also inefficient for populations at highest risk of vitamin D deficiency (29–31).
The groups most responsive to the media’s unprotected sun exposure message are those at statistically lowest risk of vitamin D deficiency: healthy, fair-skinned adolescents and young adults. Indeed, surveys in the United States show that >70% of tanning bed users are white women aged 16–49 y (16), and 95% of all users exceed the exposure levels recommended by the US Food and Drug Administration (49) to maximize vitamin D photosynthesis. The demographics and exposure habits of the sunbathing public are similar to those of tanning bed users, although the average age is probably even younger and exposures even greater. The safe-sun message promulgated by dermatologists and the American Academy of Dermatology does not target dark-skinned persons, who already have excellent endogenous sun protection in the form of epidermal melanin. Moreover, the groups at demonstrated risk of vitamin D deficiency have not embraced the “UV advantage” message (28), perhaps because this message does not target them.

The interest among the media and public in the pseudo-controversy is nevertheless real and persistent. Why? The sun protection message is old, dating back at least 23 y (50), and its intended audience views it, like the “bucket up” seatbelt message, as wimpy. Real men and rebellious, fun-loving, and spontaneous adolescents do not wear sunscreen (or seatbelts). Moreover, many persons, especially teenagers, want to sunbathe not to decrease their risk of age-associated disease decades later but to acquire a “sexy” tan (15). In addition, relaxing in the sun and making one’s own vitamin D have a back-to-nature holistic appeal for many persons. It is therefore not surprising that the print and electronic media continue to cover the pseudo-controversy; it sells. However, press releases crafted by representatives and employees of the indoor tanning industry have greatly facilitated the media’s natural tendency to pursue a controversial story, especially if it is one their audience wishes to hear.

THE INDOOR TANNING INDUSTRY

In the United States alone, the indoor tanning business earns $5 billion per year (51, 52) and has >50 000 tanning facilities, 28 million customers annually (53), and >1 million visits per day (54). In some regions of the United States, more than half of all teenage girls have visited a tanning facility at least 3 times in the previous year (55).

Because professional groups such as the American Academy of Dermatology have requested stricter guidelines and better enforcement of existing regulations governing indoor tanning, over the past decade, at least 29 states have enacted legislation restricting access to tanning parlors for teenagers, and at least 3 additional states are considering similar legislation (R Bohannon, State Affairs Division, American Academy of Dermatology Association, personal communication, May 2006). The indoor tanning industry vigorously opposes such legislation through paid lobbyists and a well-orchestrated media campaign. The cornerstone of the industry’s argument to curtail proposed restrictions on teenage use and general overuse is that more UV exposure is healthy, indoor tanning is safer than natural tanning, and UV radiation exposure reduces the risk of multiple diseases (56). The message that the tanning industry has shared with state legislatures and the media and that the UV Foundation’s website publicizes is: “Vitamin D from UV exposure is free and easy to get—why pass up the simplest way to improve your odds of preventing cancer?” (57). Similar websites describe industry funding of research by the principal and perhaps only academically based proponent of UV exposure to increase vitamin D concentrations (58).

The indoor tanning industry’s concern for the public’s health would be more credible if its coverage of the issues were more balanced and a decade or so of extolling the virtues of UVA lamps (not the UVB lamps that it now touts as healthful) had not preceded the campaign (2, 59, 60). Before publication of the epidemiologic studies questioning the adequacy of conventional vitamin D recommendations, the industry argued strenuously that indoor tanning was superior to natural sun exposure precisely because people could tan with less UVB exposure (and, of course, less vitamin D photosynthesis; 61). Indeed, a review of the industry’s public positions over the 30 y of its dramatic growth in annual revenues (12) reveals a series of opportunistic, contradictory positions. There can be no doubt that the business of the tanning industry is to sell tanning sessions, not to safeguard the public’s health.

THE APPEAL OF NATURAL SOLUTIONS

Over millions of years, life has adapted beautifully to the earth’s environment. Nature has devised elegant, efficient, and often surprising solutions to complex problems, and humans are rarely able to improve on them. Exceptions to this rule occur when the rate of change that civilization has imposed outpaces evolution by modifying the environment in ways that create previously nonexistent downsides for the natural solution. Vitamin D photosynthesis is a prime example.

Humans evolved as relatively hairless, darkly pigmented beings in a highly insulated tropical subsistence environment. Their abundant epidermal melanin absorbed most of the incident UV photons, allowing them to avoid painful sunburns while hunting and gathering food. However, sunlight’s high UVB content permitted epidermal photochemistry, including conversion of membrane lipids to vitamin D, the biologically inactive precursor of the hormone 1,25(OH)2D that requires hydroxylation steps in the liver and kidney before acquiring the ability to modulate genes in cells throughout the body (26). Metabolic spillover pathways that convert excess vitamin D to inactive metabolites in the skin during prolonged UV exposure prevented overproduction of the precursor molecule. The ability to photosynthesize vitamin D avoided the requirement for dietary vitamin D in this environment with its unpredictable and often inadequate food supply. Life expectancy was far <40 y (62) and therefore there was an enormous priority for health in the first decades, a time sufficient to permit reproduction. Very gradually, humans migrated away from the equator to far less insulated climates and skin color gradually lightened, giving rise, for example, to the fair-skinned, blue-eyed, blond populations of Scandinavia and northern Europe. Although quite speculative, one appealing explanation for the complexion change in humans who migrated north is that acute UV damage became less problematic and maintaining adequate vitamin D concentrations became a priority.

