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THE EFFECTS OF ULTRAVIOLET RADIATION ON OCCUPATIONALLY EXPOSED WORKERS

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This review includes an introduction on the nature of UV light and on the effects of ultraviolet radiation (UVR) on cell constituents. The effects of UVR on humans are discussed from the point of view of occupational exposure particularly when exposure to UVR is combined with exposure to photosensitizing chemicals. At the end a short summary on protective measures in exposure to UVR is presented.

The UVR spectrum may be divided into three major portions which induce significantly different biological effects: UVA-wavelengths from 400 to 320 nm (synonyms: long wave UVR, near UVR, black light) UVB — wavelengths from 320 to 280 nm (synonyms: middle UVR, »sunburn radiation«) and UVC wavelengths from 280 to 200 nm (synonyms: short wave UVR, germicidal radiation) (1, 2).

SOURCES OF EXPOSURE AND MODES OF ACTION

Exposure to UVR may originate from natural and artificial sources. The sun is the principal natural source. Practically only UVA and UVB radiations reach the earth surface. Artificial UVR sources are widely used in industry and because of the germicidal properties of certain portions of the UVR spectrum they are also used in hospitals, biological laboratories and schools. Numerous man-made sources include high-intensity mercury vapour lamps, xenon lamps, medical photography lamps, sunlamps and even fluorescent lamps (2).

All photobiological responses to UVR and visible radiation are dependent on the energy of the incident photons. Absorption of UVR photons by a molecule results in the conversion of radiant energy into rotation-vibrational energy and a change in the electronic configuration inside the molecule.

Deoxyribonucleic acid (DNA) is one of the most important target molecules for the photobiological effect. The most common changes produced in DNA are damages to bases and to the polynucleotide chains. Damages to the bases may be unimolecular or bimolecular. Since pyrimidine bases are ten times more sensitive to UVR than purine bases, the most important effect is the formation of cyclobutane-type pyrimidine dimers. Normally they are produced by UVB but can also be produced by UVA with photosensitizers. Product additions to DNA bases are very numerous. Cross-links between DNA and proteins have been found in bacteria (3, 4) cell cultures (5) and also *in vivo* in the epidermis after irradiation with UVC or UVB (6).

If the features of the DNA macromolecules and the universality of the cell structure of living organisms in which DNA represents the genetic heritage are considered, it can be anticipated that any lesion inflicted on DNA, even a slight one, may have serious consequences. The lesions which prevent the functioning of DNA can be recognized by repair enzymes or may act as a signal for other biological processes to intervene. Several kinds of repair mechanisms exist but some lead to changes, even in undamaged sequences of DNA (3, 4, 7). If the lesions are not repaired they may produce cell death, genetic recombination, mutagenesis or even carcinogenesis (8).

THE EFFECTS OF UVR ON HUMANS

The effects of UVR on humans may be beneficial or detrimental depending on a number of circumstances (9, 10). Table 1 gives a survey on these effects. Harmful effects may be acute or chronic. Eyes and the skin are primarily involved, because UVR does not penetrate deeply the body tissues. UVR absorption by the mucous membranes of the eyes and eyelids can cause conjunctivitis (commonly known as »ground glass eye ball« or »welder's flash«). Lesions may also be formed on the cornea (photokeratitis) at high exposure levels. Such injuries usually manifest themselves six to twelve hours after exposure. They may be very painful and incapacitating but, as a rule, impairment is temporary (11). Chronic effects of UVR on the eye may cause the development of pterygium and squamous cell cancer of the conjunctiva and possibly cataracts (12).

The human skin serves as a filter for UVR. The shorter is the radiation wavelength, the greater is filtration. A considerable variation in the energy received by the different epidermal layers explains the very different biological effects as well as the irradiation accidents (13). Acute effects on the skin consist of solar erythema and sunburn. Sunburn, which is severe enough, may result in blistering and destruction of the surface of the skin with a secondary infection and systemic

Table 1
Effects of ultraviolet radiation on humans

Effects	UVB (285 — 320 nm)	UVA (320 — 400 nm)
Beneficial	mineral metabolism	can augment biological effects of UVB
	vitamin D synthesis	
	phosphorus and calcium metabolism	
	bone forming processes	
	defensive power to diseases including dental caries	
Harmful (primarily eyes and skin)	chronic pterygium and squamous cells cancer of the conjunctiva and cataracts	doses which alone demonstrate no biological effects can — in presence of certain environmental chemicals — result in:
	solar elastosis (aging of the skin)	phototoxicity photoallergy
	premalignant and malignant skin tumours (actinic keratoses)	enhancement of photocarcinogenesis
	non-melanoma and melanoma skin cancers	

adapted from Greiter and co-workers (10)

effects similar to a first or second degree heat burn. Fortunately, the skin has natural adaptative protection mechanisms consisting of increased production of the skin pigment melanin and thickening of the outer horny layer (14, 15).

