European Sunlight Association

EUROPEAN SUNLIGHT ASSOCIATION

Voice of the European Indoor Tanning Industry

SUNLIGHT AND SUNBEDS

Natural and Artificial UV Radiation

REVISED WITH NEW SCIENTIFIC EVIDENCE, 2022–2026

2026 EDITION

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Preface

Sunlight, with its infrared, visible, and UV light components, is of central importance to all of us. Without it, the evolution of life on Earth as we know it would not have been possible.

The Earth would be a lifeless ball of ice-coated rock without the sun. It warms our seas, generates our weather patterns, and gives energy to the plants that provide food and oxygen for life on Earth. We know the sun through its heat and light, but other, less obvious aspects of the sun affect our society too: solar flares, for example, can cause blackouts of long-distance radio communications or disturbances in the magnetic field that lead to power outages in cities.

This White Book provides an overview of the scientific knowledge on the effects of exposure to the sun and sunbeds, with a focus on UV radiation. It explains the effects on human health and offers guidance on behaviour and awareness around sun exposure, aiming to inform policymakers and industry stakeholders as well as anyone who wants to understand the complex effects of natural and artificial ultraviolet radiation on human health.

Public health messaging on this topic has often lagged behind the evidence, particularly for studies on sunbeds and their connection with skin cancer. This 2026 edition has been substantially revised to reflect the scientific literature published since the White Book was last released in 2022 and to respond to newer regulatory positions with current evidence.

There are other, often neglected, benefits of UV radiation, as well as the visible and infrared light spectrum, that deserve consideration before forming a judgment. This White Book aims to contribute to a more balanced public message, one that addresses not only the risks but also the often-disregarded health benefits of sun exposure. Today's health messages often condemn sun exposure to the point of advising against it altogether. As later chapters explain, this carries real consequences for human well-being, including higher mortality and an increased risk of developing chronic diseases such as hypertension, diabetes, and Alzheimer's disease.

One of sunlight's main benefits, vitamin D synthesis in the skin, is strongly affected by these public health messages, leaving a majority of people with deficient or insufficient serum levels and the health consequences that follow. Emerging research suggests vitamin D serum levels may be as much an indicator of overall sun exposure as a cause of benefit in their own right, and that UV exposure in general may prove more beneficial than supplementation alone, a distinction this White Book returns to throughout. With modern lifestyles, many of us spend most of the day indoors, which, combined with travel between climate zones or a sudden tropical holiday, is a particular problem: our skin often doesn't get the chance to adapt gradually to increased sunlight. The result is sunburn, the main risk factor for skin cancer.

A Note on the Use of This White Book

This White Book is intended for policymakers, regulators, researchers, and public health professionals as a review of the scientific evidence on sunlight and UV exposure. It summarizes population-level epidemiological findings and their policy implications. It is not a marketing document, and nothing in it constitutes medical advice or a health claim for any specific product or service.

Findings described here, including those on solarium and sunbed use, are associations observed in large population studies. They do not establish that any individual using a sunbed will experience the same outcome, and they should not be presented to consumers as a guarantee or promise of health benefit. Health claims made directly to consumers, including in advertising and on social media, are subject to separate and specific legal requirements in most European jurisdictions, and are not authorized by anything in this document.

Sunbed operators and industry members should not extract or quote sections of this White Book to advertise health benefits of sunbed use to customers. Doing so may create legal exposure for the operator and does not reflect the purpose for which this document was prepared. Members with questions about what can and cannot be said to customers should seek their own legal advice on applicable advertising and health-claim regulation, rather than relying on this document.

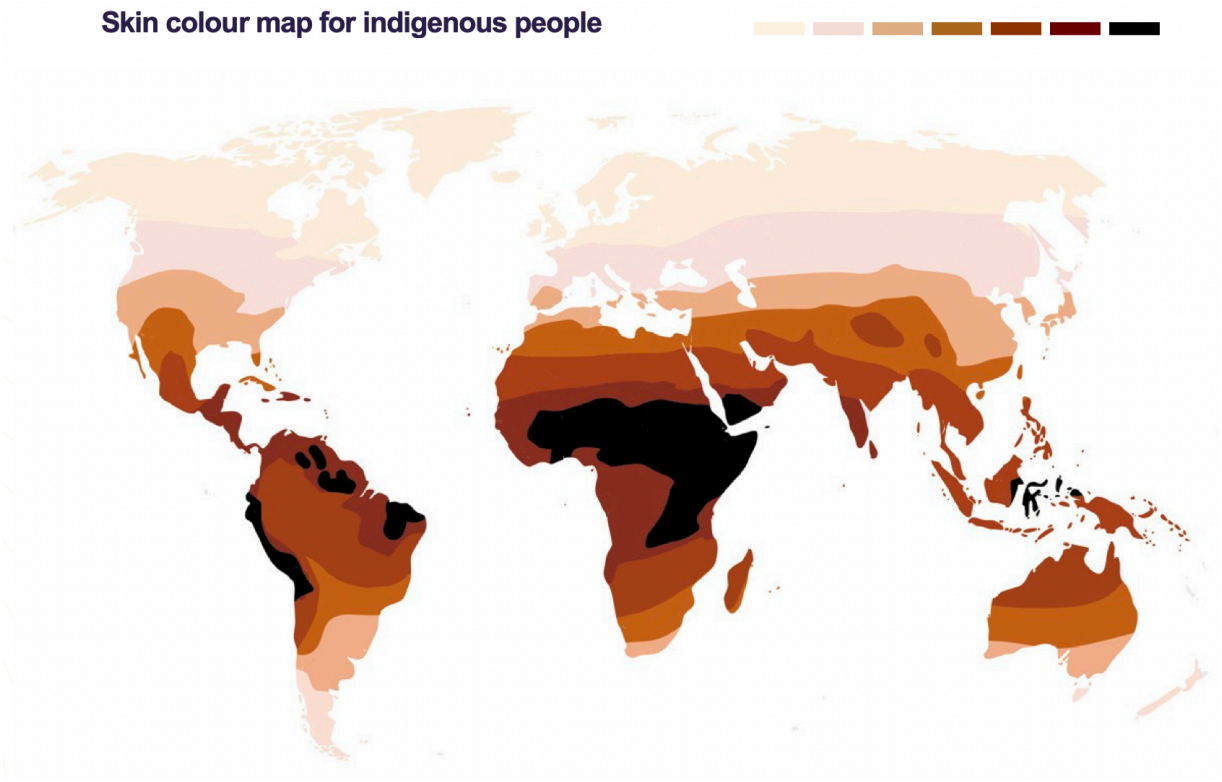
The Evolution of the Human Skin

For a better understanding of the effects of sunlight on the human body, it is necessary to look a bit closer into the evolution of the human skin.

Human skin colour reflects an evolutionary balancing act tens of thousands of years in the making. There's a logical explanation for why human skin tone varies around the globe, with the darkest populations living close to the equator and the lightest ones near the poles: a dark skin is an advantage in sunnier places, whereas pale skin is needed in regions with less sun.

Several million years ago, our ancestors lost their fur and gained pigment in their skin. Many researchers agree the loss of fur helped them stay cool in the sunny, open habitats of equatorial Africa, but the trade-off was bare skin exposed to intense sunlight. Dark, thick skin offered protection from sunburn and, as a consequence, skin cancer. As the human lineage moved to higher latitudes with less sunlight, vitamin D became a problem: the vitamin facilitates calcium absorption, necessary for healthy bones and immunity, and can only be made in the skin when triggered by UVB wavelengths. Away from the tropics, for most of the year there is simply not enough UVB for skin cells to form vitamin D.

So, at high latitudes, people rely on vitamin D stores built up over summer, or acquire the nutrient through food or supplements, or expose themselves to artificial UVB. The darker the skin, the harder it is to maintain adequate vitamin D. Studies comparing dark- and light-skinned residents of northern cities found that paler people had higher vitamin D levels year-round. This adaptability is why skin colour and thickness decrease systematically from south to north: in Europe most of the population has fair skin, while in Africa, with more sunlight, skin is darker.



The Sun

From modern science we have learned that the sun is a big (the Earth fits 1.3 million times into it), very hot (5,500°C on the surface), and old (4.6 billion years) ball of mostly hydrogen (73%) and helium (25%) at the centre of our solar system, around which all the planets orbit.

