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Review

In defense of the sun

An estimate of changes in mortality rates in the United States if mean serum 25-hydroxyvitamin D levels were raised to 45 ng/mL by solar ultraviolet-B irradiance

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Key words: cancer, cardiovascular diseases, melanoma, respiratory infections, skin cancer, vitamin D, ultraviolet-B

Emerging scientific evidence strongly supports the beneficial role of vitamin D in reducing the risk of incidence and death from many chronic and infectious diseases. This study estimates increases in melanoma and nonmelanoma skin cancer mortality rates and decreases in chronic and infectious disease mortality rates in the US from the standpoint of approximately doubling population doses of solar UVB to increase mean serum 25-hydroxyvitamin D levels from 16 ng/mL for black Americans and 25 ng/mL for white Americans to 45 ng/mL. The primary benefits are expected to come from reductions in cancer and cardiovascular diseases. Although a few thousand excess deaths per year might occur from melanoma and skin cancer, the avoided premature death rate could be near 400,000/year, with most of the avoided deaths coming late in life. While oral sources of vitamin D could be used instead of UVB or when UVB irradiance is not available, public health policies do not yet recommend the 3,000–4,000 IU/day required to raise serum 25-hydroxyvitamin D levels to the levels required for optimal health, which would be required before vitamin D fortification levels in food can be raised. Until then, moderate solar UVB irradiance remains an import source, and the health benefits greatly outweigh the risks.

Introduction

Solar UVB (290–315 nm) irradiance correlates with reduced risk of about 14 types of cancer.[1-4] It is hypothesized to explain the latitudinal variation of multiple sclerosis,[5] the seasonality of epidemic influenza,[6] the epidemiology of septicemia,[7] and case fatality rates during the 1918–1919 influenza pandemic.[8] The beneficial effect of ultraviolet irradiance (UVR) arises from production of vitamin D. Serum 25-hydroxyvitamin D [25(OH)D] level also inversely correlates with incidence and/or mortality rates of other diseases such as type 2 diabetes mellitus,[9,10] coronary heart disease (CHD)[11] and congestive heart failure.[12]

Let us put vitamin D production into the context of human history on Earth. The human species originated in the eastern portion of tropical Africa. Skin pigmentation in that region was very dark to protect against the adverse effects of solar UVR, primarily free radical production and DNA damage leading to melanoma and other skin cancer.[13] Because UVB doses were high and clothes were not worn, sufficient UVB penetrated the epidermis to produce adequate vitamin D. As people migrated poleward from the tropics, skin pigmentation lightened to become very pale in northern Europe because those with dark skin had lower survival rates because of rickets and both chronic and infectious diseases.[14]

One underlying reason for concern about skin cancer and melanoma today is that many people with skin that has adapted for life at high latitudes are now living at lower latitudes, where their skin pigmentation does not afford adequate protection against the adverse effects of solar UV. Conversely, many with dark skin have moved poleward and have chronically low serum 25(OH)D levels and, as a result, higher disease rate.[15,16]

This report will estimate the health benefits and risks of increasing solar UVB irradiance and oral intake of vitamin D to increase mean serum 25-hydroxyvitamin D levels from 16 ng/mL for black Americans and 25 ng/mL for white Americans[17,18] to 45 ng/mL, a level that seems to be in the range required for optimal health,[19,20] which requires the production from UVB irradiance or oral intake of about 3,600 IU/day.[21,22] The indices used for this study are mortality rates for diseases affected by either vitamin D or UVR leading to death. Although incidence and prevalence rates and the economic burden could also be used, they should yield similar results.

Results

The index used to estimate the changes in health due to increased serum 25(OH)D levels is mortality rates of UV- and vitamin D-sensitive diseases. It is assumed that raising mean serum 25(OH)D levels for white Americans from 25 ng/mL to 45 ng/mL would take 2–2.5 times the current solar UVB irradi-

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Table 1 **Death rates, white males, 2005**[23]

