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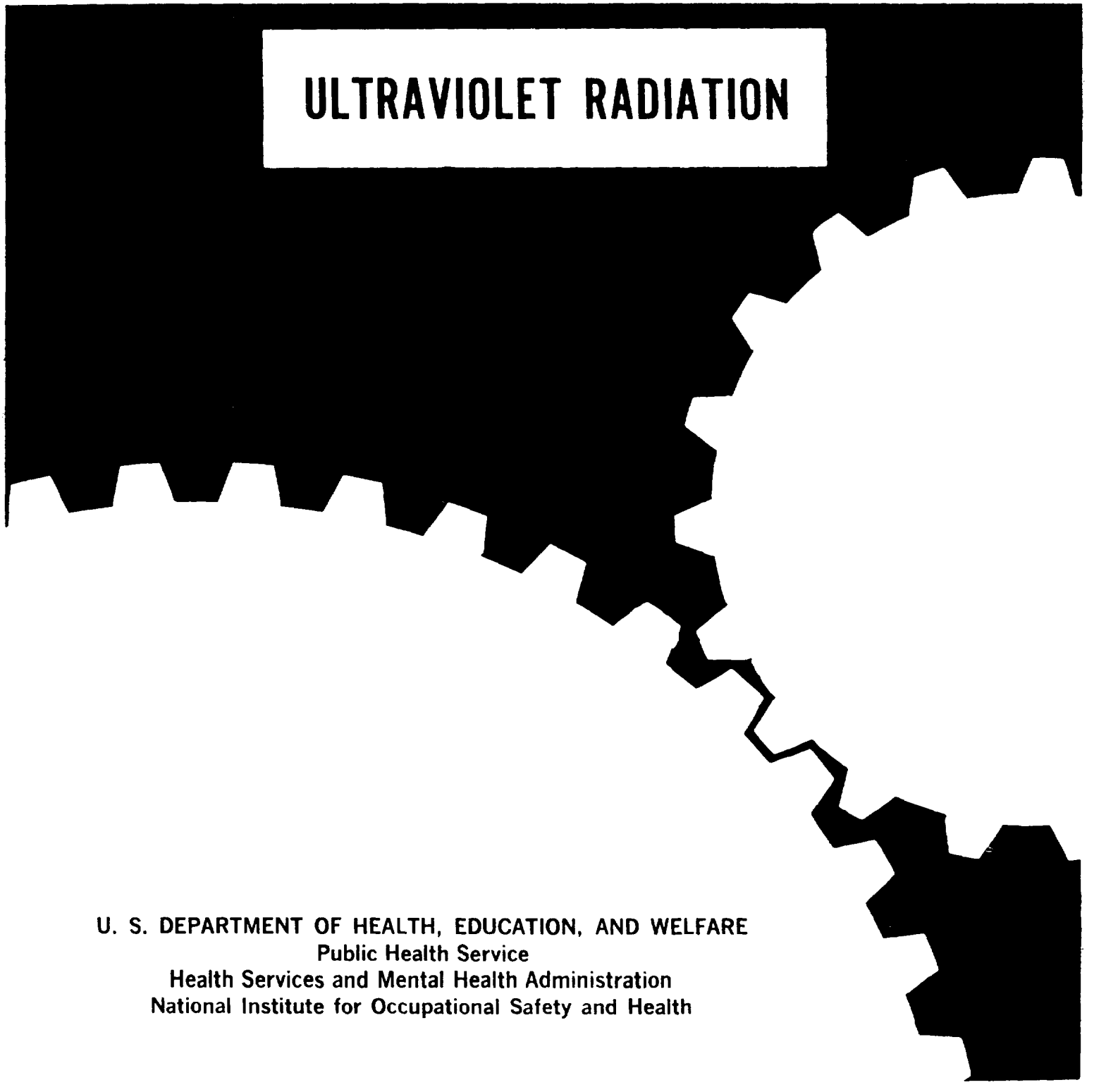

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Health Services and Mental Health Administration
National Institute for Occupational Safety and Health

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OCCUPATIONAL EXPOSURE TO ULTRAVIOLET RADIATION

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Health Services and Mental Health Administration
National Institute for Occupational Safety and Health

1972

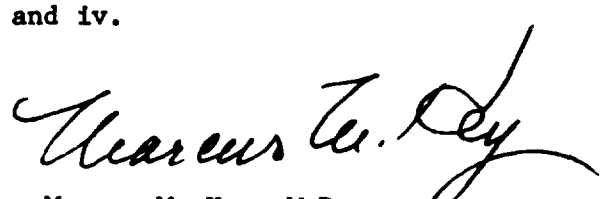
HSM 73-11009

PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. To provide relevant data from which valid criteria and effective standards can be deduced, the National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the workers.

I am pleased to acknowledge the contributions to this report on ultraviolet radiation by my staff and the valuable, constructive comments by the Review Consultants on Ultraviolet Radiation, an ad hoc committee of the American Industrial Hygiene Association, and the American Medical Association Committee on Occupational Toxicology. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on ultraviolet radiation. A list of the NIOSH Review Committee members and of the Review Consultants appears on page iii and iv.



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The Office of Research and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for ultraviolet radiation. The Franklin Institute Research Laboratories developed the basic information for consideration by NIOSH staff and consultants under contract No. HSM-049-71-36. Robert E. Seiter served as criteria manager.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR ULTRAVIOLET RADIATION

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I. RECOMMENDATIONS FOR AN ULTRAVIOLET RADIATION STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that occupational exposure to ultraviolet energy in the workplace be controlled by compliance with the following sections. Ultraviolet radiation (ultraviolet energy) is defined as that portion of the electromagnetic spectrum described by wavelengths from 200 to 400 nm. (For additional definitions and conversion factors, see Appendix II.) Adherence to the recommended standards will, it is believed, prevent occupational injury from ultraviolet radiation, that is, will prevent adverse acute and chronic cutaneous and ocular changes precipitated or aggravated by occupational exposure to ultraviolet radiation.

Sufficient technology exists to prevent adverse effects on workers, but technology to measure ultraviolet energy for compliance with the recommended standard is not now adequate, so work practices are recommended for control of exposure in cases where sufficient measurement or emission data are not available.

These criteria and the recommended standard will be reviewed and revised when relevant information warrants.

Section 1 - Exposure Standards

(a) For the ultraviolet spectral region of 315 to 400 nm, total irradiance incident on unprotected skin or eyes, based on either measurement data or on output data, shall not exceed 1.0 mW/cm^2 for periods greater than 1000 seconds, and for exposure times of 1000 seconds or less the total radiant energy shall not exceed 1000 $mW \cdot sec/cm^2$ (1.0 J/cm^2).

(b) For the ultraviolet spectral region of 200 to 315 nm, total irradiance incident on unprotected skin or eyes, based on either measurement data or on output data, shall not exceed levels described below.

Measurement techniques are discussed in Appendix I.

(1) If the ultraviolet energy is from a narrow-band or monochromatic source, permissible dose levels for a daily 8-hour period can be read directly from Figure I-1, or, for selected wavelengths, from Table I-1.

(2) If the ultraviolet energy is from a broad-band source, the effective irradiance (I_{eff}) relative to a 270-nm monochromatic source shall be calculated from the formula below. From I_{eff}, the permissible exposure time in seconds for unprotected skin or eyes shall be computed by dividing 0.003 J/cm^2, the permissible dose of 270-nm radiation, by I_{eff} in W/cm^2.

$$I_{eff} = \sum I_\lambda S_\lambda \Delta\lambda$$

where I_{eff} = effective irradiance relative to a monochromatic source at 270 nm.

I_λ = spectral irradiance in $W/cm^2/nm$.

S_λ = relative spectral effectiveness (unitless); see Table I-1 for values of S_λ at different wavelengths.

Δ_λ = band width in nm.

Table I-2 lists permissible exposure times corresponding to selected values of I_{eff} in $\mu W/cm^2$.

If radiation intensity from a point source is known at some distance from the worker, for example, from measurement at another point or from output data at a known distance from the ultraviolet source, attenuation of radiation from that point to the worker can be calculated from the principle that radiation decreases with the square of the distance it must travel. For

example, an object 3 feet away from a radiation source receives 1/9 the energy of an object 1 foot away. This assumption is conservative in some instances, since ultraviolet radiation, especially at very low wavelengths, may be absorbed by some components of the atmosphere. Where information on atmospheric absorption of ultraviolet radiation is known, further correction may be applied. The calculation of intensity of radiation at any given point by use of the inverse square formula explained above does not take into consideration reflected energy.

The recommended standard is not proposed for application as a standard to lasers. It should be recognized that significant non-occupational exposure to ultraviolet radiation can occur from exposure to sunlight, particularly during the summer months.

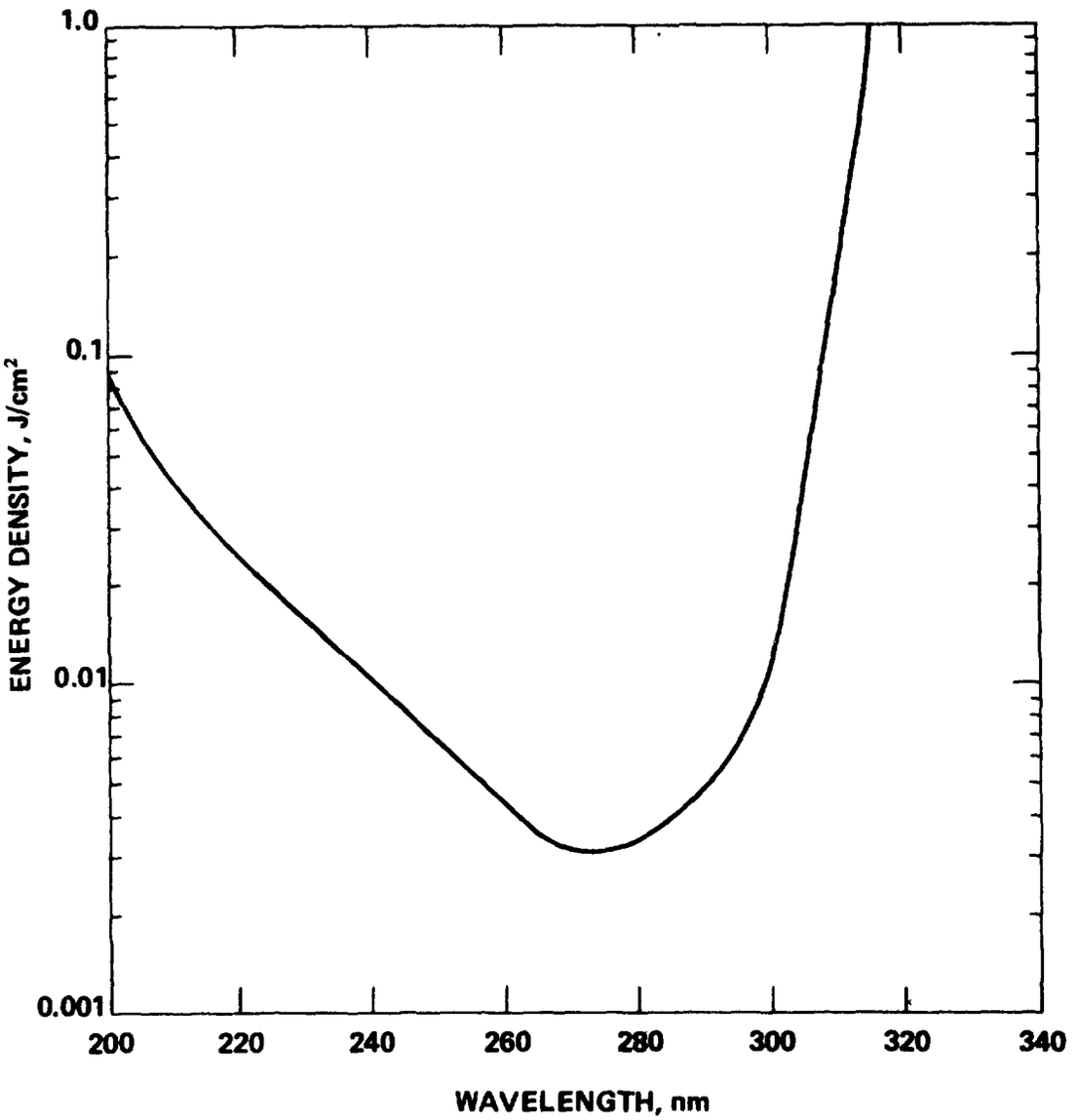

Figure I-1. Recommended Ultraviolet Radiation Exposure Standard
This figure was adapted from a figure developed and published by the American Conference of Governmental Industrial Hygienists in "Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1972".

