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Influence of solar radiation on skin

Solar radiation has been a fundamental component of the earth's environment for more than 4.5 billion years. It is a type of electromagnetic radiation which consists of visible light as well as ultraviolet and infrared radiation.

Infrared radiation (IR) penetrates the deepest skin layers. It influences blood vessel dilatation and consequently causes erythema and a burning sensation during exposure to sunlight. The effect of prolonged influence of infrared radiation on skin is known as *eryhema ab igne* (2, 4).

In view of different wave lenghts, one can distinguish the following types of ultraviolet radiation:

- UVA: 320–400 nm – constitutes 95% of ultraviolet radiation which reaches the Earth. It penetrates through clothes and windows; its intensity does not depend on the time of the day or on the season. Therefore, we are constantly exposed to its activity. In view of the wave length, it penetrates deeply into the skin. The effects of its activity are visible even after years. We distinguish two types of UVA radiation: UVA 1 with wave length of 340–400 nm and UVA 2 – 320–340 nm
- UVB: 280–320 nm – constitutes only 5% of UV radiation on the Earth's surface. As opposed to UVA, UVB does not penetrate through clouds or windows. The highest intensity of UVB is seen on sunny days between 10 a.m. and 3 p.m. We gain on average 70% of the yearly dose of UVB during the summer time. This type of rays has very strong erythematogenic features
- UVC: 100–280 nm – does not reach the surface of the Earth since it is absorbed by the ozone layer. Therefore, it has no significant influence on our skin (1, 9).

The influence of solar radiation on the skin depends on a number of factors: wave length, radiation intensity, exposure time, exposure frequency, exposure conditions, eg. angle of incidence for rays or skin phototype. The longer is the time of exposure, the higher is the intensity of radiation and the paler is the complexion, the more intensified is the erythematous reaction. The parts of the body which are most sensitive to solar radiation include: head, cleavage, palms and internal surfaces of thighs (9).

Sunlight is a source of light and warmth, both of which are essential for the existence of life on the Earth. The proven and favourable effect of UVB radiation on the skin is to enable the synthesis of vitamine D3, which is essential for proper calcium and phosphorus assimilation, and thus it prevents rickets in children and osteomalacia in adults. Ultraviolet radiation, thanks to increased serotonine synthesis, has an influence on the general well-being and prevents depression. It is therapeutically effective in many dermatological diseases.

Negative effects of UV radiation include: photo-ageing, impairment of the immune system, phototoxic and photoallergic reactions, and stimulation of pigmentary changes. The skin has a few

mechanisms which protect it against harmful effects of UV radiation. The corneous layer of the epidermis has the ability to reflect some of the rays which fall on the skin. The main defence line against solar radiation is melanin in the epidermis which absorbs and disperses UV radiation and stabilizes free radicals.

The most common reaction to solar radiation is an acute erythematous reaction caused mainly by UVB radiation. High energy of radiation reaches only epidermis and is responsible for immediate erythematous reactions and sun burns. Erythema appears within about 2–3 hours after exposure to sunlight and gets more intensive gradually until it reaches its culmination after 6–9 hours. It disappears after 24–48 hours.

The natural mechanism which protects us from excessive erythematous reactions is the production of melanin. The synthesis of melanin is a dual phenomenon. The first phase takes place during the exposure to ultraviolet radiation. It consists in a photo-oxidation reaction which leads to immediate release of the pigment. In the other phase (after 72 hours) of melanogenesis, an increase in the synthesis of melanin in the epidermis takes place as well as an increase in the number of melanosomes. Repeated exposures to sunlight may cause an increase in the number of melanocytes in the epidermis.

Table 1. Fitzpatrick skin phototype

Skin type	Tan	Burns
I	-	+++
II	+	++
III	+	+
IV	++	+
V	++	+/-
VI	+++	-

Frequent, long-term exposures to solar radiation makes the skin dry, scaling, yellow in colour and thickened. Moreover, pigmentary changes and blood vessel dilatations (telangiectasia) are observed. In the process of skin ageing due to sun exposure, the phenomenon of elastosis is observed. It consists in the degeneration of elastic fibres and their compaction into a shapeless mass. The number of collagen fibres decreases whereas the number of fibroblasts increases. The amount of mucopolysaccharides also decreases. The dermoepidermal junction gets flattened. In the process of photo-ageing, the epidermis gets thickened as opposed to the skin aging, which refers to the natural process of chronologic aging. In the epidermis, changes in sizes and shapes of keratinocytes and melanocytes take place and the number of Langerhans' cells decreases. As a result of UV radiation, blood vessel obliteration also takes place (8, 11, 12).

