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Occupational Skin Diseases Caused by UV Radiation

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SUMMARY This overview highlights the risk of skin diseases arising in workers exposed to ultraviolet radiation (UVR) at their workplace. There is a plethora of skin manifestations in outdoor workers such as seamen, fishermen, farmers after acute intense or long-term exposure to solar UVR, but some cutaneous diseases may also develop in indoor workers exposed to artificial sources. In recent years, investigations of the biological effects and damage caused by UVB and UVA on the skin have improved our understanding of the cellular and molecular mechanisms of photoaging, skin cancer and other skin diseases caused by UVR exposure. The necessity of primary prevention in workers exposed to UVR is emphasized.

KEY WORDS: ultraviolet radiation, outdoor work, indoor work

INTRODUCTION

Ultraviolet radiation (UVR) is a form of non-ionizing rays as part of the broad electromagnetic spectrum. UVR has greatest energy of all types of optical radiation, and these rays can be reflected, scattered, transmitted or absorbed. These rays can be subdivided into three main bands: 320-400 nm band designated as UVA or long-wave, 290-320 nm band designated as UVB or medium wave, and 200-290 nm band designated as UVC or short-wave (1,2). Wavelengths below 200 nm called vacuum UV are absorbed by the air, thus being of no biological significance. The energy of each part of the radiation is inversely related to the wavelength. UVR makes up approximately 5% of all rays emitted from the sun to the earth, nevertheless, they have a large amount of energy and can affect the skin, eyes and immune system considerably (3,4).

UVB rays make about 5% of all UVR reaching the earth as solar UVR of highest energy, and are mostly absorbed in the epidermis. Window glass filters out the major part of UVB. Biologically, they are most active in damaging the skin and eyes. UVB exposure provokes vasodilatation with erythema and inflammation, probably due to the release of mediators such as prostaglandins, histamine and cytokines, e.g., IL-1, IL-6 and IL-8 (5,6). Causing damage to nuclear DNA, they are mutagenic, immunosuppressive, and can induce premalignant lesions, non-melanoma skin cancer, and have also been suggested to play a role in causing melanoma. In the last decades, ozone depletion in the stratosphere has led to an increase in UVB at the ground level (7). UVA is the predominant component of solar UVR (about 95%) that reaches the earth's surface. The energy

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of these rays is relatively small, but they are less affected by altitude and atmospheric conditions, can penetrate deeply into the skin, and can cross window glass and fabrics. They cause quick tanning and premature skin aging, and are involved in phototoxic and photoallergic reactions. Today, it is confirmed that these rays can also be carcinogenic (5,7).

Solar UVC rays are completely filtered out by the ozone layer in the stratosphere, 15-50 km above the sea level (7,8). Terrestrial UVC rays can cause erythema, are mutagenic and germicidal. Visible light can also cause photodermatoses, while infrared rays can contribute to chronic skin damage and aging from UVA rays (5).

The effect of solar radiation or artificial UVR can be beneficial as in case of vitamin D3 photosynthesis, influencing our mood maybe by affecting the endogenous opioid system, and in the treatment of diseases, or can cause unwanted and deleterious effects such as skin diseases, eye diseases (keratoconjunctivitis, cataract, pinguecula, pterygium and tumors), and local and systemic immunosuppression (9,10). So, intense acute or chronic exposure to UVR of solar origin or from artificial sources may overcome the natural cutaneous photoprotection mechanisms (stratum corneum, melanin, urocanic acid, antioxidants, DNA repair) and cause occupational skin diseases (7,9,10). We therefore thought it worthwhile to give a survey of occupational skin diseases caused by exposure to solar or artificial UVR in outdoor or indoor workers.

SKIN DISEASES DUE TO SOLAR EXPOSURE

Occupational health risks like environmental hazards can be physical, chemical, biological and psychosocial. Among physical ones, the most important is UVR exposure, and the biological effects depend on the quality (spectrum) and intensity of exposure (8). Despite their biological differences, both UVB and UVA can cause occupational skin diseases. Acute or chronic exposure to solar UVR is of significance in outdoor workers, and exposure to UVR from a wide variety of artificial sources in indoor workers (9). The UVB and UVA radiation can cause skin alterations alone, act synergistically or in combination with endogenous or exogenous substances (11). Some occupational skin diseases are of minor medical significance, e.g., pigmentations, telangiectasias or other stigmata, which do not affect working capacity, whereas others may cause major trouble and concern (11).