Life on Earth as we know it would not exist without sunlight, which has shaped evolution as the fundamental source of energy, with photosynthesis as the clearest example. It wasn't until the 1930s that astrophysicists Chandrasekhar and Bethe developed the theoretical concept of nuclear fusion, finally explaining how the sun generates its energy.

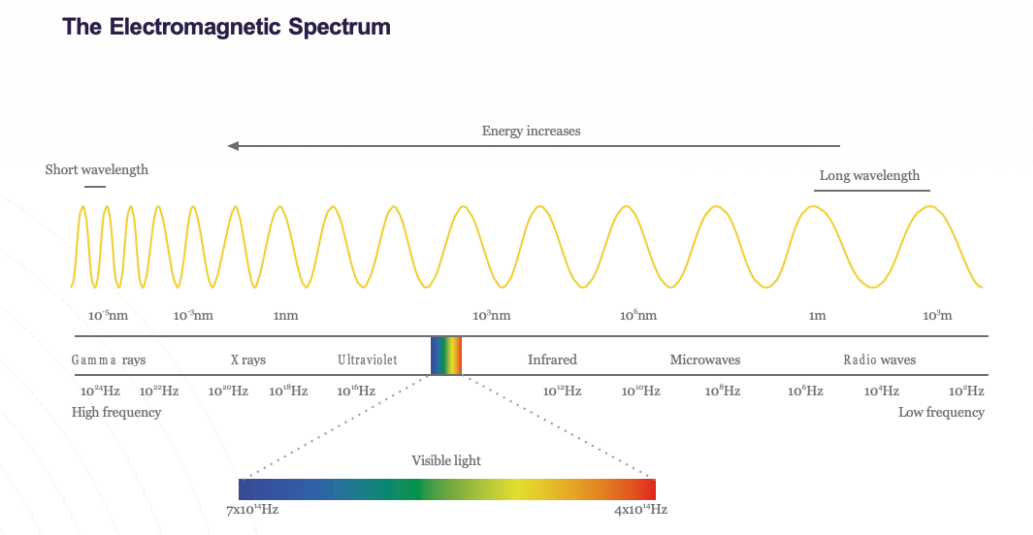
The sun has often played a central role in human history, worshipped as a god in many cultures; Stonehenge and the Pyramids were both used to mark its position over the year. The first accurate measurement of the Earth-sun distance was made by the Greek philosopher Anaxagoras, who was threatened with death for proposing the sun was a burning ball of fire rather than a god. It was Nicolaus Copernicus who first proposed a sun-centred solar system, a theory later supported by Galileo. Historically, the relationship between the sun and human health has shifted between benefit and risk and back again. The ancient Greeks coined the term heliotherapy for healing uses of sunlight. During the Middle Ages the sun fell out of favour and “noble paleness” became fashionable among the wealthy. Light therapy was rediscovered at the start of the 20th century by Niels Ryberg Finsen, who won the Nobel Prize in 1903 for the work.

The Effects: Physical Parameters of the Sun

A short introduction to some of the basic physical parameters of the sun, and some useful explanations to help understand the complexity of this topic.

The Electromagnetic Spectrum

For most of history, visible light was the only known part of the electromagnetic spectrum. The spectrum consists of electromagnetic waves ranging from below one hertz to above 10^{25} hertz, corresponding to wavelengths from thousands of kilometres down to a fraction of the size of an atomic nucleus. The shorter the wavelength, the higher the energy transported. The bands of interest here: 100–380 nm is ultraviolet light, 380–780 nm is visible light, and 780 nm–1 mm is infrared light. Ultraviolet light therefore carries more energy than visible or infrared light, and neither UV nor infrared is visible to the human eye.



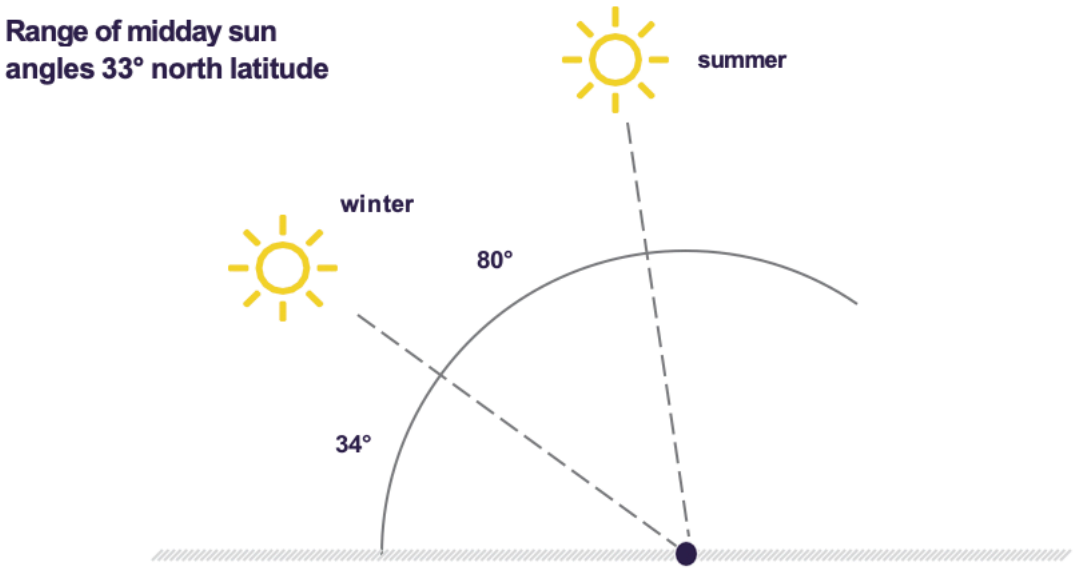
Ultraviolet Radiation

The shorter the wavelength, the higher the energy, but the lower the penetration into the skin. UVA and UVB play the most important roles in tanning and the build-up of natural protection. UVC, the shortest and most powerful UV wavelength, fortunately does not reach the Earth's surface in daylight and is not present in sunbeds. Of UVA and UVB, UVB has the shorter wavelength and is the more effective at causing sun erythema.

The Sun's Altitude, or Angle of Incidence

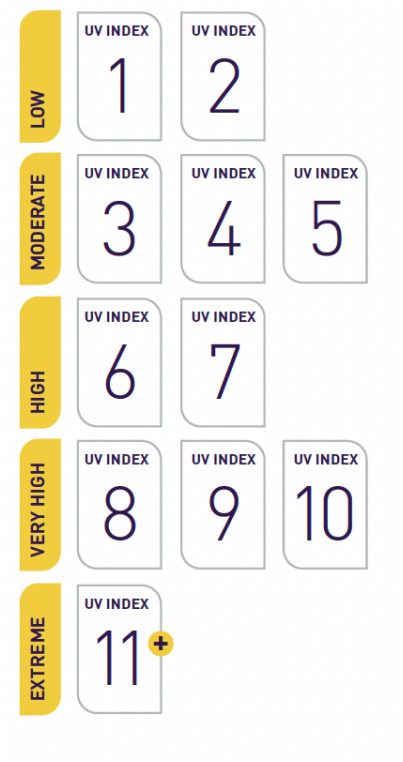
Roughly 50% of solar rays reach Earth as visible light and 45% as infrared; only around 5% is UV radiation, and that share varies widely by location. Of that 5%, at noon on a sunny summer day, roughly 95% is UVA and 5% UVB, a small share but biologically very active. The amount and UVA/UVB ratio of UV reaching the surface depends mainly on the sun's altitude (its angle of incidence) and the thickness of the ozone layer. UVB in particular increases through the morning,

peaks between noon and 3pm in summer, and declines afterward. Seasonally, it is also stronger in central-European summer than winter, when the sun sits low and its rays travel further through the atmosphere.



UV Index

The UV index is a communication tool: an international system for measuring UVB solar radiation for a given day and location, developed jointly by the World Health Organization, the UN Environment Programme, the World Meteorological Organization, and the International Commission on Non-Ionising Radiation Protection in 1994. It is a linear scale: higher values mean greater sunburn risk. An index of 0 is essentially night-time; an index of 12 corresponds roughly to a clear midday summer day in the Mediterranean. A person who would start to burn in 30 minutes at UV index 6 would be expected to burn in about 15 minutes at UV index 12, twice the UV and twice as fast.