Disease	Vit D (%)	Death rate by age (y)								
		20–24	25–29	30–34	35–39	40–44	45–49	60–64	70–74	80–84
Melanoma			0.7	0.9	1.1	2.2	3.1	9.0	18.3	28.3
Melanoma and NMSC increases			0.9	1.2	1.4	2.9	4.0	11.7	23.8	36.8
Septicemia	25	0.4	0.6	0.6	1.1	2.0	3.6	15.8	41.8	113
Cancers										
Esophageal						1.8	3.9	22.0	40.4	54
Gastric				0.4	0.6	1.3	2.0	8.6	20.7	39
Colorectal			0.5	1.0	2.1	4.1	7.6	40.0	92.9	180
Pancreatic					0.7	2.2	5.1	30.4	62.0	96
Renal					0.4	1.2	2.8	15.7	29.6	44
Bladder					0.3	0.5	1.4	7.1	19.8	22
Lymphoma		0.5	0.7	0.7	1.4	2.5	3.5	16.3	43.5	87
These cancers	35	0.5	1.2	2.1	5.5	13.6	26.3			
Total cancer	20	5.4	7.0	10.0	17.1	38.7	79.2	470	1083	1895
Diabetes	15	0.6	0.9	1.9	3.5	6.5	11.3	51.0	112	235
Ischemic heart		0.8	1.9	5.0	12.7	32.7	64.1	282	647	1799
Acute myocardial infarction		0.4	0.8	1.9	4.9	13.1	26.3	114	241	573
Atherosclerotic cardio			0.3	0.9	2.7	7.0	14.6	51.2	82.8	195
Total coronary heart	15	1.4	3.0	7.8	20.3	59.3	105.0	447	971	2567
Heart failure	20					0.8	1.5	12.5	48.4	218
Cerebrovascular	15	0.5	0.8	1.6	2.8	5.5	9.5	41.8	138	505
Influenza, pneumonia	30	0.6	0.7	1.0	1.6	2.7	4.5	16.1	56.5	254
Asthma	15	0.2	0.4	0.3	0.4	0.6	0.6	0.9	1.4	4.1
Falls	30	1.4	1.4	1.3	1.9	2.8	3.7	8.4	22.4	85
Total avoided death rate		1.7	2.5	4.2	8.8	20.0	34.1	189	445	1050
Ratio of avoided to increased mortality rates			2.8	3.5	6.3	6.9	8.5	16.2	18.7	28.5

ance and that the increase in serum 25(OH)D is achieved through increased time in the sun. The only adverse effects of increased UVR listed in Tables 1–4,[23] are melanoma mortality rates. However, NMSC mortality rates would also increase. In 2009, the American Cancer Society estimates that 8,650 melanoma deaths will occur and 2,940 NMSC deaths (34% as many as for melanoma).[24] To first order, total UVR-related deaths for white Americans could increase by a factor of 1.34 times the 2009 mortality rates. However, some of the increase in serum 25(OH)D can be achieved by exposing more body surface area and by taking supplements, so this value should be considered an upper limit.

The beneficial effects of increasing serum 25(OH)D levels from a population mean of 25 ng/mL to 45 ng/mL for white Americans and from 15 ng/mL to 45 ng/mL for black Americans as a function of age range are estimated based on the data presented in Table 5. The values are given as a column in Tables 1 and 2 for white Americans and Tables 3 and 4 for black Americans. The values used in this study are similar to those used in a study of vitamin D deficiency in western Europe.[31] However, the estimate for CHD is somewhat lower than in that study. The reason is that the epidemiological studies on vitamin D and CHD and precursor metabolic disease to date are primarily cross-sectional[32-34] or observational[11,35-37] in nature, with no ecological studies and limited randomized controlled trials (RCTs), but with low vitamin D doses.[38] The lack of ecological studies showing an inverse correlation with UVB indices for CHD indicates that other factors such as diet, genetics and smoking are more important risk factors for incidence and mortality than vitamin D. Several proposed mechanisms explain the beneficial roles of vitamin D for CHD, such as reduced risk of calcification of the arteries,[39] negative influence on the renin-angiotensin-aldosterone system,[40] and increased insulin sensitivity.[41] On the other hand, there are many non-vitamin D risk-modifying factors for cancers, yet ecological studies have usefully demonstrated links to UVB.[42-44] Again, many proposed mechanisms seem to explain the beneficial role of vitamin D in reducing the risk of cancer.[45,46] This study assumes a 15% reduction for CHD for whites and 20% for blacks. Until results of RCTs with sufficient vitamin D doses are reported, considerable uncertainty persists in these values.