The quantity of solar UVR that reaches the earth depends on the season, time of the day, latitude, altitude, surface reflection, ozone layer, and cloud cover (7). It is estimated that 5% to 10% of the population in Europe are working outdoor (12). Occupational photodermatoses occur mostly in outdoor workers with fair complexion, i.e. with phototype I and II according to Fitzpatrick (13). Exposure to solar UVR in workers engaged in outdoor work characteristically provokes lesions in sun exposed areas: face, neck, décolletage, and extensor surface of the upper limbs, particularly forearms, and hands (14,15). Both acute and chronic effects of exposure to solar UVR are linked to DNA damage. Already 0.1-0.3 MED can activate p53 and p21, and cause immunosuppression and apoptosis of keratinocytes, i.e. sunburn cells (3). Acute exposure to sunlight causes sunburn, immediate and delayed tanning, immune alteration, and participates in phototoxic and photoallergic reactions, whereas chronic exposure causes actinic keratoses, actinic cheilitis, immunosuppression, premature aging of the skin, and malignant tumors of the skin such as basal cell cancer, keratoacanthoma, squamous cell cancer, melanoma, and probably Merkel cell carcinoma (16). Some workers exposed to UVR can rarely develop diseases such as solar urticaria or polymorphous light eruption.

Photoaging (premature skin aging or extrinsic aging) and skin cancer are steadily increasing in the population. This is so because people live longer and due to depletion of the ozone layer in the stratosphere caused by the use of chlorofluorocarbons. Photoaging is characterized by the development of xerosis, coarseness, wrinkling, deep furrows, yellow discoloration (actinic elastosis), irregular pigmentation (actinic lentigo, guttate hypomelanosis), senile purpura, and telangiectasias in the sun exposed areas (17,18). The described changes (dermatoheliosis) are caused by cumulative exposure to UVR, particularly UVA. UVA induces the formation of reactive oxygen species (ROS) like singlet oxygen, hydrogen peroxide, hydroxyl radical and others, and so indirectly damages DNA. ROS also cause lipid peroxidation. UVA causes changes not only in the epidermis (pigmentation) but even in the dermis (degeneration of collagen and elastic fibers). Studies in animals and humans have shown that exposure of the skin to UVR provokes activation of transcription factors (AP-1, NF-kB), which results in an increase of matrix metalloproteinases (MMPs) produced by kera-

tinocytes and fibroblasts, like collagenase (MMP-1), gelatinase (MMP-9) and stromelysin (MMP-3) (18,19). Recent *in vitro* and *in vivo* studies have demonstrated that UVR induces ROS, which also damage mitochondrial DNA. It is known that mitochondrial DNA has a reduced repair capacity, and so alteration in the mitochondria may contribute to photoaging (20).

Colloid pseudomilium, poikiloderma of Civatte, and Favre-Racouchot syndrome characterized by the presence of brown-black papules and open comedones in periorbital region may also develop occasionally in workers exposed to sunlight, mostly in men. So, Cellini and Offidani found dermatosis in 2.5% of agricultural workers but not in the control group (21).

Frequently, photoaging, actinic keratoses and skin cancers can be observed in outdoor workers such as seamen, fishermen, farmers, asphalters, roofers, horticultural workers, construction workers, and others (8,9) (see Table 1). In some professions such as fishermen and seamen the quantity of UVR reaching the skin is increased not only by direct exposure to solar UVR but also by the rays reflected (5%-20%) from the sea (15). In addition, some reports point to the importance of sun exposure as a health risk in professional cyclists, mountain guides and policemen (22,23). So, Moehrle *et al*. demonstrated in mountain guides carrying dosimeters that the daily dose of UV exceeded the limits sixfold or more, which is suggestive of an increased risk of skin tumors (23). Recent studies in gardeners demonstrated the dose of UVR received to depend not only on sun exposure but also on the time of the day, possible shade during the work, and clothing habits (24).

