Acute Skin Sun Damage in Children and Its Consequences in Adults

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ABSTRACT

Children spend more time outdoors than adults and there is compelling evidence that childhood is a particularly vulnerable time for the photocarcinogenic effects of the sun. The negative effects of solar radiation are accumulated during the entire lifetime; however 80% of total lifetime sun exposure is taking place before the age of 18 years. Child skin is more sensitive than adult skin because natural defense mechanisms are not fully developed. A short exposure to midday sun will result in sunburns. Epidemiologic studies show a higher incidence of malignant melanoma in persons with a history of sunburns during childhood and adolescence. Sun exposure among infants and pre-school children is largely dependent on the discretion of adult care providers. Sun protective habits of mothers may predict the level of sun exposure in children. It is very important to transfer the knowledge and positive habits of proper sun protection to children. The purpose of sun-safety behavior is not to avoid outdoor activities, but rather to protect the skin from detrimental sun effects. Proper sun protection of children includes protection from excessive sun exposure, sunburns and other forms of skin damage caused by sun, which may lead to the future development of skin cancers. This paper reviews acute skin reactivity to sun in childhood and adolescence that causes damage in skin structure and function and produces undesirable chronic changes in adults.

Key words: skin sun damage, sun protection, sun education

Introduction

Children need sun for growth and development. Yet, tender child skin is very sensitive to the negative effects of solar ultraviolet (UV) radiation, because of its natural defence mechanisms are not fully developed. The effect of UV rays upon skin has a cumulative character, meaning that it adds up over the lifetime. It has been shown that between 50% and 80% of the total lifetime accumulation takes place precisely in the first 18 years of life. Numerous studies have proven the association between excessive, uncontrolled skin sun exposure in childhood and skin damages in adulthood.

The behaviour of children is determined by the behaviour of their social environment: parents, carers, etc. Maternal sun habits are predictive of the level of the child sun exposure. The transfer of knowledge and positive sun habits to children is of high importance. The purpose of sun protection is not to avoid the outdoors activities, but to protect the skin from excessive, harmful sun effect. A correct sun protection means a protection of sunburns today but also a protection from other forms of skin damage, especially skin carcinoma, in the future.

Acute Skin Damage in Children

The most common acute skin damage under the influence of sun are sunburns, or solar dermatitis, unfortunately still a common occurrence in children, caused primarily by inadequate prevention. Solar dermatitis is acute primary dermatosis caused by UVB rays (wave length from 295 to 315 nm). It is characterized by intense redness that appears 4–6 hours following the sun exposure, and it is accompanied by the feeling of burning and, in severe cases, pain. Severe cases of solar dermatitis may lead to the formation of vesicles and blisters, along with the general symptoms of malaise, fever, headache, and, in the most severe cases, circulatory collapse. Symptoms usually lessen around 72 hours after the sun exposure and pigmentation develops. After several days,
sunburns are succeeded by the desquamation (peeling) of the skin\textsuperscript{10}. Histological signs, apparent 12–72 hours after UVB light effect, include sunburn cells in the Malpighi cellular layer, occasional vacuolization of basal cells, and, in severe cases, focal cellular necrosis. Blood vessels in the upper dermis are dilated\textsuperscript{11}. The development of solar dermatitis and its intensity depend on the skin type, the length of the sun exposure, season, altitude, reflection of sun rays from the surface of water, snow, sand and the like\textsuperscript{10,12}. In most cases, the history of outdoors activities, along with the clinical picture, suffices for the correct diagnosis. Occasionally it is necessary to exclude phototox and photoallergic reactions\textsuperscript{12}.

Phototoxic and photoallergic sun-sensitive reactions are less common in children than in adults. They arise from an abnormal response of the organism to ordinary UV sun exposure, and they are mediated by photosensitizing substances of either endogenous or exogenous origin\textsuperscript{13–15}. Thus, the emergence of these reactions necessitates a chemical substance in the skin that has either been ingested (medication) or applied to the skin (for instance, soap, a cream or a plant substance). Most photosensitising reactions are activated within the UVA range of the sun spectrum\textsuperscript{10}. Phototoxic reactions are more common and they are caused by direct interaction between the substance and light, followed by a skin reaction that resembles sunburns. Photoallergic reactions are extremely rare in children, and they require, in addition to the sunlight and photosensitizer, immunological mechanisms, or the late cellular T-cell mediated hypersensitivity type\textsuperscript{12}.

Here I shall not discuss primary idiopathic photosensitive reactions (polymorphous light eruption, chronic actinic dermatitis, actinic prurigo, hydroa vacciniforme and solar hives) and photosensitive reactions linked to genetic and metabolic disorders. Although exceptionally strong, these reactions are abnormal skin responses to a normal level of skin sun exposure; they are furthermore rare\textsuperscript{16}; and they cannot be prevented by the usual sun protection preventive measures.