Tables 1–4 give estimates of the changes in mortality rates for those aged 20–24 to 71–74 years. For white males and females, the ratio of avoided deaths to melanoma deaths rises from a factor of 2.8 for males and 4.2 for females in the 25- to 29-year age range to a factor of 28.4 for males and 52.4 for females in the

Table 2 **Death rates, white females, 2005**[23]

Disease	Vit D (%)	Death rate by age (y) (deaths/100,000/year)								
		20–24	25–29	30–34	35–39	40–44	45–49	60–64	70–74	80–84
Melanoma			0.4	0.6	1.0	1.3	2.1	4.7	7.4	10.9
Melanoma and NMSC increases			0.5	0.8	1.3	1.7	2.7	6.1	9.6	14.2
Septicemia	25	0.3	0.4	0.8	1.3	1.8	2.9	12.4	33.9	89
Cancers										
Esophageal						0.3	0.6	3.6	8.4	12
Gastric					0.5	0.9	1.5	3.9	9.7	20
Colorectal			0.3	1.1	1.5	3.4	6.0	24.8	59.2	127
Pancreatic					0.5	1.5	3.6	19.3	44.0	82
Breast			0.6	2.4	6.7	13.3	20.7	61.1	88.6	134
Endometrial					0.5	0.8	1.8	10.6	18.0	28
Ovarian			0.3	0.5	1.1	2.7	5.7	24.4	41.6	58
Renal					0.3	0.7	1.2	6.7	13.1	23
Bladder						0.3	0.5	3.2	9.1	22
Lymphoma		0.3	0.3	0.8	0.6	1.0	2.1	10.8	27.0	61
These cancers	35	0.3	1.5	4.8	11.7	24.9	43.7			
Total cancer	25	3.9	5.5	11.7	23.3	47.3	84.8	362	728	1143
Diabetes	15	0.4	0.7	1.2	2.5	3.3	5.9	33.2	80.7	176
Ischemic heart			0.5	1.8	4.1	9.2	18.1	105	312	1092
Acute myocardial infarction				0.7	1.8	3.9	7.7	44.3	121	372
Atherosclerotic cardio				0.3	0.7	1.5	3.4	16.6	39	130
Total coronary heart	15		0.5	2.8	6.6	14.6	29.2	166	472	1594
Heart failure	20					0.5	1.0	8.6	37.2	177
Cerebrovascular	15	0.5	0.7	1.4	2.7	5.2	8.6	31.1	112	482
Influenza, pneumonia	30	0.4	0.7	0.9	1.1	2.1	3.2	11.4	39.2	173
Asthma	15		0.4	0.5	0.6	0.9	1.3	2.2	3.0	7.9
Falls	30	0.3			0.4	0.8	1.3	3.7	13.6	55
Total		1.2	2.1	4.2	8.4	16.7	26.9	139	312	744
Ratio of avoided to increased mortality rates			4.2	5.3	6.5	9.8	10.0	22.8	32.5	52.4

80- to 84-year age range. No estimates are given for black Americans because of lack of data for melanoma at various ages; they have lower melanoma rates than white Americans but comparable NMSC mortality rates.[47]

The data in Tables 1–4 along with Census Bureau data could be used to estimate the total avoided death rates. However, it is also possible to use total mortality rate for each vitamin D-sensitive disease. Work in progress estimates that increasing mean serum 25(OH)D levels in the US to 45 ng/mL would avoid 400,000 premature deaths per year, with cancers and cardiovascular diseases providing the largest shares (Grant et al. in preparation).

