UNIVERSITY OF WARMIA AND MAZURY IN OLSZTYN

Polish Annals of Medicine

Journal homepage: https://www.paom.pl

COLLEGIUM MEDICUM

Review Paper

Do we know how much we can help ourselves by using preventive exposure to solar radiation?

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Article info

Article history
Received: March 20, 2025
Accepted: September 3, 2025
Available online: May 20, 2026

Keywords
Ultraviolet radiation
Vitamin D
Immune system
Skin

Doi
https://doi.org/10.31648/paom/210224

Abstract

Introduction: The role of ultraviolet radiation (UVR) in processes occurring in ecosystem is significant, and the effects of its actions on human health are both positive and negative. UV radiation mainly affects the eyesight and skin. Prolonged exposure of the eyes to ultraviolet radiation, especially without protection can lead to serious diseases. On the other hand, insufficient exposure to UVR may also be a serious public health issue.

Aim: The aim of this paper is to present the health advantages that come from a preventive dose of solar radiation with an overview of their mechanisms of action.

Material and methods: This narrative review summarizes 36 peer-reviewed studies on vitamin D, sunlight exposure, and health outcomes, identified in PubMed, Scopus, and Google Scholar from 2002 to 2024 through screening of titles, abstracts, and full texts.

Results and discussion: Current sun protection guidelines often emphasize avoidance of exposure, which may contribute to vitamin D deficiency and related health problems. UV requirements vary with factors such as skin type, location, and season, underscoring the need for individualized recommendations that balance the risks and benefits of sunlight.

Conclusions: To reap the maximum benefits, it is important to find a balance between protecting oneself from the harmful effects of UVR and ensuring an adequate amount of exposure, which is necessary for vitamin D production and overall well-being.

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1. INTRODUCTION

Ultraviolet radiation (UVR) represents a minor part of the solar radiation reaching the Earth. Its role in processes occurring in the ecosystem is significant, and the effects of its actions on human health are both positive and negative. UV radiation mainly affects the eyesight and skin. Prolonged exposure of the eyes to ultraviolet radiation, especially without protection (such as sunglasses with a filter) can lead to serious diseases, including cortical cataracts, photokeratitis, conjunctivitis, pterygium, and even melanoma. Excessive exposure to solar radiation significantly accelerates the process of skin aging and the development of wrinkles (photoaging), which results from damage to collagen and elastin fibers, blood vessels and the structure of connective tissue. It also triggers the secretion of free radicals, contributes to hyperpigmentation, causes oxidative stress, disrupts the protective barrier, and can even lead to DNA alterations in the skin. It is associated with a higher likelihood of proliferative processes, including basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and malignant melanoma (MM). On the other hand, insufficient exposure to UVR may also be a serious public health issue. Scientific studies have shown that there is a correlation between deficient exposure to ultraviolet radiation and a higher risk of autoimmune or degenerative diseases; or even cancers (breast cancer, colorectal cancer).

2. AIM

The aim of this paper is to present health advantages that come from preventive doses of solar radiation with an overview of their mechanisms of action.

3. MATERIAL AND METHODS

The present narrative review was based on a literature search concerning the role of sunlight exposure and vitamin D in human health. Publications were identified in PubMed, Scopus, and Google Scholar databases, covering the years 2002–2024. The search strategy included the following keywords and their combinations: *vitamin D*, *sunlight exposure*, *health effects*, *autoimmune diseases*, *skin*, *mental health*, *cardiovascular*, and *cancer*. Only peer-reviewed articles written in English or Polish were considered, including original research papers, randomized clinical trials, meta-analyses, and review articles. The selection process involved screening titles and abstracts, followed by full-text evaluation of relevant studies. Finally, 36 publications were included and analyzed for the purpose of this review.