Fine wrinkles are an early sign of skin ageing due to sunlight and the wrinkles transform into creases with the passage of time. The wrinkles appear due to the loss of support fibres.

Under the influence of UV radiation many pigmentary changes appear, i.e. freckles, lentigos, melanocytic naevi and even malignant melanoma. Freckles are small (1–2 mm in diameter) benign pigmentary patches, localized in the places exposed to solar radiation. Long-term exposure to sunlight makes them darker and more numerous. As opposed to lentigos, the concentration of melanocytes in freckles is lower than in the surrounding skin. These cells are however bigger, more active and develop more dendritic processes. Lentigos are acquired brown patches, frequently localized on the face, arms and forearms. This benign form of melonocyte proliferation is seen in 90% of white elderly people. Lentigo maligna, also known as Hutchinson's freckle, is big, brown and usually

localized on the cheek. When histological structure is taken into account, lentigo maligna is the same as melanoma *in situ*, but this change is characterized by a low malignancy potential and may remain unchanged for a long period of time. When it acquires invasive features, it becomes as malignant as any melanoma.

Table 2. Drugs causing photosensitivity

Used generally	Used locally
Antibiotics: sulphonamides, tetracyclines, fluorochinolons	Tar preparations
NSAID: ibuprofen, ketoprofen, naproxen, piroxicam	Psoralens
Diuretics: thiazides, furosemide	Antiseptics: chlorhexidine
Phenotiazine derivatives: chlorpromazine, promethazine, thioridazine	Sulphadiazine
Niphedipine, verapamil, dilitazem	
Beta-blockers: atenolol, bisoprolol, propranolol	
Fibrates and statins: clofibrate, phenofibrate, benzafibrate, atorvastatine, simvastatine	
Psoralens	
Sulphylurea derivatives: glibenclamide, glipizide, tolbutamide	
Isoniazide	
Antiarrhythmic drugs: amiodarone, quinidine	
Antifungal drugs: gryzeofulvine, ketoconazole	
Chlorodiazepoxide	
Tricyclic antidepressive drugs	
Cytostatics: busulfan, 5-fluoruracil	
Methotrexate	
Phenytoin	
Zidovudine	
Antimalarial drugs: chloroquine, hydroxychloroquine	
Hormones: estrogens, progesterone	

In women taking hormonal drugs, poikiloderma of Civatte (mottled heper-/depigmentation and superficial atrophy in a reticular pattern) may occur on the skin of the face and neck. In periods of disturbed hormonal balance (pregnancy, menopause) and in people using photosensitizing drugs, characteristic pigmentary changes called chloasma (melasma) develop.

In elderly people exposed to long-term solar radiation, walls of capillary vessels become fragile. It is a symptom of solar purpura (Bateman's disease).

It seems that both long-term exposure and sun burns, especially in children younger than 12 years old, predispose to the development of malignant melanoma. It is one of the most malignant skin neoplasms which develops in the unchanged skin or within pigmentary patches. Melanoma malignum is characterized by: uneven colour of the pigmentary naevus or patch with its simultaneous growth, asymmetrical shape and irregular borders, slight inflitration at the base, inflammatory edge. The risk of melanoma occurrence increases in people who have had melanoma diagnosed previously or who give family history of the disease. Factors predisposing towards the development of melanoma include: atypical naevi, congenital and numerous pigmented naevi (more than 50) and light complexion.

UV radiation modifies the local and systemic immune response. Even small doses of UVB decrease the immune barrier of the skin, and thus diminish its immunity to infections. Higher doses of UVB disable the systemic immunity. Langerhans' cells (LCs) are especially sensitive to UVB

radiation (280–320 nm). As the result of skin exposure to UVB, the number of those cells decreases and their functions get disturbed. Researches have proved the presence of antigen-specific Ts lymphocytes which appear after the application of the antigen onto the skin previously exposed to UVB. Urocanic acid (UCA), which has two forms *trans* and *cis*, may play an important role in the production of specific Ts lymphocytes after the exposure of skin to UVB. *Trans*-UCA constitutes the main compound which absorbs UV radiation in the corneous layer of the skin. After exposure to sunlight, the *cis*-UCA form with its immunosupressive features is created. It has been proved that *cis*-UCA given locally onto the unexposed skin inhibits reactions of late hypersensitivity against various antigens (i.e. herpes simplex type I, contact allergens), which depends on the production of specific Ts lymphocytes. The mechanism of action of UCA is probably connected with the indirect disturbance of the functioning of LCs presenting antigen (i.e. through TNF induction) (13).