Shadow Rule

A simple way to estimate UV exposure is by observing shadow length: a taller shadow generally means lower UVB exposure, a shorter one suggests higher UVB levels.

The Human Skin

To understand how the sun's parameters interact with the skin, it helps to understand the skin's structure and characteristics.

The Skin Layers

Human skin consists of three layers: the epidermis (the outer layer), the dermis beneath it, and the hypodermis, which extends to bone or muscle depending on location.

The Dermis

The dermis is structurally quite different from the epidermis and mostly consists of connective tissue, a fibrous network. It has two layers of its own: the upper dermis contains blood capillaries that nourish the epidermis and a finely woven network of collagen fibres, while the deeper dermis consists of thicker collagen bundles.

Skin Thickness

The epidermis consists of an outer layer of dead, horny cells (the stratum corneum) and, beneath it, a living layer of squamous cells, with a single layer of basal cells adjacent to the dermis. The epidermis renews roughly every 28 days: a dividing basal cell produces one new basal cell and one squamous cell, which gradually cornifies on its way outward to form the horny layer, which has a genuine protective function, including against UV radiation. The epidermis has no blood capillaries of its own (only nerve endings), is about 0.1mm thick on average, with the horny layer just a tenth of that. The entire tanning process takes place within it. Total skin thickness, excluding the hypodermis, ranges from 2–4mm depending on location; the thinnest skin is around the eyes and genitals, the thickest on palms, soles, and the scalp.

Skin Functions

The skin's structure supports several distinct functions: overall protection of underlying organs against mechanical and chemical threats and infection, aided by Langerhans cells that signal the immune system; acting as a large sensor for pain, heat, cold, and pressure; regulating body temperature through dilating or constricting blood vessels and evaporative cooling via sweat glands, which also excrete waste products; and synthesizing vitamin D and other photoproducts in response to sun exposure.

The Tanning Process

Photoadaptation (or Tanning)

The skin adapts to UV exposure by increasing melanin and thickening the horny layer, protecting DNA by reducing the damaging effect of UVA and UVB, together roughly equivalent to an SPF of 10. Regular exposure to sub-erythemal doses of solar-simulating artificial UV over three weeks decreases ultraviolet sensitivity for erythema by an average of 75%, and reduces formation of cyclobutane pyrimidine dimers (CPD, a marker of UV DNA damage) by around 60%, critically with virtually no CPDs found in the basal and suprabasal layers, where DNA damage carries the most consequence given those cells' proliferative capacity.

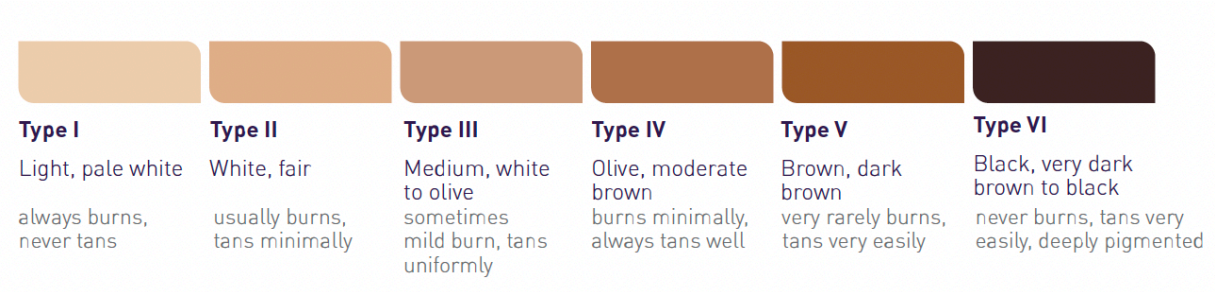
Two Types of Melanin

Tanning occurs in two phases. Immediate pigment darkening begins during UV exposure itself, caused by oxidation of melanin already present in the skin, and is most visible in already-pigmented skin; depending on exposure time it can fade within minutes or persist for days, blending into the second phase. Delayed tanning, induced mostly by UVB, results from increased epidermal melanin production and becomes visible 48–72 hours after exposure.

Skin colour is largely determined by the balance of two pigment types: pheomelanin (brown/red), found mainly in skin types I and II, and eumelanin (brown/black), more abundant in skin types III and IV. Eumelanin absorbs UV radiation effectively and protects the skin; pheomelanin protects less effectively, and can become less effective still when free radicals form. These proportions, and the relative capacity of melanocytes to produce pigment, are largely determined by genetics.

The Fitzpatrick Scale

Developed by Thomas B. Fitzpatrick in 1975 to estimate how different skin types respond to UV light. It was originally used to help dose UVA correctly for light therapy, later refined using patients' own reports of how their skin responds to the sun. It remains a recognised dermatological research tool and is used by professional sunbed salons today:



Positive and Negative Effects on the Human Body

Effects on the Hormone Balance

Sunlight has long-lasting effects on our hormone balance. Under its influence, the brain forms several key hormones: endorphins, produced by the central nervous system and pituitary gland, which act on opiate receptors to reduce pain and boost feelings of well-being; serotonin, a neurotransmitter popularly linked to well-being and happiness, though its actual role is complex, modulating cognition, reward, learning and memory; and melatonin, released by the pineal gland to regulate the sleep-wake cycle.

Vitamin D

Despite its name, vitamin D is actually a fat-soluble hormone (more precisely a secosteroid) responsible for increasing intestinal absorption of calcium, magnesium and phosphate, among other benefits. It affects risk for several cancers and plays a role in hypertension, cardiovascular disease, diabetes, depression and multiple sclerosis, among other conditions. UVB exposure is the main natural route to vitamin D, providing an estimated 80–90% of a person's daily requirement comes from it.

In the skin, UVB irradiation of 7-dehydrocholesterol (concentrated in the stratum basale) converts it into pre-vitamin D3 through a photochemical process; this transforms into vitamin D3, which enters the bloodstream bound to vitamin D binding protein and travels to the liver, where it is hydroxylated into 25(OH) vitamin D3. This circulates to tissues throughout the body, many of which can convert it locally into the active hormone, calcitriol, which binds an intracellular vitamin D receptor and alters the transcription of hormone-sensitive genes.

Sunbeds and Vitamin D

Because sunbeds and the sun have a similar UVA/UVB ratio, sunbeds can raise vitamin D levels. In one experiment using a sunbed compliant with EN 60335-2-27 [16], researchers from Leiden University found vitamin D blood levels in sunbed users rose from 25 to 44 ng/ml over eight weeks, compared with a rise from 23 to 37 ng/ml among supplement users [3]. The researchers concluded that sub-sunburn sunbed treatment was more effective at raising 25(OH)D than 1,000 IU/day of oral supplementation. A separate observational study of 1,433 patients in Edmonton, Alberta found that periodic or regular tanning bed use was associated with higher vitamin D levels [80].

Vitamin D Deficiency

Vitamin D deficiency (serum 25-hydroxyvitamin D below 20 ng/ml) is common across Europe and the Middle East: under 20% of the population in Northern Europe, 30–60% in Western, Southern and Eastern Europe, and around 80% in the Middle East [5]. A 2014 meta-analysis of 73 studies covering more than 800,000 participants found even higher rates, and estimated that 9.4%

of all deaths in Europe and 12.8% in the United States could be attributable to vitamin D insufficiency [6].

Can Diet Alone Solve Vitamin D Deficiency?

For most people, food is not the primary source of vitamin D. Recommended intake ranges from 400–800 IU/day depending on age, though some research suggests 1,000–4,000 IU/day or more may be needed to maintain optimal levels. Vitamin D-rich foods, including fatty fish such as salmon or mackerel, eggs, and fortified milk, orange juice or cereal, can contribute meaningfully, but for many people this still falls short of reaching 20 ng/ml. UV light, natural or artificial, together with supplements where appropriate, remains key to improving vitamin D status at a population level.

Cardiovascular Health

Skin exposure to UV radiation also triggers release of nitric oxide from dermal storage sites into the bloodstream. A growing body of evidence links nitric oxide to cardiovascular health: it expands blood vessels, improving circulation and the supply of oxygen and nutrients to tissues, and can prevent or partially reverse arteriosclerosis.