4. RESULTS AND DISCUSSION

4.1. Vitamin D

There are two ways to increase the level of vitamin D in the human body: the first is through its endogenous synthesis, and the second is via dietary intake. Approximately 90% of the vitamin D requirement is covered by its synthesis in the skin.[1] Under the influence of UVB radiation, in the keratinocytes of the basal layer of the epidermis, provitamin D_3 (7-dehydrocholesterol) is converted into previtamin D_3, which, under the influence of heat, undergoes isomerization to vitamin D_3 (cholecalciferol). Cholecalciferol, in combination with the vitamin D-binding protein, is transported to the liver, where it is converted to $25(OH)D_3$ (calcidiol). It acts as a prohormone and does not exhibit biological activity. Calcidiol, in combination with the vitamin D-binding protein, is transported to the proximal tubules of the kidneys, where, with the help of CYP27B1, the active form of vitamin D – $1{,}25(OH)_2D_3$ (calcitriol) – is produced.[2] Nowadays, it is known that calcitriol is also synthesized in other organs and tissues, such as the prostate gland, mammary glands, colon, lungs, and in the B cells of the pancreatic islets.[3] The blood level of calcidiol is used to determine vitamin D concentration, as it reflects the amount derived from both the diet and synthesis in the skin. Even though calcitriol is an active form of this vitamin, it is not typically measured.[1,3]

Vitamin D_3 regulates calcium-phosphate balance, mainly by influencing its absorption in the digestive system. Its optimal level ensures the absorption of approximately 30% of dietary Ca^{2+}, and during periods of increased demand, such as growth, pregnancy or lactation, it can reach as high as 60–80%. An insufficient level of vitamin D significantly reduces the absorption of calcium even to 10–15%.[3] In response to hypocalcemia, parathyroid hormone is secreted from the parathyroids, stimulating calcium release from bones, causing their demineralization, promoting calcitriol production in the kidneys, and increasing phosphate excretion in the urine. As a result, a deficiency of vitamin D leads to insufficient levels of Ca^{2+} in the blood, which consequently increases the secretion of parathyroid hormone. This negatively affects the skeletal system, causing rickets in children and osteomalacia or osteoporosis in adults.

4.2. Prevention of deficiencies

Moderate UVR exposition may cause some health benefits without increasing the risk of skin cancers. Short sessions of solar radiation, especially in the summer, can provide sufficient synthesis of calcidiol.[4] An adult wearing a swimming suit, who is exposed to minimal erythemal dose (MED, minimal amount of UV radiation that causes noticeable redness of the skin that lasts for 24 hours after exposure) can synthesize the equivalent of vitamin D from an oral dose of 20 000 IU, while exposure of the upper and lower limbs to 0.5 MED is equivalent to taking 3 000 IU.[5] Skin production of $25(OH)D_3$ is affected by various factors – such as season and time of day, geographic latitude, skin pigmentation level, age, and the use of sunscreen. An SPF (sun protecting factor) of 30 absorbs about 98% of UVR and reduces the synthesis of vitamin D_3 by 98%.[5] Melanin, which is a pigment present in the epidermis, decreases the penetration of ultraviolet radiation through the skin, weakening the endogenous synthesis of calcidiol. As a result, individuals with a darker skin phenotype require longer sun exposure compared to those with lighter skin to achieve the same level of vitamin D.[6]

4.3. Impact on the immune system

Vitamin D and its analogs play a role in modulating the immune system by regulating both innate and acquired immune responses, including the induction of antibacterial responses, influencing antigen presentation, and promoting T cell differentiation.[7] The ligand $1,25(OH)_2D_3$ binds to the nuclear transcription regulator, the vitamin D receptor (VDR). The result of this is the expression of the CAMP (cathelicidin antimicrobial peptide) genes, which encode the LL-37 peptide and human beta defensin 2 (DEFB4/HBD2), both of which have antimicrobial properties. Gene expression encoding the vitamin D receptor (VDR) has been detected in immune cells, including CD4+ and CD8+ T cells, B cells, neutrophils, as well as in antigen-presenting cells such as dendritic cells (DC) and macrophages. Cathelicidin LL-37 is an antimicrobial protein primarily produced in tissues that are particularly exposed to microorganisms, such as the epithelium of the oral cavity, tongue, esophagus, lungs, intestines, cervix, and vagina, as well as in salivary and sweat glands.[8] It promotes wound healing, angiogenesis and has antiseptic properties. Increased expression of the CAMP gene has been demonstrated as a result of skin damage, infections, as well as in the course of skin cancers (especially BCC) and inflammatory diseases such as psoriasis, while decreased expression has been observed in atopic dermatitis.[9]

4.4. Autoimmune diseases

Vitamin D regulates the differentiation and activity of CD4+ T cells, exerting a suppressive effect on the autoimmune response of the body. It inhibits antigen presentation by dendritic cells, reduces the polarization of Th cells towards Th1, and consequently decreases the production of pro-inflammatory cytokines (IL-1B, IL-6, IFNγ).

