# ULTRAVIOLET RADIATION RELATED EXPOSURES: BROAD-SPECTRUM ULTRAVIOLET (UV) RADIATION, UVA, UVB, UVC, SOLAR RADIATION, AND EXPOSURE TO SUNLAMPS AND SUNBEDS

## INTRODUCTION

Solar radiation and exposure to sunbeds and sunlamps were first listed in the Ninth Edition of the Report on Carcinogen*s* as *known to be human carcinogens*. Broad-spectrum ultraviolet radiation (UVR) and its components ultraviolet A radiation (UVA), ultraviolet B radiation (UVB), and ultraviolet C radiation (UVC) were reviewed for listing in the Tenth Edition of the Report on Carcinogens. Because exposure to solar radiation and to sunbeds and sunlamps involves exposure to broad-spectrum radiation, these listings have been combined into one profile. Much of the evidence for listing the various exposures applies to more than one type of UVR. Evidence for the carcinogenicity of broad-spectrum UVR comes from studies on solar radiation and exposure to sunbeds and sunlamps. Similarly, animal and mechanistic studies to evaluate the carcinogenicity of solar radiation involve exposure to broad-spectrum UVR or its UVA, UVB, or UVC components. Use of sunbeds and sunlamps entails exposure to ultraviolet radiation. The listings for the exposures related to ultraviolet radiation are as follows:

- Broad-spectrum ultraviolet radiation is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans, which indicates a causal relationship between exposure to broad-spectrum ultraviolet radiation and human cancer.

- Solar radiation is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans, which indicates a causal relationship between exposure to solar radiation and human cancer.

- Exposure to sunbeds and sunlamps is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans, which indicates a causal relationship between exposure to sunbeds and sunlamps and human cancer.

- Ultraviolet A radiation is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals.

- Ultraviolet B radiation is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals.

- Ultraviolet C radiation is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals.

## CARCINOGENICITY

Broad-spectrum UVR, solar radiation, or exposure to sunbeds and sunlamps are *known to be human carcinogens* based on sufficient evidence of carcinogenicity from studies in humans, which indicates a causal relationship between exposure to broad-spectrum UVR, solar radiation, or sunbeds and sunlamps and human cancer. Epidemiology studies have demonstrated that exposure to broad-spectrum UVR induces skin cancer (both melanocytic and non-melanocytic). Studies of humans exposed to solar radiation, artificial devices emitting broad-spectrum UVR, or devices emitting predominantly UVA or UVB all contribute to this conclusion. Evidence for the role of the broad-spectrum UVR component of solar radiation in carcinogenicity comes from studies of human cancer associated with exposure to devices that emit artificial broad-spectrum UVR, the fact that tumors develop at the same sites both in humans exposed to sunlight and in animals exposed to broad-spectrum UVR from artificial sources, and human mechanistic studies using artificial sources of broad-spectrum UVR.

Solar radiation is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans, which indicate a cause-and-effect relationship between exposure to solar radiation and skin cancer (both cutaneous malignant melanoma and non-melanocytic skin cancer) in humans. Some studies suggest that solar radiation also may be associated with melanoma of the eye and non-Hodgkin's lymphoma (IARC 1992).

Exposure to sunlamps or sunbeds is *known to be a human carcinogen*, based on sufficient evidence of carcinogenicity from studies in humans, which indicate a cause-and-effect relationship between exposure to sunlamps or sunbeds and human cancer. Sunlamps and sunbeds emit primarily UVA and UVB radiation. Epidemiological studies have shown that exposure to sunlamps or sunbeds is associated with skin cancer (cutaneous malignant melanoma) (Autier *et al*. 1994, Swerdlow *et al*. 1988, Walter *et al.* 1990, 1999, Westerdahl *et al*. 1994, 2000, Chen *et al.* 1998). The longer the exposure, the greater the effects, which are especially pronounced in people younger than 30 and people who have been sunburned. Malignant melanoma of the eye also is associated with use of sunlamps. There is little support for an association between exposure to sunlamps or sunbeds and non-melanocytic skin cancer (IARC 1992).

