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A mathematical model for seasonal variability of vitamin D due to solar radiation

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ABSTRACT

It is widely recognized that vitamin D deficiency has detrimental health consequences. The ultraviolet (UV) B radiation increases the serum vitamin D level, expressed by 25-hydroxyvitamin-D₃ [25(OH)D]. An analytical model is presented to calculate the serum 25(OH)D changes throughout a year, caused by the solar exposure variability due to geophysical and habitual factors. The model is tuned by taking into account recent experimental results of serum 25(OH)D changes, after a series of artificial (by fluorescent tubes) UV exposures. The model uses the erythemal and vitamin D weighted irradiances, inferred from the Brewer spectrophotometer and the Kipp and Zonen broad-band meter measurements, carried out in Belsk (52°N, 21°E), Poland, in 2010. The modeled seasonal pattern of the serum 25(OH)D concentration in Polish indoor workers is only slightly different, than in subjects with typical outdoor activity habits, and in those with sun-seeking behavior. A deep minimum in the serum 25(OH)D concentration appears in late winter, regardless of outdoor activity habits. An extra sunbathing to boost the vitamin D level is not worth taking, because of a minor improvement of the vitamin D status, and because of a greater erythema risk. It would be much safer and more effective to maintain an adequate vitamin D level through diet supplements, even in summer, for non sun-seeking subjects.

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1. Introduction

A skin exposure to the solar ultraviolet (UV) radiation is a basic source of vitamin D for humans [\[1,2\]](#page--1-0). Many recent papers demonstrated health problems related to a low level of vitamin D. It is well established that vitamin D is essential for the bone and muscle status [\[3,4\]](#page--1-0). It has been suggested in literature that the vitamin D insufficiency might also be related to a higher risk of colorectal cancer, prostate cancer, multiple sclerosis, type-1 diabetes, and cardiovascular diseases [\[5–9\].](#page--1-0) Otherwise, the increased UV exposure could induce several diseases, such as nonmelanoma skin cancers, cataracts [\[10\],](#page--1-0) DNA damage [\[11\]](#page--1-0), immune suppression [\[12\],](#page--1-0) etc. Thus, balancing beneficial and harmful solar UV effects is of a special importance for health status [\[13\].](#page--1-0)

The typical measure of the vitamin D status is a serum concentration of the 25-hydroxyvitamin D [25(OH)D]. Several studies indicated the vitamin D insufficiency across Caucasian population, based on measurements of the serum 25(OH)D levels [\[14–16\].](#page--1-0) The studies applied a statistical approach for finding a relationship between changes of the serum 25(OH)D level and the solar UV exposure. The vitamin D synthesis in a human body depends on a number of causes, including geographical factors (solar elevation, ozone, cloudiness, albedo, etc.), as well as on individual human factors (skin phototype, outdoor activity habits, age, genetic factors, etc.). Mathematical modeling is a promising tool to analyze the vitamin D changes, related to all these factors. Such modeling has already begun [\[17\].](#page--1-0) An alternative model is presented here for an estimation of the seasonal changes in serum 25(OH)D, for various scenarios of outdoor behavior. The model is constructed taking into real UV exposures, measured in Poland in 2010, and findings of recent laboratory experiments with the UV light, emitted by fluorescent tubes [\[18–21\]](#page--1-0).

2. Methods and data

2.1. Ambient UV radiation

The surface UV radiation at the Central Geophysical Observatory of the Institute of Geophysics, Polish Academy of Sciences, in Belsk (52-N, 21-E, central Poland), has been measured by various broadband instruments since 1975, and by the Brewer spectrophotometer (BS) since 1992. The UV series has been recently homogenized and used for the trend detection for the period of 1976–2008 [\[22\]](#page--1-0). The biologically effective (BE) spectral irradiances for the erythema appearance, and production of pre-vitamin D3 from the 7 dehydrocholesterol, were obtained by using the CIE action spectra for erythema [\[23\]](#page--1-0), and the vitamin D production [\[24\]](#page--1-0), respectively, as the weighting functions for all of the BS spectra, collected in 2010. The ShicRIVM algorithm [\[25\]](#page--1-0) was applied to extend the spectra from the measured 290–325 nm range, up to 400 nm.

To run our model and compare it with the previous model [\[17\],](#page--1-0) we calculated the BE fractional daily doses for 1 h or 2 h period, on

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each day of 2010. Normally, there are 1–4 Brewer UV measurements, performed every hour throughout the day. Such time resolution gives only a crude approximation of 1 h doses, as clouds may affect the surface UV irradiance on much shorter time scales. Thus, the BE fractional daily doses have to be reproduced from proxy variables, correlated with the UV irradiance. The following proxies were selected: the output (in Volts) of the collocated Kipp and Zonen UV broad-band meter, the UV–S–EA–T, solar elevation from an astronomical formula, and the amount of ozone in the whole column of the atmosphere (from the nearest BS ozone measurements). A multiple regression model of the BE irradiance, measured by the BS during clear-sky conditions, on the proxy values at the time of BS measurement, was constructed. A standard least-squares procedure was applied to find the regression constants. Subsequently, the 1 min BE irradiances were calculated throughout the whole day, using a linear combination of regression constants, and the time series of the regression proxies. Similar procedures were commonly used in the past reconstructions of the ground level solar UV [\[26–28\]](#page--1-0).