Effects on the Immune System

UV radiation has potent immuno-modulatory properties affecting outcomes across malignant, inflammatory, autoimmune and infectious disease. This immunomodulation operates both locally and systemically, involving multiple mechanisms across the innate and adaptive immune systems.

Negative Effects on the Human Body

"All things are poison, and nothing is without poison; the dosage alone makes it so a thing is not a poison." This principle, credited to Paracelsus, is the basic tenet of toxicology: a substance is only harmful once it reaches sufficient concentration in the body, even substances as essential as water or oxygen. It underlies public health standards for maximum acceptable concentrations of contaminants in food, water and the environment.

The skin's reaction to sun exposure depends on intensity, duration, and the UVA/UVB ratio: more intense radiation produces a faster, more pronounced response; shorter wavelengths produce a more intense reaction (which is why UVB output in sunbeds is limited, even though it is also what initiates vitamin D production); and longer exposure produces a more intense reaction.

Acute Damage: Sunburn

Sunburn is an irritation and inflammatory reaction triggered by overexposure to UV light, mainly from UVB rays. Symptoms vary by person and by exposure intensity and duration; redness may not appear for several hours, peaking at 12–24 hours. Minor sunburn typically causes slight redness and tenderness; more serious cases can blister, and severe cases can be debilitating enough to require hospital care. In tanning salons, exposure duration is calculated and personalized per customer specifically to avoid sunburn. If sunburn does occur, further sun or UV exposure should stop immediately; cooling and moisturizing with an aloe vera lotion generally supports healing over a few days, and a doctor should be consulted if blistering occurs.

Chronic Damage: Premature Skin Ageing

The causes of skin ageing are not fully understood, but genetics, lifestyle factors (smoking, alcohol, diet), environmental influences and UV exposure are all implicated. UV radiation, mainly UVA, can accelerate collagen breakdown beyond normal ageing, both by promoting abnormal elastin build-up and by generating free radicals. Natural photoprotection helps reduce this risk by limiting how much UVA reaches the dermis.

Skin Cancer

An increased risk of non-melanoma skin cancer (rarely fatal) can be among the more serious consequences of chronic sun exposure; in some studies, regular, moderate sun exposure has shown an inverse association with melanoma specifically, the most dangerous form. UV exposure effects accumulate over a lifetime: responsible behaviour as an adult does not undo the effect of frequent childhood sunburns. Recognised risk factors for skin cancer include:

- Fair, light-sensitive skin
- MC1R variant (red hair)
- A history of sunburn, particularly in childhood
- Excessive or burning UV exposure
- Many (>50) or unusual moles
- A family history of skin cancer or melanoma
- Poor diet, obesity, or low vitamin D

Other factors, including certain inherited conditions and a weakened or suppressed immune system, also play a role. Anyone with one or more of these risk factors is advised to avoid direct or intensive sun exposure.

Allergic and Toxic Reactions

Photoallergic reactions are caused by drugs whose structure is altered by UV exposure such that the immune system treats them as invaders, producing eczema-like, often long-lasting inflammation in sun-exposed skin; common culprits include some sunscreens, antimicrobials, painkillers, chemotherapy drugs and fragrances, and once triggered the reaction is often permanent. Phototoxic reactions, by contrast, don't cause lasting UV sensitivity and can be triggered by certain foods (citrus, parsley) or cosmetic products that accelerate tanning; these artificially heighten UV sensitivity and should be avoided.

Because chronic effects like skin ageing or skin cancer can take decades to appear, establishing a clear causal link to any single period of exposure is difficult. Since the possibility cannot be ruled out, however, responsible, moderate exposure remains important. Extra caution is warranted around midday in midsummer or in southern latitudes, using shade, clothing, or sunscreen as appropriate.

> *"The advantage of a sunbed is that exposure to UV light can be controlled more precisely than casual sun exposure."*
> — Dr. Reinhold Vieth, Department of Laboratory Medicine and Pathobiology, University of Toronto, Canada

The Difference Between the Sun and Sunbeds

Much of the evidence in this White Book points to the same conclusion: most people need more sunlight than they get. At certain latitudes it simply isn't available year-round, which raises the question of whether sunbeds can compensate, and how artificial and natural tanning differ.

A sunbed's irradiance spectrum is constant, while the sun's varies by geographic location and season, as the Earth's axis tilts toward or away from the sun. A sunbed's composition is broadly similar to the sun's, though the UVA/UVB ratio differs by location on Earth; unlike the sun, a sunbed's output stays stable, and its "strength" does not vary by time of day, season, or location the way sunlight's does. Alongside supporting vitamin D production, this gives sunbeds a measured, controllable exposure, since the irradiance intensity doesn't change, so dosage can be monitored carefully to help prevent overexposure and burns. Depending on skin type, a sunbed can also help prepare and adapt skin ahead of increased natural sun exposure, such as before a holiday, reducing sunburn risk.

To limit the risk of burns and long-exposure skin damage, the EU adopted a mandatory standard in 2007 (EN 60335-2-27) [16] capping sunbed irradiance at 0.3 W/m^2. In practice, a sunbed session cannot exceed the UV output of the midday Mediterranean sun (UV index 12).

Scientific Evidence

There is an overwhelming amount of research covering both adverse and positive effects of sunlight on human health. While overexposure to UV radiation can have serious consequences such as skin ageing and skin cancer, moderate, non-burning sun exposure is associated with decreased risks of all-cause mortality, several types of cancer, cardiovascular disease, Alzheimer's disease, and type 2 diabetes, among others.

Disclaimer: the statements in this chapter do not constitute medical advice. Consult a general practitioner or specialist for individual guidance.

Prevention of Rickets

Rickets was common among children in sun-deprived societies in the late 19th century and is caused by a deficiency of vitamin D, calcium, or phosphate, leading to insufficient calcification of the growth plate in bones. Due to sun-avoidance policies and lifestyle changes, the disease, marked by softened, weakened bones and poor growth and most common in children aged 6–24 months, has seen a resurgence. NHS data recorded 101,136 UK hospital admissions in 2017–2018 where vitamin D deficiency was a primary or secondary factor, a 34% rise on the previous year, alongside 474 admissions where rickets itself was the main or secondary cause.

Doctors discovered in the early 20th century that cod liver oil and UV exposure could cure rickets, a finding that led to the discovery of vitamin D itself in 1921. Modern prevention focuses on maternal vitamin D status and adequate calcium intake in areas with low dietary calcium. As one review of vitamin D's skeletal and non-skeletal effects put it, vitamin D plays a central role in maintaining calcium and phosphate balance and in normal bone growth and mineralization. [8]

Osteomalacia & Osteoporosis

In adults, the same underlying process is called osteomalacia: bones normally consist of a soft inner matrix covered by a hard mineral shell, and when mineralization fails, the bone softens without proper covering. It is most common in people who stay indoors, live in low-sunlight climates, have darker skin pigmentation, or use strong sunscreen consistently; other conditions such as cancer, kidney failure, or liver disease can also cause it.

Osteoporosis, literally meaning "porous bone," is a related condition in which bone density and quality decline, raising fracture risk. Bone loss is silent and progressive, often symptomless until the first fracture. The International Osteoporosis Foundation estimates that 1 in 3 women and 1 in 5 men over 50 are at risk of an osteoporotic fracture, and that one such fracture occurs globally every three seconds.

Whether vitamin D and calcium supplements meaningfully improve bone density remains debated: one study found a significant 15% reduction in total fractures with combined daily vitamin D and calcium, while others found no benefit. [9,10] Since the skin's capacity to synthesize vitamin D declines with age, researchers generally recommend that older adults either supplement or spend more time outdoors; opinions differ on the optimal supplement dose. [11]

Cardiovascular Diseases (CVD)

Cardiovascular diseases remain the leading cause of death globally, responsible for an estimated 17.9 million deaths per year according to the World Health Organization, a figure that dwarfs the roughly 1 million non-melanoma skin cancer diagnoses and 300,000 melanoma diagnoses recorded worldwide each year, though incidence and mortality figures are not directly comparable. Vitamin D status has long been correlated with cardiovascular risk in observational studies, and low serum vitamin D has been associated with higher rates of hypertension and cardiovascular mortality. However, large randomized trials of vitamin D supplementation have not consistently confirmed a protective effect on cardiovascular outcomes, which has shifted scientific attention toward vitamin-D-independent pathways: UVA exposure mobilizes nitric oxide stored in the skin, producing measurable vasodilation and short-term reductions in blood pressure in controlled irradiation studies, and population data continue to show blood pressure and cardiovascular mortality rising with distance from the equator and during winter months.