Broad-spectrum UVR is absorbed by DNA and causes direct and indirect DNA damage with the potential to result in mutations, as demonstrated by mechanistic studies using human tissue. Mutations found in the *p53* tumor suppressor gene of human skin cancer are specific for broad spectrum UVR-induced damage.

The findings in humans are supported by evidence in experimental animals. Exposure to broad-spectrum UVR induces skin tumors (papilloma and squamous cell carcinoma) and eye tumors (spindle-cell sarcoma) in albino rats and skin tumors (fibrosarcoma and/or squamous-cell carcinoma) in mice, hamsters, and opossums.

Results from epidemiological studies on the effects of sunlight or artificial broad-spectrum UVR cannot be subdivided into effects specifically for UVA, UVB, or UVC. However, information regarding the specific effects of UVA, UVB, and UVC can be inferred from the results of human epidemiology studies of mixed broad-spectrum UVR exposure together with the results of studies on the effects of specific broad-spectrum UVR components in experimental animals and human tissues.

UVA is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of

carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals. Studies in which UVA was the major portion of UVR exposure (solar radiation and UVA-emitting sunbeds) have demonstrated an excess of skin cancer. Westerdahl *et al*. (2000) reported an association between melanoma and exposure to sunbeds when most of the exposures studied were from sunbeds emitting mainly UVA (with 0.1% to 2.1% UVB). The finding in humans is supported by evidence in experimental animals; exposure to UVA induced skin tumors in mice (squamous-cell carcinoma and papilloma) and fish (melanoma).

UVB is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans and sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals. Mechanistic studies in humans have demonstrated that the UVB component in solar radiation is responsible for the mutagenic photoproducts that lead to the signature *p53* mutations observed in human skin cancer. However, epidemiologic studies linking these exposures to skin cancer are limited because they lack information on the specific wavelengths of UVR to which the individuals were exposed. Although increased skin cancer is clearly associated with exposure to UVB, as a component of solar radiation or from sunlamps used before the early 1970s, these human exposures also involved other components of broad-spectrum UVR; therefore, the studies cannot eliminate these other confounding components. One study found an association between exposure to sunlamps used in the early 1970s, which produced significant amounts of UVB (22% to 40%), and cutaneous malignant melanoma (Chen *et al*. 1998). The finding in humans is supported by evidence in experimental animals. Prolonged exposure to devices emitting primarily UVB caused skin tumors in rats (papilloma), mice (squamous-cell carcinoma, fibrosarcoma, papilloma, and keratoacanthoma), guinea pigs (fibroma and trichofolliculoma), and opossums (melanocytic hyperplasia and melanoma).

UVC is *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from human mechanistic studies and sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors in multiple species of experimental animals. Studies of human tissue have demonstrated that both *in vivo* and *in vitro* exposure to UVC causes DNA damage. UVC is absorbed by DNA and induces mutagenic photoproducts that cause damage similar to that caused by UVB; however, no epidemiological studies have adequately evaluated UVC carcinogenicity in humans. UVC is absorbed by the ozone layer and does not contribute to solar exposure, and studies using artificial devices emitting UVC also emitted wavelengths of other components of broad-spectrum UVR. Exposure to high doses of radiation from devices emitting primarily UVC caused skin tumors in rats (keratoacanthoma-like skin tumors) and mice (squamous-cell carcinoma and fibrosarcoma) (IARC 1992).

## ADDITIONAL INFORMATION RELEVANT TO CARCINOGENESIS OR POSSIBLE MECHANISMS OF CARCINOGENESIS

Broad-spectrum UVR causes skin cancers via DNA damage, immunosuppression, tumor promotion, and mutations in the *p53* tumor suppressor gene. Broad-spectrum UVR induces mutations in cultured human cells; the type of damage depends on the specific wavelength applied and whether the affected cells can repair the damage without

error. DNA is a major cellular chromophore absorbing broad-spectrum UVR (mainly UVB and UVC), yielding intermediates that react with free radicals and various photoproducts with mutagenic potential. UVB causes the following four major DNA base modifications in humans: cyclobutane-type pyrimidine dimers, (6-4) photoproducts, the corresponding Dewar isomers, and thymine glycols. Both UVA and UVB induce 8-hydroxydeoxyguanosine production from guanosine by the action of singlet oxygen (Griffiths *et al.* 1998).