Outdoor workers can develop photodamage by contact with plants, coal tar derivatives, drugs, dyes and exposure to sunlight; these interactions are phototoxic and cause photoallergic reactions. Phototoxic reactions (phytophotodermatitis) are more common and occur without involvement of immune mechanisms (11,25). They are mostly caused by contact with plants that contain photosensitizers such as furocoumarins. Furocoumarins are tricyclic compounds that plants synthesize in defense from fungi, and can be linear (psoralens) or angular (angelicin and pimpinellin). Their photoactivation leads to the formation of adducts with pyrimidine bases and consequential DNA damage and cross strand links. This leads to erythema, vesiculobullous lesions, burning, and can occur in farmers, florists, gardeners, horticulturists, botanists, grocery store workers, food handlers, and others (26). Most of the plants causing phototoxic reactions belong to the family *Umbelliferae* (*Apiaceae*) like celery (*Apium graveolens*), parsnip (*Pastinaca sativa*), parsley (*Petroselinum crispum*), false bishop's weed (*Ammi majus*), cow parsnip (*Heracleum sphondylium*) that causes trimmer dermatitis, and angelica (27-29). Plants of other families can also cause phytophotodermatitis, e.g., figs (*Ficus carica*), garden rue (*Ruta graveolens*), and others (30). It is important to differentiate this reaction from pseudophytophotodermatitis caused by contact with plant insecticides and herbicides (31). In workers, the use of certain drugs (sulphonamides, antidiabetics, thia-

Table 1. Outdoor workers potentially exposed to UV radiation

Fishermen	Farmers
Seamen	Gardeners
Watermen	Green-keepers
Lifeguards	Horticultural workers
Construction workers	Oilfield workers
Road workers	Pipeline workers
Brick masons	Military personnel
Postal carriers	Ski instructors
Railroad track workers	Professional cyclists
Policemen	Surfers
Outdoor maintenance workers	Landscapers

Modified from Epstein JH *et al*. Occupational skin cancer. In: Adams RM, ed. Occupational skin diseases, 3rd ed. Philadelphia: Saunders; 1998.

zides, nonsteroidal anti-inflammatory drugs, phenotiazines) or contact with oils and tars can also induce sensitization to solar UVR (11,32). Phototoxic and photoallergic dermatitis can be induced by airborne allergens and irritant substances in the form of solid particles, gases or droplets (33).

Sun exposure probably also causes chronic actinic dermatitis (actinic reticuloid), which develops mostly on the face in older men working outdoor while photoprotected sites as upper eyelids, submental region and behind the ears are spared (34). Exposure to sunlight in workers can also worsen a pre-existing skin disease such as lupus erythematosus, dermatomyositis, pityriasis rubra pilaris, Darier's disease and rosacea (35).

INDOOR WORK AND PHOTODAMAGE

There are numerous sources that emit different types of UVR, which are used for a wide range of application at workplace. The potential risk of indoor exposure to UVR is very high in welders, depending on the material being welded, and particularly when using electric arc welding. The risk is diminished in workers using oxyacetylene welding, laser welding, and electron beam welding (36-38). In electric arc welding temperature can be very high (2000 °C and more), and according to Wien's law the radiation peaks to short waves if temperature increases.

Non-welders in the vicinity of welding are also exposed to high doses of UVR (39). The effects of welding on the skin include erythema, small scars and keratoconjunctivitis on the eyes (arc eye); some studies have demonstrated an increase of eye cancer and melanoma in welders (40). In our region as in other maritime regions welding can pose a problem in ship building industry (41). Sometimes welding is also used by mechanics, electricians, sculptors, and others, who are not aware of all problems associated with this process and so can be injured by UVR.

UVR is used in sterilization of operating rooms, dermatologists use it in diagnosing and treatment of numerous skin diseases, and pediatricians use phototherapy for neonatal hyperbilirubinemia. UVR is used in lighting offices too. Indoor UVR can cause lesions in dentists who use UVA for polymerization of resins (42,43). UVR is used for curing of printing inks, curing of paints, curing of metal decorating in packaging industry, inspection of printed circuit board in electronics industry, and by the staff of tanning pools (42) (Table 2). UVR lamps and transilluminators are used by many research laboratories for studies of photobiology (animal experiments), in photochemistry, genetics (visualization of DNA and RNA) and molecular biology (visualization of subcellular structures), where researchers or students are engaged (44,45). UVR is used for identification of substances, analytical and diagnostic processes, or for polymerization of chemicals in biochemical laboratories. In microbiological laboratories UVC rays (germicidal lamps with a peak around 260 nm) are employed for sterilization of surfaces, liquids and spaces (42). Similar lamps are used to kill fly (electric fly killers) and for disinfection of water, milk, wine, beer, juices, and pools. Recently, 26 medical students suffered skin and eye injuries due to exposure to a malfunctioning germicidal lamp (46). From the above one can conclude that there is a necessity to prevent UVR exposure in workers.