The increase in calcidiol levels, resulting from factors such as sun exposure, influences the reduction of rheumatoid arthritis (RA) activity.[10] A cohort study involving 29 368 women aged 55–69 years showed a lower incidence of rheumatoid arthritis (RA) among those with higher vitamin D levels.[11] A lowered $25(OH)D_3$ concentration was associated with high disease activity, the presence of rheumatoid factor (RF) and the need to use at least three disease-modifying drugs.

White describes the correlation between insufficiency of vitamin D_3 and increased incidence of Leśniowski-Crohn disease.[12] The key factor seems to be the effect of vitamin D on reducing the synthesis of the pro-inflammatory cytokine IL-1B, suppressing antigen presentation to T lymphocytes by dendritic cells, as well as inducing the transcription of genes encoding the NOD2 receptor (nucleotide oligomerization domain protein 2). It regulates both directly and indirectly the innate immune system response, of which excessive activation is the basis of the disease's pathogenesis. Nerich et al. studied the relationship between sun exposure and the risk of developing inflammatory bowel diseases (IBD).[13] Increased UVR incidence was associated with a significantly reduced risk of Crohn's disease, but not ulcerative colitis. Subsequent cohort studies conducted on a population of women living in France confirmed this relationship. However, oral intake of vitamin D had no effect on the incidence of the aforementioned intestinal diseases.[14] Therefore, the beneficial effect of sunlight is not solely due to vitamin D levels, but may be linked to other biological mechanisms triggered by it. This suggests that vitamin D supplementation alone does not replace all the potential benefits of natural UVR exposure. It is also suggested that reduced sunlight exposure not only increases the risk of developing diseases but is also associated with a worse progression of the disease itself.[15]

Mohr studied the prevalence of type 1 diabetes (DM1 – diabetes mellitus type 1) among residents of 51 regions worldwide with varying levels of sun exposure (assessed based on the region's distance from the equator).[16] The conducted studies showed a correlation between increased UVB radiation intensity and a decreased incidence of DM1. The results of this study correlate with previous analyses of the effect of oral vitamin D intake on the incidence of type 1 diabetes. Vitamin D_3, by influencing the function of antigen-presenting cells, promotes the formation of regulatory T cells that produce anti-inflammatory cytokines (TGF-β, IL-4, IL-10), instead of effector T cells. This suppresses the production of pro-inflammatory cytokines (TNF-α, IFN-γ, IL-2, IL-17A, IL-21), which helps limit the autoimmune response to insulin.

4.5. Dermatological diseases

Psoriasis is a chronic and widespread dermatosis in which there is an increased immune response mediated by Th1 and Th17 lymphocytes. This leads to excessive proliferation and decreased differentiation of keratinocytes in the epidermis. Vitamin D, through its inhibitory effect on the formation of Th1 lymphocytes, stimulation of differentiation, and suppression of keratinocyte proliferation, may offer benefits in the treatment of moderate forms of this disease.[7] Moosazadeh et al., in their conducted meta-analysis, observed lower levels of calcidiol in patients with psoriasis compared to the healthy population.[17] However, they point to the lack of definitive studies determining whether low levels of $25(OH)D_3$ occurred before the onset of the disease and contribute to the increased incidence, or if they are a result of the disease. Vitamin D increases the expression of LL-37 and HBD2, thereby controlling the inflammatory process. Additionally, it inhibits the production of psoriasin and koebnerisin, which are antimicrobial peptides produced by leukocytes and act as mediators of the systemic immune response in psoriasis.

Increased polymorphism of vitamin D receptors in patients with atopic dermatitis (AD) compared to the healthy population suggests an important role of vitamin D in the pathogenesis of this disease.[18] Under normal conditions, skin damage leads to an increase in the expression of the CAMP gene, which stimulates reepithelialization. However, in patients with AD, this expression is reduced. Vitamin D, by binding to the VDR receptor, increases the levels of LL-37 and HBD2 proteins and modulates the innate immune response. Therefore, it may be used to alleviate the symptoms of the disease.

