

Systemic and Hormonal Effects of Ultraviolet Filters in Sunscreens

Güler Dilara Solmaz¹, Oytun Erbaş¹

The sun is the primary source of energy for our planet. Although solar radiation plays a crucial role in essential functions such as heat regulation and vision, it can also have adverse effects, including sunburn and the development of cancer.^[1] Solar radiation consists of three main categories of electromagnetic waves: visible light (VL), ultraviolet radiation (UVR), and infrared (IR) radiation. Visible light, ranging from 380 to 750 nanometers, is the portion of the electromagnetic spectrum that can be perceived by the human eye. Ultraviolet radiation, which has shorter wavelengths than VL, is subdivided into three types: UVA (320-400 nm), UVB (280-320 nm), and UVC (100-280 nm). Approximately 95% of the UVR that reaches the skin is UVA, while the remaining 5% consists of UVB. UVC radiation, on the other hand, is absorbed by the ozone layer and therefore does not reach the Earth's surface.^[2]

The skin, serving as the interface between the body and the external environment, is the largest organ of the human body and is continually exposed to external factors such as UVR, microorganisms, chemicals, and numerous environmental pollutants.^[3]

The acute effects of UVR on the skin include erythema, sunburn, and photoimmunosuppression,

ABSTRACT

The sun serves as a fundamental source of light and heat for the Earth and for all living organisms. While sunlight confers numerous physiological benefits- including facilitating vitamin D synthesis, supporting immune function, contributing to psychological well-being, and driving key photochemical and photobiological processes-prolonged or uncontrolled exposure to ultraviolet radiation (UVR) poses significant health risks. The UVR is considered harmful to human skin due to its capacity to induce DNA damage and mutations, promote skin carcinogenesis, accelerate photoaging, and cause acute injuries such as sunburn. With growing public awareness of the importance of sunscreen use, the potential biological effects of the chemical compounds contained in these products have become a matter of public and scientific interest. Although current evidence regarding the health effects of UV filters in sunscreens remains limited and inconclusive, the increasing prevalence of sunscreen use has encouraged further investigation within the medical and scientific communities. This review provides an overview of sunlight and the pathways through which UVR induces cutaneous responses. It also examines UV filters-the primary active components of sunscreens-as the most accessible and widely used method of photoprotection. In addition, we summarize the major classes of UV filters currently in use and discuss recent research findings and ongoing debates concerning their safety profiles and potential biological effects.

Keywords: Endocrine disruptors, solar radiation, sunlight, sunscreen, ultraviolet filters, vitamin D.

¹ERBAS Institute of Experimental Medicine, Illinois, USA & Gebze, Türkiye

Correspondence: Güler Dilara Solmaz. Institute of Experimental Medicine, 41470 Gebze-Kocaeli, Türkiye

E-mail: diilarasolmaz@gmail.com

Cite this article as: Solmaz GD, Erbaş O. Systemic and Hormonal Effects of Ultraviolet Filters in Sunscreens. JEB Med Sci 2025;6(2):45-52.

doi: 10.5606/jebms.2025.1110

Received : June 18, 2025

Accepted : June 25, 2025

Published online : November 29, 2025

©2025 Journal of Experimental and Basic Medical Sciences. All rights reserved.

whereas its chronic effects comprise photoaging and skin cancer.^[4,5] Seeking shade, wearing protective clothing, using hats and sunglasses, and applying sunscreen can be highly effective in preventing photodamage and photodermatoses caused by UVR.^[5,6]

The level of UVR reaching the Earth's surface depends on variables such as latitude, altitude, season, geographic location, weather conditions, and time of day, whereas individual UV exposure is influenced by factors including clothing, time spent outdoors, age,

skin color, and skin type.^[4] Personal sensitivity to UVR can be assessed either by measuring the minimal erythema dose or by classifying skin types using the method developed in 1975 by Dr. Thomas Fitzpatrick. The Fitzpatrick classification remains a widely used and practical approach. In this system, skin color is categorized into six types: Type I-always burns, never tans; through Type VI-never burns.^[7] Individuals with fair skin and infants are considered high-risk groups and should exercise caution between 10:00 a.m. and 4:00 p.m.^[8]

Exposure to solar UVR is the most important preventable risk factor for skin cancer, with 80–90% of cases associated with UV exposure.^[9] In Türkiye, the number of diagnosed cutaneous melanoma cases increased from 631 in 2016 to 752 in 2020.^[10] In the United States, the incidence of melanoma has increased by more than 320% since 1975.^[11] Following this rise, public awareness has grown, which has indirectly led to increased use of UV filters.