Although numerous skin diseases can occur in workers exposed to UVR during outdoor or indoor work, as described above, the primary long-term risk of exposure to solar or artificial UV light, particularly in fair-skinned people (melanocompro-

Table 2. Indoor workers potentially exposed to UV radiation

Welders	Printers
Physicians	Lithographes
Nurses	Painters
Dentists	Wood curers
Cosmetologists	Plastic workers
Laboratory workers	Food irradiation
Plasma torch operators	Kitchen workers
Maintenance workers	Pipecutters

Modified from Epstein JH *et al*. Occupational skin cancer. In: Adams RM, ed. Occupational skin diseases, 3rd ed. Philadelphia: Saunders, 1998.

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mised, types I and II according to Fitzpatrick), is the development of precancerous lesions, non-melanoma skin cancer and melanoma. As early as 1894, Unna described degenerative changes in the epidermis and dermis, found in the sun exposed area of the skin in seamen, and linked these alterations with the development of cancer (47). In the same year, Enziere described cancer of the lower lip in peasants working outdoor, while being rare among town people (48). In 1928, Findlay first proved experimentally the development of skin cancer in mice irradiated with UVR from mercury arc (49). It was confirmed by the Argentinian Roffo, who demonstrated that sunlight and UVR from artificial sources can induce skin cancer in rodents. He also demonstrated that the major carcinogen was UVB (50,51). Later investigations demonstrated that UVR induced mutation of the genetic material, i.e. DNA, directly on pyrimidine bases (thymine and cytosine) in particular forming photoproducts that led to C-T or CC-TT transitions (52), or indirectly by ROS, i.e. oxidation of the purine guanine (53). Most of these DNA damages are quickly enzymatically corrected (nucleotide excision repair). These "signature mutations" are present particularly in the suppressor genes such as p53 or PTCH. If not corrected, their protein product cannot control cell cycle properly, thus favoring cell growth, proliferation and survival. More recently, Kripke and Fisher (54) and Kripke and Morison (55) demonstrated experimentally that UV irradiation of rodents significantly depressed their immune defense, thus allowing for carcinogenesis. These studies confirmed the clinical and epidemiological data that skin cancer develops after intensive and prolonged exposure to UVR, and permitted better understanding of the cellular and molecular mechanisms in photocarcinogenesis.

Numerous epidemiological, clinical, and experimental studies assessed the importance of UVR exposure in the development of skin cancers and melanoma (56-58). Basal cell cancer seems to be connected with cumulative sun exposure and also to intermittent exposure (on the trunk), while the more aggressive squamous cell carcinoma correlates better with lifelong cumulative exposure. Melanoma seems to be associated with intense episodic, intermittent UVR exposure (56). Epidemiological studies from Australia and USA suggest the relevance of outdoor work for the development of basalioma, squamous skin cancer and melanoma, and most of the workers also have evidence of chronic actinic damage (56,59,60). Data from Europe are variable; so, Cherry *et al.* found in UK that 96% of skin cancers were caused by UV exposure, so that outdoor work represents an important factor in the development of skin tumors (61), whereas in Finland and Sweden there was no association of outdoor work in agricultural workers, forestry workers and construction workers with increased skin cancers (62,63). An explanation could be that in these countries cumulative exposure is low because of the high latitude. Different health systems and legislation across European countries may also make the data difficult to compare (12). Data from Japan also show that cancer is increased in outdoor workers (64).

