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Solar UV Doses of Young Americans and Vitamin D_3 Production

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Abbreviations:

AF, Age Factor
ASCF, Action Spectrum Conversion Factors
FBE, Fraction Body Exposure
GCF, Geometric Conversion Factors
25(OH)D, 25-hydroxyvitamin D
1, $25(OH)_2D$, 1, 25-dihydroxyvitamin D
IU, International Units
MED, Minimum Erythemal Dose
SED, Standard Erythemal Dose
SPF, Sun Protection Factor
STF, Skin Type Factor
SVD, Standard Vitamin D Dose
US, United States
UV, 290-400 nm
UVB, 290-315 nm
VDD, Vitamin D_3 Dose

ABSTRACT

Background: Sunlight contains UVB radiation (290-315 nm) that affects human health in both detrimental (skin cancers) and beneficial (vitamin D_3) ways. Serum 25-hydroxyvitamin D concentrations of young Americans (≤19 yr) show many have deficient (<50 nmol/L, 20 ng/ml) or insufficient (<75 nmol/L, 30 ng/ml) vitamin D levels, indicating they are not getting enough sun exposure. Those findings are in conflict with some calculated, published values that suggest people make "ample" vitamin D_3 (~1,000 IU/day) from their "casual," or everyday, outdoor exposures even if they diligently use sunscreens with sun protection factor 15. **Objective:** We estimated how much vitamin D_3 young Americans (~2,000) produce from their everyday outdoor UV doses in the north (45°N) and south (35°N) each season of the year with and without vacationing. **Methods:** To do these vitamin D_3 calculations properly, we used geometric conversion factors that change planar to whole body doses, which previous calculations did not incorporate. **Results:** Our estimates suggest that American children may not be getting adequate outdoor UVB exposures to satisfy their vitamin D_3 needs all year, except some Caucasians during the summer if they do not diligently wear sunscreens except during beach vacations. **Conclusion:** These estimates suggest most American children may not be going outside enough to meet their minimal (~600 IU/day) or optimal (≥1,200 IU/day) vitamin D requirements.

INTRODUCTION

The UV radiation (290-400 nm) in sunlight can affect peoples' health in both detrimental and beneficial ways. Short-term detrimental health effects include sunburn and immune suppression, while long-term detrimental health effects include cataracts, photoaging, and DNA damage with mutations that can lead to non-melanoma and melanoma skin cancers. Beneficial health effects of UV radiation include medical detection and treatments of diseases such as cancers, possible reduction in the mortality from some cancers including colon (Garland et al. 1989), breast, prostate (Freedman et al. 2002) and melanoma (Berwick et al. 2005), and vitamin D_3 production (Holick et al. 1980).

Children may need more vitamin D_3 than those recently recommended (600 IU/day) by the Institute of Medicine (2011) to maintain healthy muscles (Pfeifer et al. 2002), bones (Holick 2007), and general health (Holick 2011; Heaney and Holick 2011). For example, the risk of type 1 diabetes may be significantly reduced in children, if they take 2,000 IU/day of vitamin D supplements early in life (Ponsonby et al. 2009; Zipitis and Akobeng 2008) Conversely, there is some evidence that infants who develop rickets, a consequence of vitamin D deficiency, are at increased risk of type 1 diabetes mellitus (Hyppönen et al. 2001).

For most children, the major source of vitamin D_3 comes from exposing their skin to sunlight (Glerup et al. 2000). In animals, yeast, fungi, and plants, sunlight forms either vitamin D_3 or D_2, both of which may be equally effective in maintaining human serum levels of 25-hydroxyvitamin D (25(OH)D; where the 'D' represents both D_2 and D_3; Holick et al. 2008). Vitamin D_3 production occurs in human skin when UVB (290-315 nm) photons convert the 7-dehydrocholesterol, or provitamin D_3, that keratinocytes make to previtamin D_3, which thermally isomerizes to vitamin D_3 (Holick et al. 1995). Vitamin D_3 that forms in the skin, and vitamin D_2 or D_3 from dietary sources, is carried into the blood stream by the vitamin D binding protein, an $alpha_1$ globulin. The liver enzyme, vitamin D-25-hydroxylase, hydroxylates it to 25(OH)D and then the kidney enzyme 25(OH)D-1-alpha-hydroxylase further hydroxylates it to the hormonally active form, 1, 25-dihydroxyvitamin D (1, $25(OH)_2D$). In addition to liver and kidney cells, most cells in the body can convert either vitamin D or 25(OH)D to 1, $25(OH)_2D$ including colon, breast, lung, prostate, keratinocytes and melanoma cells (Reichrath et al. 2007; Zehnder et al. 2001).

Most people in the United States (US) do not get sufficient vitamin D from dietary sources [fortified foods and drinks (milk and orange juice), and supplements] (Moore et al. 2004), so that sunlight-derived vitamin D is their primary source. However, because the incidence of skin cancers is increasing at an alarming rate, public health organizations have warned people, especially children, to stay out of the sunlight whenever possible, and to wear protective clothing, sunglasses, and sunscreens with sun protection factor (SPF) 15 or higher while outdoors from 10 a.m. to 4 p.m. (Goldman 2002; American Academy of Dermatology. 2011. http://www.aad.org/00PSA1.html; Cancer Society. 2011. http://www.cancer.org; Center for Disease Control. 2011. http://www.cdc.gov/cancer/nscpep/skin.htm; Environmental Protection Agency. 2011. http://www.epa.gov/sunwise/kids.html; Skin Cancer organization. 2011. http://www.skincancer.org/skincancer-facts.php [accessed 1 January 2011]). Although sunscreens with SPF 15 or higher almost completely inhibit vitamin D_3 production (Holick et al. 1995; Matsuoka et al. 1988), the American Academy of Dermatology has concluded that people will still make "ample" vitamin D_3 ($\geq$1,000 IU/day) because they get plenty of "casual," or everyday, outdoor UV exposure (Lim et al. 2005). However, an evaluation of serum 25(OH)D revealed about half of all American children have either deficient or insufficient levels (Looker et al. 2002, 2008; Mansbach et al. 2009).

