Ultraviolet radiation and melanogenesis

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ABSTRACT

Light radiation is a part of the electromagnetic (EM) radiation, and it consists of the ultraviolet (UV) radiation, visible light, and infrared radiation. UV radiation energy is absorbed in the form of photons in biomolecules (chromophores) and induces various cellular reactions, out of which photochemical and photosensitizing are the most significant. In contact with the skin UV radiation incites protection mechanisms: the most important are stratum corneum thickening and melanin synthesis (melanogenesis). Basic role of melanin is absorption and scattering of UV rays and neutralization of free radicals. In this review physical characteristics of UV radiation, its biological effects, and relation to melanogenesis and carcinogenesis are discussed.

KEY WORDS: Ultraviolet Rays; Melanins; Melanocytes; Skin Pigmentation

PHYSICAL CHARACTERISTICS OF ULTRAVIOLET RADIATION

Light is a form of radiant energy omnipresent in the environment and the essential pre-condition of the survival of man and all the life on the Earth. Light (optical) radiation is a part of the electromagnetic (EM) radiation, situated according to its wavelength between soft x-radiation and radiofrequency radiation. It consists of the ultraviolet (UV) radiation (20-60 w/m2 out of the total 550 w/m2 at the outer side of the Earth’s atmosphere (1,2)). UV radiation is a part of the electromagnetic (EM) radiation, and it consists of the ultraviolet (UV) radiation, visible light, and infrared radiation. UV radiation energy is absorbed in the form of photons in biomolecules (chromophores) and induces various cellular reactions, out of which photochemical and photosensitizing are the most significant. In contact with the skin UV radiation incites protection mechanisms: the most important are stratum corneum thickening and melanin synthesis (melanogenesis). Basic role of melanin is absorption and scattering of UV rays and neutralization of free radicals. In this review physical characteristics of UV radiation, its biological effects, and relation to melanogenesis and carcinogenesis are discussed.

BIOLOGICAL EFFECTS OF UV RADIATION

UV radiation energy is absorbed in the form of photons in biomolecules (chromophores); these molecules are then brought to an excited state, usually higher than the previous. During the process, the energy of the excited molecule can be transformed into chemical energy, with accompanying photochemical or photosensitizing reactions. The energy may also be transferred to some other molecule (the acceptor), which similarly undergoes certain changes (2).

UV radiation-induced cellular changes can be beneficial or harmful, associated with free radical release. The form of the photochemical cellular reaction depends on the UV radiation wavelength, molecular structure of the chromophore and specific conditions in the environment where the reaction takes place (5). Absorption spectrum of the cellular molecules is very diverse. Melanin has a broad absorption spectrum, ranging through the whole UV spectrum, through the whole visual part of the light spectrum, and through the part of the infrared spectrum (2,3).

Basic chromophores in the skin cells are DNA, proteins, urocanic acid and melanin, chlorones, flavins, steroids, and porphyrins (5,7). Nucleic acids are 220 times more sensitive...
MELANOCYTE BIOLOGY AND MELANIN SYNTHESIS

Melanocytes are dendritic cells situated among the keratinocytes of the basal epidermal layer and among the hair matrix cells. They are attached to the basal membrane by plates similar to hemidesmosomes. One melanocyte provides melanin for 36 surrounding keratinocytes (melanin unit). Per 1 mm² of skin there are 1100-1500 melanocytes and that number is almost the same regardless of the skin type (5,13,14). Melanocytes synthesize melanin out of tyrosine (amino-acid), with the coaction of melanocytic tyrosinase enzymes TRP-1 and TRP-2 (tyrosinase related protein), membrane lysosomal proteins LAMP (lysosome associated membrane protein), and other enzymes.