Table I-1

Total Permissible 8-Hour Doses and
Relative Spectral Effectiveness of Some
Selected Monochromatic Wavelengths

Wavelength (nm)	Permissible 8-hour dose (mJ/cm^2)	Relative spectral effectiveness ($S\lambda$)
200	100.0	0.03
210	40.0	0.075
220	25.0	0.12
230	16.0	0.19
240	10.0	0.30
250	7.0	0.43
254	6.0	0.50
260	4.6	0.65
270	3.0	1.00
280	3.4	0.88
290	4.7	0.64
300	10.0	0.30
305	50.0	0.06
310	200.0	0.015
315	1000.0	0.003

This table was adapted from a table developed and published by the American Conference of Governmental Industrial Hygienists in "Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1972".

Table I-2

Maximum Permissible Exposure Times
for Selected Values of I_{eff}

Duration of exposure per day	Effective irradiance, I_{eff} ($\mu W/cm^2$)
8 hrs	0.1
4 hrs	0.2
2 hrs	0.4
1 hr	0.8
30 min.	1.7
15 min.	3.3
10 min.	5.0
5 min.	10.0
1 min.	50.0
30 sec.	100.0

This table was adapted from a table developed and published by the American Conference of Governmental Industrial Hygienists in "Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1972".

Section 2 - Medical Recommendations

(a) The worker's past medical history should be obtained to determine if the worker suffers from any condition that is exacerbated or aggravated by exposure to sunlight.

(b) A worker who gives a history of such a condition should not be permitted to work in an area exposed to ultraviolet radiation.

(c) The worker should be advised that any blemish that appears on skin exposed to long term ultraviolet radiation should be examined by a physician.

Section 3 - Apprisal of Employees of Hazards From Exposure to Ultraviolet Energy.

(a) Each employee who may be exposed to high intensity artificial sources of ultraviolet energy shall be apprised of all hazards, relevant symptoms and precautions concerning exposure. This apprisal of hazards shall include:

(1) Information as to the proper eye protection and protective clothing to be used.

(2) Instruction on how to recognize the symptoms of eye and skin damage due to ultraviolet radiation.

(3) Information as to special caution that shall be exercised in situations where employees are exposed to toxic agents and/or other stressful physical agents which may be present in addition to and simultaneously with ultraviolet radiation.

(b) Highly susceptible (i.e. light skinned, easily sunburned) employees who regularly work out of doors and are exposed to sunlight should be apprised of possible long term effects of sun exposure and of the desirability of preventing these effects by use of protective clothing or sunscreens.

Section 4 - Labeling

All sources, work areas, and housings specified in Table I-3 shall carry the following warning:

CAUTION

HIGH INTENSITY ULTRAVIOLET ENERGY

PROTECT EYES AND SKIN

Table I-3

Radiation Source	Lamp or Instrument	Housing	Work Area	Container (Shipping or Storage)
1. Low Pressure Mercury	Yes	Yes	No	Yes
2. Sunlamp	Yes	No	No	Yes
3. Black light lamp	No	No	No	No
4. Pressure Type Arc lamps*	No	Yes	Yes	Yes
5. Open Arcs* and Incandescent Sources	No	Yes	Yes	Yes
6. Welding	Yes	—	Yes	Yes
7. Plasma Torches	Yes	Yes	Yes	Yes
8. Other artificial UV generating sources	Yes	Yes	Yes	Yes

* Lamps cannot be labeled because of their high operating temperatures.

Section 5 - Work Practices

Worker exposure to ultraviolet energy from 200 to 400 nm shall be controlled by adherence to the standard set forth in Section 1 or the preventive procedures described in this Section, as applicable. Compliance with the standard, based on measurement data or emission data, or adherence to the work practice procedures will protect against injury from ultraviolet energy.

Exposure to ultraviolet energy can be controlled by enclosures, shields, protective clothing, skin creams, gloves, goggles, or face shields. Workers shall be protected from eye or skin exposure to ultraviolet radiation.

Specific protective measures to be used for various types of ultraviolet exposure are noted below.

(a) Sunlight. Susceptible persons working outside in strong sunlight should be protected. Protective clothing, such as long-sleeved shirts, trousers or skirt, and face and neck protection will normally be adequate. Face and neck protection can be afforded by a broad-brimmed hat, by a billed hat or cap, or by a neck shield (if the neck is not protected by hair). Hard hats may have bills or face shields to protect the face, and may have neck shields. Alternatively, face and eye protection can be achieved by barrier creams and goggles or spectacles.

(b) Low-intensity ultraviolet sources. Examples of sources of low-intensity ultraviolet sources are low-pressure mercury vapor lamps, sunlamps, and black-light lamps.

Glass or plastic (1/8-inch thickness or greater) spectacles, goggles or shields provide adequate eye protection. Skin can be protected by light-weight clothing, by absorbing skin creams containing benzophenones or p-aminobenzoic acid, or by barrier creams containing titanium dioxide

or zinc oxide.

(c) High-intensity ultraviolet sources. Examples of high-intensity ultraviolet sources are high-pressure mercury vapor lamps, high-pressure xenon arcs, xenon-mercury arcs, carbon arcs, plasma torches, and welding arcs.

For eye protection, workers shall wear goggles, face shields or masks. For shade required for this eye protection, consult Section 7 of American National Standards Institute Z49.1-1967 (ANSI Z49.1). However, in some welding operations such as gas-shielded arc welding, workers with inadequate visual acuity may have to wear a shade of less absorbance (greater transmission) to facilitate their locating the electrodes and prevent starting the arc before putting their masks or goggles in place; eye protection must be used at all times while the arc is operating, and, if necessary in order to see the operation, shade 8 may be used in place of a shade of greater absorbance.

Skin must also be protected. Clothing of densely woven flannelette, poplin, or synthetic fabric will give sufficient protection. Facial skin can be protected by face shields of shades specified in ANSI Z49.1 or by barrier creams containing titanium dioxide or zinc oxide.

Because many synthetic clothing fibers can melt or catch fire and thereby cause severe thermal burns, clothing of synthetic fibers should be flame-resistant if operations involve great heat, sparks, or flame.

Welders' helpers and others working nearby may also require protection. Shielding such as the welder's booth guard against accidental exposure

of other people. Reflection from lamp housings, walls, ceilings, and other possible reflective surfaces should be kept to a minimum by coating such surfaces with a pigment-based paint of low ultraviolet reflectance. Where such shielding and non-reflective surfaces are not used, welders' helpers and others near the welding operation should wear protective clothing, skin creams, gloves, goggles, or face shields.

Additional hazards. There are other hazards from some ultraviolet sources that must also be prevented. There is a shock hazard in some operations involving arcs, because of the high starting voltages required; wiring and connections must be adequately insulated, and persons handling the equipment must wear gloves and face shields. There must be adequate ventilation to prevent build-up of ozone and oxides of nitrogen. There may also be an explosion hazard from some ultraviolet operations, and the wearing of gloves and face shields will reduce the consequences of an explosion.

Arc welding on plates wet with unsaturated chlorinated hydrocarbons (perchloroethylene and trichloroethylene) must be avoided unless well vented, because of possible production of phosgene and hydrogen chloride.

Section 6 - Recordkeeping

Because measurement of exposure of workers to ultraviolet energy is not required, records are not required.

II. INTRODUCTION

This report presents the criteria and recommended standard based thereon which were prepared to meet the need for preventing impairment of health from occupational exposure to ultraviolet radiation. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to ". . . develop criteria dealing with toxic materials and harmful physical agents and substances which will describe . . . exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health formalized a system for the development of criteria upon which standards can be established to protect the health and safety of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any criteria for a recommended standard should enable management and labor to develop better engineering controls and more healthful work practices and should not be used as a final goal.

The standard proposed is based on the results of numerous investigations of the effects of ultraviolet energy on skin and eyes, and is based on the eye as the most sensitive organ, so that protection of the eyes should result in a significant safety factor for the skin. Additionally, because of variations in pigmentation, skin thickness, normal clothing styles, and,

in the case of outdoor exposures, in insolation, an additional safety factor for skin protection is afforded many workers.

Prevention of the acute effects of ultraviolet radiation on skin and eyes should provide protection from chronic effects such as cataracts or skin cancer. However, it is believed more research into chronic effects of ultraviolet energy on skin and eyes is needed.

Because of the present difficulties in measurement of broadband ultraviolet energy pointed out in this document, evaluation for compliance is based on three different approaches: (1) utilization of available instrumentation wherever applicable with recognition of instrument shortcomings; (2) utilization of data on energy output from a specific source, such as lamps; and (3) utilization of the work practices when suitable instrumentation or energy output data are not available.

These criteria and recommended standard will be subject to review and will be revised when appropriate.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

Occupational exposure to ultraviolet radiation occurs from both natural and artificial generation of ultraviolet. The sun is the principal natural source. Artificial sources either produce ultraviolet as a byproduct, or are designed to generate ultraviolet to utilize its properties. Some industrial processes in which ultraviolet energy is a byproduct are welding, plasma torch operations, photoelectric scanning, and hot metal operations. Because of the germicidal properties of certain portions of the ultraviolet spectrum, artificial sources are used in hospitals, biological laboratories, schools, and in industry. Other common applications are illumination; advertising; crime detection; chemical synthesis and analysis; photoengraving; food, water, and air sterilization; vitamin production; and medical diagnosis. Many of these occupations are listed in Table X-1.[1] New sources, such as ultraviolet lasers and fluorescent panels, are being developed.

Table X-2 shows the best available estimate of the number of workers with industrial exposure to artificial sources of ultraviolet radiation.

Historical Reports

The light-induced, acute inflammatory reaction of the eye has been known since early times, as indicated by Xenophon's mention of "snow-blindness" in his treatise Anabasis (ca. 375 BC), quoted by Duke-Elder.[2] Although more energetic than the visible portion of the electromagnetic

spectrum, most ultraviolet radiation is not detected by the visual receptors in mammals, including man. Thus, exposure to ultraviolet may result in ocular damage without the subject's being aware of the potential danger. Cases of keratinization of the cornea and cataracts of the lens have been observed since the early part of this century[2,3] from ultraviolet radiation levels associated with welding arcs, high-pressure pulsed lamps, and reflection of solar radiation from snow, desert and water.

Effects on Humans

Reviews of the literature on the biologic effects of ultraviolet radiation have been compiled by Verhoeff et al.,[4] Buchanan et al.,[5] Christner et al.,[6] and Duke-Elder.[2] Verhoeff and his colleagues included extensive research data in their report and formulated some of the basic hypotheses regarding ocular damage by ultraviolet radiation.

The International Commission on Illumination[7] has separated the ultraviolet spectrum into 3 different wavelength bands, 315 to 400 nm, 280 to 315 nm, and 200 to 280 nm, for convenience in classification. These ranges, with slight variations, are also referred to as near, midrange, and far ultraviolet, respectively. Wavelengths below 200 nm are of little biological significance since radiation in this region (vacuum ultraviolet) is absorbed in very short pathlengths in air with associated production of ozone.[8] Ozone is produced principally at wavelengths less than 220 nm.

1. Effects on eyes

Ordinary clinical photokeratitis has been described by Pitts and Gibbons[9] as characterized by a period of latency that tends to vary inversely with the severity of exposure. The latent period may be

as short as 30 minutes or as long as 24 hours, but is usually 6 to 12 hours. Conjunctivitis follows, often accompanied by erythema of the facial skin surrounding the eyelids. There is a sensation of a foreign body or "sand" in the eyes and varying degrees of photophobia, lacrimation, and blepharospasm. These acute symptoms usually last from 6 to 24 hours with nearly all discomfort disappearing within 48 hours. The individual is visually incapacitated for varying periods of time. It is important to note that the ocular system, unlike the skin, does not develop tolerance to repeated ultraviolet exposure.[9]

Quantitative dose-response studies on eyes have been conducted in man and animals, and the two approaches have complemented each other; some of the following comments on effects on the human eye are amplified and compared with studies on animals in the section on Animal Toxicity.