In recent centuries, humans have become far more mobile, migrating thousands of miles in weeks or, more recently, hours. During this period, many people moved to cities. As a result, large populations of dark-skinned individuals now live primarily indoors in poorly insulated climates, and many fair-skinned persons live in relatively well-insulated places, spending recreation and sometimes work time outdoors and intermittently traveling to highly insulated places for business or pleasure.
these trends, life expectancy has increased dramatically in recent centuries and now approaches or exceeds 80 y in much of the developed world (63). This has led to decades of progressive photoaging and an exponential increase in annual skin cancer incidence between the 4th and 8th decades that is due, at least in part, to age-associated decreases in DNA repair capacity (24, 25).

In addition, childhood rickets has emerged among dark-skinned, inner-city ethnic minorities in the northern United States and Europe (64, 65), and fair-skinned whites now have a 1 in 3 lifetime skin cancer risk.

These facts imply that fair-skinned persons benefit enormously from regular, lifelong safe sun practices. Moreover, while wearing sunscreen with a high sun protection factor (SPF) in season, such persons probably generate vitamin D maximally in exposed areas during incidental sun exposure (1). Although some have claimed that sunscreens block all UV and hence all vitamin D photosynthesis (22), this is not the case. By definition, sunscreens allow continuous transmission of a fraction of erythemogenically weighted incident UV photons equal to 1/SPF of the total (eg, 1/15th or 7% for an SPF 15 product). Moreover, studies have shown that sunscreen users customarily apply half or less of the FDA-stipulated amount of product required to generate the stated level of protection (2 mg/cm²) and hence achieve far less protection (66). If persons require 2–8 min of unprotected summer sun exposure to optimize cutaneous vitamin D synthesis (28), they could accomplish this in ≈10–20 min of exposure after applying an SPF 15–30 sunscreen in the customary manner (66, 66). Most critically, regardless of one’s complexion or extent of UV exposure, daily oral vitamin D supplementation can completely compensate for lack of cutaneous vitamin D photosynthesis (1).

AREAS OF UNCERTAINTY AND RESEARCH NEEDS

The longstanding pseudo-controversy has led many responsible professional groups to wonder whether to recommend a safe or prudent amount of unprotected sun exposure to the public concerned about skin and overall health or even cosmetic tanning. The risk-benefit ratio of sun exposure and probably of high 25(OH)D concentrations varies enormously within the population. Moderate or even generous sun exposure might have little effect on a darkly pigmented person’s risk of subsequent photoaging and skin cancer while promoting higher 25(OH)D concentrations, but it could promote development of precancerous and even cancerous lesions in already photodamaged fair skin without increasing the already maximized vitamin D photosynthesis.

Solar UVB intensity varies enormously with latitude, altitude, time of day, and time of year, among many other variables (68). UVA radiation varies far less in intensity and is far more abundant in sunlight than UVB radiation (69), so unprotected late-summer-afternoon or midday-winter exposure might involve almost no UVB exposure (and hence no vitamin D synthesis) but might still contribute to photoaging and photocarcinogenesis. A rule of thumb might be that any sunburn dose is too much by a factor of ≥3, because maximal vitamin D synthesis is achieved after approximately one-third of a minimal erythema dose (21).

Individuals who never sunburn or who live in climates that never allow them to sunburn are relatively safe from the damaging effects of unprotected sun exposure. Persons with complexions or living circumstances associated with the possibility of frequent sunburns probably have no safe minimum unprotected exposures, because these would be only a few minutes and would almost certainly be exceeded cumulatively on a daily basis during the course of routine activities.

All clinicians, investigators, and public health officials interested in vitamin D biology, photocarcinogenesis, or skin biology and pathophysiology can probably agree that more research in overlapping areas is desirable. Perhaps the most clinically important questions are: Does an inverse cause-effect relation exist between higher 25(OH)D concentrations and cancer incidence, hypertension, diabetes, multiple sclerosis, and other conditions for which research has noted inverse epidemiologic associations (1)? Does having a higher than conventionally recommended serum 25(OH)D concentration produce a health benefit, or even a future health benefit, in healthy children and adults? If such a benefit exists, what is the minimum duration required for maintaining high 25(OH)D concentrations (eg, throughout life or only for a period of months or years)? Implicit in these questions is the fact that one cannot deduce cause-and-effect relations from epidemiologic studies, which are inevitably confounded by indirect and group-averaged measures of key variables, socioeconomic factors, racial and genetic factors, and lifestyle associations.

In contrast with these understudied areas, randomized, prospective controlled trials among frail elderly groups strongly suggest that such individuals benefit from daily oral supplementation of ≥800 IU of vitamin D, which enhances muscle strength and decreases falls, reducing bone fracture risk (1). These data imply that the present recommended daily allowance (600 IU/d) for vitamin D in those ≥70 y old (69) is probably inadequate and that increasing vitamin D intake in frail older individuals, particularly those who are housebound or institutionalized, would probably confer a health benefit. Other research has shown that doubling or tripling the standard vitamin D supplement doses or fortifying more foods with vitamin D would be very safe (45, 70). Signs of vitamin D toxicity only appear after daily doses exceeding 10 000 IU (45), so the safety margin is broad.

One area does not require further research, at least to resolve the present controversy. Overwhelming data, briefly summarized above, establish UV radiation as a carcinogen responsible for ≥1 million skin cancers per year in the United States alone (3), as well as for photoaging (71), an essentially universal problem among whites in middle age and beyond. These data also show that lifelong safe sun practices minimize both risks. With continued goodwill and enhanced communication, one can hope that the “controversy” surrounding the “sunshine vitamin” will become a thing of the past.

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REFERENCES

7. Aszterbaum M, Epstein J, Oro A, et al. Ultraviolet and ionizing radiation...
57. Westerdahl J, Ingvar C, Mabback A, Jonsson N, Olsson H. Risk of