To clarify if 'casual' sunlight exposures make ample vitamin D_3, we calculated the amounts produced from everyday outdoor UV dose estimates (Godar 2001) according to sex, age, Fitzpatrick skin type (Fitzpatrick 1988), clothing (Matsuoka et al. 1992) and season for children in the northern and southern US.

MATERIALS AND METHODS

We extracted and calculated erythemally-weighted UV doses (Godar et al. 2001) from a two-year survey of 9,386 Americans residing in the contiguous US, including about 2,000 children (≤19 yr) (Godar 2001). We converted average daily standard erythemal doses (SED, where 1 SED = 100 J/m^2; a UV dose weighted by the erythemal action spectrum, so that it is independent of the spectral output of the source and the individual's skin type) for each season to standard vitamin D_3 doses (SVD) relative to the horizontal plane using action spectrum conversion factors (ASCF; Pope et al. 2008), and then converted those planar dose estimates to human body doses using geometric conversion factors (GCF; Pope and Godar, 2010). An action spectrum shows the relative effectiveness of each wavelength (nm) toward some endpoint that can be used to weight spectral outputs of different sources like the Sun or a tanning bed in order to estimate amounts produced, e.g., erythemal response (sunburn) or vitamin D_3 production.

The ASCF account for the differences between wavelength contributions estimated by the erythemal action spectrum and the previtamin D action spectrum toward previtamin D_3 production. To derive the SVD for a given season and latitude, we multiplied the SED/day by the appropriate ASCF:

$$\text{SVD} = \text{SED/day} \times \text{ASCF} \qquad [1] .$$

ASCF for the northern (45°N) and southern (35°N) US are 1.034 and 1.104 for summer, 0.879 and 1.029 for fall, 0.565 and 0.842 for winter, and 0.9 and 1.049 for spring, respectively (Pope et al. 2008).

SVD, which represent horizontal plane or planar doses, are converted to whole body doses using GCF based on a full-cylinder model representing the human body (Pope and Godar 2010). GCF for the northern US (45°N) are 0.434 during the summer and spring and 0.508 during the winter and fall; GCF for the southern US (35°N) are 0.417 during the summer and spring and 0.484 during the winter and fall. The average vitamin D_3 dose (VDD) per day is derived by multiplying the SVD by the appropriate GCF:

$$\text{VDD} = \text{SVD} \times \text{GCF} \qquad [2].$$

To estimate the amount of vitamin D_3 a person makes from outdoor UV exposures when engaged in different activities, we first determined how much vitamin D_3 a person would make from an erythemally-weighted UV dose with uniform geometry, such as the UV dose from a tanning bed. For example, a female with Fitzpatrick skin type II (where skin type II indicates Caucasian and skin type V indicates African-American; Fitzpatrick 1988) with a whole body exposure to one minimum erythemal dose (MED), or the amount of UV needed to barely turn skin pink after 24 hr, in a tanning bed with a weighted spectral distribution similar to the midday summer sun at ~35°N produces the equivalent of an oral dose of ~15,500 IU vitamin D_2 or D_3 (Holick 2002, Holick et al. 2008). Because melanin impedes the penetration of UVB and reduces vitamin D_3 production (Clemens et al. 1982; Matsuoka et al. 1991), the UV dose required to achieve a MED and make the same amount of vitamin D varies by skin type. For Fitzpatrick skin type II (Fitzpatrick 1988), 1 MED is defined as 250-350 J/m^2, and so we refer to skin type II as

300 J/m^2 or 3 SED. For a whole body exposure, a person with skin type II with a MED of 320 J/m^2 will make ~15,500 IU/MED or ~4,900 IU/SED. Similarly, 1 MED is 300-500 J/m^2 or 4 SED (average) for skin type III, 450-600 J/m^2 or 5.25 SED for skin type IV, and 600-900 J/m^2 or 7.5 SED for skin type V. Consequently, for a given UVB dose, Caucasians with skin type II make at least 2-3 times more vitamin D_3 than light-skinned, skin type V African-Americans (Dong et al. 2010; Matsuoka et al. 1991) and 10-20 times more than dark skinned (skin type VI) African Americans (Clemens et al. 1982). The ratio of vitamin D production for someone with skin type II compared with other skin types, referred to as the skin type factor (STF), is used to adjust predictions for each skin type, such that the STF is 3.2/3 for skin type II, 3.2/4 for type III, 3.2/5.25 for skin type IV, and 3.2/7.5 for skin type V:

STF = (skin type II SED)/(other skin type SED) [3].

The amount of vitamin D_3 people make from outdoor UV exposures also depends on how much skin they expose to the sun, or the fraction body exposed (FBE). To get the best estimates of how much body area people expose or FBE during each season of the year, we used estimates for burn areas (Lund and Browder 1944). For example, we assumed young adults would expose their face (4.5-7.8%), the front half of their neck (1%), and the front and back of both their hands (5%) during all seasons of the year (FBE 10.5-13.8%; see Table 1), and would also expose their lower arms (6%) and lower legs (10-13%) during the spring and fall (FBE ~30%) and half of their upper arms (4%; short-sleeved/tee shirts) and half of their upper legs (7-9%; shorts/skirts) during the summer (FBE ~41-44%).