ULTRAVIOLET RADIATION

The solar spectrum includes UVR with wavelengths ranging from 100 to 400 nm.^[4] As the wavelength of radiation decreases, its energy increases. Conversely, the longer the wavelength of UVR, the deeper it can penetrate the human skin. UVC radiation possesses the shortest wavelength and the highest energy; therefore, it can irritate the cornea and skin and exhibits strong mutagenic potential. UVB radiation reaches the epidermis and affects keratinocytes, Langerhans cells, and melanocytes. It is primarily responsible for tanning and sunburn. UVA radiation, in contrast, has a longer wavelength and lower energy and is less erythemogenic than UVB. However, UVA penetrates more deeply, with approximately 50% reaching the papillary dermis. Consequently, it can target various cellular components, including dendritic cells, fibroblasts, matrix metalloproteinases, T lymphocytes, mast cells, and endothelial cells. Due to these effects, UVA radiation is considered a major contributor to photoaging and the initiation of phototoxic and photoallergic reactions in the skin.^[7,12]

Chromophores are molecules capable of absorbing electromagnetic waves, such as UV or IR radiation, thereby inducing biological and chemical reactions. In human skin, chromophores can be of endogenous origin-such as DNA, urocanic acid, and porphyrins naturally produced by the body-or exogenous, introduced through drugs or chemical compounds, for example, psoralens. Ultraviolet radiation exerts its biological effects by being absorbed by these

chromophores.^[13] UVB radiation is directly absorbed by the DNA chromophore, leading to the formation of abnormal covalent bonds between pyrimidine bases, particularly thymine and cytosine, known as cyclobutane pyrimidine dimers (CPDs). These dimers disrupt the normal structure of DNA, increasing the risk of mutations. This phenomenon is referred to as the “UVB signature,” as it is characteristic of DNA damage specifically induced by UVB radiation.^[14]

Under normal conditions, CPD damage is repaired by the nucleotide excision repair mechanism. However, when mutations impair the proper functioning of the DNA’s natural protective enzymatic systems, UVB-induced damage cannot be effectively repaired. This can result in early-onset photosensitivity, pigmentation disorders, precancerous lesions, and ultimately skin cancer.^[2]

UVB radiation also affects urocanic acid (UCA), a molecule naturally present in the epidermis that actively absorbs UV light, converting it to cis-UCA. This conversion triggers immunosuppression. At the cellular level, UVB exposure induces inflammatory infiltrates, apoptosis of damaged cells-commonly referred to as sunburn cells-and a reduction in the number of antigen-presenting cells, along with alterations in their surface receptors, all of which contribute to UVB-mediated skin effects.^[15]

In contrast to UVB, UVA radiation has a longer wavelength and lower energy, and therefore is not strongly absorbed directly by DNA or other cellular molecules. To date, no specific chromophore has been identified as primarily responsible for UVA absorption. Instead, UVA acts on surrounding molecules to generate reactive oxygen species (ROS), which induce oxidative stress within the cell and indirectly damage structures such as DNA and proteins. The most characteristic form of DNA damage caused by UVA is the oxidative modification 8-oxo-dG, which has been associated with carcinogenesis.^[16,17] It has also been observed that, like UVB, UVA can induce the formation of CPDs, which can vary in type. Approximately 85% of CPDs caused by UVA are thymine-thymine (T-T) dimers, whereas in UVB-induced CPDs, this proportion is around 40%. Although T-T dimers generated by UVA are genetically less mutagenic, they may still contribute to melanoma development.^[18]

When the skin is exposed to short-term but intense UVR, melanin production increases, resulting in a darker skin tone, commonly known as tanning. This response serves as a protective mechanism against sunburn rather than an aesthetic outcome.

Another important effect of UVR exposure is the synthesis of vitamin D. UVB radiation activates 7-dehydrocholesterol molecules in the skin, initiating the production of vitamin D.^[19]

SKIN PIGMENTATION

Melanin, produced by melanocytes in the epidermis, is the primary determinant of skin pigmentation. Exposure to UVR increases melanin production, resulting in enhanced pigmentation—commonly referred to as tanning—and a darkening of the skin.^[20]

Melanocytes, the cells responsible for pigmentation in the skin, are located in the basal layer of the epidermis, as well as in the eyes, hair follicles, and certain neural structures. The primary function of melanocytes is to produce melanin, thereby protecting the skin against UVR. Acting as radiation-sensing cells, melanocytes capture UVR to synthesize two main types of melanin: eumelanin and pheomelanin. Eumelanin produces pigments ranging from brown to black, whereas pheomelanin generates yellowish to reddish pigments. The type of melanin produced is genetically determined.^[21,22]