Acquired vitiligo is a condition in which there is autoimmune destruction of melanocytes in the epidermis. Patients have low levels of vitamin D_3, which may result from recommendations to avoid sunlight, as well as from the pathogenesis of the disease itself. However, it has been shown that therapy using UVA and UVB radiation, as well as the topical application of vitamin D analogs, is effective in alleviating symptoms.[18]

In systemic lupus erythematosus, a correlation has been shown between low levels of vitamin D and increased disease activity.[18] However, the exact mechanism remains unknown.

4.6. Impact on eyesight

Increased exposure to sunlight helps reduce the likelihood of developing myopia.[19] Visible light stimulates retinal cells to produce dopamine, which inhibits the elongation of the eyeball. Reduced release of this neurotransmitter impairs blood flow through the choroid and worsens retinal circulation, leading to a reduction in its volume and, consequently, the elongation of the eyeball.[20] Exposure to UVR for 2–3 hours a day and limiting activities performed in low light conditions help prevent the development of myopia, although they do not slow its progression.[20,21]

4.7. Impact on the nervous system

Sunlight, specifically daylight, has a significant impact on the functioning of the nervous system. This interaction is multifaceted and includes the regulation of the circadian rhythm, influence on mood and cognitive functions.

4.8. Regulation of the circadian rhythm

Daylight is a key factor in regulating the circadian rhythm, which is the body's natural sleep-wake cycle. Information about the light intensity is detected by photoreceptors in the retina and transmitted to the suprachiasmatic nucleus (SCN) in the hypothalamus. The SCN acts as the main biological clock, synchronizing the circadian rhythms throughout the body.[22,23] Exposure to natural light at the appropriate times of the day, especially in the morning, strengthens the signals reaching the SCN and helps maintain the proper functioning of the sleep-wake cycle. Conversely, insufficient exposure, such as during the winter months, can lead to circadian rhythm disturbances, resulting in sleep problems, fatigue, and a decrease in mood.[22,23,24]

4.9. Mood regulation

Sunlight also affects mood. Numerous studies have indicated a connection between UVR exposure and serotonin levels in the brain.[25] Serotonin is a neurotransmitter responsible for regulating mood, sleep, and appetite. Low levels of serotonin are associated with the occurrence of depression.

Exposure to daylight stimulates serotonin production, which may contribute to improving mood and reducing the risk of depression. Studies have shown that individuals who spend more time outdoors, and thus have greater UVR exposure, are less likely to experience depressive symptoms.[23,25,26] Conversely, individuals with limited access to sunlight, such as those living in countries with low sunlight or working in windowless environments, are more susceptible to developing affective disorders.[24]

4.10. Impact on cognitive functions

Sunlight exposure may also influence cognitive functions. Studies suggest that exposure to natural light has a positive impact on memory, concentration, and learning ability.[27] The mechanisms underlying this phenomenon are not yet fully understood, but they are likely related to the influence of UVR on the production of melatonin and cortisol, hormones that regulate the circadian rhythm and stress.

4.11. Impact on body mass

Studies on mice have shown that exposure to UV radiation increases appetite but prevents weight gain, even when on a high-fat diet.[28] This happens because UVR increases the level of norepinephrine, which lowers leptin levels (a hormone that regulates appetite) and increases energy expenditure by converting white adipose tissue to brown adipose tissue. Increased energy intake, driven by heightened appetite, is converted into heat and burned before it has a chance to accumulate in subcutaneous fat tissue, thus preventing weight gain. There is evidence suggesting that exposure to sunlight may reduce the risk of developing obesity and cardiometabolic disorders, such as metabolic syndrome, type 2 diabetes, and non-alcoholic fatty liver disease.[29] Epidemiological observations indicate a link between lower sunlight exposure and a higher incidence of obesity. One of the mechanisms through which UV radiation may influence these diseases is the production of vitamin D in the skin. Low levels of calcidiol are associated with obesity, liver diseases, insulin resistance, metabolic syndrome, type 2 diabetes, and cardiovascular diseases. Other mediators induced by UVR, such as nitric oxide and α-melanocyte-stimulating hormone, may also play a role in the regulation of body weight and metabolism.[29] Stevenson et al. conducted a study showing that physical activity and UVR exposure correlate with higher serum levels of vitamin D.[30] Changes in the 25(OH)D_3 levels partially mediated the effect of sunlight exposure and physical activity on fat tissue in both visceral and subcutaneous areas.[31] Interestingly, sunlight was associated with a lower amount of fat tissue in subcutaneous areas, but a higher amount of fat tissue in visceral areas. The authors suggest that UV radiation may increase the ratio of brown to white fat tissue in the visceral area, which could explain this paradoxical effect.