Because vitamin D_3 production decreases with age (MacLaughlin and Holick 1985), we must also include an age factor (AF) in the calculations. Young adults (<22 y), who have the highest ability to make vitamin D_3, are assigned an AF = 1 and older adults ≥22 yr) are assigned fractions according to their age range (Godar et al. In Press). Therefore, the final equation for calculating the amount of vitamin D_3 produced from an average everyday UV exposure during each season of the year is:

Vitamin D_3 (IU)/day = VDD x (4,900 IU for skin type II) x STF x FBE x AF [4]

For example, during the summer, a four-year-old African American girl with skin type V living near Atlanta, Georgia (~34°N) and wearing a t-shirt and short shorts (thus exposing approximately 40.8% of her body area; see Table 1) would get, on average, about 1 SED/day (Godar 2001), and would make about 397 IU of vitamin D_3/day [Vitamin D_3 (IU)/day = ((1.01 SED * 1.104) * 0.417) * (4,900 IU) * (3.2/7.5) * 0.408 * 1.0]

To account for sunscreen use, divide the estimate from equation 4 by the SPF factor (Matsuoka et al. 1988). However, this assumes that sunscreens are used correctly, i.e., that they are generously applied to the entire body prior to going outdoors, and are reapplied every 2 hours.

To account for additional sun exposure during vacations, we recalculated estimates assuming that 2-3 weeks each summer were spent vacationing at ~40°N, as estimated by Godar et al. (2001) with the use of SPF 4 sunscreen, or the equivalent only during beach vacations. In addition, we averaged exposures for four different types of vacations: beach, sightseeing, country, and home, and assumed people would wear the least amount of clothing during a summer vacation (FBE ~50-90%), depending on the type of vacation.

RESULTS

Table 1 shows estimates for the percentage of body area exposed to outdoor UV by different age groups during each season of the year, based on the data of Lund and Browder (1944). During the winter, body exposure is highest in the youngest children (≤5 yr), and during the rest of the year, body exposure is highest in the teenagers (13-19). Adults (≥22 yr) have the lowest fraction of body exposed during all seasons. Note here that levels of vitamin D_3 can increase during the summer by 30% or more (~500 IU/day) depending on clothing choice (data not shown).

Figure 1 shows estimates of the average amount of vitamin D_3 made by children with Fitzpatrick skin type II according to season, age group, sex, diligent use of SPF 15 sunscreen, and residence in the northern or southern US. According to our estimates, the minimum recommended daily dose of vitamin D (600 IU/day; Institute of Medicine 2011) is only achieved by skin type II children in the northern US during the summer if they do not wear SPF 15 sunscreen (Figure 1A). However, results suggest that most children with skin type II in the southern US can get the recommended daily dose during the spring and summer if they do not wear SPF ≥15 sunscreen (Figure 1B). Optimal vitamin D_3 production (≥1,200 IU/day) is primarily achieved by a subset of children in the southern US during the summer (Dong et al. 2010).

Figure 2 shows the average amount of vitamin D_3 produced from everyday outdoor exposure according to skin type (II –V), season, and residence in the northern or southern US for all children ≤19 yr combined (without using sunscreens). Our estimates suggest that in the northern US, the minimum recommended daily dose of vitamin D_3 (600 IU/day) is only made by skin types II, III and IV children during the summer. In addition, estimates suggest that the optimal dose of ≥1,200 IU/day is not made during any season, regardless of skin type (Figure 2A), except by a small subset of skin type II children during the summer (see Figure 1A). In the southern US, we estimate that the minimum amount (600 IU/day) is achieved during the summer by children with skin types II, III and IV and during the spring by children with skin type II only, whereas the optimum amount (≥1,200 IU/day) is achieved only by skin type II children during the summer (Figure 2B). These findings suggest that children with skin type III (e.g., olive skin tone, Hispanic or Asian) and skin type IV rarely meet their minimum vitamin D_3 need (~600 IU/day), and that dark skinned children (skin type V) may never meet their minimum daily vitamin D need from everyday outdoor exposure.

Taking a 2 or 3 week summer vacation at 40°N during the summer increases the average estimated vitamin D_3 production for children of all skin types, but still may be insufficient to meet minimum or optimum requirements in children with darker skin types ≥V (Figure 3; UV doses estimated by Godar et al. 2001).

DISCUSSION

Although our estimates assumed “optimistic” clothing scenarios for making vitamin D_3, they raise the question as to whether or not American children are going outdoors enough to meet their minimum daily vitamin D requirements (600 IU/day; for blood levels of 20 ng/ml or 50 nmol/L) recommended by the Institute of Medicine (2011). Based on the results of this analyses, it appears that most active Caucasian children of skin type II can make optimal amounts of vitamin D_3 (≥1,200 IU/day for blood levels of 30 ng/mL or 75 nmol/L) during the summer, but only if they do not use sunscreens diligently except during beach vacations.

However, children with darker skin may never achieve optimal production, while those with skin types III and IV may only meet minimum requirements during the summer, and children with very dark skin (type V) may only meet their minimum requirements during the summer if they take a 3 week beach vacation at latitudes lower than 40°N (~middle of US).

If children really need to achieve optimal blood levels of 30 ng/ml (~75 nmol/L), then they need at least twice the current recommendation or about 1,200 IU/day of vitamin D. However, two recent studies, which examined over 25 children in each different skin type and sun exposure category, concluded that children need ≥2,000 IU/day from oral supplements (Maalouf et al. 2008), equivalent UV exposure, or a combination (Hall et al. 2010) to maintain blood levels of 25(OH)D above 75 nmol/L. If children really need ≥2,000 IU/day, then almost no one in the US can make the needed amount of vitamin D_3 from everyday sun exposure all year.

Overall, males go outside somewhat more than females and the youngest age group of children (≤5 yr) go outside somewhat more than the other age groups (Godar 2001) so they consequently make a little more vitamin D_3, in agreement with the findings of three large recent US studies (Ginde et al. 2009; Looker et al. 2008; Mansbach et al. 2009). Children spend approximately 1.6 ± 0.1 h/day (weekdays and weekends averaged) outdoors during the summer (Godar 2001), giving them about two thirds of an MED for a skin type II body exposure (Pope and Godar 2010). If the children are Caucasian with skin type II and wear minimal clothing (e.g., wear a diaper, shorts or a bathing suit, or females wear tank or halter tops, or males do not wear a shirt and do wear short shorts), then they can make at least 30% more vitamin D_3 (~500 IU/day; results not shown). In contrast, if people wear sunscreen with SPF ≥15, they will make virtually no vitamin D_3 (Holick et al. 1995; Matsuoka et al. 1998).