UVA, UVB, and UVC as individual components of broad-spectrum UVR are genotoxic in several *in vitro* test systems, including prokaryotes, lower eukaryotes, non-human mammalian cells, and human cells. Moreover, exposure to each of the three components of broad-spectrum UVR causes DNA damage in humans. UVA's biological effects are indirect and largely the result of energy transferred through reactive oxygen intermediates, whereas UVB and UVC are absorbed by DNA and directly damage DNA through base modifications. Based on the number of positive genotoxic studies, UVC is the strongest and UVA the weakest genotoxin of the components of broad-spectrum UVR.

More than 90% of human squamous-cell carcinomas contain mutations of the *p53* tumor suppressor gene. These mutations were found in 74% of sun-exposed normal human skin and only 5% of unexposed skin, indicating a strong association with sun exposure. Observed *p53* gene mutations were most frequently C to T or CC to TT transitions at pyrimidine–pyrimidine sequences. These specific *p53* mutations now are considered a signature of broad-spectrum UVR carcinogenesis (Brash *et al.* 1991, Griffiths *et al.* 1998, Wikonkal and Brash 1999, Zeigler *et al.* 1993).

Exposure to solar radiation and broad-spectrum UVR alters immune function in humans and experimental animals. Evidence that immunosuppression is related to skin cancer comes from the following observations: (1) immunosuppressed organ transplant recipients showed a marked increase in skin cancer, particularly squamous-cell carcinoma, (2) broad-spectrum UVR decreased the ability to mount a delayed-type hypersensitivity response, and (3) mice exposed to low levels of broad-spectrum UVR failed to reject highly immunogenic tumor cell lines.

Exposure of human skin grafts on mice to UVB radiation after pretreatment with the carcinogen dimethylbenz(*a*)anthracene yields human skin tumors (squamous-cell carcinoma, actinic keratoses, melanocytic hyperplasia, and melanoma). Exposure of human skin grafts on mice to UVB alone yields pre-cancerous lesions (melanocytic hyperplasia).

## PROPERTIES

Solar radiation includes most of the electromagnetic spectrum (IARC 1992). Of the bands within the optical radiation spectrum, UVR is the strongest and most damaging to living things. UVR is divided into UVA, UVB, and UVC. UVA is the most abundant of the three, constituting 95% of the solar UV energy reaching the equator, with UVB accounting for the other 5%. The short-wavelength UVC rays are absorbed by ozone, molecular oxygen, and water vapor in the upper atmosphere, so measurable amounts of UVC from solar radiation do not reach the earth's surface (Farmer and Naylor 1996).

Molecules that absorb UV and visible light (photoreactive molecules) contain segments that react with light (called chromophores), in which photons of light excite electrons from the ground state to higher energy states. These molecules then generally

re-emit light on returning to lower energy or ground states (Dyer 1965). Molecules sensitive to UV light absorb and emit different wavelengths of UV light.

Photochemical and photobiological interactions occur when photons react with a photoreactive molecule, forming either a photochemically altered molecule or two separate molecules (Phillips 1983, Smith 1989). For this reaction to occur, the photons must have enough energy to alter a photoreactive chemical bond (breaking the original bond and/or forming new bonds).

UVB is considered to be the major cause of skin cancer, despite the fact that it does not penetrate the skin as deeply as UVA or react with the outer skin layer as vigorously as UVC. Its high reactivity with macromolecules, coupled with the depth to which it penetrates skin, makes UVB the most potent portion of the UV spectrum for both short-term and long-term biological effects. UVA, while possibly not as dangerous, also induces biological damage (Farmer and Naylor 1996).