Discussion

The health benefits of solar UVB are much larger than the adverse effects represented by melanoma and NMSC mortality rates. This study's conclusion is similar to that in a report commissioned by the World Health Organization: "UVR exposure is a minor contributor to the world's disease burden, causing an estimated annual loss of 1.6 million DALYs; i.e., 0.1% of the total global disease burden. A markedly larger annual disease burden, 3.3 billion DALYs, might result from reduction in global UVR exposure to very low levels."[48]

Interestingly, although the estimated benefits for females are lower than for males, the ratio of avoided premature death rates to melanoma and NMSC death rates is higher for females. That outcome could be due to males spending more time in the sun in both occupational and nonoccupational activities. Nature has recognized that women need more vitamin D than males for pregnancy and lactation[49] in that skin pigmentation is lighter for females than for males in all ethnic groups.[13]

The estimates for melanoma and skin cancer mortality rates are considered to be an upper bound. For one reason, UVA, not UVB, is the primary spectral region of risk for melanoma, and chronic solar UV irradiance is protective against melanoma.[50,51] The reasons why chronic UV irradiance can be associated with reduced risk of melanoma incidence and mortality rates is probably threefold: production of vitamin D;[52,53] tanning and thickening of the stratum corneum to reduce penetration of UVA to the lower

Table 3 **Death rates, black males, 2005**[23]

Disease	Vit D (%)	Death rate by age (y) (deaths/100,000/year)								
		20–24	25–29	30–34	35–39	40–44	45–49	60–64	70–74	80–84
Melanoma										
Septicemia	35		1.6	2.1	3.6	5.5	10.9	43.4	97.1	239
Cancers										
Esophageal						2.1	5.1	31.3	41.4	49
Gastric					1.8	3.2	5.4	24.2	48.4	79
Colorectal				2.2	2.8	5.7	13.7	57.5	101.3	198
Pancreatic						2.3	7.2	39.6	71.5	117
Renal						1.9	3.6	19.6	29.1	39
Bladder								9.1	25.0	57
Lymphoma				1.9	1.7	2.8	4.4	14.7	29.1	37
These cancers	35			4.1	6.3	15.2	35.0			
Total cancer	30	6.1	9.3	14.7	21.7	50.0	127.0	730	1395	2354
Diabetes	15	1.4	2.9	6.7	10.8	12.3	25.9	118	237	392
Ischemic heart		1.3	4.2	7.9	23.0	41.2	95.4	455	857	1887
Acute myocardial infarction			2.1	2.9	7.5	15.8	34.4	145	278	598
Atherosclerotic cardio				1.7	6.0	8.9	23.1	130	201	362
Total coronary heart	20	1.3	6.3	12.5	36.5	65.9	127.1	730	1336	2847
Heart failure	30					4.6	6.4	32.7	82.5	224
Cerebrovascular	15		2.6	4.1	7.1	17.4	32.0	133	277	655
Influenza, pneumonia	35			2.2	3.4	6.1	10.5	33.3	90.5	259
Asthma	25	1.6	1.5	1.9	1.7	2.5	3.3	4.5	6.3	53
Falls	30				1.5	1.7	4.4	9.8	14.5	20
Total		2.7	5.8	10.6	18.4	35.0	78.2	443	860	1693

epidermis;[54] and generation of elastosis, which is associated with slower growth of melanoma.[55] Also, people need increase UVB only enough to produce sufficient vitamin D, which could be a few minutes a day near solar noon in summer.[56,57] The shadow rule favored by dermatologists,[58,59] is designed to reduce the risk of erythema. However, the ratio of UVB to UVA increases with solar elevation angle, so solar UVR near solar noon is most favorable for vitamin D production.[60] The time required for erythema to occur could be about 15 minutes for fair skinned individuals in midlatitude midday summer solar irradiance.[59,61]

In addition, skin cancer screening efforts could be increased. The combined mortality rate for melanoma and NMSC has more than tripled for males between 1950–1954 and 1990–1994 (3.30x) while declining slightly for females (0.96x).[62] Evidently increased sun avoidance and use of sunscreen reduced NMSC rates, whereas the same plus increased travel increased melanoma rates. Thus, the factor of 1.34 times combined melanoma and NMSC mortality rates is considered a reasonable estimate of the increased mortality rate.