The phenomenon which leads to antigen-specific immunosupression/ immunotolerance as a response to UVB may be the induction of MHC class II antigens and ICAM-1 on keratinocytes which in normal conditions show no expression of those cells. It has been proved that keratinocytes HLA-DR+ present antigens, thus causing immunotolerance. This phenomenon is due to the process of creating lymphocyte clones which induce Ts cells (T suppressor-inducer, CD4+, CD45RA+). Additional non-specific immunosupressive factors produced as a result of exposure to UV include: IL-10, antagonist of IL-1 receptor and inhibitor of contact hypersensitivity (11, 13).

UVB radiation leads to the release of free radicals and the production of nitric oxide (NO), which may induce both physiological processes (among others: activation of guanyl cyclase, dilatation of blood vessels, processes of platelet antiaggregation, inhibition of smooth muscle proliferation, defence mechanisms of the body) and pathophysiological ones (hypotension, septic shock, contraction of blood vessels). It has been proved that NO counteracts postradiation keratinocyte damage by protecting the keratinocytes against apoptosis (10).

Even small doses of UVB decrease the immunity of the human body. The probability of viral or bacterial infections and skin neoplasms increases. It is claimed that mutagenic activity of UV radiation on keratinocytes is connected with the direct influence on DNA or antioncogens type P53 and also with the induction of immunosuppression (13).

UV radiation influences covalent bonds of adjoining pyrimidines. These ractions lead to the creation of pyrimidine dimers which disable the corrective mechanism of DNA and unable the normal transcription and replication (7).

The impairment of the immune system, mutagenic influence of UVA and UVB radiations and long-term exposure to solar radiation are responsible for neoplasmatic diseases (5). Basal cell carcinoma (BCC) is the most common. BCC foci are most often localized on the face. Squamous cell carcinoma (SCC) develops on the border of mucose membranes and skin (lower lip, area of orbital cavities, nose, and genital organs) (3).

Solar radiation may provoke various dermatological diseases. The disorders which are characterized by hypersensitivity to ultraviolet radiation are called photodermatoses. They include: idiopathic photodermatoses (summer prurigo, hydroa vacciniforme, solar cheilitis, chronic actinic dermatitis, solar urticaria, xeroderma pigmentosum), exogenous ones (phototoxic and photoallergic reactions), endogenous and congenital ones.

Summer prurigo appears in young women a few hours after the exposure to sunlight during spring time. In uncovered areas, polymorphous eruptions are observed: erythematous lesions, papulas, and even blisters. The intensity of the disease decreases with the development of the natural protection, but it may recur in the next season. Hydroa vacciniforme is frequent in children. It has the form of blisters and bullas which are localized in the uncovered areas. Secondary infections are

common. Ophthalmic reactions may coexist (conjunctivitis and keratitis) as well as high temperature. Exposure to sun radiation may lead to an inflammatory process of the lips known as solar cheilitis. The symptoms include scaling of the lips and development of erosions covered with crusts. Chronic actinic dermatitis (ChAD) is another disease due to hypersensitivity to sun rays and there are three types of the disease. The changes are localized in the uncovered areas. They have the form of acute or subacute eczema. Symptoms of another type of the disease include persisting solar reactions. Rarely does the disease involve covered areas and it may involve almost the whole skin (erythrodermia). The third form of ChAD is actinic reticuloid. It has a very severe course. It occurs in more than 90% of males older than 60 years old. Within the involved areas – with the passage of the disease – intense skin lichenification develops, and sometimes infiltrative changes similar to lymphomas are seen. In some people the changes may be preceded by photoallergic symptoms. Sometimes contact allergy to chromium, cobalt, nickel, p-phenylenodiamine, Peruvian balm, as well as to natural and artificial fragrance substances coexist.

Very rarely do we observe solar urticaria. Skin changes appear very fast (a few minutes) after the exposure to sunlight and they disappear after about 1 h. The changes are in the form of urticas. No systemic symptoms are observed.

In genetically predisposed people, UVA radiation, and to a lesser extent UVB radiation, or UVA and UVB, stress and infection may induce autoimmunological processes. It leads to the development of such diseases as: discoid lupus erythematosus (DLE), subcutaneous lupus erythematosus (SCLE), and systemic lupus erythematosus (SLE). UVA radiation during the course of SLE is not only a trigger, but also the cause of exacerbations of systemic symptoms (kidney diseases, changes in the CNS) (4).