The most substantial recent evidence comes from Stevenson et al. (2024, Health & Place; corrigendum 2025), who analyzed UK Biobank data from around 395,000 participants and found that higher UV exposure, measured through both solarium use and residential sunlight and validated against measured vitamin D levels, was associated with lower cardiovascular, cancer, and all-cause mortality [58]. A 2024 review by Weller and colleagues (Journal of Investigative Dermatology) reached a similar conclusion: UV radiation is an established skin carcinogen, yet no study has linked sun exposure to increased all-cause mortality, and UK and Swedish cohort data instead point toward reduced cardiovascular and total mortality with higher sun exposure [60].

These are observational associations, and the mechanisms discussed below under "Mechanisms Beyond Vitamin D" give them biological plausibility.

Cancer

Many public health messages focus mainly on the possible negative effects of sunlight, particularly UV radiation. Cancer development is a complex, multistep process, and it is an oversimplification to speak of single causes for most cancers; family history, diet, physical activity, and age all play a role alongside UV exposure.

Some observational studies report an association between higher sun exposure or vitamin D levels and lower incidence of certain internal cancers, and this remains an active area of research. This evidence is largely observational, and the underlying mechanisms are the subject of ongoing research.

UV radiation is classified by the International Agency for Research on Cancer (IARC) as a Group 1 human carcinogen. This classification states how strong the evidence for a carcinogenic effect is; it does not describe how large the risk is at a given level of exposure. For sunbeds, the 2016 SCHEER opinion and the fifth edition of the European Code Against Cancer take the position that no level of use is free of skin cancer risk. This White Book does not share that assessment for moderate, regulated sunbed use; the reasons are set out under "Sunbed Use and Melanoma Risk" in Chapter 7. The relationship between UV exposure and skin cancer, and the role of sunburn in particular, is covered immediately below.

Skin Cancer & Melanoma

Chronic overexposure to the sun likely plays a role in increasing the risk of skin cancers, mainly non-melanoma skin cancer (NMSC). For melanoma, intermittent sun exposure and sunburn are the more significant risk factors. While incidence is rising worldwide, many other factors contribute to this trend, as described elsewhere in this chapter.

NMSCs are rarely deadly. The overall 5-year survival rate for melanoma patients is 92% according to the American Cancer Society: 92 of every 100 people diagnosed will be alive in five years. In the earliest stages the 5-year survival rate is 99%; once melanoma has spread to the lymph nodes it falls to 64%, and to just 20% if it has spread to other parts of the body.

A 2019 study investigating vitamin D levels and melanoma risk found that both deficient and insufficient serum vitamin D were associated with melanoma, with a trend toward reduced risk as vitamin D approaches normal values. [17] Separately, a study of U.S. Navy personnel in the 1990s found outdoor workers had a lower melanoma risk than office workers. [18]

Breast Cancer

Breast cancer is the most common invasive cancer diagnosed among women and the second leading cause of cancer death in women. Risk factors include genetics as well as lifestyle factors such as alcohol intake.

A pooled analysis of data from over 5,000 women aged 55 and older, drawn from two randomized trials at Creighton University, found a 78–82% lower risk of breast cancer for women with vitamin D levels of 60 ng/ml or greater compared with those below 20 ng/ml. [19] A separate meta-analysis found that serum 25(OH)D deficiency was associated with breast cancer occurrence in the general population, and concluded that increasing sunlight exposure may be a more effective preventive strategy than diet or supplements alone. [20]

Colorectal Cancer

Colorectal cancer is the third most common cancer worldwide, with over 1.8 million new cases in 2018, and is considered one of the clearest markers of epidemiological and nutritional transition as its incidence rises alongside declining infection-related cancers in societies undergoing rapid economic change.

A meta-analysis of 15 studies across 14 countries found a clear, dose-dependent reduction in colorectal cancer risk with higher vitamin D serum concentrations: a level of 30 ng/ml was associated with a 33% lower risk, and 50 ng/ml with a 60% lower risk. [21]

Prostate Cancer

Prostate cancer is one of the most common cancers in men, with 1.3 million new cases in 2018. It usually grows slowly and remains confined to the prostate, though some forms are aggressive and spread quickly.

An inverse association between serum vitamin D and prostate cancer risk has been shown in multiple studies, with a dose-response relationship: each 8 ng/ml increment in serum vitamin D is associated with a 9% lower risk of prostate-cancer-specific mortality. [22,23]

Non-Hodgkin Lymphoma

Non-Hodgkin lymphoma (NHL) begins in lymphocytes, part of the body's immune system, and is among the 5th to 9th most common cancers globally, with almost 510,000 new cases estimated in 2018.

Significant protective associations with sunlight and UV exposure have been observed, with risk decreasing 20–33% between low- and high-exposure groups. Because dietary vitamin D intake did not improve the risk estimate in one study, the authors concluded this points to a vitamin-D-independent mechanism. [24]

Lung Cancer

Lung cancer is one of the most common and serious cancers worldwide, with few early symptoms. Smoking accounts for roughly 85% of cases, though it can occur in never-smokers too, and it carries the second-highest mortality of all malignancies.

A study led by researchers at Yangtze University in China found a dose-response relationship between circulating vitamin D and lung cancer risk: each 4 ng/ml increase in vitamin D was associated with an 8% reduction in risk and a 7% reduction in mortality. [25] A separate study following 210 lung cancer patients for up to 18 years found those with the highest vitamin D levels had the longest survival. [26]

Leukaemia

Leukaemia affects the body's blood-forming tissues, with over 400,000 new diagnoses annually. Researchers at the University of California found that people living at higher latitudes with lower sun exposure, such as the US, Australia, New Zealand, Canada, and Ireland, were at least twice as likely to have leukaemia as those in countries near the equator, such as Nigeria, Bolivia, Samoa, and Madagascar, concluding that higher UVB irradiance and vitamin D levels may help prevent the disease. [27]

Diabetes

Diabetes describes a group of conditions involving the hormone insulin, which the pancreas releases to help the body store and use sugar and fat from food. It occurs when the pancreas produces too little insulin, or when the body does not respond to it properly.

In 2017, an estimated 425 million adults worldwide were living with diabetes, a figure more recent international estimates put substantially higher. Vitamin D is believed to help improve the body's sensitivity to insulin, and observational studies have long linked low vitamin D status with a higher risk of developing type 2 diabetes.

This is now supported by randomized controlled trial evidence. A 2023 pooled individual-participant-data meta-analysis of three RCTs (the U.S. D2d trial, the Japanese DPVD trial, and a Norwegian trial) in people with prediabetes, published in Annals of Internal Medicine, found that vitamin D supplementation reduced the risk of progressing to type 2 diabetes by 15% (hazard ratio 0.85, 95% CI 0.75–0.96), with a 3.3-percentage-point absolute risk reduction over three years [65]. The effect held across prespecified subgroups.

It is worth noting that this trial evidence concerns oral vitamin D supplementation specifically, rather than sun or sunbed exposure, and the two should not be conflated when discussing mechanism, even though both raise circulating vitamin D levels.

A 2026 UK Biobank analysis (Stevenson et al., Scientific Reports) examined UV exposure directly rather than supplementation [81]. Using satellite-derived UVA estimates for 429,368 participants, the study found that higher short-term UVA exposure was associated with modestly lower blood glucose, most clearly in winter. In a second analysis of 408,531 participants, self-reported tanning salon use served as a marker of longer-term exposure, and occasional and frequent users had a 14% and 22% lower hazard of type 2 diabetes than non-users. The authors note that tanning salon use may partly mark broader sun-seeking behaviour and that, as in any observational analysis, differences between users and non-users cannot be fully excluded.

Multiple Sclerosis

Multiple sclerosis (MS) is a chronic condition affecting the brain and spinal cord: the protective coating around nerves (myelin) is damaged, causing symptoms ranging from limb numbness to paralysis or vision loss. It is two to three times more common in women, typically diagnosed between ages 20 and 50, and affects an estimated 2.3 million people worldwide, with higher incidence in colder climates.