Additional harmful effects depend on the penetration of UVR (15). One should have in mind that the skin is an organ highly overperfused with blood. At rest an equivalent of the entire blood system may pass through the skin in about 10 minutes which means that an unusually large portion of blood can be exposed to UVR (17). Alterations of many parameters of immunologic reactivity can be observed (18).

To measure the intensity of the UVR effects on the skin, the minimum erythemal dose (MED) is used. It is defined as the minimum UVR dose (290—320 nm) that produces definite but barely perceptible redness, 24 hours after exposure. The maximum amount of solar UVR to which an individual could be exposed in one day is about 25 minimum erythemal skin doses.

After many years of repeated exposure to UVR, the skin of susceptible individuals becomes leathery wrinkled and discoloured. Skin tumours (non-melanoma and melanoma skin cancers) may develop (19). The degree to which these changes develop depends not only on the UVR dose but on the individual genetic background and particularly on the ability of the skin to become pigmented. For these reasons »ageing« changes (solar elastosis, actinic keratosis and skin cancer) are much less common in genetically heavily pigmented individuals (20).

Table 2 shows a partial list of possible occupational exposures to UVR.

Table 2

Partial list of potential occupational exposures to ultraviolet radiation

Outdoor Sun Exposure	Agricultural workers
	Gardeners
	Construction workers
	Lifeguards
	Fishermen
	Open-pit miners
	Seamen
	Oilfield workers
	Pipeline workers
	Railroad track workers
Welding Arc Exposure	Welders
	Foremen
	Maintenance workers
	Pipeline workers
	Metal cutters
Germicidal Source Exposure	Medical profession
	Hospital employees
	Bacteriology laboratory workers
	Barbers
	Nurses
Plasma Torch Exposure	Plasma torch operators
Ultraviolet Laser Exposure	Laboratory workers
Curing Processes	Food irradiators
	Wood curers
Printing Processes	Lithographers
	Printers
Drying and Curing Paint	Paint curers
Nondestructive Testing	Metal casting inspectors

adapted from Emmett (19)

PHOTOSENSITIZATION

In exposure to UVR there is another very important effect. It relates to interactions of various UVR wavelengths, particularly UVA (320—400 nm) with natural and artificial chemical agents. This may result in a variety of deleterious effects not elicited by UVR or the chemical agents alone. The effects are due to photosensitization, a process in which the combined action of a chemical or a drug and an appropriate wavelength of UVR or visible radiation absorbed by the chemical or drug, leads to damaging effects in a biological system. Absorption of radiant energy by the photoexcited states can initiate chemical reactions with components of the biological systems in close proximity to the photosensitizer (21).

Two types of reaction may occur: phototoxic or photoallergic reactions. The phototoxic response can be divided into two subgroups: photodynamic and non-photodynamic. The difference relates to the needs for oxygen in the more complex photodynamic process. Mechanisms of these reactions are shown in Figure 1 (22).

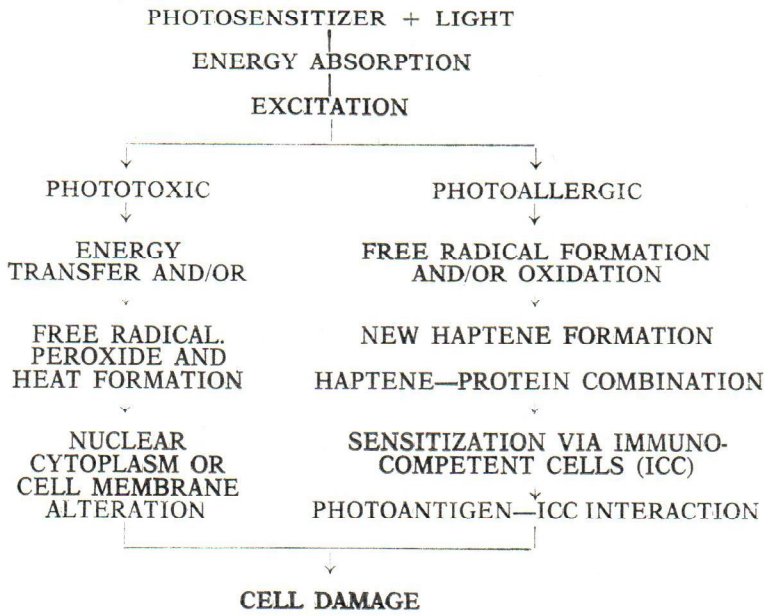

Fig. 1 — *Mechanism of phototoxic and photoallergic reactions*

adapted from Harber and co-workers (22)

Phototoxicity is common. Clinically, the responses are usually a delayed erythema, with or without oedema, followed by hyperpigmentation and desquamation. Thus they resemble the ordinary UVB radiation sunburn response.