Our estimates are consistent with reports of vitamin D deficiency (<50 nmol/L; <20 ng/ml) and insufficiency (<75 nmol/L; <30 ng/ml) among children and adolescents in the US. For example, the NHANES III (1988-1994) study showed 13% male and 29% female adolescents (12-19 yr) had deficient levels and 25% male and 47% of female adolescents had insufficient levels of 25(OH)D during the winter (Looker et al 2002). During the summer, 8% of males and 13% of females had deficient levels and 21% of males and 28% of females had insufficient levels of 25(OH)D. In addition, the percentage of children with deficient levels during the winter increased with increasing skin color: skin types I/II (8% male/15% female), skin types III/IV (18% male/41% female), and skin types V/VI (53% male/70% female). NHANES 2000-2004 data indicated that approximately 20% more Americans were vitamin D deficient compared with the previous decade, partly due to increased sun protection (Ginde et al. 2009; Looker et al. 2008; Mansbach et al. 2009). A study in Augusta, Georgia (33˚N) examined 559 adolescents and found 3.9% of male and 2.6% of female adolescents with skin type II had deficient levels while 46.9% of male and 73.8% of female adolescents had insufficient levels of 25(OH)D during the winter (Dong et al. 2010). That study also showed 84% of African Americans with skin types V and VI had deficient levels and 98% had insufficient levels of 25(OH)D during the winter while 56% had deficient and 88% had insufficient levels of 25(OH)D during the summer. A study in Pittsburg, Pennsylvania (40˚N) of 41 preadolescent African-Americans (6-10 yr) found 49% had deficient 25(OH)D levels (Rajakumar et al. 2005). The recent resurgence of rickets in breast fed African-American infants in several southern states suggests vitamin D deficiency is on the rise in the US (Kreiter et al. 2000). Insufficient and deficient levels of 25(OH)D in young adults are similar in the southern hemisphere at comparable latitudes as the US (35-46°S; Rockell et al. 2005) where they get similar UV doses

(Godar 2005). Insufficient sun exposure in adults is also a concern, particularly given that the ability to make vitamin D decreases with age, so that seniors over 70 can only make 25-50% of what a child can make (MacLaughlin and Holick 1985). Deficient and insufficient vitamin D levels from insufficient sun exposure are a worldwide problem with serious health consequences (Holick and Chen 2008).

Conversely, serious health consequences can also arise from too much sun exposure. Too much UV radiation can lead to the formation of three types of skin cancers: squamous cell carcinoma, basal cell carcinoma, and melanoma. Paradoxically, regular, moderate sun exposure may reduce the incidence of fatal melanoma (Godar et al. 2009). Melanoma cells have been shown to convert vitamin D_3 to $1,25(OH)_2D$ in vitro (Reichrath et al. 2007), and it has been reported that $1,25(OH)_2D$ can reduce tumor growth and decrease the number and size of skin tumors and melanoma xenografts in animal models (Eisman et al. 1987), and inhibit *in vivo* pulmonary metastasis (Yudoh et al. 1999) and angiogenesis (Mantell et al. 2000). Further, continual rather than intermittent outdoor UV exposure has been associated with a reduced incidence of melanoma (Gandini et al. 2005; Kennedy et al. 2003) relative to the cumulative annual UV dose (Godar 2005) and sun exposure has been associated with increased survival in melanoma patients (Berwick et al. 2005). Thus, it has been suggested that promoting protection from all mid-day UV exposures and advising the diligent use of SPF ≥ 15 sunscreens may paradoxically be promoting the incidence of melanoma (Godar et al. 2009; Gorham et al. 2007).

In conclusion, our estimates suggest that many children may not get enough sun exposure to meet their minimum daily vitamin D requirements. However, additional research is needed to confirm our estimates, and improve our understanding of the net benefits and risks of sun exposure to children's health.

REFERENCES

Berwick M, Armstrong BK, Ben-Porat L, Fine J, Kricker A, Eberle C, et al. 2005. Sun exposure and mortality from melanoma. JNCI 97:195-199.

Clemens TL, Adams JS, Henderson SL, Holick MF. 1982. Increased skin pigment reduces the capacity to synthesize vitamin D_3. Lancet 1:74-76.

Dong Y, Pollock N, Stallmann-Jorgensen IS, Gutin B, Lan L, et al. 2010. Low 25-hydroxyvitamin D levels in adolescents: race, season, adiposity, physical activity, and fitness. Pediatrics 125:1104-1111.

Eisman JA, Barkla DH, Tutton PJ. 1987. Suppression of in vivo growth of human cancer solid tumor xenografts by 1, 25-dihydroxyvitamin D_3. Cancer Res 47:21-25.

Fitzpatrick TB. 1988. The validity and practicality of Sun-reactive skin types I through VI. Arch Dermatol 124:869-871.

Freedman DM, Dosemeci M, McGlynn K. 2002. Sunlight and mortality from breast, ovarian, colon, prostate, and non-melanoma skin cancer: a composite death certificate based case-control study. Occup Environ Med 59:257-262.

Gandini S, Sera F, Cattaruza MS, Pasquini P, Picconi O, Boyle P, et al. 2005. Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. Eur J Cancer 41:45-60.

Garland CF, Comstock GW, Garland FC, Helsing KJ, Shaw EK, Gorham ED.1989. Serum 25-hydroxyvitamin D and colon cancer: eight-year prospective study. Lancet 2:1176-1178.

Ginde AA, Liu MC, Camargo CA Jr. 2009. Demographic differences and trends of vitamin D insufficiency in the US population, 1988-2004. Arch Intern Med 169:626-632.

Glerup H, Mikkelsen K, Poulsen L, Has E, Overbeck S, Thomsen J, et al. 2000. Commonly recommended daily intake of vitamin D is not sufficient if sunlight exposure is limited. J Intern Med 247:260-268.