4.12. Impact on mortality

Many studies suggest that moderate exposure to sunlight can have various health benefits, including reducing mortality, lowering the risk of cardiovascular diseases, and certain types of cancer. A cohort study conducted among Swedish women found that individuals who avoided UVR exposure had a four times higher risk of death compared to the group with the highest exposure.[32] The study suggests that avoiding sunlight may be more harmful than moderate exposure. An analysis of data from the United Kingdom, involving over 395,000 participants, also showed that individuals with higher UV exposure had a lower risk of death from cardiovascular diseases and cancer.[30] It is believed that UVA radiation plays a protective role in cardiovascular diseases. It stimulates the production of nitric oxide in the skin, which has a vasodilatory effect and helps lower blood pressure.[30] A study conducted in Scotland found that higher UVA exposure was associated with a lower risk of myocardial infarction.[34] In another study involving over 29,000 women, it was found that avoiding sunlight was associated with an increased risk of hypertension.[35] Although excessive UVR exposure is a known risk factor for skin cancer, including melanoma, some studies suggest that vitamin D, produced in the skin under the influence of UVB radiation, may have a protective effect against certain internal organ cancers.[36] A study conducted on women with melanoma found that those with higher levels of calcidiol had a better prognosis.[35] Other studies suggest that vitamin D may play a role in the prevention of breast, colorectal, and prostate cancer.[33]

5. CONCLUSIONS

(1) Sunlight, although often perceived as a threat, brings many health benefits. One of the most important is the production of vitamin D, which is essential for healthy bones and the proper functioning of the immune system. Exposure to the sun positively affects our mood, improves sleep quality, and may reduce the risk of certain diseases, such as depression, type 1 diabetes, and autoimmune diseases.
(2) However, excessive exposure carries serious risks. UV radiation damages the DNA of skin cells, which can lead to the development of cancers such as melanoma, basal cell carcinoma and squamous cell carcinoma. Additionally, excessive tanning accelerates the skin aging process, causing wrinkles, discoloration, and loss of elasticity.
(3) Current recommendations for sun protection often emphasize avoiding exposure, but this approach can lead to vitamin D deficiencies and other health issues. It is important to remember that the need for UVR exposure is individual and depends on many factors, such as skin color, geographical location, season, or the type of work one does.
(4) Therefore, it is essential to find a balance between the benefits and risks associated with UV radiation exposure. Sun protection recommendations should be individualized and take into account both the risk of skin cancer and the need for vitamin D synthesis.
(5) Sunlight plays a key role in maintaining proper health. To reap the maximum benefits, it is important to find a balance between protecting oneself from its harmful effects and ensuring an adequate amount of exposure, which is necessary for vitamin D production and overall well-being.

Ethics approval
None declared.

Conflict of interest
None declared.

Funding
None declared.

Author Contributions
Study design: PT, JS, JK, BA, MF
Data collection: PT, BA
Manuscript preparation: PT, JS, JK, MF