Eumelanin functions as a biological sunscreen due to its high UV-protective properties and ability to neutralize free radicals. By eliminating ROS, eumelanin contributes to the lower incidence of skin cancer in individuals with darker skin compared to those with lighter skin. Individuals whose melanocytes predominantly produce eumelanin typically have darker skin and hair. In contrast, pheomelanin provides less UV protection. In fair-skinned individuals with a predominance of pheomelanin, melanin is insufficient to effectively shield against solar radiation, resulting in a higher risk of malignant tumors.^[23] Recent studies have shown that not only UVR but also VL and IR radiation can damage the skin.^[4,5,24] Visible light and near-IR radiation can induce pigmentation even in the absence of UV. Solar VL has been observed to cause pigmentation, particularly in individuals with darker phenotypes; however, these effects are noticeable only under high-dose VL exposure.^[25,26] Additionally, when VL and UVA1 act together, they may exacerbate the harmful effects of sun exposure: phototoxicity in fair-skinned individuals, and post-inflammatory hyperpigmentation or dark facial spots in darker-skinned individuals.^[25,27]

VITAMIN D

Vitamin D plays a crucial role in multiple physiological processes, including calcium and

phosphorus homeostasis, bone health, immune regulation, and cardiovascular protection. Its most important biological function is to promote enterocyte differentiation and facilitate intestinal calcium absorption, thereby maintaining calcium balance in the body.^[28]

Vitamin D can be obtained through two primary sources: diet or cutaneous synthesis induced by sunlight. More than 90% of the total vitamin D in the body is produced via skin photosynthesis triggered by UVB radiation. There are two main forms of vitamin D: cholecalciferol (vitamin D₃), derived from human and animal sources, and ergocalciferol (vitamin D₂), derived from plant sources.^[29,30]

In the epidermis, particularly in areas such as the palms, face, and back, 7-dehydrocholesterol molecules undergo photochemical conversion upon UVB exposure. The UVB radiation breaks a specific bond in the B-ring of 7-dehydrocholesterol, forming previtamin D₃. This transformation cannot be induced by UVA radiation. Subsequently, thermal isomerization converts previtamin D₃ into active vitamin D₃.^[31]

Both vitamin D₃ and D₂ are biologically inactive and require further enzymatic conversion to become active. Initially, vitamin D₃ is hydroxylated in the liver to form 25-hydroxyvitamin D (25(OH)D, calcidiol). This is the main circulating form of vitamin D in the bloodstream and is the primary marker assessed in clinical measurements due to its long half-life. Subsequently, 25(OH)D undergoes a second hydroxylation in the kidney by the enzyme 1 α -hydroxylase, resulting in the formation of 1,25-dihydroxyvitamin D₃ (1,25(OH)₂D₃), also known as calcitriol.^[32] This compound represents the active form of vitamin D. Calcitriol exerts multiple biological effects: it binds to enterocytes in the intestine to enhance calcium and phosphorus absorption, regulates osteoblast and osteoclast activity in bone, supports bone mineralization, increases calcium reabsorption in the kidneys, modulates the activity of monocytes, macrophages, and dendritic cells, regulates inflammation, and exerts antiproliferative effects.^[28]

In a study conducted in 1987, unprotected individuals showed a significant increase in vitamin D₃ production following UV exposure, whereas protected individuals exhibited no such increase.^[33]

The reflection or absorption of UVB light affects the penetration of solar radiation into the deeper layers of the skin, thereby inhibiting the conversion of 7-dehydrocholesterol to previtamin D₃. Both *in*

vivo and *in vitro* studies have demonstrated that even moderate sunscreen use can reduce vitamin D production. However, other research indicates that when sunscreens are applied under real-life conditions, they do not significantly affect circulating vitamin D levels.^[34] This is because not all sun-exposed areas of the body are typically covered with sunscreen. Furthermore, it should be noted that vitamin D synthesis is influenced by numerous factors, including skin type, age, anatomical site, clothing, weather conditions, latitude, and time of day. Currently, there are no real-life trials evaluating the long-term use of high sun protection factor sunscreens. It is important to remember that sunscreens protect the skin from UV damage, sunburn, photoaging, and skin cancer. A balanced approach can be achieved through regular monitoring of serum vitamin D levels and controlled, short-term sun exposure.^[29-33]

CORE INGREDIENTS OF SUNSCREENS

Sunscreens are an important tool for protecting the skin against the harmful effects of UVR. They contain UV filters, which are compounds or substances capable of absorbing, reflecting, or scattering sunlight, thereby providing protection from harmful UV rays. However, these filters are not used alone; they are combined within a carrier system to ensure uniform distribution, stability, and proper application on the skin. An ideal sunscreen should possess the following characteristics: a broad-spectrum effect against both UVA and UVB radiation, retention of efficacy and appearance upon exposure to sunlight, adherence to the skin without running, provision of adequate protection, suitability for the intended skin type, ease of spreading, and a light texture. In addition to these features, it should be tested for both safety and effectiveness.^[34-37]