Jakac, a pioneer in the study of professional skin diseases in Croatia, clearly demonstrated the relevance of solar exposure in the development of skin cancer in sailors and fishermen, but also in construction workers, agricultural workers, and shipworkers. On their skin, he often observed *cutis nautae* and *cutis agricolae* on their skin, which are not *atrophia cutis senilis* but *degeneratio cutis climatica* according to Kogoj (65). His study on the epidemiology of skin cancer in Rijeka region from 1955 to 1959 showed that nearly 90% of male patients were outdoor workers, mostly fishermen and sailors (32.9%), then farmers (29.2%), dock workers (9.6%), building and construction workers (9.3%) and other outdoor workers (7.5%). In women, skin cancer developed most frequently in peasants (34.4%) and housewives working in their vegetable gardens (23.1%). He had also found that cancer mostly developed in fair skinned workers, and proposed skin cancer as a professional disease (15,66,67). In more recent years, the prevalence of skin cancer in fishermen and sailors is dropping (68,69); these recent data are probably associated with the lower number of workers in these professions, and their later engagement in this kind of work. Fishermen now work mostly during the night, do not use tars to impregnate their nets, and perhaps use sunscreens. The high rate of skin cancer in housewives can be explained by the fact that women frequently work outdoor in their vegetable gardens or truck farms. Particular caution is needed in workers treated with immunosuppressive drugs because they are at a high risk of cancer development (70).

In conclusion, UVR exposure at workplace can induce numerous different skin diseases and also leads to precancerous lesions and malignant tumors. It is not easy to obtain verification that skin cancer is occupational because there is a long period between the exposure and its development, and sometimes because the individuals had previ-

ously worked in a tropical country (12). For this reason, it is of paramount importance to recommend effective primary prevention measures like legislation measures, education of the workers to undertake appropriate protection measures, wearing appropriate protective clothing, broad brimmed hats, eye protectors, and seek to work in shade. The use of sunscreens may pose a problem because they frequently are not used appropriately and sometimes can prolong the exposure.

References

1. Coblentz WW, Stair R, Hogue JM. The spectral erythemic reaction of the human skin to ultraviolet radiation. Proc Natl Acad Sci USA 1931;17:401-5.
2. Matts PJ. Solar ultraviolet radiation: definition and terminology. Dermatol Clin 2006;24:1-8.
3. ICNIRP. Guidelines on limits of exposure to ultraviolet radiation of wavelengths between 180 nm and 400 nm (incoherent optical radiation). Health Phys 2004;87:171-86.
4. Longstreth J, de Gruijl FK, Kripke ML, Abseck S, Arnold F, Slaper HL, *et al*. Health risks. J Photochem Photobiol 1998;46:20-5.
5. Gruber F, Peharda V, Brajac I. Influence of climatic factors on the skin. Labin 1999; Proceedings. pp. 234-40.
6. Clysdale GJ, Dandie GW, Muller HK. Ultraviolet light induced injury: immunologic and imflammatory effects. Immunol Cell Biol 2001;79:547-68.
7. Guarrera M. Fotoprotezione: come, quando e perché. G Ital Dermatol Venereol 2001;136:21-38.
8. Diffey BL. Human exposure to solar ultraviolet. J Cosmet Dermatol 2002;1:124-30.
9. Škreb Y, Šarić M. The effects of ultraviolet radiation on occupationally exposed workers. Arh Hig Rada Toksikol 1983;34:275-86.
10. Nola I, Kotrulja L. Skin photodamage and lifetime protection. Acta Dermatovenereol Croat 2003;11:32-40.
11. Jakac D. Profesionalne dermatoze. In: Jakac D, editor. Dermatologija i venerologija. Beograd-Zagreb: Medicinska knjiga; 1981. p. 198.
12. Diepgen TL, Drexler H. Hautkrebs und Berufserkrankungen. Hautarzt 2004;55:22-7.
13. Fitzpatrick TB. The validity and practicality of sun reactive skin type I through VI. Arch Dermatol 1988;124:869-71.
14. Wolf A. Fotodermatoze (aktiničke dermatoze). In: Jakac D, editor. Dermatologija i venerologija. Beograd-Zagreb: Medicinska knjiga; 1981. p. 301.
15. Jakac D. Importanza dei fattori climatici nella manifestazione degli epiteliomi cutanei nei marinai e nei pescatori. Chron Derm 1971;2:43-57.
16. Honigsmann H, Diepgen TL. UV induced skin cancer. JDDG 2005;3(Suppl 2):26-31.
17. Matsamura Y, Ananthaswamy HN. Toxic effects of ultraviolet radiation on the skin. Toxicol Appl Pharmacol 2004;195:298-308.
18. Berneburg M, Plettenberg H, Krutmann J. Photoaging of human skin. Photodermatol Photoimmunol Photomed 2000;16:239-44.
19. Fisher GJ, Wang ZQ, Datta S, Varani J, Kang S, Voorhees JJ. Pathophysiology of premature skin aging induced by ultraviolet light. N Engl J Med 1997;337:1419-28.
20. Berneburg M, Kamenisch Y, Krutmann J. Repair of mitochondrial DNA in aging and carcinogenesis. Photochem Photobiol Sci 2006;5:190.
21. Cellini A, Offidani A. An epidemiologic study on cutaneous diseases of agricultural workers authorized to use pesticides. Dermatology 1994;189:129-32.
22. Moehrle M, Heinrich L, Schmid A, Garbe C. Extreme UV exposure of professional cyclists. Dermatologica 2000;201:44-5.
23. Moehrle M, Dennenmoser B, Garbe C. Continuous long-term monitoring of UV radiation in professional mountain guides reveals extremely high exposure. Int J Cancer 2003;103: 775-8.
24. Thieden E, Collins SM, Philipsen PA, Murphy GM, Wulf HC. Ultraviolet exposure patterns of Irish and Danish gardeners during work and leisure. Br J Dermatol 2005;153:795-801.
25. Kiec-Swierczynska M, Krecisz B. Photosensitivity induced skin disease. Med Pol 2001; 52:3-7.
26. Berkley SE, Hightower AW, Beier RC, Fleming DW, Brokopp CD, Ivie GW, *et al*. Dermatitis in grocery workers associated with high natural concentration of furocoumarins in celery. Ann Intern Med 1986;105:351-5.
27. Maso MJ, Ruszkowski AM, Bauerle J, DeLeo VA, Gasparoo FP. Celery phytophotodermatitis in a chef. Arch Dermatol 1991;127:912-3.