References

1 Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. *Am J Clin Nutr.* 2004;80(6 Suppl): 1678–1688. https://doi.org/10.1093/ajcn/80.6.1678S.
2 Tuchendler D, Bolanowski M. Seasonality of vitamin D concentration changes in the human body. *Endokrynol Otyl Zab Przem Mat.* 2010;6(1):36–41. [in Polish] Available from: https://journals.viamedica.pl/eoizpm/article/view/25989 Accessed: May 12, 2010.
3 Napiórkowska L, Franek E. The role of vitamin D testing in clinical practice. *Chor Serca Naczyn.* 2009;6(4): 203–210. [in Polish] Available from: https://journals.viamedica.pl/choroby_serca_i_naczyn/article/view/12035 Accessed: November 25, 2009.
4 Holick MF. Vitamin D deficiency. *N Engl J Med.* 2007; 357(3):266–281. https://doi.org/10.1056/NEJMra070553.
5 Holick MF. The D-lightful vitamin D for child health. *JPEN J Parenter Enteral Nutr.* 2012;36(1 Suppl):9–19. https://doi.org/10.1177/0148607111430189.
6 Joh HK, Hwang SS, Cho B, Lim CS, Jung SE. Effect of sun exposure versus oral vitamin D supplementation on serum 25-hydroxyvitamin D concentrations in young adults: a randomized clinical trial. *Clin Nutr.* 2020;39(3): 727–736. https://doi.org/10.1016/j.clnu.2019.03.021.
7 Bikle D. Nonclassic actions of vitamin D. *J Clin Endocrinol Metab.* 2009;94(1):26–34. https://doi.org/10.1210/jc.2008-1454.
8 Gombart AF, Borregaard N, Koeffler HP. Human cathelicidin antimicrobial peptide (CAMP) gene is a direct target of the vitamin D receptor and is strongly up-regulated in myeloid cells by 1,25-dihydroxyvitamin D_3. *FASEB J.* 2005; 19(9):1067–1077. https://doi.org/10.1096/fj.04-3284com.
9 Fijałkowska M, Kowalski M, Koziej M, Antoszewski B. Elevated serum levels of cathelicidin and β-defensin 2

are associated with basal cell carcinoma. *Cent Eur J Immunol.* 2021;46(3):360–364. https://doi.org/10.5114/ceji.2021.109707.

10 Schwalfenberg GK. Solar radiation and vitamin D: Mitigating environmental factors in autoimmune disease. *J Environ Public Health.* 2012;2012:619381. https://doi.org/10.1155/2012/619381.

11 Hossein-Nezhad A, et al. Vitamin D for health: A global perspective. *Mayo Clin Proc.* 2013;88(7):720–755. https://doi.org/10.1016/j.mayocp.2013.05.011.

12 White JH. Vitamin D deficiency and the pathogenesis of Crohn's disease. *J Steroid Biochem Mol Biol.* 2018;175:23–28. https://doi.org/10.1016/j.jsbmb.2016.12.015.

13 Nerich V, Jantchou P, Boutron-Ruault MC, et al. Low exposure to sunlight is a risk factor for Crohn's disease. *Aliment Pharmacol Ther.* 2011;33(8):940–945. https://doi.org/10.1111/j.1365-2036.2011.04601.x.

14 Jantchou P, Clavel-Chapelon F, Racine A, Kvaskoff M, Carbonnel F, Boutron-Ruault MC. High residential sun exposure is associated with a low risk of incident Crohn's disease in the prospective E3N cohort. *Inflamm Bowel Dis.* 2014;20(1):75–81. https://doi.org/10.1097/01.MIB.0000436275.12131.4f.

15 Ardesia M, Ferlazzo G, Fries W. Vitamin D and inflammatory bowel disease. *Biomed Res Int.* 2015;2015:470805. https://doi.org/10.1155/2015/470805.

16 Mohr SB, Garland CF, Gorham ED, Garland FC. The association between ultraviolet B irradiance, vitamin D status, and incidence rates of type 1 diabetes in 51 regions worldwide. *Diabetologia.* 2008;51(8):1391–1398. https://doi.org/10.1007/s00125-008-1061-5.

17 Moosazadeh M, Damiani G, Khademloo M, et al. Comparing vitamin D level between patients with psoriasis and healthy individuals: A systematic review and meta-analysis. *J Evid Based Integr Med.* 2023;28:2515690X231211663. https://doi.org/10.1177/2515690X231211663.

18 Kechichian E, Ezzedine K. Vitamin D and the skin: An update for dermatologists. *Am J Clin Dermatol.* 2017;19(2):223–235. https://doi.org/10.1007/s40257-017-0323-8.

19 Williams KM, Bentham GC, Young IS, et al. Association between myopia, ultraviolet B radiation exposure, serum vitamin D concentrations, and genetic polymorphisms in vitamin D metabolic pathways in a multicountry European study. *JAMA Ophthalmol.* 2017;135(1):47–53. https://doi.org/10.1001/jamaophthalmol.2016.4752.

20 Biswas S, El Kareh A, Qureshi M, et al. The influence of the environment and lifestyle on myopia. *J Physiol Anthropol.* 2024;43:7. https://doi.org/10.1186/s40101-024-00354-7.