Pitts and Tredici[10] studied threshold intensities for production of photokeratitis. From animal studies, they predicted maximal sensitivity of humans to occur at 280 nm and exposed a few humans at this wavelength. From the limited results, they estimated a threshold at 280 nm of 0.05×10^{6} ergs/cm^{2}; however, while there were no symptoms reported until the light intensity was about 15% greater than this threshold, there was a reduction in visual acuity to as much as 20/40 at the "threshold". They concluded that ultraviolet induced photokeratitis is insidious and incapacitating. Most symptoms of photokeratitis did not appear for about 4 to 12 hours; it took about

8 hours for visual incapacitation to be evident.

A later report by Pitts and Gibbons[9] showed that the human threshold of response was similar to that of rabbits and primates at 260 nm and longer, while at 250 nm and shorter the human was more sensitive than animals. At 270 nm the human threshold was 0.04×10^6 ergs/cm^2.

As a result of observations at above-threshold intensities, it was felt that the reaction of the cornea to wavebands from 220 to 250 nm was different from those found with exposures from 250 to 310 nm. For exposures below 250 nm, signs and symptoms occurred soon after exposure, and subjective symptoms always returned to normal prior to completion of the experiment, approximately 14 hours later. For exposures above 250 nm, symptoms did not occur until late in the experiment, generally 9 to 11 hours after exposure, and visual acuity remained below normal for 24 hours after exposure. The observed differences were attributed to the difference in the absorption of the different wavebands. The lower wavebands were absorbed in the outer corneal epithelial layers and underwent rapid change whereas the higher wavebands were absorbed in the deeper epithelial layers and showed delayed changes because these cells were more viable. Thus, the response at shorter wavelengths was rapidly revised while at the longer wavelengths there was a delayed and more serious response.

Kinsey et al.[11] studied the production of eye damage from arc-produced ultraviolet radiation and Rieke[12] considered it to account for 40% of all injuries in engineering shops. Grim and Kusnetz[13] reported

severe pain in workers several hours after a brief (10-second) exposure to radiation from an arc torch that generated an intense flame 8 to 12 inches long. Powell et al.[14] studied hazards from both laboratory and industrial plasma torches and found the output of these sufficient to cause eye and skin irritation on long exposure. Erythema on unprotected forehead and forearms developed within an hour after exposure began.

Schall et al.[15] observed no eye lesions or erythema in "Go-Go dancers" exposed to the following maximum levels of UV energy from fluorescent "black" light bulbs: 0.2 $\mu W/cm^2$ at 253.7 nm; 1.4 $\mu W/cm^2$ at 296.7 nm; and from less than 20 to 210 $\mu W/cm^2$ at 365 nm.

2. Effects on skin

Erythema is the most conspicuous change in the skin brought about by ultraviolet radiation.[16] Erythema has been evaluated by varying the amount of ultraviolet energy to produce a different biological response and is most commonly expressed as the Minimal Erythema Dose (MED).[17]

Methods to quantitate the erythemal response have involved use of both a series of red-stained slides or color-graded modifications to which the reaction could be compared and graded[18,19,20,21] and reflectance spectrophotometry.[22,23,24]

In an attempt to standardize the definition of minimal erythema, Van Der Leun[25] prepared a conversion table for various forms of MED determinations to what he thought more likely to be a true MED.

determinations to what he thought more likely to be a true MED.

Through a series of graded determinations ranging from - to + +, the first + reaction is taken to be the MED.

Action spectra (Figure X-1) for the erythemal response have been developed by a number of investigators.[26,27,28,29] These spectra were based on data showing the relative effectiveness of equal amounts of energy at different wavelengths in producing erythema. The different curves showed close agreement from approximately 270 nm to 310 nm. From these reports, a "standard erythemal curve" (Figure X-2) was formulated in 1934 by Coblentz and Stair[30] which plotted relative erythemal effectiveness against wavelength. This standard erythemal curve has been accepted for a number of years, and shows maximum erythemal effectiveness at approximately 297 nm, least at 280 nm, and intermediate at 254 nm. Hausser and Vahle demonstrated[27] that erythema develops more slowly at 260 nm than at 300 nm. From this observation it was concluded that a true action spectrum for a simple response such as vasodilatation cannot be obtained by comparing the energy requirements of different wavelengths to elicit a given intensity of reaction.[31] Everett et al.[32] developed a spectral curve (Figure X-3) considerably different from the standard curve and showing maximum erythemal effectiveness at about 254 nm with an intermediate plateau between 280 and 300 nm, at which point it coincided with the standard curve for the higher ultraviolet wavelengths. Freeman et al.,[33] in 1966, reported a spectral curve (Figure X-3) which was intermediate

between that which was reported by Everett and co-workers and the standard curve. Berger et al.,[34] in 1967, demonstrated that different choices of time after irradiation, and whether minimal or moderate erythema was used as the endpoint, would produce action spectra (Figure X-4) resembling those reported by Everett et al. and Freeman et al. Furthermore, their results confirmed the original observations of Hausser and Vahle[27] and indicated that disagreements were due to differences in time of evaluation (8 hours vs. 24 hours) and the difficulties inherent in the delineation of "minimal erythema".

Melanin, the pigment responsible for varying degrees of skin coloration, is present in the epidermis of the skin.[35] When it is present in high concentrations, the deeper levels of the skin are protected from damaging effects of ultraviolet radiation, the melanin acting somewhat as a supplementary epidermal biological filter. The process of melanin pigmentation in the skin is believed to be initiated from pigment granules present in melanocytes with transfer of the granules to neighboring cells in the basal layers of the skin.[36] The number of melanocytes in Negro and Caucasian skin is about the same,[37] so that differences in degree of skin pigmentation result from differences in cell activities. Longer wavelengths than those required for erythema produce some suntanning, even wavelengths extending well into the visible range.[38,39]

Miescher[40] showed that the ratio of thresholds for mild sunburn was about 8 times as great for Negro skin as for Caucasian skin, and about 120 times as great for severe sunburn. Thus, though skin pigmentation does afford protection from sunburn, Fitzpatrick[41]

demonstrated that erythema nevertheless does occur in deeply pigmented skin even though it is extremely difficult to measure.

The manner in which melanin affords protection is not entirely understood. Daniels,[42] in reviewing the relation between pigment and human adaptation to environmental radiation, stated that it was unlikely that a darkly pigmented skin was required solely as a shield against the ultraviolet radiation of sunlight.

The epidermis of the skin which has been exposed to mild doses of ultraviolet radiation becomes thickened, initially due to inter- and intra-cellular edema. After approximately 72 hours, the mitotic rate has accelerated and increased cellular production contributes to the epidermal thickening. All layers of the epidermis, except for the basal layer, are thickened and remain so with further stimulation. The thickened epidermal layer affords protection against damage by ultraviolet radiation. The potential protection afforded by the thickened epidermis is illustrated by the practical impossibility of eliciting an ultraviolet erythema in the palms of the hands or soles of the feet. Calculations based on the thickness of the horny layer have shown that a dose many thousandfold that of the MED for trunk skin would be required to produce erythema in such areas as the palms of the hands.

Worthy of brief mention is Vitamin D production and two genetically inherited diseases, xeroderma pigmentosum and congenital erythropoietic porphyria. These are mentioned primarily because of the unique role played by ultraviolet radiation in their development.

The photochemical conversion of provitamin D to the active compound by ultraviolet radiation is a well established reaction. Johnson et al.[16] compared the ultraviolet energy requirement for Vitamin D synthesis to that of the MED. Gorter[43] found that with 297 nm radiation, a daily dose of 0.1 calories (4.2×10^6 ergs) was required to cure rickets in children. The radiation covered 200 cm^2 of skin and was, therefore, 2.1×10^4 ergs/cm^2. According to Coblentz et al.,[44] the MED at 297 nm, 4×10^4 ergs/cm^2, on the average, the daily dose effective in curing rickets, amounted to 5% of the MED over a skin area as small as 200 cm^2.

Xeroderma pigmentosum presents an unusual example of the effects of ultraviolet radiation on normal skin. At an early age, the victims of this disease develop freckling, depigmentation, precancerous tumors, basal and squamous cell cancers, and malignant melanomas which cause early death. When this occurs in African Negroes, the course is the same in spite of very dark pigmentation,[45] so that melanin <u>per se</u> cannot entirely account for the protection of skin from ultraviolet carcinogenesis.

Congenital erythropoietic porphyria is a rare disease in which red teeth and red urine are characteristic. Photosensitization of the skin leads to blisters, hyperpigmentation, increased hair growth, and progressive scarring and deformity of the fingers, ears, nose, eyelids, and face. The picture of a hairy scarred face, clawlike hands, and blood-red teeth in people who avoided daylight and went about by moonlight, led to the idea of werewolves.[46]

The topical application or the oral or parenteral administration of certain drugs and chemicals causes the skin to become hypersensitive to ultraviolet and visible light. In many cases, the photosensitizing

ability of a drug has been discovered only after its acceptance for clinical use. Pathak[47] listed various agents implicated in the photosensitivity reactions of skin and showed their therapeutic uses and their effect on skin in the presence of light. For specific agents, he also gave the biologic spectrum, i.e., the band of wavelengths that effectively induced erythemal response, edema, photo-allergic manifestations, and other biologic changes .

The chronic effects of repeated ultraviolet exposure in individuals not adequately protected by pigmentation or other skin mechanisms are basophilic degeneration of the connective tissue, fragmentation of the elastic tissue (senile elastosis), and carcinogenesis.[48] Sunlight, but more specifically wavelengths from about 290 nm to 325 nm,[49] is far more important than aging in producing skin changes.[50] Solar-damaged skin has markedly increased ground substance, increased elastic fibers associated with a diminution of collagen,[51,52] and epidermal atrophy with many abnormal cells in a disorderly pattern.[53]

Epidemiologic, clinical, and tumor distribution studies have clearly implicated solar ultraviolet radiation as a factor in the etiology of human skin cancer. Brodkin et al.[54] present many early findings relating the incidence of basal-cell epithelioma to specific geographic regions, areas of the body, and complexion characteristics in individuals. The following arguments have been proposed to support the belief that sunlight is a causal factor in human skin cancer:

(1) Skin cancer occurs most frequently on exposed areas of the body;[55]

(2) Pigmented races have less skin cancer than do people with white skin [42];

(3) Among Caucasians, those having outdoor work activities appear to have a greater prevalence of skin cancer than those who work indoor[56,61];

(4) Skin cancer is more common in light-skinned people living in areas where solar radiation is greater.[56,59]

The histologic and cytologic changes induced by ultraviolet radiation have been reviewed by Blum[49] and Daniels.[48] The erythema noted after exposure of the skin to ultraviolet radiation is accompanied by glycogen deposition in the basal-cell layer. Approximately 24 hours after initial exposure, the upper portion of the Malpighian layer contains pycnotic, densely nucleated cells and a glassy homogenous cytoplasm shrunken around the nucleus, leaving a clear area outside.[62] In the normal skin, the cells in the upper Malpighian layer undergo changes leading up to nuclear disappearance. It has been suggested that the latent period of ultraviolet effects is partly related to mitotic interval delays.[48] Later, the outer portion of the Malpighian layer becomes hyalinized and concentrated rather than dissolved and broken down. Mature cells seem to be withdrawn from biochemical activity, particularly the production of organ-specific mitotic inhibitors to the basal-cell layer. Thus, interrupted feed-back aspects of carcinogenesis appear to be associated with genetic changes produced in germinal cells.[48]

Lysosomes, which contain a number of hydrolytic enzymes, have been implicated by Novikoff[63] in keratinization processes and squamous

metaplasia. These lysosomal enzymes, when released, are capable of breaking down the major components of cells.