Two regression models are considered here to calculate the erythemal and vitamin D weighted irradiances with 1 min resolution. The erythemal irradiances from the reconstruction model differ only slightly, by about $2-3\% \pm 7\%$ (standard error), from the routine erythemal irradiances, detected by the Belsk broad-band meter. Moreover, the reconstructed vitamin D irradiances agree (within $a \pm 5$ % range) with those derived by the recently proposed statistical model, that converts the observed erythemal irradiances (from the Belsk's UV broadband meter) to the vitamin D weighted irradiances with an accuracy of about 10% [\[29\].](#page--1-0)

The BE doses are expressed in hundreds of BE Joules per square meter. The standard erythema dose (SED) of 100 $J_{\rm {eryt}}$ m $^{-2}$ is used as a standardized measure of erythemogenic UV radiation. The value of 100 J_{vitD3} m⁻² also approximates the standard vitamin D dose (SDD), corresponding to the UV exposure on 25% of the whole skin area, that is equivalent of an oral daily dose of 1000 IU vitamin D, necessary to keep sufficient vitamin D level in the blood. The recent estimation of the SDD value was about 106 $J_{\rm vitD3}$ $\rm m^{-2}$ for the skin phototype II [\[30\]](#page--1-0). The latest calculations suggested the lower value of SDD [\[31\].](#page--1-0) There is no common agreement on the shape of the pre-vitamin D3 action spectrum, reflecting uncertainties of the photoconversion rate of pro-vitamin D3 to pre-vitamin D3 in hu-man skin [\[32\].](#page--1-0) Hereafter, we denote 1 SDD = 100 J_{vitD3} m⁻². However, this may only be a rough estimate of the UV exposure on 25% skin area, providing an adequate vitamin D status.

Tables 1 and 2 include the monthly means of daily and fractional BE doses, corresponding to a hypothetical behavior of Polish indoor workers. The daily UV exposure are calculated from a 1 min series of the BE irradiances. The mean ratio of the vitamin D action spectrum weighed UV, and the erythemal weighted UV, reaches the late spring/summer plateau (May–August) of 1.7–1.8, and the winter (December–January) minimum of 0.8, that corresponds to the ratios found at mid-latitudinal Canadian stations [\[29\].](#page--1-0)

The average time spent outside, during working days, is supposed to be 1 h (7.30–8.00 am and 3.00–3.30 pm local time, during home–office–home travel). During weekends, it is 1 h around the local noon in the cold period of the year (16 October–14 April), or 2 h in the warm period of the year. This schedule corresponds to that found for British adults [\[14\]](#page--1-0). An alternative scenario assumes that the weekend scenario is valid also for all working days. This scenario is more appropriate for persons on maternity leaves, or for healthy pensioners.

For a standing subject in a mid-latitudinal region (latitude about 50°N), without a hat, about 10% of the whole body (including the face, neck, and hands) is normally exposed. The subject may receive between 5% and 50% of possible daily irradiation, that is incident on horizontal surfaces exposed to the direct sunlight [\[33\].](#page--1-0) Following the recent model [\[17\],](#page--1-0) we assume that a subject receives 15% of the ambient dose at Belsk. Table 1 shows that for such a person, there is a minimal erythema risk during the whole year, as the monthly means of daily erythemal doses are always less than 2 SED (i.e., below the threshold to produce the skin reddening even for a subject with the skin phototype I [\[34\]\)](#page--1-0). [Table 2](#page--1-0) shows that subjects with typical and sun-seeking habits, exposing 1/4 part of the whole body, could receive the UV doses, equivalent to the oral daily intake of 1000 IU vitamin D, in the period of June–July 2010 (normal scenario), and April–September 2010 (sun-seeking scenario), respectively. [Fig. 1](#page--1-0) indicates that there was the erythema risk on some days during late spring and summer in 2010, even for subjects with the skin phototype II, receiving only 15% of the ambient UV dose, during a 2 h solar exposure around noon in the warm period of the year.

2.2. Analytical model

Following the model proposed by Diffey [\[17\]](#page--1-0), for a description of seasonal variation of vitamin D due to the solar radiation, we define a response function $R(t)$, which provides the serum 25(OH)D concentration t days after a single exposure of 1 SDD, i.e.,

$$
R(t) = F(2^{-t/\alpha} - 2^{-t/\beta})
$$
\n(1)

where F is a normalizing factor, α is the half-life (days) for the disappearance of 25(OH)D, and β is the half-life for the synthesis of

Table 1

Monthly means of daily erythemaly weighted doses from the measurements carried out in Belsk, central Poland, in 2010 for the following scenarios: the whole day insolation (from sunrise up to sunset), scenario 1 - the outdoor activity in the periods 7.30–8.00 am + 3.00–3.30 pm in working days and 1 h (cold subperiod of the year) or 2 h (warm subperiod, 15.04–15.10) around noon in weekends, scenario 2-outdoor activity 1 h and 2 h around noon in all days in cold and warm subperiod of the year, respectively. For scenario 1 and 2, the exposure fraction (EF) of 0.15 is assumed.

Month	Mean daily dose in SED				
	Whole day	Scenario 1	Scenario 2	Scenario 1 $EF = 0.15$	Scenario 2 $EF = 0.15$
January	1.722	0.128	0.368	0.019	0.055
February	3.948	0.297	0.782	0.044	0.117
March	8.387	0.636	1.492	0.095	0.224
April	15.488	1.839	3.974	0.276	0.596
May	18.763	2.756	5.348	0.413	0.802
June	31.900	4.020	9.237	0.603	1.386
July	30.994	4.048	8.444	0.607	1.327
August	23.735	3.230	7.364	0.485	1.105
September	11.318	1.703	3.833	0.255	0.575
October	6.845	0.880	2.069	0.132	0.310
November	1.626	0.118	0.351	0.018	0.053
December	1.201	0.078	0.283	0.012	0.042

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