**Consequences in Adulthood**

Chronic changes of skin caused by the sun become apparent in adulthood and involve the development of skin carcinoma and premature aging of the skin. It is well known that cumulative sun exposure over a long time period is the key etiological factor in the development of non-melanoma skin carcinomas, especially basal cell and spindle cell cancers\textsuperscript{17,18}. Furthermore, occasional intense sun exposure and sunburns, especially in childhood and youth, are the chief etiological factor in the development of malignant melanoma\textsuperscript{18,19}. The mechanisms of the carcinogenic effect of UV rays are multiple. The most oncogenic portion of the solar spectrum is the UVB rays portion. Namely, DNA absorbs UV rays of the wavelength between 245 to 290 nm\textsuperscript{20}. This is, for the most part, UVB radiation (280–300 nm), as UVC rays (200–280 nm) do not reach the earth surface. At the molecular level, the absorbed UV radiation causes damage to the cellular DNA and induces the formation (6–4) of photoproducts (between two pyrimidines) and cyclobutane dimers (between thymine and cytosine)\textsuperscript{20}. These damages are usually repaired by DNA repair mechanisms\textsuperscript{20}. A normal cell has the ability to repair and remove DNA damages\textsuperscript{19–23}. The key element of the repair processes is the tumour suppression gene p53, an anti-oncogene located on chromosome 17 p. Tumour suppressor p53 is activated under the influence of acute UV radiation and it stops the cell cycle in G1 phase, thus enabling the repair of damaged DNA prior to its replication in S1 phase of the cell cycle. While the cell cycle is on standby, cyclobutane dimers and 6–4 photoproducts are removed using a complex process of excision repair (nucleotide excision repair – NER)\textsuperscript{21,22}. Should the repair fail, the death or apoptosis of the affected cell will succeed. Apoptosis is regulated by tumour suppressor p53 as well, and this is why p53 is often called «the guardian of the genome». Mutations in p53 gene cause the loss of UV light-induced apoptosis in keratinocytes. Later exposure to UV light selectively promotes clonal expansion of cells carrying mutated p53 gene\textsuperscript{21,22}. The emergence of malignant tumours is the consequence of the cell’s inability to repair damaged DNA\textsuperscript{22,23}. Damage to the DNA is the first step in oncogenesis. If the damage is strong or repeated, the protective mechanisms of the cell are no longer able to repair all the damage and that leads to the emergence of carcinoma.

The role of UVA radiation in the process of carcinogenesis is not as well documented as the role of UVB radiation. UVA radiation causes damage indirectly, by forming reactive oxygen free radicals\textsuperscript{19,22,23}. Another important component of UV radiation in the process of carcinogenesis is the suppression of the immunological response, which indirectly promotes carcinogenesis\textsuperscript{19,22,23}.

The process of skin ageing under the influence of sun, popularly called photoaging, refers to the external or premature ageing of skin caused primarily by UV radiation. UVA rays penetrate deep layers of skin and damage the structure of collagen and elastic fibres\textsuperscript{24,25}. They induce the synthesis of several types of collagenase enzymes named matrix metalloproteinases, and these in turn reduce collagen synthesis and increases collagen breakdown\textsuperscript{24,25}. The histological characteristics of the skin aged under the influence of the sun include elastosis, the accumulation of amorphous elastotic material within the upper and middle dermis that may be stained with elastin dyes\textsuperscript{24,25}. The exact mechanism of the formation of elastotic material is unknown. It is assumed that UV radiation induces the synthesis of non-functional elastic fibres that form the amorphous mass typical of photo-damaged skin\textsuperscript{24,25}. Elastosis is not part of the physiological process of skin ageing. UV radiation promotes the formation of free oxygen radicals in the skin, and they damage nucleic acids, lipids and proteins, including collagen fibres. These processes then result in premature skin ageing\textsuperscript{24–26}. Early clinical signs of photoaging include variations in the skin pigmentation. A skin that
has aged through a normal chronological process is uniformly pigmented. UV radiation affects the activity and proliferation of melanocytes, and this process then results in dyspigmentation (hypo- and hyperpigmentation) of the skin\(^{27}\). Skin aged under the influence of the sun becomes dry, wrinkled and rough; it has patches of irregular hyperpigmentation (ephelides, lentigines, patchy hyperpigmentations) and hypopigmentation (e.g. hypomelanosis guttata). The tonus and the elasticity of the skin diminish, the fragility of the blood vessels increases so telangiectasias, cherry angiomas and bruises (senile purpura) appear\(^{24,28}\). The process of sun-induced skin ageing first becomes evident as early as at the age of 25 years. Photoageing begins the earliest and has the strongest impact in skin type I and II persons, and it is comparatively less pronounced in darkly pigmented persons, for instance in Africans. The difference between the physiological and the premature skin ageing is best observed when comparing the skin of the face, neck and back of the hand (so called photo-exposed skin) with, for instance, the skin of the armpit or another spot that is usually protected from the impact of the sun.