Photobiological reactions of concern for skin cancer risk due to UV light exposure are the reactions with the main chromophores of the epidermis—urocanic acid, DNA, tryptophan, tyrosine, and the melanins. DNA photoproducts include pyrimidine dimers, pyrimidine-pyrimidone (6-4) photoproducts, thymine glycols, and DNA exhibiting cytosine and purine damage and other damage, such as DNA strand breaks and cross-links and DNA-protein cross-links. The various DNA photoproducts differ in their mutagenic potential (IARC 1992).

UVR-induced DNA photoproducts produce a variety of cellular responses that contribute to skin cancer. Unrepaired DNA photoproducts may result in the release of cytokines that contribute to tumor promotion, tumor progression, immunosuppression, and the induction of latent viruses (Yarosh and Kripke 1996, IARC 1992).

## USE

Broad-spectrum UVR has many uses as a natural source of energy and is important in various biological processes. Solar radiation is required for life. Plants must have sunlight to grow and to produce carbohydrates and oxygen. Broad-spectrum UVR from solar radiation helps produce vitamin D in human skin cells. Vitamin D is absorbed by the body and then used to absorb calcium in the intestinal tract. Vitamin D is essential for the growth and development of healthy bones. Brief exposure to sunlight on a regular basis is sufficient to produce all of the vitamin D most people need. This vitamin also can be obtained from dietary sources. Artificial sources of broad-spectrum UVR have many uses, including tanning, medical diagnosis and treatment, and promotion of polymerization reactions (e.g., curing of protective coatings). Tanning beds use artificially produced UVR to enable individuals to develop “suntan” for cosmetic reasons. Originally, tanning beds were built with mercury arc lamps, which emitted large quantities of UVB and UVC. Now, sunbeds and solaria emit mostly UVA (IARC 1992).

Broad-spectrum UVR has both diagnostic and therapeutic uses in medicine and dentistry. More than 30 disorders now can be treated through UVA exposure combined with compounds called psoralens (PUVA therapy). Psoriasis and eczema are the skin diseases most frequently treated with PUVA therapy. PUVA can also be used with UVB exposure to treat psoriasis patients who are not good candidates for systemic therapy with methotrexate or etretinate (Morrison 1992). Broad-spectrum UVR, but more commonly UVB, and coal-tar creams also are used to treat psoriasis (Reid 1996). In addition, UVB

is used to convert 7-dehydrocholesterol (provitamin D3) to vitamin D in the skin of vitamin D-deficient patients.

UVA has been used to treat neonatal jaundice or hyperbilirubinemia. Although treatment typically involves irradiating the infant with visible light for several hours a day, for up to one week, one commercial neonatal phototherapy unit was found also to emit UVA , UVB and UVC radiation (IARC 1992). UVA has been found to alter the molecular structure of melatonin, a hormone that helps to regulate sleep-wake cycles, to unidentified photoproducts; therefore, moderate phototoxicity of melatonin has been predicted (Kim *et al*. 1999). Broad-spectrum UVR also has been used to detect various dental disorders, such as early dental caries, dental plaque, and calculus (IARC 1992).

Broad-spectrum UVR has many industrial applications. One of the major industrial uses involves photopolymerization, which includes curing of protective coatings and inks. Broad-spectrum UVR also is used to simulate weathering of various materials, such as polymers. It is used to sterilize and disinfect tools and materials, usually in the range of 260 to 265 nm (UVC). Other uses include UV photography and UV lasers. Broad-spectrum UVR is a by-product of electric arc welding (IARC 1992).

## SOURCES

In the broadest sense, broad-spectrum UVR is formed when something is heated or when electrons that have been raised to an excited state return to a lower energy level. Broad-spectrum UVR is naturally emitted by the sun. An estimated two-thirds of the energy emitted by the sun penetrates the atmosphere. Broad-spectrum UVR constitutes approximately 5% of the solar radiation that reaches the earth's surface.