Godar DE. 2001. UV Doses of American Children and Adolescents. Photochem Photobiol 74:787-793.

Godar DE. 2005. UV Doses Worldwide. Photochem Photobiol 81:736-749.

Godar DE, Landry R, Lucas AD. 2009. Increased UVA exposures and decreased cutaneous Vitamin D(3) levels may be responsible for the increasing incidence of melanoma. Med Hypotheses 72:434-443.

Godar DE, Wengraitis SP, Shreffler J, Sliney DH. 2001. UV doses of Americans. Photochem Photobiol 73:621-629.

Godar DE, Pope SJ, Grant WB, and Holick MF. In Press. Solar UV Doses of Adult Americans and Vitamin D_3 production. Dermatoendochrinol.

Goldman EI. 2002. Skin Cancer Messages Target Teens. In Skin and Allergy News. June 33(6):1-4.

Gordon CM., DePeter KC, Feldman HA, Grace E, Emans SJ. 2004. Prevalence of vitamin D deficiency among healthy adolescents. Arch Pediatr Adolesc Med 158: 531-537.

Gordon CM, Feldman HA, Sinclair L, Williams AL, Kleinman PK, Perez-Rossello J, et al. 2008. Prevalence of vitamin D deficiency among healthy infants and toddlers. Arch Pediatr Adolesc Med 162:505-512.

Gorham ED, Mohr SB, Garland CF, Chaplin G, Garland FC. 2007. Do sunscreens increase risk of melanoma in populations residing at higher latitudes? Ann Epidemiol 17:956-963.

Hall LM, Kimlin MG, Aronov PA, Hammock BD, Slusser JR, Woodhouse LR, et al. 2010. Vitamin D intake needed to maintain target serum 25-hydroxyvitamin D concentrations in participants with low sun exposure and dark skin pigmentation is substantially higher than current recommendations. J Nutr 140:542-550.

Harkness LS, Cromer BA. 2005. Vitamin D deficiency in adolescent females. J Adolesc Health 37:75.e1-75e.5.

Harris SS. 2005. Vitamin D in type 1 diabetes prevention. J Nutr 135:323-325.

Heaney RP, Holick MF. 2011. Why the IOM recommendations for vitamin D are deficient. J Bone Miner Res. Jan 4. [Epub ahead of print]

Heaney RP, Davies KM, Chen TC, Holick MF, Barger-Lux MJ. 2003. Human serum 25-hydroxycholecalciferol response to extended oral dosing with cholecalciferol. Am J Clin Nutr 77:204-210.

Holick MF. 2007. Vitamin D deficiency. N Engl J Med 357:266-281.

Holick MF. 2011. Vitamin D: Evolutionary, Physiological and Health Perspectives. Curr Drug Targets 12:4-18

Holick MF, Biancuzzo RM, Chen TC, Klein EK, Young A, Bibuld D, et al. 2008. Vitamin D_2 is as effective as vitamin D_3 in maintaining circulating concentrations of 25-hydroxyvitamin D. J Clin Endocrinol Metab 93:677-681.

Holick MF, Chen TC. 2008 Vitamin D deficiency: a worldwide problem with health consequences. Am J Clin Nutr 87:1080S-1086S.

Holick MF, MacLaughlin JA, Clark MB, Holick SA, Potts Jr. JT, Anderson RR, et al. 1980. Photosynthesis of pre-vitamin D_3 in human skin and the physiological consequences. Science 210:203-205.

Holick MF, Tian XQ, Allen M. 1995. Evolutionary importance for the membrane enhancement of the production of vitamin D3 in the skin of poikilothermic animals. Proc Natl Acad Sci U S A. 92:3124-3126.

Holick MF, Matsuoka LY, Wortsman J. 1995. Regular use of sunscreen on vitamin D levels. Arch Dermatol 131:1337-1339.

Hyppönen E, Laara E, Reunanen A, Jarvelin MR, Virtanen SM. 2001. Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. Lancet 358:1500-1503.

Institute of Medicine. 2011. Dietary Reference Intakes for Calcium and Vitamin D. Washington, DC: The National Academies Press.

www.iom.edu/Reports/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D.aspx

Kennedy C, Bajdik CD, Willemze R, De Gruijl FR, Bouwes Bavinck JN. 2003. The influence of painful sunburns and lifetime sun exposure on the risk of actinic keratoses, seborrheic warts, melanocytic nevi, atypical nevi, and skin cancer. J Invest Dermatol 120:1087-1093.

Kreiter SR, Schwartz RP, Kirkman Jr. HN, Charlton PA, Calikoglu AS, Davenport ML. 2000. Nutritional rickets in African American Breast-fed infants. J Pediatr 137: 153-157.

Lim HW, Gilchrest BA, Cooper KD, Bischoff-Ferrari HA, Rigel DR, Cyr WH, Miller S, et al. 2005. Sunlight, tanning booths, and vitamin D. J Am Acad Dermatol 52:868-876.

Looker AC, Dawson-Hughes B, Calvo MS, Gunter EW, Sahyoun NR. 2002. Serum 25-hydroxyvitamin D status of adolescents and adults in two seasonal subpopulations from NHANES III. Bone 30:771-777.

Looker AC, Pfeiffer CM, Lacher DA, Schleicher RL, Picciano MF, Yetley EA. 2008. Serum 25-hydroxyvitamin D status of the US population: 1988-1994 compared with 2000-2004. Am J Clin Nutr 88:1519-1527.

Lund CC, Browder NC. 1944 The estimation of areas of burns. Surgery Gynecol Obstetrics 79:352-358.

Maalouf J, Nabulsi M, Vieth R, Kimball S, El-Rassi R, Mahfoud Z, et al. 2008. Short- and long-term safety of weekly high-dose vitamin D_3 supplementation in schoolchildren. J Clin Endocrinol Metab 93:2693-2701.

MacLaughlin JA, Anderson RR, Holick MF.1982. Spectral character of sunlight modulates photosynthesis of previtamin D3 and its photoisomers in human skin. Science 216:1001-1003.