A 2018 study of 151 people with MS and 386 controls examined both time spent in the sun and ambient UVB exposure. Those living in high-UVB areas before MS onset had a 45% lower risk than those in low-UVB areas, and those who spent 10 or more hours per week outdoors in summer during high-UVB years (ages 31–40) had an 82% lower risk than those spending under 10 hours outdoors in low-UVB areas. [31] Other studies support a correlation between both serum vitamin D and sun exposure and MS's clinical course. [32,33]

Alzheimer's Disease

Alzheimer's disease is the most common cause of dementia, accounting for 60–80% of cases; nearly 44 million people worldwide have Alzheimer's or a related dementia.

Low vitamin D levels appear to contribute to increased risk. A University of Bordeaux study following 916 participants aged 65 and older for 12 years found a significant association between vitamin D deficiency and faster cognitive decline, alongside a threefold increase in Alzheimer's risk; deficient and insufficient participants had roughly double the risk of all-cause dementia, and nearly triple the risk of Alzheimer's specifically, compared with those at sufficient levels. [34]

Pregnancy

Multiple studies have found that vitamin D levels above 40 ng/ml during conception and pregnancy support maternal and infant health: preterm birth risk is reduced by 60%, pre-eclampsia is almost entirely eliminated, and the risk of postnatal depression is lower. [35,36,37] Comparing low (<12 ng/ml) with higher early-pregnancy vitamin D levels, one study found a 90% increased risk of MS in offspring, and another found a 67% increased risk of type 1 diabetes in offspring linked to low maternal sun exposure. [38,39]

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) mainly refers to ulcerative colitis and Crohn's disease, long-term conditions involving gut inflammation, usually diagnosed between ages 15 and 40. More than 0.3% of people in industrialized nations have IBD. Vitamin D protects the gut barrier and modulates both innate and adaptive immunity; 60–70% of people with IBD have insufficient vitamin D in some studies. Research from the Australian National University found that children spending half an hour a day outdoors significantly reduce their IBD risk. [40]

Immune System

Vitamin D deficiency is associated with increased autoimmunity and greater susceptibility to infection. A University of Edinburgh team studying how vitamin D affects dendritic cells' ability to activate T cells, the immune cells that fight infection but can misfire in autoimmune disease, concluded that low vitamin D status is a significant risk factor for several autoimmune diseases, and that vitamin D metabolites can substantially influence immune function. [41]

Respiratory Diseases & the Flu

Respiratory diseases affect the lungs and other parts of the respiratory system and include asthma, chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, and pneumonia. A 2017 individual-participant-data meta-analysis (Martineau et al., BMJ) found that vitamin D supplementation modestly reduced the risk of acute respiratory infections overall, with the clearest benefit in people who were substantially vitamin D deficient at baseline [42].

This finding has not held up as well once further, larger trials were added. An updated 2024 meta-analysis incorporating six additional RCTs, including one trial of over 15,000 participants, found the overall protective effect was no longer statistically significant [66]. A separate 2024 dose-response meta-analysis of 43 RCTs and more than 49,000 participants likewise found no significant overall preventive effect (risk ratio 0.99, 95% CI 0.97–1.01), though a possible benefit remained for people who were both severely deficient and given daily or weekly low-dose supplementation.

The COVID-19 pandemic prompted a wave of trials testing vitamin D against SARS-CoV-2 specifically; several large randomized trials published in 2022 found no benefit for prevention or severity, and multiple Mendelian randomization studies, which sidestep confounding by using genetic variants as a proxy for lifelong vitamin D status, likewise found no causal association between vitamin D levels and COVID-19 risk or severity.

Regarding the flu, which according to the WHO results in 3–5 million severe cases worldwide each year with 290,000–650,000 deaths, seasonal patterns are driven partly by the virus surviving longer in cold, dry air and partly by lower winter vitamin D levels, though the causal contribution of the latter remains unresolved given the mixed trial evidence above.

These trials tested oral vitamin D supplements, not sunlight or sunbed exposure, which act through additional pathways (see “Mechanisms Beyond Vitamin D” below). Their null results therefore do not settle whether UV exposure itself affects respiratory infection.

Depressive Disorder

Depression is a common mental disorder affecting more than 300 million people worldwide. Sunlight exposure is thought to increase the brain's release of serotonin, associated with improved mood and focus, through light-sensitive pathways triggered via the retina. Seasonal increases in sun exposure have been associated with decreased mental health distress, and other studies show a relationship between vitamin D deficiency and depressive symptoms, though whether low vitamin D is a cause or an effect of depression remains unclear. [44,45,46]

All-Cause Mortality Due to Sun Avoidance

When recommending sun safety, public health messages often advise avoiding the sun outright, without engaging with evidence that this advice itself carries health costs.

A prospective cohort study by researchers at the Karolinska Institutet in Sweden followed 29,518 women for 20 years and assessed sun avoidance as a risk factor for all-cause mortality. Compared with the highest sun-exposure group, all-cause mortality was roughly doubled among avoiders (hazard ratio 2.0; 95% CI 1.6–2.5) and increased by around 40% among those with moderate exposure (1.4; 1.1–1.7) [47]. The same research group later found that women with more active sun-exposure habits had a lower risk of cardiovascular and other non-cancer mortality [48].

The largest and most recent addition to this evidence base is Stevenson et al. (2024, Health & Place; corrigendum 2025), who examined UK Biobank data from around 395,000 participants using two independent measures of UV exposure, solarium use and residential sunlight, both validated against measured vitamin D levels [58]. Higher UV exposure by either measure was associated with lower all-cause, cardiovascular, and cancer mortality, and solarium users additionally showed lower non-cardiovascular, non-cancer mortality. An important distinction runs through this evidence: these are studies of UV exposure itself, sunlight or sunbed use, not of oral vitamin D supplementation. Randomized trials of vitamin D pills have generally failed to replicate the mortality and cardiovascular benefits seen in these UV-exposure cohort studies, leading researchers including Weller and colleagues to propose that UV light confers benefits through vitamin-D-independent pathways, such as nitric oxide release and its effects on blood pressure and immune regulation, rather than through raising circulating vitamin D alone. Findings on sun or sunbed exposure and findings on vitamin D supplementation are therefore related but distinct bodies of evidence, and should not be treated as interchangeable.

In Focus: The Edinburgh UK Biobank Studies

Two studies from the University of Edinburgh, both built on the UK Biobank, form the most robust population-level evidence available on habitual UV exposure and mortality. Stevenson et al. (2024, peer-reviewed) followed around 395,000 white European UK Biobank participants, measuring UV exposure through two independent, validated indicators, solarium use and annual average residential shortwave radiation, both checked against participants' measured serum vitamin D [58; 59]. A larger 2026 preprint by Gu, Stevenson, Weller and colleagues extended this to 419,007 participants and introduced Sun-BEEM, a composite score combining behavioral and environmental UV indicators into low, medium, and high exposure categories, alongside a proteomic sub-cohort of 44,712 participants with blood protein profiling.

Both studies find the same directional pattern: higher UV exposure is associated with lower all-cause, cardiovascular, and non-skin cancer mortality. In the Gu 2026 preprint, medium and high Sun-BEEM exposure carried hazard ratios of 0.89 and 0.84 for all-cause mortality compared with low exposure. Skin cancer mortality showed no clear dose-response relationship with exposure category, even though incident keratinocyte cancers did rise with exposure, a dissociation between skin cancer diagnosis and skin cancer death that runs through both studies. Using g-computation counterfactual modelling, the Gu 2026 analysis estimated that, if the associations are causal, shifting the population to a uniformly high-UV exposure pattern would prevent substantially more cardiovascular and non-skin cancer deaths than it would cause in additional melanoma and keratinocyte cancer deaths.

For this White Book, these studies matter for two reasons. First, they are the largest and most methodologically sophisticated cohort studies yet conducted on this question, in a population that is white-skinned and high-latitude, directly relevant to most of Europe. Second, the proteomic mediation analysis in the Gu 2026 preprint begins to identify biological mechanisms, discussed below, rather than resting on association alone.