Six artificial sources of broad-spectrum UVR have been identified: incandescent lights, gas discharge lamps, arc lamps, fluorescent lamps, metal halide lamps, and electrodeless lamps. Incandescent sources provide visible radiation in a continuous spectrum. Gas discharge lamps produce visible radiation by passing an electrical current through a gas. The type of gas present in the lamp determines emission wavelengths; at low pressures, fine lines are produced, while higher pressures create broad bands. Arc lamps are intense sources of broad-spectrum UVR and often are used to simulate solar radiation. Fluorescent lamps create radiation from a low-pressure mercury discharge, which produces a strong emission at 254 nm. This process in turn excites the phosphor-coated lamp to produce fluorescence. Various emission spectra can be obtained by altering the makeup and thickness of the phosphor and the glass envelope. Metal halide lamps add metal to a mercury discharge lamp, creating emission lines in addition to the mercury emission spectrum. Electrodeless lamps use magnetrons to generate microwave energy, which then is absorbed by the discharge tube (IARC 1992).

Sunlamps and tanning beds emit broad-spectrum UVR. The latter chiefly emit UVA (315 to 400 nm), although before the mid 1970s, lamps that emitted UVB and UVC radiation were more common (IARC 1992). However, UVB produces a better tan than does UVA, and recently, at least in the United States and United Kingdom, use of sunlamps with more UVB radiation has become widespread (Wright *et al*. 1997). Low-pressure mercury vapor lamps, sunlamps, and black-light lamps are considered to be low-intensity UV sources. High-intensity UV sources include high-pressure mercury vapor lamps, high-pressure xenon arcs, xenon-mercury arcs, plasma torches, and welding arcs. Three different UVA phosphors have been used in sunlamps sold in the United States over the past 20 years, producing emission spectra that peak at 340 nm, 350 nm, or 366 nm. Two modern U.S. sunlamps evaluated by the U.S. Food and Drug Administration (FDA) emitted 99.0% and 95.7% UVA, with the remaining radiation as UVB (< 320 nm). A new high-pressure UVA sunbed with eighteen 1600-W filtered arc lamps emitted 99.9% UVA. An older type of sunlamp, used more than 20 years ago (UVB/FS type), emitted 48.7% UVA (Miller *et al*. 1998).

## EXPOSURE

The greatest source of human exposure to broad-spectrum UVR is solar radiation; however, the exposure varies with geographical location. Information on global broad-spectrum UVR levels has been compiled from data gathered for epidemiological studies of skin cancer and other health effects, such as premature aging of the skin, cataracts, and suppression of the immune response. Despite the large number of measurements, estimating human exposure is complex. The radiation wavelengths comprising the broad-spectrum UVR to which an individual would be exposed vary considerably with latitude, altitude, time of day, and season. People also vary in their length of outdoor exposure and parts of the body exposed. In addition, the varied shapes of the human body complicate efforts to estimate human exposure. Although broad-spectrum UVR levels were estimated for many studies, few studies were able to distinguish between UVA, UVB, and UVC (IARC 1992).

Various factors influence terrestrial levels of UVA (i.e., levels found at the earth's surface). UVA levels decrease with increasing distance from the equator and increase with increasing altitude (decreasing with distance below sea level). Terrestrial UVA levels also are decreased by stratospheric ozone, which varies with latitude and season. When there is less ozone, more UVA will reach the earth's surface. Time of day also influences daily UVA levels. Clouds reduce the amount of UVA reaching ground level. Air pollution, including tropospheric ozone, can decrease UVA exposure, especially in urban areas. Surface reflection also contributes to personal exposures to UVA (IARC 1992).

Terrestrial UVB levels are affected by the same factors that influence terrestrial UVA levels; however, because UVB is absorbed more by stratospheric ozone than is UVA, differences in latitude and altitude affect UVB exposure more than UVA exposure. Seasonal changes affect UVB levels, mostly in temperate regions. Time of day at a given latitude also affects UVB levels, as do changes in stratospheric ozone with latitude and season. Air pollution decreases UVB exposure, and clouds also affect UVB levels. Generally, cloud cover scatters less than 10% of the UVB under a clear sky; however, very heavy cloud cover virtually eliminates UVB, even in the summer. Surface reflection contributes to human UVB exposure. Exposure due to reflection is important, because body parts normally shaded are exposed to reflected radiation (IARC 1992).