MacLaughlin JA, Holick MF. 1985. Aging decreases the capacity of human skin to make vitamin D_3. J Clin Invest 76:1536-1538.

Mansbach JM, Ginde AA, Camargo CA Jr. 2009. Serum 25-hydroxyvitamin D levels among US children aged 1 to 11 years: do children need more vitamin D? Pediatrics 124:1404-1410.

Mantell DJ, Owens PE, Bundred NJ, Mawer EB, Canfield AE. 2000. 1α, 25-dihydroxyvitamin D_3 inhibits angiogenesis in vitro and in vivo. Circ Res 87:214-220.

Matsuoka LY, Wortsman J, Dannenberg MJ, Hollis BW, Lu Z, Holick MF. 1992. Clothing prevents ultraviolet-B radiation-dependent photosynthesis of vitamin D3. J Clin Endocrinol Metab 75:1099-1103.

Matsuoka LY, Wortsman J, Haddad JG, Kolm P, Hollis BW. 1991. Racial pigmentation and the cutaneous synthesis of vitamin D. Arch Dermatol 127:536-538.

Matsuoka LY, Wortsman J, Hanifan N, Holick MF. 1988. Chronic sunscreen use decreases circulating concentrations of 25-hydroxyvitamin D. A preliminary study. Arch Dermatol 124:1802-1804.

Moore CE, Murphy MM, Keast DR, Holick MF. 2004. Vitamin D intake in the United States. J Am Diet Assoc 104:980-983.

Munger KL, Zhang SM, O'Reilly E, Hernan MA, Olek MJ, Willet WC, et al. 2004. Vitamin D intake and incidence of multiple sclerosis. Neurol 62:60-65.

Pfeifer M, Begerow B, Minne HW. 2002. Vitamin D and muscle function. Osteoporos Int 13:187-194.

Pope SJ, Godar DE. 2010. Solar UV geometric conversion factors: horizontal plane to cylinder model. Photochem Photobiol 86:457-466.

Pope SJ, Holick MF, Mackin S, Godar DE. 2008. Action spectrum conversion factors that change erythemally weighted to previtamin D_3-weighted UV doses. Photochem Photobiol 84:1277-1283.

Ponsonby AL, Cameron F, Saffery R, Pezic A, Wong N, Craig J, Ellis J, Dwyer T. 2009. A case-sibling assessment of the association between skin pigmentation and other vitamin D-related factors and type 1 diabetes mellitus. Photochem Photobiol 85:1267-1270.

Rajakumar K, Fernstrom JD, Janosky JE, Greenspan SL. 2005. Vitamin D insufficiency in preadolescent African-American children. Clin Pediatr 44:683-692.

Reichrath J, Rech M, Moeini M, Meese E, Tilgen W, Seifert M. 2007. In vitro comparison of the vitamin D endocrine system in 1,25(OH)2D3-responsive and -resistant melanoma cells. Cancer Biol Ther 6:48-55

Rockell JE, Green TJ, Skeaff CM, Whiting SJ, Taylor RW, Williams SM, et al. 2005. Season and ethinicity are determinants of serum 25-hydroxyvitamin D concentrations in New Zealand children aged 5-14 y. J Nutr 135:2602-2608.

Sullivan SS, Rosen CJ, Haltman WA, Chen TC, Holick MF. 2005. Adolescent girls in Maine are at risk for vitamin D insufficiency. J Am Diet Assoc 105:971-974.

Yudoh K, Matsuno H, Kimura T. 1999. 1-alpha, 25-dihydroxyvitamin D_3 inhibits in vitro invasiveness through the extracellular matrix and in vivo pulmonary metastasis of B16 mouse melanoma. J Lab Clin Med 133:120-128.

Zehnder D, Bland R, Williams MC, McNinch RW, Howie AJ, Stewart PM, et al. Extrarenal expression of 25-hydroxyvitamin D(3)-1 alpha-hydroxylase. 2001. J Clin Endocrinol Metab 86:888-894.

Zipitis, C. S. and A. K. Akobeng. 2008. Vitamin D supplementation in early childhood and risk of type 1 diabetes: A systematic review and meta-analysis. Arch Dis Child 93, 512–517.

Table 1. Estimates of the percent body area exposed each season by age, based on the data of Lund and Browder (1944).

Age	**0 – 5 yr**	**10 yr**	**15 yr**	**Adult ****
½ head (face)	7.8%	5.5%	4.5%	3.5%
½ neck (front)	1%	1%	1%	1%
hands (front&back)	5%	5%	5%	5%
lower arms	6%	6%	6%	6%
lower legs	10%	12%	13%	14%
½ upper arms	4%	4%	4%	4%
½ upper legs	7%	8.5%	9%	9.5%
Summations†				
Winter	13.8%	11.5%	10.5%	9.5%
Spring/Fall	30%	29.5%	29.5%	15.5%
Summer	40.8%	42%	43.5%	33.5%
<u>For summer only:</u>				
upper arms	8%	8%	8%	8%
upper legs	14%	17%	18%	19%
Trunk	26%	26%	26%	26%
Feet	7%	7%	7%	7%
Bathing suit/diaper	85.6%	88.5%	89.5%	90.5%

† The ‘0-5 age range’ percent body part data was weighted by age (Lund and Browder, 1944).

**** (≥ 22yr)**

Figure Legends

Figure 1. Average estimated vitamin D_3 (IU/day) production by American children with skin type II according to age, season, sunscreen use, and residence in the (A) northern (45°N) or (B) southern (35°N) US. Lines near the origin represent the estimated amount of vitamin D_3 made by children who diligently wear SPF 15 sunscreen all day.

Figure 2. Average estimated vitamin D_3 (IU/day) produced in children (≤19 yr) from everyday outdoor UV exposures according to Fitzpatrick skin type, season, and use of SPF 15 sunscreen: (■) Skin type II, (♦) Skin Type III, (▲) Skin Type IV, (•) Skin Type V.