Ultraviolet Radiation Filters

Ultraviolet filters are classified into two main categories: organic (chemical) and inorganic (physical/mineral). Additionally, based on their ability to absorb UVR, UV filters can be categorized as UVA, UVB, or broad-spectrum filters that protect against both UVA and UVB.^[35]

Organic filters are carbon-based molecules that absorb UVR. They neutralize the absorbed energy by converting it to longer wavelengths or heat, which is then released from the skin. Most organic UV filters contain aromatic rings, which are highly effective at absorbing light and dissipating energy. In the absence of external energy, the conjugated electrons

in these compounds remain in their lowest energy state. When UVR from sunlight is absorbed by the molecule, the electrons are excited to a higher energy level, rendering the molecule temporarily electrically unstable. As the electrons return to their ground state, the molecule releases the excess energy as heat or as less harmful, longer-wavelength light, thereby rendering the UV energy harmless.^[36] Each chemical molecule has a distinct maximum absorption wavelength, which depends on its molecular structure. Therefore, a single filter is generally insufficient to provide adequate protection against both UVA and UVB radiation. Commercial sunscreens combine multiple filters to achieve broad-spectrum protection. For organic filters, a waiting period of approximately 15 minutes is usually required to achieve optimal effectiveness. Sunscreens are also formulated in various delivery forms, including lotions, gels, sprays, and sticks.^[37] Some of the commonly used organic UV filters today include butyl methoxydibenzoylmethane (avobenzone), Octocrylene, ethylhexyl methoxycinnamate (octinoxate), and ethylhexyl salicylate (octisalate).^[38]

Inorganic UV filters do not absorb UVA and UVB radiation; instead, they act as a physical barrier by reflecting and scattering the rays in different directions. For this reason, they are often referred to as “blockers.” The two most commonly used substances are titanium dioxide (TiO₂) and zinc oxide (ZnO).^[36,37] These substances are naturally occurring minerals and are chemically stable. Unlike organic UV filters, inorganic filters do not undergo chemical reactions, meaning they do not cause chemical changes in the skin. Therefore, the risk of photoallergic or phototoxic side effects is very low. They are less irritating to sensitive skin and eyes. Upon application, they provide immediate protection due to they act as a physical barrier. However, their disadvantages include being easily washed off upon contact with water and, due to the dense formulations, leaving a white residue on the skin or clothing. With technological advancements, TiO₂ and ZnO have been engineered into nanoparticles, preventing the white residue on the skin. However, the very small size of these particles raised concerns about potential systemic absorption and toxicity. Studies have shown that nano TiO₂ and ZnO do not penetrate the stratum corneum, the outermost layer of the epidermis. Therefore, these substances are considered safe.^[39,40]

Modern sunscreens contain additional components designed to mitigate UV-induced skin aging, mutagenesis, erythema, wrinkling, and

oxidative effects. The inclusion of antioxidants and DNA repair enzymes in topical sunscreens has expanded the concept of active photoprotection, enhancing the protective capacity of conventional sunscreens.^[41]

REGULATORY FRAMEWORKS

Sunscreens are classified differently across countries and are subject to varying regulatory frameworks. In the United States, sunscreens are regulated as drugs, whereas in the European Union, they are considered cosmetics.^[42,43] These differences pose challenges for international harmonization. The Food and Drug Administration classifies sunscreens as over-the-counter drugs, meaning they can be sold without a prescription, and as a result, the approval process is very strict. Due to these stringent regulations, no new UV filter has been approved in the U.S. since the 1990s.^[42] In the European Union, the EC 1223/2009 Cosmetics Regulation, which includes the list of UV filters permitted in cosmetic products, currently contains 37 UV filters.^[43] In Türkiye, according to the Cosmetics Regulation published by the Turkish Medicines and Medical Devices Agency (TITCK), the list of UV filters permitted for use in cosmetic products currently includes 34 UV filters. This regulation specifies, for each substance, its chemical name, Chemical Abstracts Service number, maximum permitted concentration, and conditions of use. The TITCK operates in alignment with the European Union's Cosmetic Regulation.^[44]

SYSTEMIC EFFECTS OF UV FILTERS

Studies have shown that UV filters used in sunscreens can penetrate the skin and enter systemic circulation, thereby potentially affecting endocrine processes.^[45] Benzophenone-3 (BP-3) and octyl methoxycinnamate (OMC)/ethylhexyl methoxycinnamate are two commonly approved sunscreen ingredients in the USA, European Union, and Türkiye, known to possess endocrine-disrupting potential. In a study by Janjua et al.,^[46] three compounds-BP-3, OMC, and 4-methylbenzylidene camphor (4-MBC)-applied topically to 32 healthy individuals were detected in both blood plasma and urine. Moreover, it was reported that some UV filters could remain in the skin even 21 days after a single application. In another study involving 200 pregnant women, benzophenone derivatives were detected in amniotic fluid, fetal tissues, umbilical cord cells, and urine.^[47]