21 Wams EJ, Woelders T, Marring I, et al. Linking light exposure and subsequent sleep: A field polysomnography study in humans. *Sleep.* 2017;40(12):165. https://doi.org/10.1093/sleep/zsx165.

22 Kent ST, McClure LA, Crosson WL, et al. Effect of sunlight exposure on cognitive function among depressed and non-depressed participants: A REGARDS cross-sectional study. *Environ Health.* 2009;8:34. https://doi.org/10.1186/1476-069X-8-34.

23 Raza A, Partonen T, Hanson LM, et al. Daylight during winters and symptoms of depression and sleep problems: A within-individual analysis. *Environ Int.* 2024;183:108413. https://doi.org/10.1016/j.envint.2023.108413.

24 Nagare R, Woo M, MacNaughton P, et al. Access to daylight at home improves circadian alignment, sleep, and mental health in healthy adults: A crossover study. *Int J Environ Res Public Health.* 2021;18(19):9980. https://doi.org/10.3390/ijerph18199980.

25 Lambert GW, Reid C, Kaye DM, Jennings GL, Esler MD. Effect of sunlight and season on serotonin turnover in the brain. *Lancet.* 2002;360(9348):1840–1842. https://doi.org/10.1016/S0140-6736(02)11737-5.

26 Hu L, Shi Y, Zou X, et al. Association of time spent outdoors with the risk of Parkinson's disease: A prospective cohort study of 329,359 participants. *BMC Neurol.* 2024;24(1):10. https://doi.org/10.1186/s12883-023-03499-7.

27 Tremlett H, Zhu F, Ascherio A, Munger KL. Sun exposure over the life course and associations with multiple sclerosis. *Neurology.* 2018;90(14):1191–1199. https://doi.org/10.1212/WNL.0000000000005257.

28 Fleury N, Geldenhuys S, Gorman S. Sun exposure and its effects on human health: Mechanisms through which sun exposure could reduce the risk of developing obesity and cardiometabolic dysfunction. *Int J Environ Res Public Health.* 2016;13(10):999. https://doi.org/10.3390/ijerph13100999.

29 Klinedinst BS, Meier NF, Larsen B, et al. Walking in the light: How history of physical activity, sunlight, and vitamin D account for body fat – a UK Biobank study. *Obesity (Silver Spring).* 2020;28(8):1428–1437. https://doi.org/10.1002/oby.22852.

30 Stevenson AC, Clemens T, Pairo-Castineira E, Webb DJ, Weller RB, Dibben C. Higher ultraviolet light exposure is associated with lower mortality: An analysis of data from the UK Biobank cohort study. *Health Place.* 2024;89:103328. https://doi.org/10.1016/j.healthplace.2024.103328.

31 Quan QL, Kim EJ, Kim S, et al. UV irradiation increases appetite and prevents body weight gain through the upregulation of norepinephrine in mice. *J Invest Dermatol.* 2024;144(10):2273–2284. https://doi.org/10.1016/j.jid.2024.05.024.

32 Lindqvist PG, Epstein E, Landin-Olsson M, et al. Avoidance of sun exposure is a risk factor for all-cause mortality: Results from the Melanoma in Southern Sweden cohort. *J Intern Med.* 2014;276(1):77–86. https://doi.org/10.1111/joim.12251.

33 Marti-Soler H, Gonseth S, Gubelmann C, et al. Seasonal variation of overall and cardiovascular mortality: A study in 19 countries from different geographic locations. *PLoS One.* 2014;9(11):113500. https://doi.org/10.1371/journal.pone.0113500.

34 Mackay DF, Clemens TL, Hastie CE, et al. UVA and seasonal patterning of 56,370 myocardial infarctions across Scotland, 2000–2011. *J Am Heart Assoc.* 2019;8(23):12551. https://doi.org/10.1161/JAHA.119.012551.

[35] Lindqvist PG, Epstein E, Nielsen K, et al. Avoidance of sun exposure as a risk factor for major causes of death: A competing risk analysis of the Melanoma in Southern Sweden cohort. *J Intern Med.* 2016;280(4):375–387. https://doi.org/10.1111/joim.12496.

[36] Alfredsson L, Armstrong BK, Butterfield DA, et al. Insufficient sun exposure has become a real public health problem. *Int J Environ Res Public Health*. 2020;17(14):5014. https://doi.org/10.3390/ijerph17145014.