A few additional caveats are involved in relying on solar UVB irradiance for vitamin D production. First, those with red hair and freckles should generally avoid any intense solar UV irradiance because of their increased risk of developing melanoma and limited ability to tan.[63] Second, vitamin D production rate decreases with age, with those older than 60 years requiring three to four times longer in the sun than those younger than 20 years.[64] Because solar UV also destroys vitamin D at wavelengths between 290 and 330 nm, spending more time in the sun does not produce more vitamin D after a certain point. Third, those with very dark skin require about five times as long to produce vitamin D as those with fair skin.[57] Fourth, it might be advisable to wear a brimmed hat when in the sun as the head and hands are generally exposed when in the sun and since UVB is highly scattered by the atmosphere, there is much diffuse UVR hitting the face.[65] Finally, avoid erythema by limiting time in the sun without protection as much of the risk of melanoma is probably due to sunburning.[66] Recent studies found that about 30% of adults become sunburned each year,[67] with sunburn frequency rising to 61% for those aged 18–24 years.[68]

Although the ratio of avoided deaths to increased melanoma deaths is low at younger ages, it does not mean that people should avoid moderate UV irradiance from the sun in early life. A European study found that the number of sunburns, but not the age at which they occurred, was an important risk factor for melanoma[69]—recently repeated in another study.[66] On the other hand, nevi, which develop in early childhood from UV irradiance,[70-72] are an important risk factor for melanoma.[73,74] No evidence has been presented that use of artificial UV sources

Table 4 **Death rates, black females, 2005**[23]

Disease	Vit D (%)	Death rate by age (y) (deaths/100,000/year)								
		20–24	25–29	30–34	35–39	40–44	45–49	60–64	70–74	80–84
Melanoma										
Septicemia	35		1.4	2.6	3.7	5.5	9.2	34.4	70.3	185
Cancers										
Esophageal							1.6	6.5	41.4	49
Gastric						1.5	2.8	6.8	17.5	46
Colorectal					2.7	5.6	11.6	44.5	85.1	180
Pancreatic						2.2	5.3	28.9	63.0	99
Breast			1.6	5.6	14.7	25.4	43.0	83.9	101.5	140
Endometrial							2.5	26.9	38.3	42
Ovarian						2.7	4.3	19.7	37.7	48
Renal								6.2	9.5	19
Bladder								3.4	9.3	25
Lymphoma						2.0	2.1	8.9	14.7	29
These cancers	40		1.6	5.6	17.4	39.4	73.2			
Total cancer	35	4.1	9.0	16.3	33.8	70.0	131.0	452	774	1180
Diabetes	15	1.7	2.6	3.6	5.7	9.5	16.2	91	194	369
Ischemic heart				3.0	8.8	20.5	38.6	217	489	1322
Acute myocardial infarction					3.6	8.4	15.3	79	175	447
Atherosclerotic cardio					1.8	4.3	8.9	47	82	214
Total coronary heart	20			3.0	14.2	33.2	62.8	343	746	1983
Heart failure	30					1.6	4.0	22	60	212
Cerebrovascular	15		1.9	3.2	6.5	16.8	29.3	73	200	588
Influenza, pneumonia	35			1.4	1.8	3.9	5.7	17.7	53	168
Asthma	25	1.3		1.9	1.8	3.1	5.3	6.7	10.4	13
Falls	30							3.0	8.4	22
Total		2.0	4.3	9.2	18.9	31.3	72.8	279	545	1151

generates nevi, although the possibility does exist. Vitamin D has important health benefits at all ages, and several studies report early-life UVB irradiance associated with significant reduction for diseases later in life (e.g., multiple sclerosis[75] and prostate cancer[76]).

Because most modern sunbeds have spectral outputs with 3%–5% of the energy in the UVB spectral region, and raise serum 25(OH)D levels,[77-79] sunbed use should afford the same benefit-risk results as solar UVR. In fact, a recent study in Sweden found that women using sunbeds more than three times per year reduced their hazard ratio (HR) of endometrial cancer by 50% (0.5, 95% CI 0.3-0.9) and those women who were sunbathing during summer reduced their risk by 20% (HR, 0.8; 95% CI, 0.5–1.5) compared with women who did not expose themselves to the sun or to sunbeds.[80] If white Americans were to obtain their vitamin D through use of artificial UVB sources in the US, such as in winter, when producing vitamin D from solar UVB is often impossible,[57,60] using them once a week would produce 10,000–15,000 IU, sufficient to raise serum 25(OH)D levels by 6–15 ng/mL, so it would take about two visits per week to increase by 20 ng/mL.