In a study examining breast milk from mothers using products containing UV filters, 46 out of 54 samples (85.19%) were found to contain UV filters. The two most frequently detected UV filters were 4-MBC and octocrylene, and a significant correlation was observed across the entire group of UV filters.^[48]

The UV filters used in sunscreens also affect the hormonal system. Studies have shown that these filters can exhibit both estrogenic and anti-androgenic activities. Some filters mimic hormone effects by activating these receptors, while others bind to the receptors and inhibit the action of natural hormones.^[49]

Schlumpf et al.^[50] reported that BP-3 and 4-MBC exhibited estrogen-like effects in immature Long-Evans rats by increasing uterine weight in uterotrophic assays. In the same study, compounds such as BP-3, homosalate, 4-MBC, and OMC demonstrated estrogenic activity *in vitro*, particularly enhancing cell proliferation in the Michigan Cancer Foundation-7 (MCF-7) breast cancer cell line. These cells are estrogen-sensitive, and their proliferation can be considered an indicator of estrogenic activity. A study published in 2016 examined the effects of environmental chemicals with endocrine-disrupting properties on serum total testosterone levels in 588 children and adolescents. The study found that BP-3 exposure in males was significantly associated with decreased serum testosterone levels, indicating that this compound exhibits anti-androgenic activity.^[51] These findings may raise concerns about the safety of sunscreen use, particularly in male children during critical developmental periods.

Beyond these effects, BP-3 has also been observed to impact the nervous system. Studies have reported that BP-3 can induce neurotoxicity and suppress apoptosis and autophagy. Additionally, BP-3 alters the expression of certain receptors.^[52] These effects may be particularly significant during pregnancy and developmental periods. In a study by Jin et al.,^[53] zebrafish larvae and human neuroblastoma cells exposed to high doses of ZnO exhibited nervous system effects such as oxidative stress and dopaminergic cell damage, which are also observed in Parkinson's disease. However, according to the safety opinion published by the European Commission's Scientific Committee on Consumer Safety (SCCS), the amount of ZnO absorbed systemically from topical sunscreen application is considered negligible.^[54] However, this study suggests that ZnO, one of the most commonly used inorganic filters, could potentially trigger neurodegenerative processes.

The UV filters also affect the thyroid system. Thyroxine (T4) and triiodothyronine (T3) perform numerous functions in the body. In a study by Schmutzler et al.,^[55] BP-3 and OMC were reported to activate gene expression by stimulating the thyroid hormone receptor, suggesting that these compounds have the potential to mimic thyroid hormone activity. In another study, OMC was administered as a UV filter to groups of pregnant Wistar rats during gestation and lactation. A decrease in T4 levels was observed in both the mothers and male offspring; however, this reduction did not result in the expected behavioral impairments in the rats.^[56]

Whether sunscreens are carcinogenic is also a concern for consumers. Alamer et al.^[57] investigated UV-absorbing compounds commonly added to personal care products, including BP-1, BP-2, BP-3, OMC, 4-MBC, and homosalate. Using an estrogen-sensitive cell line, long-term exposure to these six chemicals resulted in increased cell motility and invasion. Similarly, in the estrogen-insensitive cell line (MDA-MB-231), exposure to these compounds also led to enhanced cell movement. These findings suggest that prolonged exposure to UV filters may increase the metastatic potential and aggressiveness of breast cancer cells. Additionally, a 1985 study exposed rats to TiO₂ particles at doses higher than typical human exposure, resulting in the development of lung tumors in the rats.^[58] As a result of this study, the International Agency for Research on Cancer classified TiO₂ as a potential human carcinogen; however, this effect is considered a potential risk only from high and chronic inhalation exposure. The SCCS does not recommend the use of spray sunscreen products containing nano-TiO₂ and nano-ZnO due to the risk of inhalation.^[54,59] Currently, data and studies regarding the carcinogenicity of UV filters are insufficient.

In conclusion, the sun is the primary energy source for life on Earth, but daily exposure to UVA and UVB rays poses risks such as skin cancer, photoaging, and hyperpigmentation. Sunscreens are the most practical protection against these effects; however, the safety of UV filters-their main active components-remains under debate. Studies show that UV filters can penetrate the skin and may affect the endocrine system, particularly in developing children, and potentially influence neurodegenerative and thyroid-related processes. Despite these concerns, current evidence is insufficient to determine significant health risks. Therefore, the benefits of sunscreen use must be balanced against potential