The Langerhans' cells, containing light-sensitive organelles, are considered among the melanocyte series.[64] These Langerhans' cells are accessible in basal-cell locations in vitiligo (failure of the skin to form melanin), possibly suggesting a feedback inhibition of the activity of melanocytes. Damage to this feedback mechanism would then be consistent with the upward melanin migration and increased melanization after sunburn.[48]

Evidence for feedback regulatory mechanisms in cancer production has been demonstrated by a number of reports and mathematical models.[65-70] The predisposition of an atrophic skin to cancer formation is more consistent with a decrease in regulatory factors produced by an inadequate supply of normal tissues and cells than it is with an irritation of hyperplasia phenomenon.[48]

Epidemiologic Studies

Epidemiologic studies clearly implicate solar ultraviolet radiation as a factor in the etiology of human skin cancer.[49,56,57,71-73] In addition, the role of sunlight in skin cancer has been documented in a number of clinical investigations and tumor distribution studies.[54,55,60,74]

Gellin et al.[57,75] demonstrated a statistically significant tendency for patients with light complexions, light eyes, blond or red hair, and who spend a greater amount of time outdoors to have

a greater incidence of basal cell epithelioma and malignant melanoma than control groups. There was a 25 percent greater incidence of basal cell epithelioma among men than among women, most likely because men spend more hours outdoors for work or sport. Ninety-one percent of the basal cell epitheliomas were on sun-exposed parts.

Silverstone and Searle [58] studied the influence of age, sex, susceptibility to sunburn, complexion, eye color, ancestry, occupation, clothing habits, and residential district in the etiology of skin cancer and solar keratosis in Queensland, Australia. These investigators reported that genetic factors, as reflected in susceptibility to sunburn, complexion, etc., were of much greater importance than environmental factors such as district and occupation. With reference to susceptibility, they concluded that it is better to make a detailed investigation of a patient's response to sunlight, such as erythemal reaction, degree of burning, and ability to produce pigmentation, than simply to ask questions about ancestry or observe skin, eye, and hair coloration. Silverstone [73] had earlier observed a significant excess of tumors in Celtic people in three areas of Queensland over that expected on the basis of distribution of the local population.

MacDonald[59] found that the prevalence of carcinoma in El Paso County, Texas, where the sun shines during 80 percent of the daylight hours, was eight times higher than in Hartford, Conn., where the sun shines 50 percent of the daytime. While concluding that the incidence of skin cancer in Rhode Island is less than in Southern states, Winkler[76]

found that the sun also plays a role in the North--particularly in individuals with light eyes, light skin, and inability to tan. Similarly, Jakac[77] observed that the majority of skin cancers in Yugoslavia occurred in light-skinned persons.

Swanbeck and Hillstrom[60] analyzed the distribution of squamous cell carcinoma on the arm and hand from medical records of the 154 cases reported in Sweden during the period 1958-1965. There were 129 patients with skin cancer on the hands (mainly dorsal parts) and only 24 with cancer on the arm. Outdoor workers formed the largest group with squamous cell carcinoma on the hands, and the incidence of this cancer was higher for subjects in southern than in northern Sweden. The amount of ultraviolet radiation reaching the ground is greater in the southern part of the country.

Studies by Davis and Herron[78] produced conflicting evidence on the role of sunlight in malignant melanoma. The tumor was more common in persons spending long periods of time outdoors and in those who burn easily on exposure to the sun. Against this evidence was the fact that the distribution of melanoma on the body was vastly different from that of squamous carcinoma. These findings led the investigators to conclude that sunlight may exert both a direct and indirect effect on Caucasians.

In investigations in Rumania, Nicolau and Balus[61] observed that chronic actinic cheilitis was the precancerous disorder responsible for most of the epitheliomas occurring on the lower lip.

A large number of his subjects spent most of their time outdoors, and all were from areas with long summers and a high rate of exposure to sunlight. Monnich[79] reported a high incidence of skin cancer among agricultural workers in Potsdam due to actinic radiation.

Animal Toxicity

The experimental evaluation of ultraviolet-induced keratitis has been conducted mainly by animal experimentation, primarily in rabbits and guinea pigs.[80-82] Pitts and Tredici,[10] and Pitts and Gibbons[9] included human subjects along with rabbits and primates to establish a comparative experimental threshold for photokeratitis.

Cogan and Kinsey[82] determined the threshold dose necessary to produce keratitis in the eyes of albino rabbits. Utilizing a double monochromator and 1 mm entrance and exit slits, the spectral region from 240 to 316 nm was evaluated with band widths approximately 20 nm wide. Threshold response was determined by a granular appearance (50 to 200 individual granules) within the corneal epithelium. The granules were of uniform size, each being approximately the size of a single epithelial cell. With severe reactions above threshold, the number of granules increased, ultimately forming a mosaic. The peak sensitivity to ultraviolet radiation was reported to be about 288 nm with a corneal threshold reaction of 0.15×10^6 ergs. This compared with 2.0×10^6 ergs as reported by Duke-Elder[2] utilizing a broad ultraviolet spectrum.

Quantitative determinations of ultraviolet absorption by different structures of the eye were reported by Kinsey[80] to clarify

questions concerning various pathologic conditions such as cataract,[83] retinal damage,[84] and functional visual disturbances.[85] The limit of ultraviolet transmission for the whole eye was found to be approximately 330 nm; that for the lens, 310 nm, and approximately 280 nm for the aqueous and vitreous humors and cornea. It was concluded from calculations that the eye would have to be exposed to three times the dose necessary to produce minimal damage to the cornea before minimal injury to the lens could be encountered. This finding confirmed the conclusions of Verhoeff and co-workers[4] from studies of men and animals, that damage to the lens could result only after severe injury had been produced to the cornea.

Bachem,[81] using low pressure and medium pressure mercury arc ultraviolet sources on the eyes of albino rabbits and guinea pigs, concluded that (1) the ultraviolet radiation most effective in causing eye irritation is that near 300 nm; 288 nm for the cornea and 297 for lens; (2) shorter wavelengths of ultraviolet radiation are relatively harmless to the eye (they produce no lens injury, but may cause corneal and conjunctival inflammation) and (3) ultraviolet radiation of longer wavelengths can cause cataracts through the cumulative effect of repeated excessive dosage.

Pitts and Tredici[10] sought to establish experimental thresholds for photokeratitis in rabbits, monkeys, and humans. Ocular changes were determined in the animals from ultraviolet exposures at 10 nm waveband steps from 210 to 320 nm. Observations were made 12 to 18 hours after exposure since previous work had shown that threshold signs were just as evident as observations made directly after the

the latent period. Procedures for determining the threshold in human subjects were identical to those used for the animal experiments except that, after exposure, the subjects were examined at 30 minute intervals for the first 6 hours and hourly thereafter and asked to describe verbally any symptoms which they had experienced.

To describe clinical photokeratitis at least 9 criteria were used: tearing, stippling, hyperemia, haze, photophobia, discharge, pain, blepharospasm, and exfoliation. The criteria used to determine the photokeratitic threshold were the production of granules and epithelial haze for both animals and humans. Threshold exposure was defined as the presence of 50 to 200 granules as used in the study reported by Cogan and Kinsey.[82]

The photokeratitic threshold (maximum sensitivity) for both rabbits and monkeys occurred at 270 nm, being 0.05×10^6 erg/cm^2 for rabbits and 0.04×10^6 ergs/cm^2 for monkeys.

The ultraviolet photokeratitic thresholds for the cornea were felt by the authors to be accurate to $\pm$10%. Human and primate data corresponded surprisingly well.

In experiments with chinchilla rabbits, Sherashov[86] studied the spectral sensitivity of the cornea to ultraviolet radiation by measuring the ultraviolet pulses with a semiconductor thermoelectric calorimeter. His report indicates that there are two clearly defined maxima of sensitivity of the cornea. The first peak corresponds to the wavelength 289.4 nm and the second is in the region of 253.7 nm.

Between them only an insignificant fall in sensitivity was observed. Ultraviolet radiation at wavelengths greater than 330 nm had practically no photochemical effects.

Studies on ultraviolet absorption in nucleopeptides and ultraviolet-induced alteration of RNA and DNA synthesis[87-90] indicate that ultraviolet effects on corneal tissue are caused by absorption within the nucleoprotein.

The experimental production of cancer by ultraviolet radiation has been reviewed by Blum[49] and Epstein[89] in 1966. According to Epstein,[89] although there is some question about the carcinogenic spectrum in human skin cancer, there is no controversy about experimental cancer produced by ultraviolet radiation. Action spectrum studies have established that carcinogenic effects are limited to wavelengths shorter than 320 nm[49,90] and are significantly more effective between 280 nm and 320 nm.[91-93] This is the same wavelength spectrum in which solar radiation induced phototoxic sunburn responses. Under ordinary conditions, longer ultraviolet radiation and visible light are not carcinogenic; however, repeated long wavelength exposures in the presence of photosensitizers, which include many chemical carcinogens, have resulted in a high incidence of cancer.[49,94,95]

Action spectrum studies involving monochromatic radiation have shown that solar radiation at the wavelengths evoking sunburn response in man, 290 nm to 320 nm, also induces cancer in mice. However, Freeman et al.[90] determined that the wavelengths between 290 nm and 320 nm are not equally effective in inducing skin cancers. A weekly dose of 1 x 10 $\mu W\ sec/cm^2$ was given to two groups of albino mice. Tumors developed in

the group exposed to 300 nm radiation but not in the group exposed to 310 nm. Winklemann[96] found that 280 nm to 310 nm induced squamous cell carcinoma in the skin of hairless mice.

Prior to 1960, sarcoma was the primary tumor produced experimentally by ultraviolet radiation on the ears of albino mice and rats.[49] With the development of hairless mice, squamous cell carcinomas were reported[97] and further studies established that squamous cell carcinomas could be produced almost to the exclusion of connective tissue (sarcoma) growths.[96,98] The hairless mouse has also provided an experimental model for demonstrating that benign pigmented lesions could be stimulated to develop into malignant melanomas by ultraviolet radiation.[99] This further emphasizes that exposure to the sun may play an important role in human malignant melanoma formation.[100]

Although one cannot make quantitative extrapolation from the induction of cancer in laboratory animals to the environmental situation of men exposed to ultraviolet radiation, it seems likely that the mechanism of cancer induction in the mouse is basically similar to that of cancer induction in man.[101] Ultraviolet exposure alone must be repeatedly applied in order to induce an observable cancer. Blum[49,101] suggests from a series of calculations and experimental observations in mice that the rate of growth of a tumor is increased with each dose of ultraviolet radiation. It has been shown by using croton oil, a substance which increases the rate of proliferation of cells but does not by itself cause cancer, that a single dose of ultraviolet light may suffice to produce a tumor. That tumors are not observed follow-

ing single doses of ultraviolet radiation may be explained by the postulate that a single dose produces some fast growing, genetically changed clones but not in sufficient quantity to form a tumor within the lifetime of a mouse. Successive doses of ultraviolet radiation progressively expand and accelerate the tumorigenic process. Cancer induction depends not only upon somatic mutations from each dose, but also upon a progressive acceleration which is speeded up by each successive dose of ultraviolet radiation. The important bearing to the problem of ultraviolet-induced human cutaneous cancer is that the process of cancer induction is cumulative and hence the total amount of exposure is the important factor rather than a single or a few severe exposures. The induction of cancer by ultraviolet radiation is inferred to be irreversible in that there is no evidence for a "precancerous" condition.[49,101,102]

Correlation of Exposure and Effect

A summary of threshold values, presented as the minimum erythema dose (MED) in humans for six independent investigations, is listed in Table X-3. The MED's were determined at approximately 300 nm, the wavelength region of maximum erythemal effectiveness according to the standard erythema curve (Figure X-2). Differences existed between investigators as to the skin site, duration, and endpoint for erythemal testing which resulted in reported MED values ranging from 1.14 to 6.4 x 10^4 $\mu Wsec/cm^2$. If the value reported by Olson et al.[103] of 2.42 x 10^4 $\mu Wsec/cm^2$ is considered to be representative of a minimum erythema dose, then the value, converted to 24.2 mJ/cm^2, is shown

to be 2.4 times that of 10 mJ/cm^2 at 300 nm proposed as a minimum hazard level by Sliney[8] in 1972. This indicates a 2.4-fold safety factor at 300 nm from the minimum hazard level for the production of minimum erythema as determined by Olson et al.[103] If the lowest MED from Table X-3 is considered, 1.14×10^4 $\mu W/cm^2$ (11.4 mJ/cm^2), the proposed minimum hazard level of 10 mJ/cm^2 still seems acceptable.

The dose of ultraviolet radiation necessary to give a threshold erythema reaction at 253.7 nm wavelength is only about 50% of that necessary at 300 nm. A limit of 0.1 $\mu W/cm^2$ (8.6 mJ/cm^2) for 24 hours has been established.[104] Again, the proposed minimum hazard level is satisfactory at the 254 nm wavelength (6.0 mJ/cm^2).