Figure 3. Average estimated vitamin D_3 made by US children (≤19 yr) during the summer according to skin type and vacation (no vacation, 2-week summer vacation, or 3-week summer vacation at latitude ~40˚N in the continental US). We assumed people use the equivalent of SPF 4 sunscreen during beach vacations.

Figure 1.

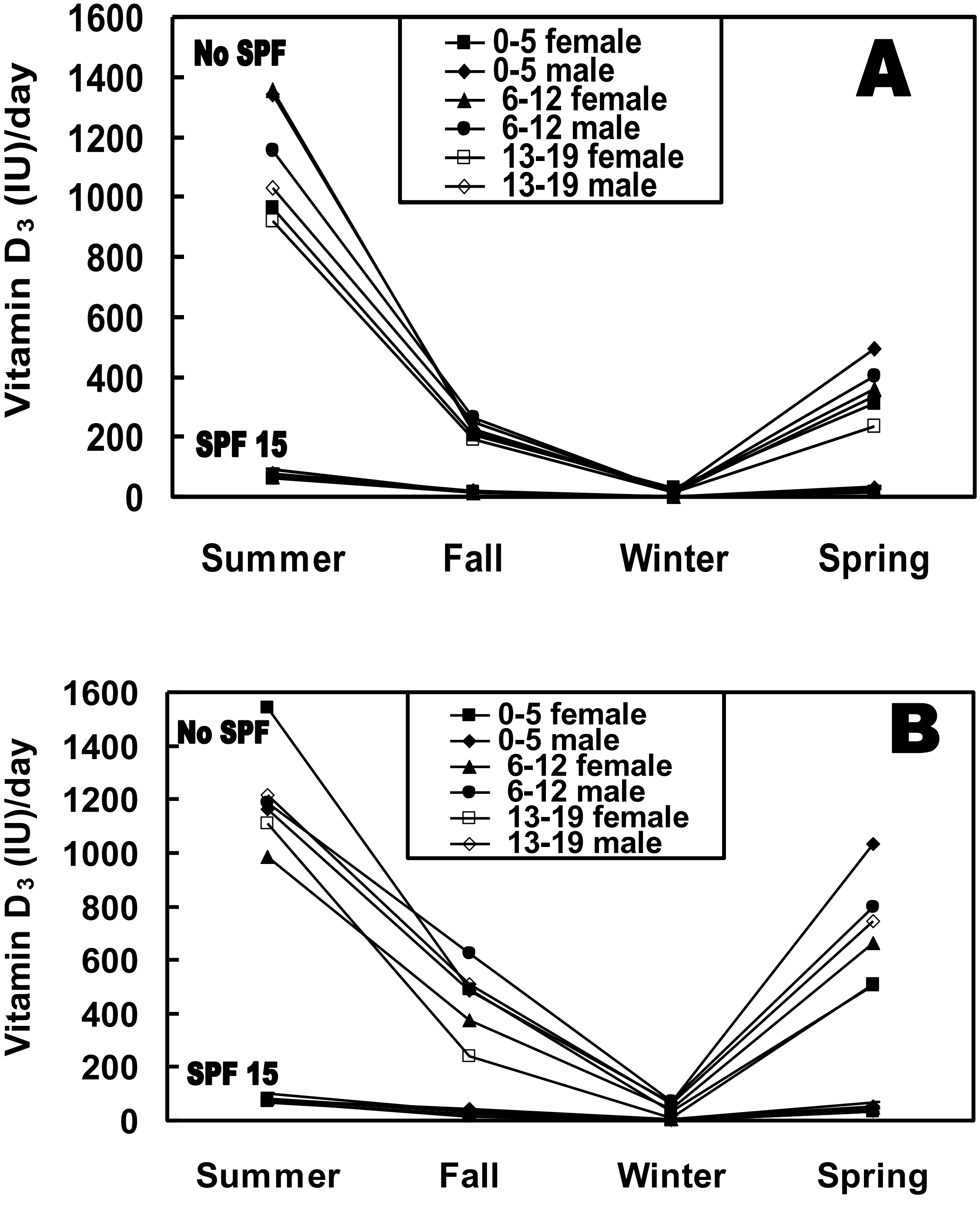

1600
1400
1200
1000
800
600
400
200
0
Vitamin D$_3$ (IU)/day
No SPF
SPF 15
0-5 female
0-5 male
6-12 female
6-12 male
13-19 female
13-19 male
A
Summer
Fall
Winter
Spring
B

Figure 2.