28. Ippen H. Phytophotodermatitis caused by plant trimming (edger's rash). Derm Beruf Umwelt 1989;38:190-2.
29. Milavec-Puretić V, Zečević J. Kontaktni alergijski i nealergijski dermatitisi uzrokovanim biljkama. Acta Dermatovenerol Iug 1983;10:25-30.
30. McGowern TW. Botanical briefs: The Fig-Ficus carica. Cutis 2002;69:339-40.
31. Stoner JG, Rasmussen JE. Plant dermatitis. J Am Acad Dermatol 1983;9:1-15.
32. Jeanmougin M. Exogenous photosensitivity reactions. Retinoids 1993;30:9-13.
33. Goosens A. Airborne dermatosis. Acta Dermatovenerol Croat 2006;14:153-5.
34. Ferguson J, Ibbotson S. The idiopathic photodermatoses. Semin Cutan Med Surg 1999;18:257-73.
35. Morison WL,Towne LE, Honig B. The photoaggravated dermatoses. In: Hawk JLM, editor. Photodermatology. London: Arnold,1999:219-60.
36. Tenkate TD. Optical hazards of welding arcs. Rev Environ Health 1998;13:131-46.
37. Garcia-Guinea J, Carrecher V, Lambardero M, Gonzales-Martin R. Study of the ultraviolet emission of the electrode coating of arc welding. Int J Environ Health Res 2004;14:285-94.
38. Emmett EA, Horstman SW. Factors influencing the output of ultraviolet radiation during welding. J Occup Med 1976;18:41-4.
39. Okuno T, Ojima J, Saito H. Ultraviolet radiation emitted by CO2 arc welding. Ann Occup Hyg 2001;45:597-60.
40. Currie CL, Monk BE. Welding and non-melanoma skin cancer. Clin Exp Dermatol 2000;25:28-9.
41. Szarmach H, Synoradzka-Nakonieczna H. Evaluation of the effect of welding on the occurrence of dermatoses in ship industry. Przegl Dermatol 1970;57:195-202.
42. Diffey BL. Human exposure to ultraviolet radiation. Semin Dermatol 1990;9:2-10.
43. Bruzell-Roll EM, Jacobsen N, Hensten-Pettersen A. Health hazards associated with curing light in the dental clinic. Clin Oral Invest 2004;8:113-7.
44. Cazzuli O, Giroletti E. Rischio di esposizione a radiazione UV nei laboratori biochimici. G Ital Lav Erg 2002;24:56-65.
45. Akbar-Khanzadeh F, Jahangir-Blourchian M. Ultraviolet radiation exposure from UV-transilluminators. J Occup Environ Hyg 2005;2:493-6.
46. Trevisan A, Piovesan S, Leonardi A, Bertocco M, Nocolosi P, Pellizzo MG, *et al*. Unusual high exposure to ultraviolet-C radiation. Photochem Photobiol 2006;82:1077-9.
47. Unna P. Histopathologie der Hautkrankheiten. Berlin: Hirschwald, 1894.
48. Enziere JP. Du cancer des levres. Thesis. Montpellier, 1894.
49. Findlay GM. Ultraviolet light and skin cancer. Lancet 1928;215:10715.
50. Roffo AH. Cancer et soleil. Carcinomes et sarcomes provoques par l'action du soleil in toto. Bull Cancer 1934;23:590-616.
51. Roffo AH. Über die physikalische Aetiologie der Krebskrankheit. Strahlenther1939;66:328-50.
52. Beukers R, Berends W. Isolation and identification of the irradiation product of thymine. Biochim Biophys Acta 1960;41:550-1.
53. Kvam E, Tyrrell RM. Induction of oxidative DNA base damage in human skin cells by UV and near visible irradiation. Carcinogenesis 1997;18:2379-84.
54. Kripke ML, Fisher MS. Immunologic parameters of ultraviolet carcinogen. J Natl Cancer Inst 1976;57:211-5.
55. Kripke ML, Morison WL. Studies on the mechanism of systemic suppression of contact hypersensitivity by ultraviolet B radiation. Photodermatology 1986;3:4-14.
56. Armstrong BK, Kricker A. The epidemiology of UV induced skin cancer. J Photochem Photobiol B 2001;63:8-18.
57. de Gruijl FR, Sterenborg HJCM, Forbes PD, Davies RE, Cole C, Kelfkens G, *et al*. Wavelength dependence of skin cancer induction by ultraviolet irradiation of albino hairless mice. Cancer Res 1993;52:53-60.
58. Wikonkal NM, Brash DE. Ultraviolet radiation induced signature mutations in photocarcinogenesis. J Invest Dermatol Symp Proc 1999;4:6-10.
59. Vitasa BC, Taylor HR, Strickland PT, Rosenthal FS, West S, Abbey H, *et al*. Association of nonmelanoma skin cancer and actinic keratoses with cumulative solar ultraviolet exposure in Maryland watermen. Cancer 1990;65:2811-7.