Photoallergy is uncommon. It is an altered reactivity which is probably acquired, presumably dependent on an antigen-antibody or cell-mediated hypersensitivity. The reactions include immediate urticarial responses and delayed lesions from a papular to eczematous nature (23).

UVA radiation markedly accentuates the injury produced by UVB radiation extending the wavelength range at which damages occur. The UVA region is responsible for the vast majority of exogenous photosensitized reactions that occur in the skin (24, 25).

The photosensitizing chemicals may be endogenous or exogenous. A type of photosensitizing chemicals is made in the body. These are the porphyrin molecules which are responsible for the clinical manifestations of the porphyrias.

Exogenous chemical photosensitizers may arrive on the skin topically or through the circulation. Their number is immense. Actually more than 400 have been identified belonging to the following categories: cosmetics, medications: advertent or inadvertent, and plants.

Compounds of industrial origin constitute an important source of topical photoactive chemicals. These agents are primarily polycyclic hydrocarbons that can act as photosensitizers or carcinogens.

Table 3 shows chemicals that induce contact photosensitivity reactions in humans.

The commonly used systemic photosensitizers may be ranged in the next five categories: antibacterial sulphonamides, thiazide diuretic medications, sulphonylurea antidiabetic medications, phenothiazines, antibiotics.

Table 4 shows a more detailed list of chemicals acting as systemic photosensitizers.

Acute phototoxic responses are by far the most common reactions to these agents (21). But patients treated with such drugs may be at increased risk to develop cancer. A great part of the working population may be at risk when exposed to industrial contaminants and radiations from the sun, such as oil field workers and the like. Industrial contaminants and air pollutants that contain both mutagenic carcinogens and tumour promoters accelerate cutaneous cancer formation in the skin similarly to the acceleration of cutaneous cancer formation noted experimentally (25).

Agents capable of producing phototoxic effects have been identified by use of a variety of *in vitro* and *in vivo* systems. The mechanism of this chemical phototoxicity includes a direct action on the DNA mo-

Table 3

Contact photosensitizers: Chemicals that induce photosensitivity reactions in humans[a]

Name[b]	Use	Reported clinical observations
Halogenated salicylanilides; 3,3',4',5-tetrachlorosalicylanilide; 3,4',5- and 3,3'5-trichlorosalicylanilide; 3,4',5- and 3,3',5-tribromosalicylanilide; 3,5- and 4,5'-dibromosalicylanilide	Deodorant, bacteriostatic agents in soaps	Phototoxic and eczematous photoallergic reactions, burning, itching, crossphotosensitivity reactions
Hexachlorophene	Antimicrobial, antiseptic	Phototoxic reactions
Bithionol or bis (2-hydroxy-3,5-dichlorophenyl) sulphide	Antimicrobial, antiseptic	Photoallergic reactions
Fentichlor (2,2'-dihydroxy-5,5'-dichlorodiphenylsulphide); multifungin (bromochlorosalicylanilide); Jadit (4-chloro-2-hydroxybenzoic acid N-n-butylamide)	Antifungal	Phototoxic and photoallergic reactions
5-Fluorouracil	Antineoplastic	Acceleration of inflammatory process
PABA and esters of PABA	Suncreen	Photoallergic reactions
4,4'-Bis(3-phenylureido)-2,2'-stilbenedisulphonic acid or blankophor	Fluorescent brightening agent for cellulose, nylon, or wool fibers	Phototoxic and photoallergic reactions
Cadmium sulphide	In tattoos	Erythema
Furocoumarins: psoralen, 8-MOP, 5-methoxypsoralen, TMP	In vitiligo for increased pigment formation and sun tolerance	Marked erythema, vesicles, bullae, hyperpigmentation
Essential oils: bergamot oil, lime oil, cedar oil, lavender oil, citron oil, sandalwood oil	Cosmetics and beauty aids	Phototoxic reactions and post-inflammatory hyperpigmentation
Plants: Umbelliferae, Rutaceae	Used in perfumes of flavorings or as spices	Phytophotodermatitis, hyperpigmentation, vesicles, bullae
6-Methylcoumarin	Used in cosmetics	Photoallergic reactions
Musk ambrette	Used in cosmetics	Photoallergic reactions
Dyes: fluorescein, rose bengal, eosin, erythrocine, trypaflavin, orange red, paraphenylenediamine. methylene blue, toluidine blue, trypan blue, anthraquinone	Cosmetics and dye industry	Erythema, edema, vesicles, pigmentation, phototoxic reaction
Coal tar and coal tar derivatives containing anthracene, phenanthrene, naphthalene, thiophene, and many phenolic agents; pitch; acridine	In therapy for psoriasis and chronic eczema; in hair shampoos	Smarting, exaggerated sunburn, urticarial wheals, tar melanosis