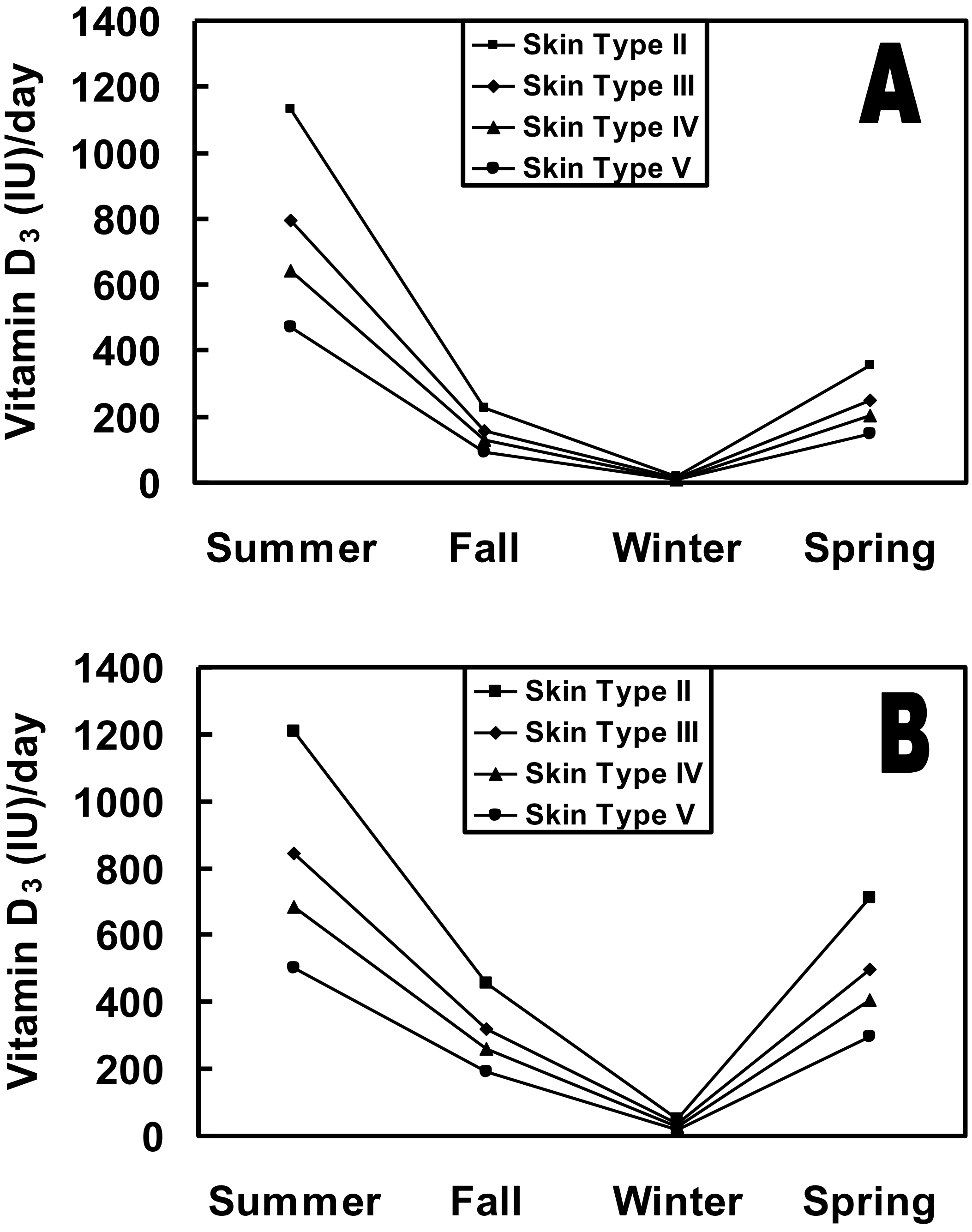
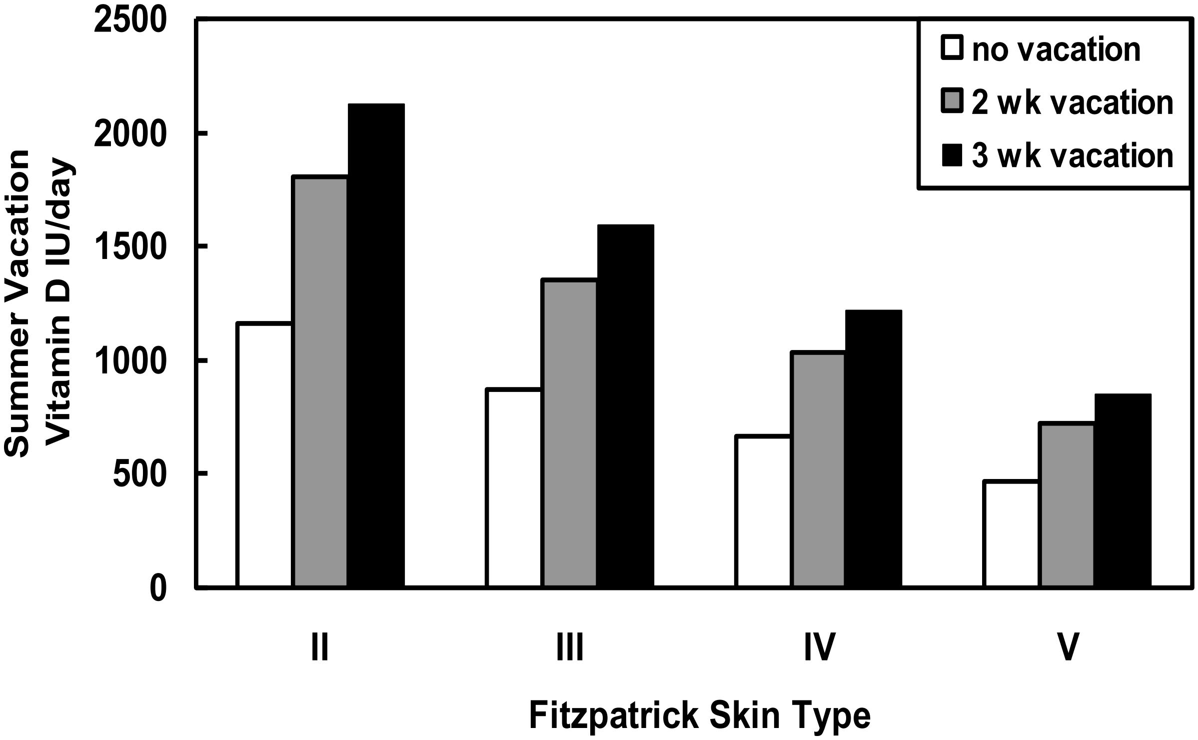

1400
1200
1000
800
600
400
200
0
Vitamin D3 (IU)/day
Skin Type II
Skin Type III
Skin Type IV
Skin Type V
A
Summer
Fall
Winter
Spring
1400
1200
1000
800
600
400
200
0
Vitamin D3 (IU)/day
Skin Type II
Skin Type III
Skin Type IV
Skin Type V
B
Summer
Fall
Winter
Spring

Figure 3.


2500
2000
1500
1000
500
0
Summer Vacation
Vitamin D IU/day
no vacation
2 wk vacation
3 wk vacation
II
III
IV
V
Fitzpatrick Skin Type