There are three types of melanin:
- Eumelanin, black-brownish colored
- Pheomelanin, yellow-reddish colored
- Neuromelanin, black colored and present in nerve cells

UV radiation exposure is the stimulant for melanin synthesis, or the synthesis is hormonal-stimulated in certain endocrine diseases (5,15,16). UV radiation exposure is the stimulant for eumelanin synthesis. Melanin synthesis commences with the entry of tyrosine into the melanocyte through a specialized transport channel (P-locus). Enzyme tyrosinase catalyzes tyrosine all the way to dihydroxyphenylalanine (DOPA) and DOPA choline. DOPA-to-DOPA choline oxidation co-factor is TRP1. Further cyclisation produces cyclo-DOPA and DOPA-chrom. With TRP coaction dihydroxyindol-2-carboxylic acid (DHICA) is produced, which with further oxidation produces eumelanin pigment (17-20).

Pheomelanin is synthesized in smaller amounts compared to eumelanin. It is the product of nucleophilic oxidation of L-cystein in the presence of DOPA choline. L-cystein is an important regulator of melanogenesis. If its level is decreased in the cells, the activity of tyrosinase and eumelanogenesis are inhibited (21,22). Chemically, melanin is a biopolymer composed of red melanin (which contains sulphur and is soluble at pH 7.2) and black melanin (water-insoluble).

UV radiation and skin tanning

Immediate skin tanning occurs during the UV radiation and it is the consequence of the pigment already present in melanophages. It occurs only in individuals that constitutionally have at least medium dark complexion. It can be stimulated by UVA and UVB radiation and also by visible light. It is the result of oxidation and redistribution of the existent melanosomes towards peripheral melanocyte dendrites.

UV radiation and carcinogenesis

In addition to its stimulatory effects on melanogenesis, UV radiation, especially UVB rays, damage melanocytes prolonging the G1 phase of their cellular cycle. Thus the time required for damaged DNA repair is prolonged and carcinogenic effect of UV radiation is reduced (27,28). Melanocytes at their disposal have also other numerous mechanisms (such as LAMP1) to neutralize free radicals and other harmful compounds produced in response to UV radiation. Protective effects on irradiated melanocytes also have keratinocytes and fibroblasts (29). The key effect in UV damage protection is the blockade of the cellular cycle while DNA damage is being repaired (30). However, aMSH, in addition to its synergistic effect on melanogenesis, stimulates proliferation of irradiated melanocytes, reducing thus the time required for damaged DNA repair (30,31). UVB rays stimulate the release of aMSH and ACTH from keratinocytes and MC1 receptors for these hormones. These hormones have immunosuppressive action and it is possible that aMSH helps the accumulation of mutations in irradiated melanocytes and their further malignant transformation (32). The invasive behavior of melanocytes is partly the consequence of altered expression and affinity of integrin-receptors for extracellular matrix (32,33).

UV radiation and skin tanning

The immediate effect of skin exposure to UV radiation is the stimulation of melanogenesis, i.e. skin pigmentation (tanning). Skin pigmentation occurs in two phases (4,14):
1. Immediate tanning/pigmentation of the skin
2. Delayed tanning/pigmentation of the skin
The effects of this type of skin pigmentation disappear quickly after the UV exposure; if delayed melanogenesis does not take place the effect may disappear completely (1,13).

This type of pigmentation does not significantly contribute to the skin protection from erythema and sunburns. Delayed skin pigmentation is the consequence of increased activity of melanocytic tyrosinase, branching of the melanocyte dendritic portions, increase of the number and size of melanosomes and melanin granules in the cells and accelerate the transfer of melanin into keratinocytes. In case of uneven resorption, freckled tanning occurs (15,34).

This delayed pigmentation has less importance in skin protection from acute erythema because it occurs (4,10). Certain antioxidants antagonize skin pigmentation process - they disturb the course of melanogenesis and melanin production by alpha-chinone reduction. Moreover, they transform the black into light brown melanin. Similar effects are expressed by some substances such as magnesium-ascorbil-2-phosphate (VC-PGM). Due to these characteristics, these and similar substances are added to the preparations the purpose of which is to reduce pigmentation and remove sun freckles from the skin (35,36).

REFERENCES