60. Geies P, Wright J. Measured solar ultraviolet radiation exposure of outdoor workers in Queensland in the building and construction industry. Photochem Photobiol 2003;78:342-8.
61. Cherry N, Meyer JD, Adisesh A, Brooke R, Owen-Smith V, Swales C, *et al*. Surveillance of skin diseases: epiderma and OPRA. Br J Dermatol 2000;142:1128-34.
62. Hakansson N, Floderus B, Gustavsson P, Feychting M, Halln N. Occupational sunlight exposure and cancer incidence among Swedish construction workers. Epidemiology 2001;12:552-7.
63. Hannuxela-Svahn A, Pukkala E, Karvonen J. Basal cell skin carcinoma and other non-melanoma skin cancers in Finland from 1956 through 1995. Arch Dermatol 1999;135:781-6.
64. Anzai S, Anan T, Kay Y, Goto M, Arakawa S, Shimizu F, *et al*. Skin cancer screening on a fishing island and in an inland agriculture area of Japan. J Dermatol 2005;32:875-82.
65. Kogoj F. Cutis nautae – koža pomoraca. Pomorski zbornik 1966;4:625-42.
66. Jakac D. Haufigkeit und berufliches Charakter des Lichtkrebses in den Küstengebieten Jugoslawiens. Berufsdermatosen 1961;9:1-14.
67. Jakac D. Actinic carcinoma in the coast region of the north-east Adria. Acta Fac Med Fluminensis 1973;1:85-92.
68. Mohar N. The frequency and importance of skin carcinoma in the region of Rijeka. Acta Derm Iug 1976;3:19-31.
69. Stašić A, Lenković M, Gruber F, Grgurev Z. Frequency of skin cancer in seamen. Proceedings, I Hrvatski kongres pomorske, podvodne i hiperbarične medicine, Split, 1998:106-12.
70. Periš Z. Skin malignancy – a side effect of continuous immunosuppressive therapy. Lijec Vjesn 1988;110:15-7.

By rain and bad weather use Nivea cream; year 1935.
(from the collection of Mr. Zlatko Puntijar)