systemic effects, highlighting the need for ongoing research and regulatory oversight.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Ahmed B, Qadir MI, Ghafoor S. Malignant Melanoma: Skin Cancer-Diagnosis, Prevention, and Treatment. *Crit Rev Eukaryot Gene Expr*. 2020;30:291-7.
2. Osmola-Mańkowska A, Silny W, Dańczak-Pazdrowska A, Olek-Hrab K, Mańkowski B, Osmola K, et al. The sun--our friend or foe? *Ann Agric Environ Med*. 2012;19:805-9.
3. Zerveatçı C, Kılıçoğlu RB, Akbulut MC, Erbaş O. Stem Cells in Dermatology and Anti-Aging Care of the Skin. *JEB Med Sci* 2024;5:81-7.
4. Schalka S, Steiner D, Ravelli FN, Steiner T, Terena AC, Marçon CR, et al. Brazilian Society of Dermatology. Brazilian consensus on photoprotection. *An Bras Dermatol*. 2014 Nov-Dec;89:1-74.
5. Mancuso JB, Maruthi R, Wang SQ, Lim HW. Sunscreens: An Update. *Am J Clin Dermatol*. 2017 Oct;18:643-50.
6. Lionetti N, Rigano L. The new sunscreens among formulation strategy, stability issues, changing norms, safety and efficacy evaluations. *Cosmetics*. 2017;4:15.
7. Sachdeva S. Fitzpatrick skin typing: applications in dermatology. *Indian J Dermatol Venereol Leprol*. 2009 Jan-Feb;75:93-6.
8. Ladizesky M, Lu Z, Oliveri B, San Roman N, Diaz S, Holick MF, et al. Solar ultraviolet B radiation and photoproduction of vitamin D3 in central and southern areas of Argentina. *J Bone Miner Res*. 1995 Apr;10:545-9.
9. Sander M, Sander M, Burbidge T, Beecker J. The efficacy and safety of sunscreen use for the prevention of skin cancer. *CMAJ*. 2020 Dec 14;192:E1802-8.
10. T.C. Sağlık Bakanlığı Halk Sağlığı Genel Müdürlüğü Kanser Daire Başkanlığı. Birinci Ş, Demirkol ME, Bağcı HH, editörler. Türkiye Kanser İstatistikleri 2020. Ankara: T.C. Sağlık Bakanlığı Halk Sağlığı Genel Müdürlüğü Kanser Daire Başkanlığı; 2025. https://hsgm.saglik.gov.tr/depo/birimler/kanser-db/Dokumanlar/Istatistikler/Turkiye_Kanser_Istatistikleri_2020.pdf
11. Saginala K, Barsouk A, Aluru JS, Rawla P, Barsouk A. Epidemiology of Melanoma. *Med Sci (Basel)*. 2021 Oct 20;9:63.
12. Hortu I, Karadadas E, Ozceltik G, Tavmergen E, Tavmergen Goker EN, Yigitturk G, et al. Oxytocin and cabergoline alleviate ovarian hyperstimulation syndrome (OHS) by suppressing vascular endothelial growth factor (VEGF) in an experimental model. *Arch Gynecol Obstet*. 2021 Apr;303:1099-108.