Xeroderma pigmentosum is the disorder which is characterized by remarkable hypersensitivity to sunlight and which is connected with a defect of DNA endonuclease. There are skin lesions in the uncovered areas such as lentigos, freckles, discolourations, pigmentary atrophies and telangiectasias. They appear in the early childhood after the first exposure to sunlight. The development of cancerous changes (papillomas, spinocellular carcinomas, sarcomas, melanomas) is early . Ophthalmic and neurologic symptoms coexist.

Phototoxic and photoallergic reactions take place under the influence of ultraviolet radiation and external photosensitizing substances included in cosmetics, drugs and some plants.

Phototoxic skin inflammation is the result of damage to some cellular structures by chemical substances which become active under the influence of UVA and UVB. The clinical picture includes: erythema, oedema and blisters. The changes appear in the places of chemical contact and exposure to sunlight. Photoallergic reactions are caused by UVA radiation, but contrary to phototoxic reactions, they do not depend on the dose of the drug or on the intensity of radiation. The immune system plays an important role. The effects are postponed and may appear only after the next exposure to sunlight. The clinical symptoms include changes of eczematous type.

UV radiation induces genetically determined disturbances in the metabolism of porphyrins. *Porphyria cutanea tarda* belongs to the previously mentioned dieases. The aethilogical factors of the disease include: deficiency of uroporphynogen decarboxylase (congenital form) or hepatotoxic drugs and alcohol (acquired form). It appears in family members, especially in grown-up men. The changes are most often localized on the back of the hand and on the face. Excessive skin sensitivity is observed. Subepidermal blisters, partially haemorrhagic, appear after injuries, and they leave scars after the healing process. When the changes accumulate, one can observe scars, hyperpigmentation, excessive hair growth and symptoms of chronic solar damage, i.e. solar elastosis, cysts and comedos. Local changes coexist with systemic symptoms, i.e. reddish brown colour of the urine, increased

level of iron in the blood serum, diabetes mellitus. Other disorders which belong to this groups include: erythropoietic porphyria (Gunther's disease) and also erythropoietic protoporphyria.

Congenital diseases with hypersensitivity to sunlight include: Bloom's syndrome, Rothmund-Thompson's syndrome, Cockayne's syndrome, Hartnup's syndrome.

The human body is still exposed to omnipresent UV radiation (UVA and UVB). One should bear in mind that apart from a positive influence on the human body (synthesis of vitamine D3, antidepressive function and treatment in some dermatological diseases), it has many negative effects (solar burns, photo-ageing, skin neoplasms). There are many disorders which show hypersensitivity to UV radiation (endo- and exogenous photodermatoses, SLE, SCLE, DLE, xeroderma pigmentosum, porphyrias). In order to protect our skin against acute and chronic reactions to UV radiation, we should sunbathe cautiously and protect the skin with appropriate clothing and with cosmetics containing sunblocks. It is recommended to avoid sun exposure during the therapy with photosensitizing and phototoxic drugs.

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SUMMARY

Solar radiation has been a fundamental component of earth's environment. The infrared and ultraviolet (UVA, UVB) radiation reach the surface of the globe. Thus they have a significant influence on human being. Defence mechanisms of the body which are being triggered by ultraviolet radiation and influence of sunlight on cells and DNA have been described. The purpose of this report is to review both positive (synthesis of vitamine D3, prevention of depression) and negative (photoaging, impairment of immune system, photodermatoses, skin cancer) effects of solar radiation based on published studies. The paper presents clinical symptoms of diseases proceeding with hypersensitiveness to sunlight.

Wpływ promieniowania słonecznego na skórę

Promieniowanie słoneczne jest nieodłącznym składnikiem środowiska ziemskiego i dlatego ma istotny wpływ na organizm ludzki. W artykule omówiono mechanizmy obronne organizmu wyzwalane w odpowiedzi na nadmierną ilość promieniowania słonecznego, a także opisano mechanizmy oddziaływania promieni słonecznych na komórki oraz na DNA. Na podstawie wybranego piśmiennictwa dokonano przeglądu zarówno pozytywnych, jak i negatywnych skutków promieniowania UV na skórę. W pracy przedstawiono obraz kliniczny chorób związanych z nadwrażliwością na promieniowanie słoneczne.