[a]This table has been adapted from Fitzpatrick and co-workers (14)

[b]*PABA* = p-aminobenzoic acid.

Table 4

Systemic photosensitizers: Chemicals that induce photosensitivity reactions in humans[a]

Name	Uses	Clinical observations	Action spectrum, nm
Sulphonamides: sulphanilamide, sulphathiazole, sulphapyridine, sulphamethazine, sulphaguanidine, sulphisoxazole, monochlorphenamide	Chemotherapy, antibacterial agents	Phototoxic and photoallergic reactions	290—320
Sulphonylurea: carbutamide, tolbutamide (Orinase), chlorpropamide (Diabinese)	Hypoglycemic or antidiabetic drugs	Phototoxic reactions	290—360
Chlorthiazides: 6-Choro-7-sulphamyl-3,4-dihydro-1,2,4-thiodiazine 1,1-dioxide (Hydrodiuril)	Diuretics, antihypertensive	Papular and edematous eruption, plaques	290—320
Quinethazone (Diuril)	Antihypertensive	Phototoxic and photoallergic reactions	320—400
Phenothiazines: chlorpromazine (Thorazine), promethazine (Phenergan), mepazine, Stelazine, trimeprazine, Compazine, promazine (Sparine)	Tranquilizer, nematode, infestation agent, urinary antiseptic, antihistamine	Exaggerated sunburn, maculopapular and urticarial eruptions, gray-blue hyperpigmentation	290—400
Antibiotics: Demethylchlortetracycline (Declomycin), chlortetracycline, oxytetracycline, doxycycline	Broad-spectrum antibiotic	Exaggerated sunburn, phototoxic reaction	320—400
Griseofulvin	Antimycotic	Exaggerated sunburn, phototoxic and photoallergic reactions	320—400
Nalidixic acid (NegGram)	Antibacterial	Erythema, bullae	320—400
Furocoumarins: TMP (trixsalen), 8-MOP (methoxsalen), psoralen	In photochemotherapy of psoriasis and vitiligo; for sun tolerance and increased pigment formation	Erythema, bullae, hyperpigmentation	320—400
Chlordiazepoxide (Librium)		Eczematous eruption	290—360
Triacetyldiphenolisatin	Tranquilizer, psychotropic	Eczematous photoallergic reaction	290—320
Cyclamates, calcium cyclamate, sodium cyclohexylsulphamate	Laxative Artificial sweeteners	Phototoxic and photoallergic reactions	290—360

[a]This table has been adapted from Fitzpatrick and co-workers (14)

lecule. Monofunctional and bifunctional adducts may be formed which lead to the inhibition of DNA synthesis, cell damage and cell death. Among them, furocoumarins have found important application in the treatment of certain skin diseases, particularly psoriasis (24).

Photoinduced DNA cross-links have been reported with coal tar extract and anthracene. Lysosomal labilization has been accomplished with anthracene as well as with porphyrins. The production of a phototoxic product also can cause a membrane damage which results morphologically in an erythematous cutaneous response. Phototoxic reactions caused by UVR exposure combined with actinomycin D on cells in culture were also observed (26, 27).

However, much more information is needed to clarify the mechanisms leading to the changes that occur in/or during chemically induced phototoxic cutaneous response.

To detect photosensitivity several techniques are used (28): systemic administration, topical application with occlusion, topical application after the stratum corneum is stripped off, intradermal injection of the molecules.

As an example of the use of those techniques a study in workers engaged in formulating UV cured inks (29) can be mentioned. UV cured inks consist of several conventional pigments dispersed in a varnish. The varnish has a great number of components. Workers weighing, mixing and milling ingredients complained of a sharp burning sensation on the exposed arena of the body while in sunlight. The response suggested photosensitivity and a phototoxic response. After testing, it was demonstrated that the observed phototoxicity was due to exposure to a mixture of amyl-o-dimethylaminobenzoic acid and amyl-p-dimethylaminobenzoic acid.