13. Young AR. Chromophores in human skin. *Phys Med Biol*. 1997;42:789.
14. Jin SG, Padron F, Pfeifer GP. UVA Radiation, DNA Damage, and Melanoma. *ACS Omega*. 2022 Sep 8;7:32936-48.
15. Hart PH, Norval M. The Multiple Roles of Urocanic Acid in Health and Disease. *J Invest Dermatol*. 2021 Mar;141:496-502.
16. Aykan K, Altuntaş İ, Erbaş O. DNA repair mechanisms: DNA repair defects and related diseases. *D J Med Sci*. 2022;8:130-40.
17. Wondrak GT, Jacobson MK, Jacobson EL. Endogenous UVA-photosensitizers: mediators of skin photodamage and novel targets for skin photoprotection. *Photochem Photobiol Sci*. 2006;5:215-37.
18. Jiveskog S, Ragnarsson-Olding B, Platz A, Ringborg U. N-ras Mutations are Common in Melanomas from Sun-Exposed Skin of Humans but Rare in Mucosal Membranes or Unexposed Skin. *Journal of Investigative Dermatology* 1998;111:757-61.
19. Bikle DD. Vitamin D metabolism, mechanism of action, and clinical applications. *Chem Biol*. 2014 Mar 20;21:319-29.
20. Tran TT, Schulman J, Fisher DE. UV and pigmentation: molecular mechanisms and social controversies. *Pigment Cell Melanoma Res*. 2008 Oct;21:509-16.
21. Cao W, Zhou X, McCallum NC, Hu Z, Ni QZ, Kapoor U, et al. Unraveling the Structure and Function of Melanin through Synthesis. *J Am Chem Soc*. 2021 Feb 24;143:2622-37.
22. Del Bino S, Duval C, Bernerd F. Clinical and Biological Characterization of Skin Pigmentation Diversity and Its Consequences on UV Impact. *Int J Mol Sci*. 2018 Sep 8;19:2668.
23. Addor FAS, Barcaui CB, Gomes EE, Lupi O, Marçon CR, Miot HA. Sunscreen lotions in the dermatological prescription: review of concepts and controversies. *An Bras Dermatol*. 2022 Mar-Apr;97:204-22.
24. Zastrow L, Meinke MC, Albrecht S, Patzelt A, Lademann J. From UV Protection to Protection in the Whole Spectral Range of the Solar Radiation: New Aspects of Sunscreen Development. *Adv Exp Med Biol*. 2017;996:311-8.
25. Yeager DG, Lim HW. What's New in Photoprotection: A Review of New Concepts and Controversies. *Dermatol Clin*. 2019 Apr;37:149-57.
26. Regazzetti C, Sormani L, Debayle D, Bernerd F, Tulic MK, De Donatis GM, et al. Melanocytes Sense Blue Light and Regulate Pigmentation through Opsin-3. *J Invest Dermatol*. 2018 Jan;138:171-8.
27. Narla S, Kohli I, Hamzavi IH, Lim HW. Visible light in photodermatology. *Photochem Photobiol Sci*. 2020 Jan 22;19:99-104.
28. Kolanko M, Brzezińska-Wcisło L. Vitamin D and its receptor – role and activity in the human body. Anomalies of metabolism and structure associated with psoriasis. *Post Dermatol Alergol*. 2011;3:212-6.
29. Kulda V. Metabolizmus vitaminu D [Vitamin D metabolism]. *Vnitr Lek*. 2012 May;58:400-4.
30. Gürler A, Erbaş O. The Role of Vitamin D in Cancer Prevention: A Review of Molecular Mechanisms. *JEB Med Sci* 2022;3:79-83.
31. Heath KM, Elovic EP. Vitamin D Deficiency: Implications in the Rehabilitation Setting. *Am J Phys Med Rehabil*. 2006;85:916-23.
32. Erbaş O, Solmaz V, Aksoy D, Yavaşoğlu A, Sağcan M, Taşkıran D. Cholecalciferol (vitamin D 3) improves cognitive dysfunction and reduces inflammation in a rat fatty liver model of metabolic syndrome. *Life Sci*. 2014 May 17;103:68-72.
33. Matsuoka LY, Ide L, Wortsman J, MacLaughlin JA, Holick MF. Sunscreens suppress cutaneous vitamin D3 synthesis. *J Clin Endocrinol Metab*. 1987 Jun;64:1165-8.
34. Libon F, Courtois J, Le Goff C, Lukas P, Fabregat-Cabello N, Seidel L, et al. Sunscreens block cutaneous vitamin D production with only a minimal effect on circulating 25-hydroxyvitamin D. *Arch Osteoporos*. 2017 Dec;12:66.
35. Jesus A, Sousa E, Cruz MT, Cidade H, Lobo JMS, Almeida IF. UV Filters: Challenges and Prospects. *Pharmaceuticals (Basel)*. 2022 Feb 22;15:263.
36. Schneider SL, Lim HW. A review of inorganic UV filters zinc oxide and titanium dioxide. *Photodermatol Photoimmunol Photomed*. 2019 Nov;35:442-6.
37. Bennett SL, Khachemoune A. Dispelling myths about sunscreen. *J Dermatolog Treat*. 2022 Mar;33:666-70.
38. Breakell T, Kowalski I, Foerster Y, Kramer R, Erdmann M, Berking C, et al. Ultraviolet Filters: Dissecting Current Facts and Myths. *J Clin Med*. 2024 May 19;13:2986.
39. Wang SQ, Balagula Y, Osterwalder U. Photoprotection: a review of the current and future technologies. *Dermatol Ther*. 2010 Jan-Feb;23:31-47.
40. Maier T, Korting HC. Sunscreens - which and what for? *Skin Pharmacol Physiol*. 2005 Nov-Dec;18:253-62.
41. Kern J, Wood E, Almukhtar R, Angra K, Lipp M, Goldman M. Evaluation of an SPF50 Sunscreen Containing Photolyase and Antioxidants for its Anti-Photoaging Properties and Photoprotection. *J Drugs Dermatol*. 2022 May 1;21:517-20.
42. Narla S, Lim HW. Sunscreen: FDA regulation, and environmental and health impact. *Photochem Photobiol Sci*. 2020 Jan 22;19:66-70.
43. European Parliament; Council of the European Union. Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products (recast). *EUR-Lex*. [updated 2023 Dec 1; cited 2024 Mar 24]. Available from: <https://eur-lex.europa.eu/eli/reg/2009/1223/2023-12-01>
44. Türkiye Büyük Millet Meclisi. Kozmetik Yönetmeliği. Resmi Gazete. 2005 Ekim 06; Sayı: 25957. [Mevzuat.gov.tr](http://www.mevzuat.gov.tr). [cited 2025 Jul 15]. Available from: <https://www.mevzuat.gov.tr/mevzuat?MevzuatNo=40405&MevzuatTur=7&MevzuatTertip=5>
45. Klammer H, Schlecht C, Wuttke W, Schmutzler C, Gotthardt I, Köhrlé J, et al. Effects of a 5-day treatment with the UV-filter octyl-methoxycinnamate (OMC) on the function of the hypothalamo-pituitary-thyroid function in rats. *Toxicology*. 2007 Sep 5;238:192-9.
46. Janjua NR, Mogensen B, Andersson AM, Petersen