For sunbed use, there are other caveats. First, the lamps used should have about 3%–5% of the UV in the UVB range. Second, the time required to produce 10,000–20,000 IU with whole-body UVB irradiance in a sunbed can be as short as a one to a very few minutes depending on the luminosity of the bulbs. In the US, bulbs are several times brighter than midlatitude midday solar UV; however, in Europe, lamp intensity is limited to midday Mediterranean solar UV, and the UVB to UVA ratio may be lower than in the US. Third, for sunbeds that employ high-pressure lamps near the head, the head should be covered. These bulbs emit only UVA, which oxidizes and darkens melanin; UVB and slightly longer wavelengths induce production of melanin. Ideally, these UVA lamps should be removed. Covering the groin area might also be advisable. Those with red hair and freckles and the type 1 Fitzpatrick skin phenotype should avoid using sunbeds.

Although an increase in melanoma and NMSC mortality rates from increased UVR is lamentable, the mortality benefit-risk ratio for all age groups combined is approximately 5–10 for males and 12–24 for females. The advantage of solar UVB is that it is free and not subject to government regulation. Supplements would be the most efficient way to obtain vitamin D, but obtaining

Table 5 **Recent results from the literature regarding disease outcome with measures of serum 25(OH)D or oral intake of vitamin D**

Disease incidence	Finding with respect to serum 25(OH)D level or vitamin D supplementation	Study type	Reference
Septicemia	Those with septicemia had mean serum 25(OH)D level = 16 ng/mL vs 26 ng/mL for healthy controls		25
Cancer (all)	35% reduction by increase from 73 nmol/L to 95 nmol/L	RCT	26
Breast cancer	OR = 0.56 (95% CI, 0.41–0.78) for ≥100 nmol/L vs. <50 nmol/L	CC	27
Colorectal cancer	50% reduction for 34 ng/mL vs. 6 ng/mL	Meta-analysis	28
Diabetes mellitus	A combined daily intake of >1,200 mg calcium and >800 IU vitamin D was associated with a 33% lower risk of type 2 diabetes with RR of 0.67 (0.49-0.90) compared with an intake of <600 mg and 400 IU calcium and vitamin D, respectively	Cohort	9
Acute myocardial infarction	RR1, 2.09; 95% CI, 1.24-3.54; P = .02 for trend for 25(OH)D (>30 ng/mL) vs <15 ng/mL	Cohort	11
Cardiovascular disease (death)	HR = 5.38 (95% CI, 2.02–14.34; p = 0.001) for first when compared to the upper three 25(OH)D quartiles	Cohort	29
Influenza, common cold	RR = 0.4 for 800 IU/day vs. placebo; =0.1 for 2,000 IU/day vs. placebo	CC	30
Pneumonia as a complication of influenza	Adjusted r^2 for case-fatality rate with respect to UVB index = 0.59 following incidence of A/H1N1 influenza in the U.S. in 1918	Ecologic study	8
All-cause mortality	HR = 1.97 (95% CI, 1.08–3.58; p = 0.027) for first when compared to the upper three 25(OH)D quartiles	Cohort	29

*WHO; CC, case-control; CI, confidence interval; FEV1, forced expiratory volume in the first second; FVC, forced vital capacity; COPD, chronic obstructive pulmonary disease; HR, hazard ratio; NHANES III, National Health and Nutrition Examination Survey III; OR, odds ratio; RCT, randomized controlled trial; RR, risk ratio; RR1, relative risk; SE, standard error.

high-dose vitamin D in European countries is difficult, and many people do not take supplements. Food can be fortified with vitamin D. In the US people obtain 250–300 IU/day of vitamin D from dietary sources.[81] The US National Academy of Sciences' Institute of Medicine has convened a Vitamin D and Calcium Dietary Requirements Committee to evaluate and revise the guidelines;[82] however, the committee seems to be restricting its sources of evidence unduly to clinical trials, downgrading observational and ignoring ecological studies in an attempt at evidence based medicine, i.e., RCT data,[83] of which there are very few with 1,000 IU/day or more. Until the recommended guidelines are changed, vitamin D fortification of food is highly unlikely to change.