Most bulbs sold in the United States for use in sunbeds emit "substantial doses of both UVB and UVA" (Swerdlow and Weinstock 1998). Many of the home and salon devices in the 1980s emitted both UVA and UVB radiation, but current devices emit predominantly UVA (FTC 1997, Sikes 1998).

FDA scientists calculated that commonly used fluorescent sunlamps would deliver 0.3 to 1.2 times the annual UVA dose from the sun to a typical tanner requiring 20 sessions at 2 minimal erythemal doses (MED) per session. The common sunlamps would deliver to a frequent tanner (100 sessions at 4 MED/session) 1.2 to 4.7 times the UVA received annually from solar radiation. The recently available high-pressure sunlamps would deliver 12 times the annual solar UVA dose to the frequent tanner (Miller *et al*. 1998).

In 1987, an American Academy of Dermatology survey found that although 96% of the U.S. population surveyed knew that sun exposure causes cancer, one-third of the adults who responded to the survey said they develop tans. By 1987, the indoor tanning industry was one of the fastest growing industries in the United States (Sikes 1998). New York State alone was estimated to have 1,300 commercial tanning facilities in 1993 (Lillquist *et al*. 1994). By 1995, indoor tanning facilities were a $1 billion industry serving one million patrons a day (Guttman 1995). More recent estimates indicate approximately one to two million patrons visit tanning facilities as often as 100 times per year (Sikes 1998).

Up to 25 million persons per year in North America are estimated to use sunbeds. Teenagers and young adults are prominent among users. A study of high school students in St. Paul, Minnesota, found that 34% had used commercial sunbeds at least four times in the past year, and 59% of the users reported some skin injury. A 1995 U.S. survey found that of commercial tanning salon patrons, 8% were 16 to 19 years old, and 42% were 20 to 29 years old, and 71% were female (Swerdlow and Weinstock 1998).

Occupational exposure to solar broad-spectrum UVR occurs for anyone working outside (for example, agricultural, construction, and road work laborers). For a group of

more than 800 outdoor workers in the United States at 39° N latitude (the latitude of Philadelphia), personal annual exposure doses for the facial area were estimated at 30 to 200 MED (Rosenthal *et al*. 1991). However, this estimate may be low because Rosenthal and colleagues assumed facial exposure to be only 5% to 10% of ambient exposure, whereas other data suggested that it could be as high as 20%. Using this higher estimate, the annual facial exposure doses for these outdoor workers would be 80 to 500 MED (IARC 1992).

Occupational exposure to artificial broad-spectrum UVR occurs in industrial photo processes, principally UV curing of polymer inks, coatings, and circuit board photoresists; sterilization and disinfection; quality assurance in the food industry; medical and dental practices; and welding (IARC 1992). UV lasers, such as those used in cornea shaping and coronary angioplasty, are another potential source of occupational exposure, with relative risks that could be comparable to risks for individuals in outdoor professions (Sterenborg *et al*. 1991). Electric arc welders are the largest occupational group with exposure to artificial broad-spectrum UVR. More than 500,000 welders in the United States are estimated to have been occupationally exposed to broad-spectrum UVR. Occupational exposure to artificial broad-spectrum UVR depends on both the source of exposure and the protective methods used to decrease exposure. Some artificial broad-spectrum UVR sources are self-contained, such as germicidal lamps in some uses, and present no risk to workers. Other occupational uses, such as use of UVR in laboratories, UV photography, and UV lasers, inevitably lead to broad-spectrum UVR exposure in which short-term and intense exposures may occur (IARC 1992).

## REGULATIONS

### Solar Radiation

No regulations were found pertaining to solar radiation.

### Broad-Spectrum UV Radiation

The FDA regulates UVR, establishing safe uses for irradiation in the production, processing, and handling of food. The FDA also sets forth labeling requirements for drugs containing coal tars for use with UVR. The FDA regulates various devices that emit UVR, such as sunlamps, sunbeds, medical lamps, and purifiers.

The Occupational Safety and Health Administration (OSHA) regulates UVR exposure among welders and cutters; regulations cover safety precautions, guidelines, and treatment.