**Discussion and Conclusion**

Numerous studies have shown that the excessive sun exposure in the first 10 to 20 years of life increases the risk of skin carcinomas\(^{4,5,29}\). Children spend much more time outside than the adults\(^{30,31}\), and the childhood is an especially vulnerable time of life with respect to the photocarcinogenic sun activity\(^{32}\). An average child receives a yearly dose of UVB radiation three times larger than an adult\(^{32}\). As childhood sunburns are the chief etiological factor in the development of malignant melanoma\(^{24-6}\), protective measures should be introduced from the youngest age. Sun protective measures are simple and should become part of everyday life from the first days of life. The use of sun-protection products is not recommended in infants under the age of 6 months, because their physiologic systems for metabolism and excretion of absorbed agents may not be fully developed\(^{31}\). Infants of this age group should be kept away from the direct sun exposure, and the chief protective measures should be clothing and staying in shade\(^{32}\). All others should avoid staying outdoors at the time of the day when the UV rays’ concentration reaches its peak; wear protective clothing and UVA and UVB protected sunglasses; and use sunscreen products that incorporate protection against both UVA and UVB spectrums. These protective measures may successfully prevent irreparable skin damages in adulthood\(^{31}\). The awareness of the harmful effect of the sun and the correct protection from the early age may prevent 4 out of 5 cases of skin carcinoma. According to the literature, only the regular use of sunscreen products with the protection of factor 15 during the first 18 years of life results in a 78% reduction of the incidence of non-melanoma skin tumours\(^{34,35}\).

The sun exposure of preschool and early school age children depends on the behaviour of their social envi-
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To the elderly. The message was unambiguous and clear: correct sun protection prevents skin carcinoma47. Education should start in the earliest days of life, as early as delivery suites48. The mother should receive the first information on the sun protection in the form of written educational material, together with other instructions, for instance on the care for healthy infant skin and nutrition. At kindergarten age, children should be acquainted with the good and the bad sides of the sun through play and games, and they should learn about proper sun protection, seeking shade, wearing protective clothes, hat and sunglasses, and applying sunscreen products49. The prevention of acute and chronic damage in uncontrolled exposure, and correct sun protection44. Health personnel-paediatricians, general practitioners, dermatologists, nurses and pharmacists-must be the source of information on correct sun protection41. Along with their usual parental advisory activities, paediatricians should include advice on the sun protection and the prevention of irreplaceable damage in later age50,51. Teaching children to protect themselves from the sun, and so prevent sun damage, means adopting healthy habits and attitudes and, in relation to that, understanding the association between behaviour and health.
AKUTNA OŠTEĆENJA KOŽE POD UTJECAJEM SUNCA U DJECE I POSLJEDICE ZA ODRASLU DOB

SAŽETAK

Djece provode puno više vremena na otvorenom nego odrasli, a dokazano je da su djetinjstvo i adolescencija posebno osjetljivo razdoblje u fotokarcinogenom djelovanju sunca. Negativni učinci sunčevog zračenja akumuliraju se tijekom života, a i do 80% ukupne životne akumulacije događa se upravo u djetinjstvu i adolescenciji. Nježna dječja koža osjetljivija je od kože odraslih jer prirodni mehanizmi obrane nisu u potpunosti razvijeni. Dovoljno je kratkotrajno izlaganje podnevnom suncu da se razviju sunčane opekline. Epidemiološki podaci pokazuju veću učestalost malignog melanoma u osoba koje su u djetinjstvu i mladišti pretpjevali sunčane opekline. Ponašanje djece određeno je ponašanjem njihove okoline, roditelja, odgajatelja itd. Majčine navike o zaštitit od sunca mogu predvidjeti stupanj izlaganja suncu u djece. Vrlo je važno znanja i pozitivne navike o pravilnoj zaštiti od sunca prenijeti djeci. Svrha zaštite od sunca nije izbjegavanje aktivnosti na otvorenom, nego zaštitit kože od prekomjernog štetnog djelovanja sunca. Pravilna zaštita od sunca znači obranu od sunčevog zračenja, sunčanih opekлина danas te drugih oblika oštećenja kože, osobito kožnih karcinoma, u budućnosti. U ovom radu su prikazana akutna oštećenja kože pod utjecajem UV zračenja tipična za dječju i adolescentnu dob, a dovode do oštećenja u strukturi i funkciji kože, što za posljedicu ima nastanak kroničnih oštećenja kože u odrasloj dobi.