The comparative photokeratitic thresholds for the cornea shown in Figure X-5 generally indicate a greater sensitivity in the human than in the rabbit or primate over the ultraviolet spectrum from approximately 220 to 310 nm. At 270 nm, the point of peak absorption, thresholds are about equivalent, 0.4×10^{-2} $\mu W/cm^2$ (4 mJ/cm^2) for humans and primates, and 0.5×10^{-2} $\mu W/cm^2$ (5 mJ/cm^2) for rabbits.[9,10] These data are in general agreement with those reported by Cogan and Kinsey[82] from studies of the rabbit. At the extremes of the ultraviolet spectrum studied, the human photokeratitic threshold is 4.6 times lower than that for the rabbit and 3.4 times lower at 310 nm. Interestingly, the human photokeratitic threshold appears to show a rather straight-line relationship through the ultraviolet spectrum studied from 220 to 300 nm. Above 300 nm, a trend toward decreased sensitivity is noted and would be expected to be quite marked as seen in the rabbit. The photo-

[illegible] nm, a trend toward decreased sensitivity is noted and would

keratitic thresholds for both animals and humans show good agreement with the proposed minimum hazard level curve.

Hart[105] reported the extensive use of bactericidal ultraviolet radiation in hospital rooms. Ultraviolet radiation at 253.7 to 290.0 nm was delivered at the operating site with an intensity of 18 to 30 $\mu W/cm^2$. A subsequent report[106] described lamp installations in which the upper portions of operating rooms were exposed to an average irradiance of 50 $\mu W/cm^2$ while maintaining the desired intensity at the operative site (24 to 30 $\mu W/cm^2$). These levels of ultraviolet radiation reduced post operative infections by as much as 85%[106] but required personnel to have skin and eye protection to prevent erythema and photokeratitis.

Schall and co-workers[15] reported the maximum energy level recorded at the working level for entertainers exposed to black-light radiation was 210 $\mu W/cm^2$ at 365 nm. In addition, small exposures of 1.4 and 0.2 $\mu W/cm^2$ were reported for 296.7 nm and 253.7 nm, respectively. No significant clinical evidence was revealed of skin or eye damage from the exposures studied. It was felt, however, that exposures for several hours at short distances from black-light sources could conceivably cause erythema and dermatitis as well as eye irritation.

High pressure arcs and plasmas produce ultraviolet-induced ocular damage considered by some investigators[11,12] to be the most common accident in engineering shops, accounting for 40% of all injuries. Irradiance in excess of 250 $\mu W/cm^2$ at 253.7 nm have been reported.[13] A 10-second exposure to this intensity produced severe ocular pain which required strong analgesics. Powell and co-workers[14] reported development of

sunburn reactions on unprotected forehead and forearms in plasma torch operators within an hour after exposure to levels sometimes in excess of 1000 $\mu W/cm^2$ at 253.7 nm and 400 $\mu W/cm^2$ at 365.0 nm. The sunburn was followed by desquamation and pigmentation.

Cases of dermatitis and erythema have been reported from ultraviolet radiation below 320 nm produced by fluorescent lamps used for general lighting purposes.[107,108] Irradiance levels were not known.

IV. ENVIRONMENTAL DATA

Although there is much information on industrial applications of ultraviolet energy, there is little information on exposure levels. The following discussion relates various ultraviolet-emitting devices with several parts of the ultraviolet spectrum, and thereby offers an impression of the nature of the hazards.

Low-pressure mercury vapor lamps emit several narrow bands; the lower the pressure of mercury vapor the fewer lines emitted. Much of this energy is of 253.7 nm wavelength, which is near the peak of germicidal effectiveness of 265 nm, hence its usefulness in control of microorganisms in operating rooms,[105,106] in control of airborne infection,[109,110] in control of bacteria in meat processing,[111] in the prevention of product contamination in pharmaceutical houses and biological laboratories,[112] in irradiation of air-conditioning ducts,[113] and in making water potable.[114]

High-pressure mercury vapor lamps are used in photochemical reactions, mineral identification, to produce fluorescence, and for diagnosis of dermal and scalp disorders, including porphyria.

Quartz-mercury arcs emit radiation over much of the ultraviolet spectrum, and can cause erythema and conjunctivitis from radiation over the range of 200 to 320 nm.

Fluorescent-type ultraviolet lamps also emit germicidal radiation similar to low-pressure mercury vapor lamps. While there is little evidence that they are significant sources of ultraviolet-induced injury,

it is believed that they may cause skin and eye effects, since a small part of their output is below 320 nm.[15,115] Fluorescent lamps used for general lighting purposes emit a negligible amount of energy below 320 nm. Although rare, skin photosensitization from these lamps has been reported.[107,108]

High-pressure xenon arcs emit a spectrum like that of sunlight in a continuous spectrum. Carbon arcs emit a continuous spectrum from the incandescent electrodes, upon which a broad-band spectrum from the luminous gases is superimposed.

Incandescent sources emit very little ultraviolet energy except at temperatures above 2500 K.[116] Open oil and gas flames are normally less than 2000 C. Oxyhydrogen and oxyacetylene flames are much hotter, so solids heated by these two flames may radiate ultraviolet.

The plasma torch can produce temperatures over 6000 K, the temperature at the surface of the sun, and intense ultraviolet radiation can result. Exposure to radiation from plasma torches can result in keratoconjunctivitis and sunburn if skin and eyes are not protected.[14]

Welding produces ultraviolet radiation in broad bands which often appear as a continuous spectrum. The intensities of the various bands depend on many factors; materials used in the electrodes, discharge current, gases surrounding the arc.[117] A common source of ultraviolet damage is from arc welding.[118,119]

V. DEVELOPMENT OF STANDARDS

Basis for Previous Standards

The production of erythema has been the most commonly used endpoint in the evaluation of the biological activity of ultraviolet. Early investigators[26-29] produced a series of erythema action spectra (Figure X-1). These workers all based their curves on the production of moderate erythema, contending that the threshold for minimal erythema was too difficult to determine and too variable among individuals.[8,34] Coblentz and Stair[29,30] proposed a "standard" erythema action curve which was the average of the curves previously developed. This curve has been widely used and accepted as the "true" erythema action curve (Figure X-2).

In 1948 the Council on Physical Medicine of the American Medical Association[104] recommended an ultraviolet exposure guide. The following criteria were recommended for safe exposure to radiant energy from germicidal lamps, which produce an almost monochromatic emission in the 253.7 nm line: "The total intensity of ultraviolet radiation ... incident on the occupant for seven hours or less should not exceed five-tenths microwatt per square centimeter ($0.5\ \mu W/cm^2$) and for continuous exposure (twenty-four hours a day) should not exceed 0.10 microwatt per square centimeter of wavelength 2,537 A." (253.7 nm). The criteria were based on that dose which would not produce erythema. According to the "standard" curve of Coblentz and Stair, moderate erythema occurs at a dose of 20 mJ/cm^2 at the most effective wavelength of 296.7 nm. The 253.7 nm line, however, is only 50% effective, and the dose at this shorter wavelength ultraviolet necessary to produce moderate erythema is 30 mJ/cm^2. The 7-hour and 24-hour doses

recommended by the AMA Council are 12 mJ/cm^2 and 8.6 mJ/cm^2, respectively. Both of these values are substantially below the "standard" action spectrum to protect against erythema.

Recognizing that many factors affect individual responses, Matelsky[119] suggested, based on the "standard" erythema action curve, the following threshold doses weighted on the basis of their action spectra:

(1) Minimum erythemal dose for previously non-exposed skin: 20 to 25 mJ/cm^2 of erythemally-weighted ultraviolet.

(2) Minimum erythemal dose for previously exposed skin: 25 to 35 mJ/cm^2 of erythemally-weighted ultraviolet.

(3) Minimum keratitic dose: 1.5 mJ/cm^2 of keratitically-weighted ultraviolet.

The American Conference of Governmental Industrial Hygienists[120] has proposed Threshold Limit Values, for 320 to 400 nm, of 1.0 J/cm^2 for periods greater than, and 1.0 mW/m^2 for periods less than, 1000 seconds. For the actinic spectral region of 200 to 315 nm, the Conference proposed limits described by a curve (see Figure I-1) in which the maximum permissible doses range upward at both longer and shorter wavelengths from 3.0 mJ/cm^2 at 270 nm.

Basis for Recommended Standard

The environmental exposure standard recommended in this document is the same as that proposed by the American Conference of Governmental Industrial Hygienists.[120] The ACGIH has not published the documentation or reasoning behind their proposed standard. The NIOSH rationale for recommending the same environmental exposure standard is as follows:

The results of the early investigators were quite consistent in the 280 to 315 nm range, but were somewhat divergent in the lower wavelengths

studied (Figure X-1). Part of this divergence could be due to difference in body location tested and time after exposure at which erythema was determined. As pointed out by Berger, Urbach and Davies[34] for 254 nm, the erythema produced by the shorter wavelengths is relatively transient compared to that produced by wavelengths of 280 to 315 nm, so that time after irradiation is a major factor contributing to the degree of erythema observed.

Other workers[121-124] between 1946 and 1964 reported quantitative data on energy requirements for erythema production. In each case cited, in contrast to expectations from the "standard" erythema curve, less energy was required to produce erythema at shorter wavelengths of 250 to 260 nm than at longer wavelengths. Nevertheless, it was not until recently that the "standard" erythema action spectrum was seriously challenged. An erythema action curve published in 1965 by Everett, Olson, and Sayer[32] was continuous, requiring larger amounts of energy for production of effects as longer wavelengths were employed. In 1966, Freeman et al.[33] developed an erythema action spectrum which was intermediate between the "standard" and the Everett, Olson, and Sayer curve (Figure X-3).

Two basic differences in experimental technique apparently are responsible for the differences in action spectra. Data for the "standard" erythema action curve were based on moderate erythema determined at various times (usually 24 hours) after exposure. The data of Everett and of Freeman and their collaborators were based on minimal perceptible erythema, determined 8 hours after ultraviolet radiation was applied. Berger, Urbach, and Davies demonstrated (Figure X-4) that by varying these two factors, one can produce erythema action spectra resembling either the "standard" or the Everett et al. action spectra.

There has been little uniformity in the choice of body sites irradiated by the different investigators. Olson, Sayre, and Everett[103] have shown the trunk to be more sensitive than either the head or the extremities, and the abdomen (used by them to develop their action spectrum[32]) the most sensitive of three trunk locations tested. After exposing abdominal skin to ultraviolet radiation at nine wavelengths between 250 and 310 nm, they reported that, while erythema response was well developed for all wavelengths after 8 hours, the response had substantially decreased at the shorter wavelengths after 24 hours. Furthermore, the energy requirements for minimal perceptible erythema at 254 and 280 nm were lowest at 8 hours after irradiation and nearly twice as much at 24 hours. The energy requirements for minimal perceptible erythema at 297 nm, however, decreased about 5% between 8 and 24 hours. Thus, it is apparent that the recent erythema action spectra, indicative of minimal perceptible erythema doses determined 8 hours after irradiation of a sensitive part of the body should at least reflect lower energy requirements and possibly other differences as well, when compared to the traditional curve.

Relatively minor damage to the conjunctiva or cornea from ultraviolet results in photophobia, pain, epiphora, and blepharospasm. Although the response is acute and incapacitating at the time, it regresses after several days leaving no permanent damage.[119] The action spectrum for photokeratitis developed by Pitts and Tredici,[10] based on animal and human data, is slightly more conservative than the recent skin erythema curves and reflects maximum efficiency at 270 nm rather than 250 nm. Nevertheless, this curve and the recent erythema action spectra are in reasonably good agreement. This is in keeping with previous statements that the action spectrum for conjunctivitis

is the same as that for skin erythema.

Sliney[8] compared the action spectra, both for erythema and for photokeratitis, with the "standard" erythema action curve. Plotting energy versus wavelength, the recent action spectra are at considerably lower energies than is the traditional curve. Additionally, these action spectra are, in general, similarly distributed. Therefore, Sliney[8] drew a minimum hazard curve which conformed to the general distribution of the new data. This curve, recommended herein as the standard for the 200 to 315 nm range, was drawn with several considerations in mind.