A diagnostic approach using preliminary *in vitro* screening to establish phototoxic potential followed by limited *in vivo* testing, seems particularly adaptable to the study of industrial phototoxicity apparently resulting from skin contact (17).

CONCLUSION

Following the recommendations of the World Health Organization it may be concluded that exposure of both the eye and the skin to UVR should be kept to a minimum (1). Whenever possible, excessive exposure should be prevented by proper engineering design of UVR installations and suitable enclosure. Protection should be also afforded by providing to workers close-fitting goggles and if necessary face shields, suitable UVR-opaque clothing and gloves. To workers exposed to UVR adequate instructions should be given concerning hazards

involved and precautions to avoid excessive exposure to radiations. Finally, the different types of sensitivity to UVR presented by people with different qualities of skin must also be taken into consideration.

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Sažetak

UČINAK PROFESIONALNE IZLOŽENOSTI ULTRAVIOLETNOM SVJETLU

Uvodno su prikazana svojstva UV svjetla, izvori izloženosti i način djelovanja. Glavni prirodni izvor UV svjetla je Sunce. Zemljinu površinu dosegnu praktički samo zrake UVA (valne dužine od 400 do 320 nm) i UVB (valne dužine od 320 do 280 nm) spektra. Umjetni izvori UV svjetlosti imaju više-

struku primjenu u industriji a zbog germicidnih svojstava nekih dijelova UV spektra upotrebljavaju se u bolnicama, biološkim laboratorijima, školama. Od umjetnih izvora UV svjetla treba spomenuti živine lampe visokog intenziteta, ksenonske lampe, fotografske i medicinske lampe, lampe za sunčanje, fluorescentne lampe. Tablično je prikazana parcijalna lista moguće profesionalne izloženosti UV svjetlosti.

Učinak UV svjetlosti na čovjeka može biti koristan ili štetan, ovisno o različitim okolnostima. Štetni učinci mogu biti akutni i kronični, a zbog slabe penetracije odnose se uglavnom na oči i kožu. Sluznice oka i vjeđa apsorbiraju UV svjetlost, što može uzrokovati konjunktivitis. U visokoj izloženosti može doći i do oštećenja na korneji (fotokeratitis). Kronični učinci UV svjetlosti na oko mogu uzrokovati pterigij a i skvamozni rak konjunktive i, vjerojatno, kataraktu. Akutni učinci na koži očituju se solarnim eritemom i opeklinama. Nakon dugogodišnje izloženosti mogu se razviti tumori kože (melanom i karcinom). Stupanj kroničnih promjena ne ovisi samo o UV dozi nego i o individualnom genetskom nasljeđu, a posebno o sposobnosti kože da proizvede pigmente. Iz tih razloga promjene »starenja« kože (solarna elastoza, aktinički keratitis i rak kože) mnogo su rjeđe u osoba s genetski više pigmenta.

U ekspoziciji UV svjetlosti vrlo je važan još jedan učinak: fotoosjetljivost. Taj se učinak odnosi na interakciju različitih UV valnih dužina, posebno UVA (320—400 nm) s prirodnim ili umjetnim kemijskim spojevima. Moguće su dvije reakcije: fototoksična i fotoalergična. Klinička posljedica fototoksične reakcije je obično eritem, koji nastaje nakon latencije, s edemom ili bez njega, praćen hiperpigmentacijom i ljuštenjem. Fotoalergija je rjeđa a nastaje vjerojatno ovisno o reakciji antigen-antitijelo ili reakciji preosjetljivosti posredovanjem stanica (cell-mediated). Posljedica je nagla pojava urtikarije te, nakon latencije, oštećenje od papula do ekcema. Tablično su prikazane tvari koje uzrokuju kontaktnu preosjetljivost kod ljudi te tvari koje djeluju kao sistemski fotosenzibilizatori.

Istodobna izloženost onečišćenjima koja sadržavaju mutagene, karcinogene tvari ili promotore tumora i sunčevim zrakama odnosno UV svjetlosti ubrzava stvaranje raka kože. To je npr. slučaj s radnicima na naftnim bušotinama.

Ukratko su prikazane metode za otkrivanje fotoosjetljivosti. U zaključku su sumirane mjere zaštite, odnosno sprečavanje mogućih nepovoljnih učinaka profesionalne izloženosti UV svjetlosti.

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