- JH, Henriksen M, Skakkebaek NE, et al. Systemic absorption of the sunscreens benzophenone-3, octyl-methoxycinnamate, and 3-(4-methyl-benzylidene) camphor after whole-body topical application and reproductive hormone levels in humans. *J Invest Dermatol*. 2004 Jul;123:57-61.
47. Krause M, Frederiksen H, Sundberg K, Jørgensen FS, Jensen LN, Nørgaard P, et al. Presence of benzophenones commonly used as UV filters and absorbers in paired maternal and fetal samples. *Environ Int*. 2018 Jan;110:51-60.
 48. Schlumpf M, Kypke K, Wittassek M, Angerer J, Mascher H, Mascher D, et al. Exposure patterns of UV filters, fragrances, parabens, phthalates, organochlor pesticides, PBDEs, and PCBs in human milk: correlation of UV filters with use of cosmetics. *Chemosphere*. 2010 Nov;81:1171-83.
 49. Kunz PY, Fent K. Multiple hormonal activities of UV filters and comparison of in vivo and in vitro estrogenic activity of ethyl-4-aminobenzoate in fish. *Aquat Toxicol*. 2006 Oct 12;79:305-24.
 50. Schlumpf M, Cotton B, Conscience M, Haller V, Steinmann B, Lichtensteiger W. In vitro and in vivo estrogenicity of UV screens. *Environ Health Perspect*. 2001 Mar;109:239-44. Erratum in: *Environ Health Perspect*. 2001 Nov;109:A517.
 51. Scinicariello F, Buser MC. Serum Testosterone Concentrations and Urinary Bisphenol A, Benzophenone-3, Triclosan, and Paraben Levels in Male and Female Children and Adolescents: NHANES 2011-2012. *Environ Health Perspect*. 2016 Dec;124:1898-904.
 52. Wnuk A, Kajta M. Is the commonly used UV filter benzophenone-3 a risk factor for the nervous system? *Acta Biochim Pol*. 2021 Aug 5;68:557-63.
 53. Jin M, Li N, Sheng W, Ji X, Liang X, Kong B, et al. Toxicity of different zinc oxide nanomaterials and dose-dependent onset and development of Parkinson's disease-like symptoms induced by zinc oxide nanorods. *Environ Int*. 2021 Jan;146:106179.
 54. Scientific Committee on Consumer Safety SCCS OPINION On Homosalate. https://ec.europa.eu/health/sites/health/files/scientific_committees/consumer_safety/docs/sccs_o_244.pdf
 55. Schmutzler C, Gotthardt I, Hofmann PJ, Radovic B, Kovacs G, Stemmler L, et al. Endocrine disruptors and the thyroid gland--a combined in vitro and in vivo analysis of potential new biomarkers. *Environ Health Perspect*. 2007 Dec;115 Suppl 1:77-83.
 56. Axelstad M, Boberg J, Hougaard KS, Christiansen S, Jacobsen PR, Mandrup KR, et al. Effects of pre- and postnatal exposure to the UV-filter octyl methoxycinnamate (OMC) on the reproductive, auditory and neurological development of rat offspring. *Toxicol Appl Pharmacol*. 2011 Feb 1;250:278-90.
 57. Alamer M, Darbre PD. Effects of exposure to six chemical ultraviolet filters commonly used in personal care products on motility of MCF-7 and MDA-MB-231 human breast cancer cells in vitro. *J Appl Toxicol*. 2018 Feb;38:148-59.
 58. Lee KP, Trochimowicz HJ, Reinhardt CF. Pulmonary response of rats exposed to titanium dioxide (TiO₂) by inhalation for two years. *Toxicol Appl Pharmacol*. 1985 Jun 30;79:179-92.
 59. Driscoll KE. Review of Lung Particle Overload, Rat Lung Cancer, and the Conclusions of the Edinburgh Expert Panel-It's Time to Revisit Cancer Hazard Classifications for Titanium Dioxide and Carbon Black. *Front Public Health*. 2022 Jul 28;10:907318.