Materials and Methods

The estimates of reductions in mortality rates are based on the available ecological, observational and cross-sectional studies and, where available, RCTs that used more than 400 IU/day of vitamin D. Table 5 lists the studies used in these determinations. Although many of these studies are considered nondefinitive (only RCTs seem to be considered definitive for vitamin D), those for many types of cancer are considered generally reliable, generally satisfying Hill's criteria for causality in a biological system.[84] Additional analysis of the evidence for a beneficial effect of vitamin D in reducing the risk of cancer is presented in ref. 85. Those for CHD are either observational or cross-sectional, with some laboratory support for mechanisms, and are considered less reliable currently.

The values for white Americans in this study are similar to those used in deriving an estimate for the economic benefit of higher serum 25(OH)D levels for western Europe. The values for black Americans are based on increasing serum levels from 15 ng/mL to 45 ng/mL, so they are higher than those for white Americans.

One can use the following approach to estimate the increased risk of melanoma from increased UV irradiance. In the US, about 90% of vitamin D is derived from solar UVB irradiance, with dietary sources providing about 250–300 IU/day.[81] The mean serum 25(OH)D for white Americans is about 25 ng/mL,[17,18] which decreased by 1–10 ng/mL between the early 1990s and the early 2000s. Similar effects have been found in Australia[86] and the UK.[87] The likely reason is increased fear of skin cancer and melanoma[88-90] and increased use of sunscreen. The role of sunscreen use in reducing serum 25(OH)D levels was recently disputed,[91] but the paper was based on insufficient serum 25(OH)D levels (<20 ng/mL) in sunny countries and did not consider the trends in these three countries. In addition, use of sunscreen has been linked to increased risk of melanoma at latitudes above 40, with the likely reason that sunscreens provide little protection against UVA, the spectral region of greatest risk for melanoma.[92] Casual irradiance in summer by those aged 45 years in the UK is sufficient to increase serum 25(OH)D from the winter value of 15 ng/mL to the summertime value of 30 ng/mL in a nearly sinusoidal manner.[93] Thus, raising mean serum 25(OH)D levels for white Americans from 25 ng/mL to 45 ng/mL would take 2–2.5 times the current solar UVB irradiance—less in the Southern states, more in the northern states, less in summer, more in winter, although there is a vitamin D winter for several months above the latitude of about 35° N,[57] in which either artificial UVB or supplements would have to be used to obtain vitamin D.

Doubling UVB irradiance could involve simple steps such as going into the sun at midday, exposing more skin area to the sun, and not applying sunscreen until after 10–20 minutes of solar UVB irradiance.

Summary and Conclusion

The analysis presented here finds that the health benefits of increasing mean population serum 25(OH)D levels in the US to 45 nm/mL solely through natural or artificial ultraviolet irradiance that contains 3%–5% of the UV in the UVB spectral region (290–315 nm) could reduce the rate of premature deaths by about 400,000/year while increasing the death rate from melanoma and skin cancer by at most 12,000/year. The beneficial role of UVB irradiance for mortality rates outweighs the risks in terms of melanoma and NMSC mortality rates at all ages considered. Although more research is required to evaluate the findings in this study, the message that UVR should be avoided is counterproductive.

The public health policies regarding solar UV irradiance and vitamin D have swung back and forth like a pendulum. In the 1920s and 1930s, public health policies supported both measures as a means to reduce the burden of disease,[94] but starting in the 1970s and 1980s, concerns regarding the risk of skin cancer turned public health policies against UVB irradiance.[95] The rapidly expanding scientific evidence of health benefits of vitamin D are pushing the pendulum back towards favoring solar UVB irradiance.

It seems worthwhile to reiterate a statement by Jörg Reichrath: "Well-balanced recommendations on sun protection have to ensure an adequate vitamin D status, thereby protecting people against adverse effects of strict sun protection without significantly increasing the risk of developing UV-induced skin cancer."[96]

Note

A paper was recently published by Martin Weinstock from Brown University, chair of the American Cancer Society's Skin Cancer Advisory Committee with Arnold M. Moses, in which the need for vitamin D was recognized.[97] They pointed out that while solar UVB is the natural source of vitamin D, those with light skin should consider obtaining vitamin D from supplements, and that as a public health measure, food fortification should be increased. This paper represents an important step forward for the US dermatological community and, hopefully, the American Cancer Society.

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