In the 300 to 315 nm range, the Pitts and Tredici[10] data seemed overly conservative since, when weighted against the ultraviolet spectrum of indirect daylight in the tropics, it indicates that almost everyone there would develop keratoconjunctivitis in a few hours outdoors. Therefore, Sliney's curve in the 300 to 315 nm region excluded the Pitts and Tredici data and paralleled the "standard" erythema curve, although displaced slightly below it.

In the 200 to 300 nm range, the curve was drawn to include all action spectra while a general shape was maintained that would lend itself to the construction of a practical instrument for measuring the entire range from 200 to 315 nm. Constructing an instrument capable of following this smooth curve is more feasible than attempting to track a curve, such as the traditional erythema action spectrum, with several high and low points.

Human photokeratitic thresholds recently determined by Pitts and Gibbons[9] do not vary greatly between 220 and 310 nm, i.e. an almost flat curve results. Like the Pitts and Tredici[10] animal data, the reported human thresholds at 300 and 310 nm are more conservative than the recommended standard. Similarly,

the energy requirements for human thresholds are lower at and below 240 nm than is the recommended standard. The reported threshold values for 250 to 290 nm are all higher than the recommended standard.

While the Pitts and Gibbons data are informative, they should not be given great weight in establishing a standard for several reasons. First, the Pitts and Gibbons data are based upon exposures of relatively few individuals. Additionally, the reported thresholds are the threshold response of a single individual at each waveband tested since the experiment was terminated at each waveband as soon as a threshold response was observed in one subject.

Second, the curve is, as mentioned, almost flat. This is in contrast to the animal photokeratitic curves reported by Pitts and Tredici[10] and again in the Pitts and Gibbons[9] paper. While it may well be true that the human response varies slightly, if at all, with wavelength, the present results alone are not strong enough to support such a conclusion. Consequently, the curve drawn by Sliney[8] is believed not to be invalidated by the data of Pitts and Gibbons.

Assigning a relative spectral effectiveness of 1.0 to 270 nm, the low point of the recommended standard, the relative spectral effectiveness of other wavelengths can be calculated (Table I-1). The formula required for determining the effective irradiance of a broad-band source assumes a single erythema mechanism rather than a combination of different mechanisms for different wavelengths.

As discussed by Johnson, Daniels, and Magnus,[31] the shape of the "standard" erythema action spectrum suggests two mechanisms in erythema

production, one with peak efficiency at 297 nm and the other at 250 nm. This is supported by differences in the latent period, duration, and appearance of erythema produced by the shorter (260 nm) and the longer (297 nm) erythematic ultraviolet radiation. On the other hand, the action spectra of Everett and associates[32] and of Freeman and associates[33] suggested a single erythema mechanism since these action spectra reflect a steadily decreasing efficiency with increasing wavelength above the single peak of efficiency at 250 nm. Using 254, 280, and 297 nm ultraviolet radiation, Sayre, Olson, and Everett[125] have demonstrated experimentally that minimal perceptible erythema can be produced by subthreshold doses of two wavelengths acting in combination when the sum of the fractional doses equal one. These results support the idea of a single erythema mechanism.

The recommended standard (Figure I-1) is based upon action spectra both for erythema and for keratoconjunctivitis and is intended to protect the skin and eyes against acute effects. Therefore, separate skin and eye standards are not recommended. The American Conference of Governmental Industrial Hygienists proposed the limits expressed in this recommended standard and they specified its applicability for protection of both eyes and skin. The recommended standard is more readily applicable to the eye since the eye, unlike the skin, does not acquire protective capabilities after repeated exposures.

On the other hand, the skin does acquire resistance to ultraviolet damage after repeated exposure. Additionally, individual variations in threshold response are great. Matelsky[119] states that, despite the extensive industrial exposures to ultraviolet radiation, no cases of industrially-induced skin cancer or keratosis have been reported and concludes that protection against the painful acute effects adequately

protects against tumorigenic doses. Nevertheless, it is believed that there is not enough information to be completely sure that industrial exposures to ultraviolet energy will not cause chronic effects on eyes or skin, such as cataracts or skin tumors.

While erythematic and carcinogenic activity is limited to wavelengths shorter than 320 nm,[33,90,119] the lens of the eye absorbs strongly in the 300 to 400 nm range. "Black-lights" have a powerful emission line of 366.3 nm, which can cause the lens to fluoresce. This apparently causes some people, when looking at "black-lights", to experience a "tired" feeling, blurred vision, discomfort, and sometimes headache, but apparently no permanent damage ensues.[126]

There is some evidence from animal studies to implicate ultraviolet in this range as contributing to cataract formation.[81] While few industrial sources emit strongly in this range, the standard for 320 to 400 nm is recommended to prevent occupational exposures in this range from exceeding levels normally encountered in the out-of-doors.

Normal individuals should be adequately protected by these standards. Photosensitive individuals, however, may respond at extremely low energy levels and over very wide wavelength ranges, even into the visible wavelengths.[122] Therefore, these standards may not be adequate for photosensitive individuals. More research is needed before the adequacy of these standards in protecting against chronic effects on skin and eyes can be assured.

VI. PROTECTION AND CONTROL MEASURES

Skin and eyes can be protected from the effects of ultraviolet radiation by shielding of sources of radiation, by goggles or face shields, by clothing, and, for special purposes, by absorbing or reflecting skin creams.

Principles and procedures in selecting suitable protection are summarized in this section, and studies of various protective measures are reviewed. Specific topics discussed are (1) sunscreens, (2) protective clothing and barrier creams, (3) transparent material for skin and eye protection and (4) reflection of ultraviolet radiation.

(1) Sunscreens

Sunscreening preparations are usually classified as chemical or physical. The former include para-aminobenzoic acid and its esters, cinnamates, and benzophenones, all of which act by absorbing radiation so that the energy can be dissipated as radiation of lower energy. The physical agents act as simple physical barriers, reflecting, blocking, or scattering light. They include titanium dioxide, talc, and zinc oxide. Largely because of cosmetic objections, the physical barriers are infrequently used in sunscreen formulations.

Sunscreen protection from absorbing chemicals depends on maintenance of film thickness. Robertson[127] reported that a series of sunscreens of 0.01 mm thickness protected fair skin during four to five hours of sunshine if the protective layer was fully maintained for the whole period. When the thickness of the layer was halved, erythema occurred within a maximum of one hour.

Pathak, Fitzpatrick and Frenk[128] produced evidence suggesting that para-aminobenzoic acid and its esters in ethanol afforded protection against the sunburn range (290 to 320 nm) for several hours with one application and that the protective action was unaffected by bathing, swimming, or vigorous exercise. MacLeod and Frain-Bell[129] confirmed the effectiveness of para-aminobenzoic acid in ethanol and observed that protection was provided for up to seven hours after the initial application. They found, however, that the agent was easily removed as a result of bathing or exercising. Katz[130] also noted that para-aminobenzoic acid is not water-resistant; he found that a consistently satisfactory protection against the erythematogenic rays of the sun was lost after a 10-minute swim.

Goldman and Epstein[131] reported that a commercial sunscreening agent containing the ultraviolet-absorbing chemical glyceryl para-aminobenzoate was a photosensitizer and that it had produced severe dermatitis in a patient who applied it prior to exposure to sunlight. The agent was an ordinary contact allergen as well as a photosensitizer. Turner, Barnes and Green[132] found that a preparation containing vitamin A and calcium carbonate reduced the unpleasant effects of solar radiation without affecting normal tanning. The beneficial effect was most marked in subjects with blond hair. This observation could not be repeated, according to Findlay.[133]

Red veterinary petrolatum is cosmetically less acceptable than other agents, but has a long history of effective protection of normal skin from the damaging effects of the ultraviolet sunburn spectrum. Like the benzophenones, it also gives some protection in the long ultraviolet waveband (MacEachern and Jillson[134] ; Luckiesh, Taylor, Cole and Sollman[135]).

benzophenones, it also gives some protection in the long ultraviolet

Fusaro and his coworkers[136,137] approached the problem of protection against sunlight by altering the stratum corneum chemically so that the keratin had new ultraviolet transmittance characteristics. They believed that this could be accomplished with a dihydroxyacetone/napthoquinone mixture (DHA/Lawsone) made up in a vanishing cream base rather than in an isopropyl alcohol/water solution. This preparation was thought to be effective in patients with erythropoietic protoporphyria, but Donaldson et al.[138] doubted its efficacy with their patients.

For individuals with chronic photosensitivity diseases, it is desirable to add a light-scattering and reflecting agent (e.g., titanium dioxide, talc, and zinc oxide) in combination with a light absorber in a hydrophilic ointment.

(2) Clothing and Barrier Creams

Protective clothing consists of long-sleeved garments to protect the arms while a small cape sewed to the cap protects the back of the neck and the sides of the face. Flannelette and poplin give maximum protection, while other materials give less protection (Table X-4).

Where it is impossible to shield the skin by clothing, polyvinyl chloride gloves, masks, shields or by redirecting the radiation by suitable reflectors, a barrier cream should be applied to the skin before irradiation. Ordinary soft paraffin is an excellent barrier, but its greasiness will often preclude its use on hands. Barrier creams contain ingredients which absorb ultraviolet radiation. The benzophenones are the best compounds for this purpose because of their great absorption capability throughout most of the near and far ultraviolet spectrum (Parrish et al.[139]).

(3) Transparent Materials for Eye and Skin Protection

Protection of the eyes in industrial applications such as welding requires materials which are strong absorbers of ultraviolet radiation. A large number of protective glasses have been developed for this purpose. Many of them also absorb strongly in various portions of the visible and infrared regions. The earliest of these glasses was developed almost 60 years ago, and subsequently, many others have been developed. Their characteristics are described in "Spectral-Transmissive Properties and Use of Eye Protective Glasses" by R. Stair.[140] The transmission of Noviol, slightly yellow glasses which cut off sharply at about 400 nm, is shown in Figure X-6 and Table X-5.

For protection of the eyes and skin from limited exposure to ordinary ultraviolet lamps, common window glass is usually adequate. Ordinary window glass in thickness of 2 mm or more is practically opaque to ultraviolet radiation of wavelengths shorter than 300 nm. Thus an ordinary window pane, although it emits much of the incident visible radiation, excludes practically all the ultraviolet wavelengths of the erythemal and therapeutic ranges. Figure X-7 shows the percent transmission as a function of wavelength for two thicknesses of window glass.[141] As can be seen from the curve, the transmission falls off rapidly with wavelength below 360 nm. Window glass 1/8 in. in thickness is adequate protection for the eyes and skin against ultraviolet radiation from ordinary ultraviolet sources. In the case of very intense sources of ultraviolet radiation, it may not be sufficient.

Full protection against 253.7 nm radiation is provided by shields of clear ultraviolet-absorbing plexiglass, ordinary (glass) spectacles, crookes glass, and similar ultraviolet-absorbing materials. Crown glass, an alkali-lime silicate glass, (2 mm-thick) will significantly reduce exposure hazards. Flint glass, a heavy glass containing lead oxide, (2 mm-thick) affords essentially complete protection at all wavelengths. Noviol glasses or Polaroid ultraviolet filters can be used where high intensity ultraviolet is anticipated, as in welding. If an individual is working in a room with an ultraviolet source for any length of time, he should wear protective glasses or a face shield because many materials reflect 253.7 nm radiation (Table X-6).

Glass workers, arc welders and people engaged in similar types of work may be exposed to infrared radiation as well as ultraviolet radiation, and may need eye protection from both types of radiation. Such people should wear goggles with an infrared absorbing glass and an infrared reflecting surface. Ordinary glass, plastics and other materials are usually transparent to infrared rays which can cause thermal damage to the eye. A glass that absorbs in both the ultraviolet and infrared regions of the spectrum will be needed in such cases. For listings of absorbing glasses refer to ANSI-Z 49.1.[142]

(4) Reflection of Ultraviolet Radiation

When a number of ultraviolet generators are operating in one room, protection of personnel poses several problems. In many applications, little difficulty is encountered in properly shielding the source so that most, or all, of the output is restricted to the exposed material. Stray radiation can be reduced, but reflection from glass, polished metal, and

high-gloss ceramic surfaces can be harmful to people working in the room. Absorption of ultraviolet radiation therefore becomes an important item to consider in planning a safe work environment. Since painted walls and ceilings can be a significant source of ultraviolet reflection, it is necessary to consider the ultraviolet reflective properties of the paint used.