Mechanisms Beyond Vitamin D

A recurring theme across this chapter is that the health associations linked to UV exposure do not reliably reproduce when vitamin D is given as a pill rather than obtained from sunlight or a sunbed. Weller (2024, Journal of Investigative Dermatology) proposed an explanation that has become an organizing framework for this field: sunlight benefits health through multiple pathways, of which vitamin D synthesis is only one, and arguably not the most important for cardiovascular outcomes.

The best-characterized alternative pathway is nitric oxide. UV, and UVA in particular, mobilizes nitric oxide stored in the skin, causing arterial vasodilation and measurable drops in blood pressure independent of vitamin D status. A 2025 international expert conference report (Riedmann et al., Photochemical & Photobiological Sciences) reached a consistent conclusion, cataloguing evidence for UV effects on inflammatory and immune pathways that also operate independently of vitamin D [62]. The proteomic mediation analysis in the Gu 2026 preprint adds a further, more granular layer, implicating circulating proteins involved in immune regulation, mucosal barrier function, and cardiorenal-neuroendocrine signalling as potential mediators linking UV exposure to lower cardiovascular and cancer mortality. The Riedmann report also notes that visible and infrared light may have biological actions as well, although most mechanistic studies to date have looked at UV alone.

This vitamin-D-independent framework has practical implications for how the evidence in this White Book should be read throughout: a negative or null result from a vitamin D supplementation trial does not by itself undermine the case for sensible UV exposure, because supplementation and sunlight may not work through the same biological route.

This list highlights only the most prominent conditions studied. Further evidence links sufficient vitamin D levels to celiac disease, headache, dental caries, and Parkinson's disease.

Questions & Evidence

UV Exposure, Sunbeds and Tanning: a section-by-section look at the claims most often raised about sunbeds and sunlight, weighed against the current evidence.

A Suntan Is Natural

This is not the public health message we are getting today, as sunlight and tanning are often portrayed as something to be avoided at all costs. Sun avoidance has been repeatedly shown to carry real costs of its own: sunlight helps maintain healthy vitamin D levels and supports other photoproducts such as nitric oxide, which counters chronic conditions like hypertension and arteriosclerosis. The all-cause mortality rate is roughly twice as high among women who avoid sun exposure compared with those who are more regularly exposed.

A 2019 commentary, "Sun Exposure Public Health Directives," argued that public health guidance should be adjusted to reflect current scientific knowledge, recommending that people without extraordinary sun sensitivity get enough sun exposure to maintain a serum 25(OH)D level above 20 ng/ml, ideally 30–60 ng/ml, while taking care to avoid sunburn. [50]

Sunbeds Are Basically Like the Sun: Enjoy It in Moderation

UV emitted from sunlight and sunbeds is more similar than generally thought: midday summer sunlight is roughly 95% UVA and 5% UVB, and most sunbeds emit a similar composition, with maximum intensity equalling the midday Mediterranean sun. This is enforced by the European standard EN 60335-2-27 [16], which limits sunbed UV output to 0.3 W/m². The key difference is that sunbed UV can be controlled to avoid overexposure by trained, certified operators following professional standards.

Weller and Gu (2025, British Journal of Dermatology) have separately argued that the data underpinning UK melanoma-attributable-risk estimates are weak, and that sun-protection advice modelled on Australian guidance does not transfer well to the very different UV environment of the UK and similar northern European countries [61]. Combined with the finding of the 2026 MISS cohort, discussed below, that solarium use without accompanying patterns of intermittent solar overexposure was not associated with increased melanoma risk, the argument that a well-regulated sunbed session is comparable to safe, moderate sun exposure has more recent empirical support than it did in 2022.

> *"The dogma, now fossilized in print, is that any tan is a sign of skin damage. Tell that to Darwin. Even if there was hard evidence that melanoma was UV-induced, it would be all the more important to keep a protective tan."*
>
> — Prof. Dr. Sam Shuster, Emeritus Professor of Dermatology, Newcastle University, UK

Regular UV Exposure Actually Lowers Melanoma Risk

The relationship between UV exposure and melanoma depends on the pattern of exposure. Melanoma risk is driven strongly by intermittent, intense exposure and sunburn, particularly at a young age, rather than by cumulative, moderate exposure alone. Some studies of outdoor workers

have found lower melanoma rates than in indoor workers [18], a pattern often attributed to adaptive tanning and lower burn frequency with regular, non-burning exposure. The clearest and most consistent risk factor across the literature is sunburn, not moderate exposure. The evidence on sunbed use specifically is discussed under "Sunbed Use and Melanoma Risk" below. Total sun avoidance, meanwhile, is not risk-free either, given the mortality evidence discussed in Chapter 6.

This distinction is now backed by direct empirical data. A 2026 analysis of the Swedish MISS cohort (Lindqvist et al., Photochemical & Photobiological Sciences) followed ~29,000 women for up to 34 years and separated solarium users with indicators of intermittent solar overexposure from those without [63]. Solarium use without overexposure indicators was not associated with increased melanoma risk; risk was concentrated in users who also showed patterns of intermittent solar overexposure (hazard ratio 1.60, 95% CI 1.30–2.00), and roughly a third of melanomas among solarium users were attributable to that overexposure rather than to sunbed use itself. Both solarium-user groups had around 18% lower all-cause mortality than non-users, with no difference in melanoma-specific mortality between them. The authors conclude that melanoma risk among solarium users appears driven by intermittent solar overexposure rather than solarium use per se, and that prevention efforts should target overexposure behavior specifically.

Sunbed Use and Melanoma Risk

Several systematic reviews pool observational studies on sunbed use and melanoma. For ever-use compared with never-use they report summary estimates of 1.20 (Boniol et al., 2012), 1.16 (Colantonio et al., 2014) and 1.19 (Burgard et al., 2018) [71, 72, 52]. These are weak associations from mostly case-control studies, a design that can show that two things occur together but not that one causes the other [51, 73].

Two of these reviews describe weaknesses in the underlying data. Colantonio et al. rate the quality of the evidence as poor to mediocre and note that bias was potentially present in all included studies [72]. Of the 31 studies included by Burgard et al., 11 did not adjust for a single confounder, and only eight adjusted for sun exposure and eight for sunburn [52]. Reichrath et al. add that some studies counted dermatological phototherapy as sunbed use and that several chose their controls in a way that left out people with presumably high earlier UV exposure. In their assessment, such biases inflate the estimate rather than distort it in either direction [51].

The main problem is confounding by sun exposure. People who use sunbeds also sunbathe more, with odds ratios between 2 and 7 for sunbathing among sunbed users compared with non-users, and sunburn from the sun is a recognized risk factor for melanoma [73]. Reichrath et al. describe a scenario in which sunbed use has no effect at all and sunbathing carries a true odds ratio of 1.2. Since sunbathers also visit tanning salons more often, the pooled studies would then yield the same estimates that Boniol et al. and Colantonio et al. report [51]. A study that does not separate the two exposures cannot tell these cases apart.

The Swedish MISS cohort, published in 2026, did separate them [63]. It followed about 29,000 women for up to 34 years. Solarium use without indicators of intermittent solar overexposure, such as sunburn or sunbathing holidays, was not associated with melanoma (hazard ratio 1.07; 95% CI 0.90–1.30). Solarium use together with such indicators was (1.60; 1.30–2.00). Without the

separation, the hazard ratio was 1.28 (1.09–1.51), almost identical to the 1.27 cited in the fifth edition of the European Code Against Cancer [63, 70]. The authors point out that exposure was self-reported and that only women aged 25 or older took part [63].

Other features of the pooled data limit what they can say about sunbed use under today's European rules. Reichrath et al. note that many studies included people of skin type I, who are advised against sunbed use in Europe, and devices that are no longer permitted there [51]. Colantonio et al. and Burgard et al. both found no statistically significant association in the European studies (odds ratios of 1.10; 95% CI 0.98–1.24 and 0.95–1.27) [72, 52]. Colantonio et al. suggest that the European intensity limit (UV index 12, equivalent to 0.3 W/m²) may explain why the European studies show no significant association [72]. Burgard et al. also found no significant association in studies with a low risk of bias (1.15; 0.94–1.41) [52]. According to Reichrath et al., both the SCHEER and the WHO reports ignore the meta-analyses that show no association between sunbed use and melanoma in Europe [51].