### Exposure to Sunlamps or Sunbeds

The FDA Center for Devices and Radiological Health (CDRH) has promulgated regulations concerning sunlamp products and UV lamps intended for use in sunlamp products. Manufacturers must notify CDRH of product defects and repair and replacement of defects. CDRH issues written notices and warnings in cases of noncompliance. Several performance requirements must be met by sunlamp products (21 CFR 1040.20), including irradiance ratio limits, a timer system, protective eyewear to be worn during product use, compatibility of lamps, and specific labels. The label should include the statement “DANGER—Ultraviolet radiation” and warn of the dangers of exposure and overexposure.

OSHA requires extensive UVR protective measures for employees engaged in or working adjacent to arc welding processes. Arc welding emits broad-spectrum UVR. Workers should be protected from the UVR by screening, shields, or goggles. Employees in the vicinity of arc welding and cutting operations should be separated from them by shields, screens, curtains, or goggles. If possible, welders should be enclosed in individual booths. Production of UVR by inert-gas metal-arc welding is 5 to 30 times more intense than that by shielded metal-arc welding. OSHA-required protective measures in shipyard employment and marine terminals include filter lens goggles worn under welding helmets or hand shields and protective clothing that completely covers the skin to prevent UVR burns and other damage.

The American Conference of Governmental Industiral Hygienists (ACGIH) has set various threshold limit values for skin and eye exposures. TLVs for occupational exposure are determined by these parameters:

1. "For the near UV spectral region (320 to 400 nm), total irradiance incident upon the unprotected eye should not exceed 1.0 $mW/cm^2$ for periods greater than $10^3$ seconds (approximately 16 minutes) and for exposure times less than $10^3$ seconds should not exceed 1.0 $J/cm^2$."
2. Unprotected eye or skin exposure to UVR should not exceed 250 $mJ/cm^2$ (180 nm) to $1.0x10^5$ $mJ/cm^2$ (400 nm) for an 8-hour period. The TLVs in the wavelength range 235 to 300 nm are 3.0 (at 270 nm) to 10 $mJ/cm^2$.
3. Effective irradiance for broad-band sources must be determined with a weighting formula.
4. "For most white-light sources and all open arcs, the weighting of spectral irradiance between 200 and 315 nm should suffice to determine the effective irradiance. Only specialized UV sources designed to emit UV-A radiation would normally require spectral weighting from 315 to 400 nm."
5. The permissible UVR exposure for unprotected eye and skin exposure may range from 0.1 $\mu W/cm^2$ (8 hours/day) to 30,000 $\mu W/cm^2$ (0.1 sec/day).
6. "All of the preceding TLVs for UV energy apply to sources which subtend an angle less than 80°. Sources which subtend a greater angle need to be measured only over an angle of 80°."

The ACGIH added that even though conditioned (tanned) individuals may not be any more protected from skin cancer, they can tolerate skin exposure in excess of the TLV without erythemal effects. The National Institute for Occupational Safety and Health criteria for a recommended standard for occupational exposure to UVR are practically identical to those given in ACGIH items 1 and 2 above.

The Federal Trade Commission investigates false, misleading, and deceptive advertising claims about sunlamps and tanning devices.

The American Medical Association passed a resolution in December 1994 that called for a ban of the use of suntan parlor equipment for nonmedical purposes. Dermatologists have urged the FDA to take action to discourage use of suntan parlors and suntan beds. Currently, the FDA CDRH and the Centers for Disease Control and Prevention encourage avoidance of sunlamps and sunbeds. Although 27 states and municipalities had promulgated some regulations on indoor tanning facilities by late 1995, they seldom are enforced. The American Academy of Dermatology's Tanning Parlor Initiative provides a manual giving examples of regulatory legislation and instructions on petitioning state, regional, and local governments on this issue.

**UVA, UVB, and UVC**

No regulations were found for the specific wavelengths of UVA, UVB, or UVC. All regulations are generalized under broad-spectrum UVR. Regulations are summarized in Volume II, Table 183.

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