The reflection of incident ultraviolet radiation from pigments can range from negligible to more than 90%. A given material's ability to reflect visible light is no indication of its ability to perform similarly with ultraviolet. Table X-7 gives the reflection from a number of white pigments and other materials at several wavelengths in the ultraviolet. The table shows that ordinary white wall plaster has a reflection of 46% at 253.7 nm, whereas zinc and titanium oxides, which are equally good reflectors for visible light, reflect only 2.5% and 6%, respectively, at this wavelength.

Oil-vehicle paints usually have low reflectances because of the absorption by the oil. However, some paints using synthetic plastic vehicles with high ultraviolet transmission may have high reflectances. Walls surfaced with gypsum products tend to have high reflectances.

Table X-8 shows the ultraviolet reflectance of a number of dry white pigments in the region between 280 and 320 nm. These measurements were made with the unresolved radiation from a S-1 lamp as a source and a cadmium phototube as a detector. These measurements may be assumed to be predominantly at the wavelength 302.4 nm.

No assumptions regarding the reflections of white pigments should be made without investigating their composition. The reason for this is demonstrated by the difference between two white pigments, zinc oxide and white lead. Although both of the pigments are very good reflectors of visible radiation, zinc oxide reflects only 3% of the ultraviolet, whereas white lead reflects about 60%. Colored pigments are almost invariably poor reflectors of ultraviolet. Stutz[143] studied 38 colored pigments and found that only turquoise blue had a reflectance of as much as 25% at 331.1 nm. At 253.6 nm turquoise blue had a reflectance of 22%, whereas none of the others exceeded 7.5%.

Table X-9 shows the ultraviolet reflectance of a number of paints with different white pigments suspended in silicone.

The basic requirements which determine the reflecting power of an ultraviolet-reflecting paint have been given by Koller[141] :

1. Particles of the pigment must be low in absorption (except metallic pigments), so that a large portion of the incident radiation is returned by multiple reflection and refractions.

2. The binder or vehicle must be transparent to the radiation to be reflected.

3. The difference in refractive index between pigment and medium must be large so that reflection and refraction at pigment-medium interfaces will be appreciable.

The properties of a paint depend upon the nature and amount of the pigment and the state of its aggregation. The addition of a small amount of colored pigment to a white paint may result in a large decrease in the ultraviolet reflection. The reflectance decreases with increase in amount of added colored pigment.

Two materials with a high reflectance in the visible and the ultraviolet are magnesium oxide and magnesium carbonate. Reflection curves are shown in Figure X-8. Tellex and Waldron[144] reported that for a sufficiently thick coating (8 mm) the reflectivity of magnesium oxide is about 98% and is almost independent of wavelength over the visible spectrum. For thinner coatings the reflection decreases slightly and there is a rather flat maximum of 98% at 540 nm.

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VIII APPENDIX I

MEASUREMENT OF ULTRAVIOLET ENERGY

The ultraviolet portion of the electromagnetic spectrum has been basically divided into several bands. While these bands were arbitrarily determined by physicists and are not directly related to the biological action spectrum of ultraviolet radiation, they are important for two primary reasons: (1) The development of artificial sources of ultraviolet energy to accomplish specific tasks, and (2) the availability of measuring devices to cover these specific areas of the ultraviolet spectrum.

The band between 320 to 280 nm is referred to as the erythemal region with 295 to 298 nm being the wavelengths of maximal effect. This area is also the one which has been identified as having a carcinogenic effect upon the skin.

Slightly overlapping these wavelengths is a germicidal band between 280 and 220 nm with a maximum germicidal effective wavelength at 265 nm with some erythemal effect noted between 250 and 260 nm.

The last band is between 220 and 170 nm and is only partially covered by the recommended environmental limit suggested in this criteria document. This is generally known as the ozone region and includes wavelengths that result in the most effective production of atomic oxygen. The absorption coefficient of ultraviolet by oxygen for wavelengths below 200 nm becomes very large; and therefore, emissions in this region have little biological significance except as related to the production of ozone.

There are several major classes of instruments for the detection of ultraviolet energy: physical, chemical, and biological. Concerning this recommended standard, only the physical methods of measurement are considered. These depend upon photosensitive elements to convert electromagnetic emissions into electrical energy.

The simplest of detection or measurement devices is the barrier layer, a photovoltaic cell which is normally insensitive at lower levels of ultraviolet energy and is sensitive to a limited or a narrow band of ultraviolet energy.

Some instruments which have been considered more reliable and sensitive for routine industrial hygiene use have relied upon vacuum phototubes and where extremely low levels of energy were to be measured have utilized photomultiplier tubes to develop the sensitivity required. Most commercially available ultraviolet measuring devices, with the exception of the thermopile, are wavelength selective. Special filters or phosphors are required to isolate the portion of the ultraviolet spectrum where specific emissions occur with any given exposure or industrial process. This results in two basic types of measurement that are necessary in determining potential exposure to hazardous levels of ultraviolet energy. In many industrial operations, such as in welding, the ultraviolet emissions are across the entire band of ultraviolet energy and into other portions of the spectrum as well. These types of exposures require measuring devices that will integrate the intensity of the ultraviolet energy over the frequency range covered by the

standard. Secondly, exposures from artificial sources that give specific emissions in limited wavelengths may require filters that can measure only in those specific wavelengths.

As a consequence of the variety of conditions of measurement required to assess the hazard from ultraviolet energy and the limited availability of ultraviolet measuring devices, care must be taken in the selection of the available instrumentation or in the calculation of the energy output of the specific source being considered.

In order to avoid errors of major magnitude in assessing ultraviolet energy, the following must be given serious consideration:

1. The spectral output of the specific source being evaluated and the spectral response of the phosphor or phototube that is being utilized in measurement of ultraviolet energy. The selection of a meter or phototube should be one that is sensitive in the range most nearly covering that part of the spectrum under consideration. Response curves of various phototubes are shown in Figure X-9.

2. Solarization and aging of lenses, tube envelopes, or cells. This can be accomplished only by calibration against a source of known wavelengths and intensity.

3. Water vapor in the atmosphere may cause absorption of ultraviolet energy as well as affecting the electronic circuitry.

4. The directionality of the meters. This is specifically true with the use of phototubes.

5. The reflection of ultraviolet from nearby surfaces or from high intensity visible light can affect most of the phototubes and

5. The reflection of ultraviolet from nearby surfaces or from

cells that are presently used for measurement of ultraviolet energy. These factors are of particular importance when measuring an intense wide-band source of ultraviolet energy.

There are several sources of commercial measuring devices available. These devices are primarily designed to measure output from specific sources. They are not in frequency ranges that can be satisfactorily utilized for purposes of evaluating exposures from wide-band sources. An attempt to use these measuring devices with specific phototubes or phosphors can be extremely hazardous and give erroneous results when attempts are made to utilize them for wide-band ultraviolet energy.

Most of the devices are marketed as being sensitive at a specific wavelength. However, it should be pointed out that the ultraviolet response may be much wider than the one wavelength indicated; and the relative spectral response of each filter or phototube must be known to reasonably assess the exposure to ultraviolet energy. Examples of spectral response of several phototubes and filters are included in Figures X-9, X-10 and X-11.

An ultraviolet device to measure broad-band ultraviolet energy is not presently available. However, it is possible to construct through a series of filters and phototubes a reasonable assessment of the levels of ultraviolet energy to which a worker may be exposed.

Narrow-band interference filters are commercially available for 254 nm, 280 nm, 297 nm, and 313 nm. When the emitted ultraviolet radiation is known to be at one of these wavelengths, single

interference filters can be used for evaluating the hazard. Filters with peak spectral response (see Table X-10) corresponding to that of the emitted radiation should be used. Care should be taken so that visible light does not affect the measurement.

IX. APPENDIX II

DEFINITIONS AND CONVERSION FACTORS

Action Spectrum - An action spectrum is a range of wavelengths in which biological effectiveness can be defined.

Biological Effectiveness - The biological effectiveness is a measure of the effectiveness of radiation at different wavelengths (within a defined range or action spectrum) in carrying out a specific reproducible photobiological process.

Irradiance - The unit of radiant power per unit area (Watt/cm^2) is the irradiance.

MED - Minimal erythema dose.

Radiant Exposure (Dose) - The unit of radiant energy per unit area (joules/cm^2) is the radiant exposure.

Relative Biological Effectiveness - The relative biological effectiveness is an

experimentally determined ratio of an absorbed dose of radiation to an absorbed dose of a reference radiation required to produce an identical biological effect in a particular organism or tissue.

CONVERSION FACTORS

Radiant Energy Units

	erg	joule	W sec	μW sec
erg=	1	10^{-7}	10^{-7}	0.1
joule=	10^{7}	1	1	10^{6}
W sec=	10^{7}	1	1	10^{6}
μ W sec=	10	10^{-6}	10^{-6}	1

Radiant Exposure (exposure dose) Units

	erg/cm^2	joule/cm^2	W sec/cm^2	μW sec/cm^2
erg/cm^2=	1	10^{-7}	10^{-7}	0.1
joule/cm^2=	10^{7}	1	1	10^{6}
W sec/cm^2=	10^{7}	1	1	10^{6}
μW sec/cm^2=	10	10^{-6}	10^{-6}	1

Irradiance (exposure dose rate) Units

	erg/cm^2·sec	joule/cm^2·sec	W/cm^2	μW/cm^2
erg/cm^2·sec=	1	10^{-7}	10^{-7}	0.1
joule/cm^2·sec=	10^{7}	1	1	10^{6}
W/cm^2=	10^{7}	1	1	10^{6}
μW/cm^2=	10	10^{-6}	10^{-6}	1

TABLE X-1

Occupations Potentially Associated with
Ultraviolet Radiation Exposures

Aircraft workers
Barbers
Bath attendants
Brick masons
Burners, metal
Cattlemen
Construction workers
Cutters, metal
Drug makers
Electricians
Farmers
Fishermen
Food irradiators
Foundry workers
Furnace workers
Gardeners
Gas mantle makers
Glass blowers
Glass furnace workers
Hairdressers
Herders
Iron workers
Lifeguards
Lithographers
Metal casting inspectors
Miners, open pit
Nurses
Oil field workers
Pipeline workers
Plasma torch operators
Railroad track workers
Ranchers
Road workers
Seamen
Skimmers, glass
Steel mill workers
Stockmen
Stokers
Tobacco irradiators
Vitamin D preparation makers
Welders

From Reference[1]

TABLE X-2

Number of Workers Exposed to Ultraviolet Radiation (Estimate from Chicago Metropolitan Survey Extrapolated to U.S. Population)

Manufacturing	
Standard Industrial Classifications 19-39	211,000
Transportation & Communication	
Standard Industrial Classifications 40-49	49,000
Wholesale, Miscellaneous Retail, Service Stations	
Standard Industrial Classifications 50,59,55	17,000
Services	
Standard Industrial Classifications 70-89	41,000
Total	320,000*

Sources: Welding (Arc)
Air Purifiers
Sanitizers

*Not equal to sum across Standard Industrial Classification because of rounding.