SCHEER (2016) and the WHO (2017) concluded that sunbed use accounts for a noticeable share of melanoma and other skin cancers and that there is no safe limit of irradiance [74, 75]. SCHEER described the evidence for an increased melanoma risk with ever-use as strong. Graded by the criteria of the Oxford Centre for Evidence-Based Medicine, Reichrath et al. rate it as very weak (level 3a−, grade of recommendation D) and call SCHEER's own classification of evidence levels highly questionable [51]. They also fault both documents for ignoring the beneficial health effects of UV radiation and the consequences of vitamin D deficiency, which this White Book covers elsewhere [51]. The WHO report states that first use before age 35 raises the risk of melanoma by 59% [75]. That figure comes from the Boniol review, which first reported 87% and corrected it after publication [71]. The review also counted studies with age limits of 25 to 30 years as first use before 35 [51].

The IARC classification does not settle the question of risk. IARC placed UV-emitting tanning devices in Group 1, carcinogenic to humans, in 2009 [76]. Its categories "refer only to the strength of the evidence that an exposure is carcinogenic and not to the extent of its carcinogenic activity (potency)" [78]. A Group 1 listing therefore says nothing about the risk that comes with a particular pattern of use. The 75% figure that IARC quoted in 2009 for first use before age 30 came from a 2007 review by an IARC working group [76, 77]. Colantonio et al. found apparent typographical errors in that review, in the number of controls reported for two studies. They also observed that data extracted for one review tended to be copied by later reviewers without checking the original articles. The errors reappeared in two later reviews, one of them the Boniol review [72].

ESA maintains its assessment that the SCHEER opinion and the WHO report do not rest on sufficient evidence and are not a sound basis for policy on sunbeds. The evidence they rely on is observational, of poor to mediocre quality and confounded by sun exposure. It does not show that moderate sunbed use under today's European rules causes melanoma. That question needs studies that record sunbed use and solar overexposure separately, as the MISS cohort did, and that capture device type, irradiance, frequency of use and place of use. Colantonio et al. also call for large prospective cohorts [72]. Place of use shows how much pooled data can hide. The Boniol review

did not separate commercial from home use. In the three North American studies that did, commercial salons were not significantly associated with melanoma (odds ratio 1.05; 95% CI 0.83–1.32), whereas home tanning was (1.53; 1.23–1.89) [79]. ESA also disputes the IARC classification of tanning devices, because the 2007 review it draws on pooled studies that mostly did not separate sunbed use from sun exposure and contained data errors [72, 77].

Age Restrictions and Risk Group Warnings

Regulation of sunbed use varies across Europe. The clearest rule is a legal ban on use by minors: most, though not all, European countries prohibit sunbed use by anyone under 18. The EU's harmonized safety standard, EN 60335-2-27 [16], additionally requires operators to warn against use by several higher-risk groups, a separate obligation from verifying age:

- People with sensitive skin that burns easily and rarely tans (skin type I)
- People with a tendency to freckle, or with natural red hair
- People with many, large, or irregularly shaped moles
- People with a history of frequent or severe sunburn, particularly in childhood
- People with a personal or family history of skin cancer
- People taking photosensitizing medication, or under medical care for a photosensitivity condition

Operator training and consistent enforcement across member states support the uniform application of these requirements.

Vitamin D Deficiency Is a Global Public Health Issue

Over one billion people worldwide are either vitamin D deficient or insufficient, a scale that amounts to a global epidemic. Current research shows that low vitamin D levels are associated with as many as seventeen varieties of cancer, cardiovascular disease, and bone health outcomes.

Sunlight is the body's natural route to vitamin D synthesis, and the body limits its own production so sun exposure cannot cause vitamin D toxicity. Vitamin D deficiency is, in an important sense, a sunlight deficiency, given how much time modern populations spend indoors. Sunbeds were originally developed to support vitamin D production in light-deprived Northern European populations, and continue to offer that benefit.

How Much Sun Exposure Is Needed to Synthesize Sufficient Vitamin D?

Webb et al. (2011) combined controlled experiments using simulated solar UV radiation with modelling of the sunlight reaching the skin. They estimated that around 30 minutes of midday summer sun, three times a week, in summer clothing, would be enough for 90% of Caucasians to reach vitamin D levels above 20 ng/ml at latitudes of 30–55 degrees [53].

Longer exposure is needed to reach the more desirable 30–60 ng/ml range, in other seasons, at other times of day, at higher latitudes, or for people with darker skin. The essential caveat throughout: never burn: at the first sign of burning, further exposure should be avoided.

Sunscreen Use Protects Against Skin Cancer

Sunscreen is a key component of public health campaigns for skin cancer prevention, but epidemiological studies have raised doubts about its effectiveness at a population level: one meta-analysis found no significant association between sunscreen use and skin cancer risk, meaning the results do not confirm sunscreen's expected protective benefit in the general population. [54] A separate study found that plasma concentrations of four common chemical sunscreen ingredients (avobenzone, oxybenzone, octocrylene, and ecamsule) exceeded the FDA's safety threshold. [55] Among 500 couples trying to conceive, one 2014 study found that male partners with higher concentrations of benzophenone-type UV filters had a 30% lower chance of conception each cycle. [56]

Beyond these questions of efficacy and chemical safety, sunscreen carries a now more clearly demonstrated physiological cost: it lowers vitamin D synthesis in the skin. A 2025 randomized controlled trial (Tran et al., British Journal of Dermatology) tested this directly, the highest quality of evidence available [67]. Over one year, Australian adults randomized to routine daily application of SPF 50+ sunscreen had significantly lower serum 25-hydroxyvitamin D than controls (adjusted between-group difference −5.2 nmol/L, 95% CI −7.2 to −3.2), and were more likely to become vitamin D deficient by the end of the trial (46% vs 37%, prevalence ratio 1.33). A 2025 systematic review and meta-analysis (Gatta & Cappelli, Endocrine Practice), pooling both observational and interventional studies, reached the same conclusion: sunscreen users have significantly lower 25(OH)D levels than non-users [68]. Public health advice that recommends both sun avoidance and consistent sunscreen use should account for this trade-off rather than treating sunscreen as a cost-free intervention.

Conclusion

Natural sunlight affects several aspects of human health and well-being.

Sunburn is the risk to avoid. Sunburn and repeated overexposure are established risk factors for skin cancer, and avoiding them is common ground between public health authorities and the sunbed industry. Sunburn is the clearest and most consistent risk factor for melanoma across the literature and needs to be avoided at all costs. Protective measures such as clothing should be combined with shade when the intensity gets too high, especially on holidays in tropical climates, to which our skin is not adapted. Protection should come alongside sensible sun exposure at safer times and intensities, not instead of it.

Sun avoidance has costs of its own. Cohort evidence, most recently the 2024 UK Biobank analysis by Stevenson and colleagues alongside earlier Swedish and UK cohort studies, associates higher UV exposure with lower all-cause, cardiovascular and cancer mortality. This evidence is observational and consistent across several large cohorts, but it cannot establish causation on its own. Randomized trials of vitamin D supplements have not consistently replicated these associations, so part of the benefit of UV exposure may run through pathways other than vitamin D. Public health messages that focus on skin cancer risk alone leave out these documented costs.

The sunbed studies do not show that moderate use causes melanoma. This holds for non-burning use under today's European rules. The SCHEER opinion and the WHO report rely on the same studies and do not change that (see "Sunbed Use and Melanoma Risk").

Exposure advice cannot be the same for everyone. Depending on personal skin type, age, latitude, time of year and time of day, there is no "one size fits all" recommendation for how much time a person can spend outside before burning. The sun exposure needed for a sufficient vitamin D level varies from person to person for the same reasons.

To keep the message simple and honest, authorities should recognize that the evidence reviewed in this White Book supports a more balanced message than blanket sun avoidance. The evidence ranges from large UK and Swedish cohort studies to the 2024–2026 UK Biobank analyses and a growing understanding of vitamin-D-independent mechanisms. In practice, the balanced message is:

Sunburn, and intermittent overexposure specifically, is the risk factor that matters most, not moderate exposure.

Sensible, non-burning sun exposure is a health opportunity worth recommending, not only a hazard to be managed.

The nuance public health guidance already applies to diet, alcohol and physical activity should be extended to sunlight.

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- World Cancer Research Fund
- American Cancer Society
- GrassrootsHealth

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