TABLE X-3

Summary of Minimum Erythema Dose (MED) Values in Humans

Investigators	Wavelength nm	MED	
		$\mu W\ sec/cm^2 \times 10^4$	mJ/cm^2
Luckiesh, Holladay, and Taylor, 1930[28]	297	4.3	43
Coblentz, Stair, and Hogue, 1932[44]	297	1.9-6.4	19-64
Olson, Sayre, and Everett, 1966[103]	300	2.42	24.2
Freeman, Owens et al., 1966[33]	300	1.4	14
Berger, Urbach, and Davies, 1968[34]	297	1.14	11.4
Cripps and Ramsay, 1970[145]	300	1.16	11.6

TABLE X-4

Ultra-Violet Transmissivity of Fabrics*

Material	Transmissivity, %
Batiste, white (Muslin)	50
Cotton voile	37-43
Kapron	31
Crepe de Chine (l. grey)	32.5
Kapron and Nylon	26.6
Nylon	25-27
Silk stockings	25
Cotton stockings	18
Stockinet	14-16.5
Linen, white, coarse	12
Rayon stockings	10.5
Satin, beige	10
Linen cambric	8-9.5
Rayon (linen type)	3.8-5.3
Wool stockinet	1.4-2.8
Flannelette	0.3
Poplin	0

*Data based on Morikofer,[146] Pfeiderer[147] and Voznesenskaia[148]

TABLE X-5

TRANSMISSION OF NOVIOL GLASSES

Wavelength in Angstroms	CG 338 Yellow Noviol C	CG 038 Lt Yellow Noviol A	CG 306 Noviol O 2 mm
		Fraction Transmitted	
3400			
3600			0
3800			0.120
4000			0.473
4200		0	0.635
4400	0	0.55	0.745
4600	0.38	0.702	0.795
5000	0.765	0.787	0.835
6000		0.825	0.880

Koller[141]

TABLE X-6

Reflectance of 253.7 nm Radiation From Various Surfaces (Summer[149])

Material	Reflectance*, %
Aluminum, etched	88
Aluminum foil	73
Chromium	45
Nickel	38
Stainless steel	20-30
Silver	22
Tin-plated steel	28
White wall plaster	40-60
White paper	25
White cotton	30
White oil paints	5-10
White porcelain enamel	5
Glass	4
Water paints	10-30

*Values obtained at normal incidence. The percentage reflectance increases rapidly at angles greater than 75%.

TABLE X-7

REFLECTION OF WHITE PIGMENTS AND OTHER MATERIALS*

	2537 Å in Percent	2967 Å in Percent	3650 Å in Percent	Visible Light in Percent
Pressed zinc oxide	2.5	2.5	4	88
Barytes	65	70	77	86
Titanium oxide	6	6	31	94
Pressed magnesium oxide	77	86	87	93-95
Smoked magnesium oxide	93	93	94	95-97
Pressed calcium carbonate	78	83	86	96
White wall plaster	46	65	76	90
S.W. white Decotint paint	33	41	58	79
Kalsomine white water paint	12	20	40	70
Albastine white water paint	10	14	45	78
White porcelain enamel	4.7	5.4	63	80
Flat black Egyptian lacquer	5	5	5	5
Five samples of wallpaper	18-31	21-40	33-50	55-75

*M. Luckiesh: Applications of Germicidal, Erythemal and Infrared Energy. New York, D. Van Nostrand Co., 1946, p. 383.

TABLE X-8

ULTRAVIOLET REFLECTANCE OF DRY WHITE PIGMENTS*

Pigment	Ultraviolet Reflectance Factor in Per Cent
Lead-free zinc oxide	3
35% leaded zinc oxide	4
Zinc Sulfide	6
Titanox B	6
Lead titanate	6
Titanium dioxide	7
Titanox C	7
Lithopone	8
Antimony oxide	17
Zirconium oxide (commercial)	41
Diatomaceous silica (Celite 110)	45
Basic sulfate white lead	48
China clay	54
Aluminum oxide	55
Basic carbonate white lead (Dutch process)	62
Aluminum hydroxide	67
Zirconium oxide, C.P.	78
Magnesium carbonate (commercial)	81

*D. F. Wilcock and W. Soller: Ind. Eng. Chem. 32: 1446, 1940

Note: Lead-based pigments must not be applied where their use might result in ingestion; lead-based pigments will be limited in paints for the home by Food and Drug Administration regulations.

TABLE X-9

REFLECTANCE OF PAINTS WITH WHITE PIGMENT SUSPENDED IN SILICONE*

Pigment	Reflectance at 3000 Å	Reflectance at 4000 Å
Zinc sulfide†	5%	58%
Antimony oxide†	8%	70%
Calcium carbonate†	22%	33%
China clay†	5%	27%
Basic white lead carbonate†	15%	65%
Leafing aluminum flake	63%	66%

*From W.A.D.D. Technical Report 60-703, Part III F.M. Noonan, A.L. Alexander, J.E. Cowling, U.S.N.R.L.
†30% pigment volume.

Note: Lead-based pigments must not be applied where their use might result in ingestion; lead-based pigments will be limited in paints for the home by Food and Drug Administration regulations.

TABLE X-10

Properties of Typical Ultraviolet
Interference Filters

Peak Spectral Response	Half Power Bandwidth
254 nm $\pm$ 1.5 nm	15 $\pm$ 2.5 nm
280 nm $\pm$ 1.0 nm	10 $\pm$ 2.5 nm
297 nm $\pm$ 1.0 nm	10 $\pm$ 2.5 nm
313 nm $\pm$ 0.8 nm	8 $\pm$ 2.0 nm

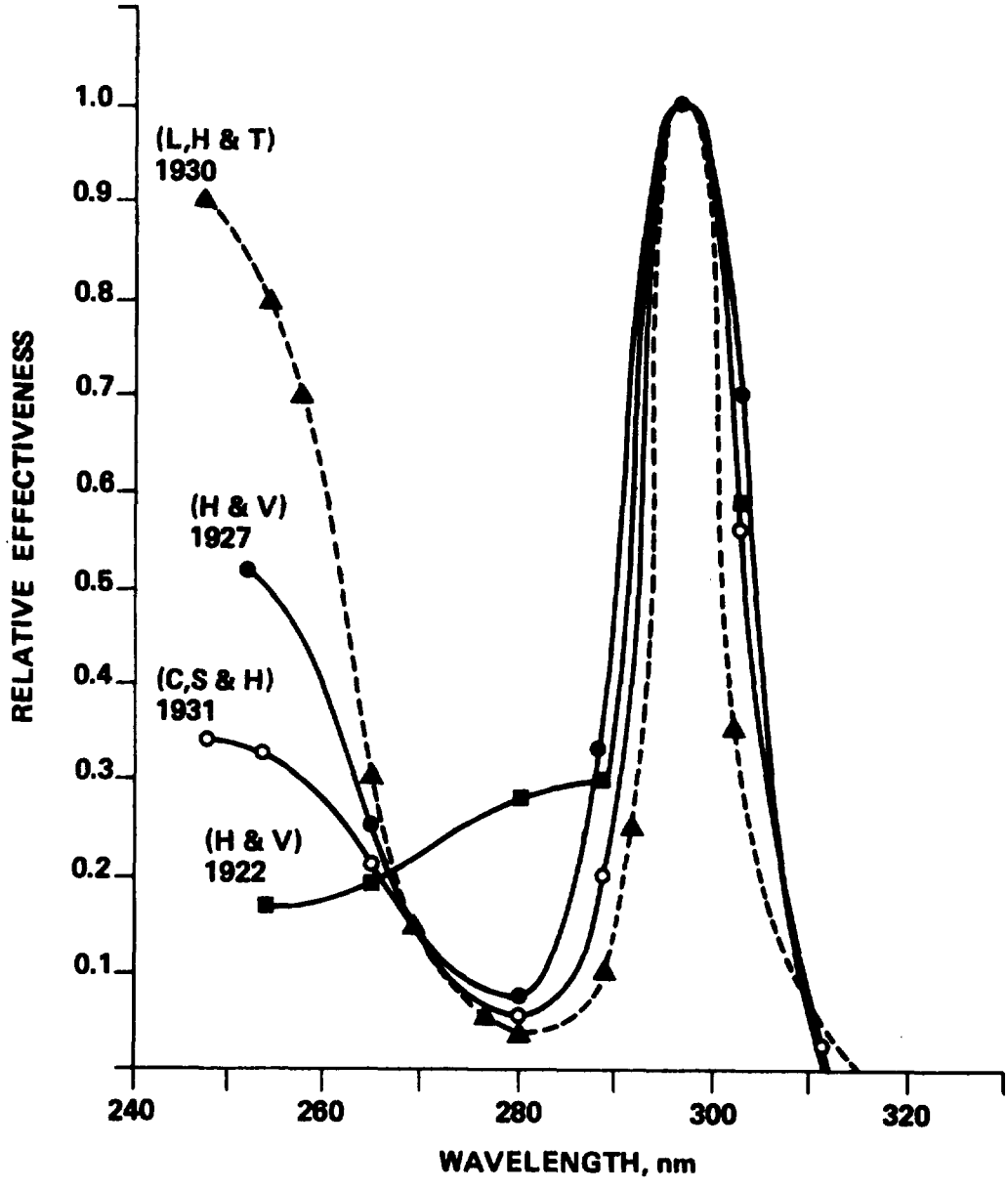

Figure X-1. Erythema action spectra (previous observers). From Everett, Olson, and Sayer.[32]

Luckiesh, Holladay, and Taylor[28] (L, H, & T)
Hausser and Vahle[26] (H & V)
Coblentz, Stair, and Hogue[29] (C, S, & H)

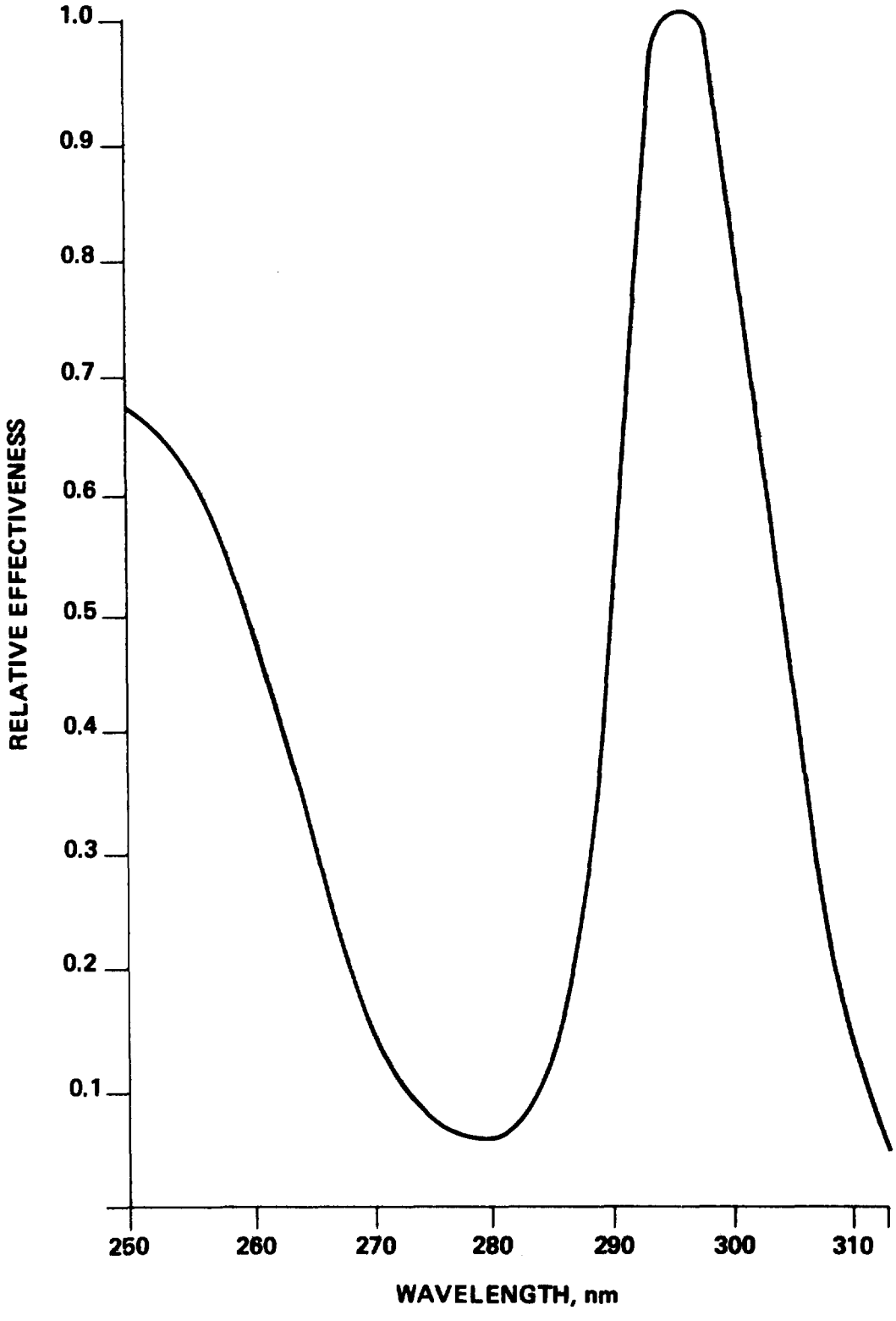

Figure X-2. "Standard" curve for erythemal effectiveness.

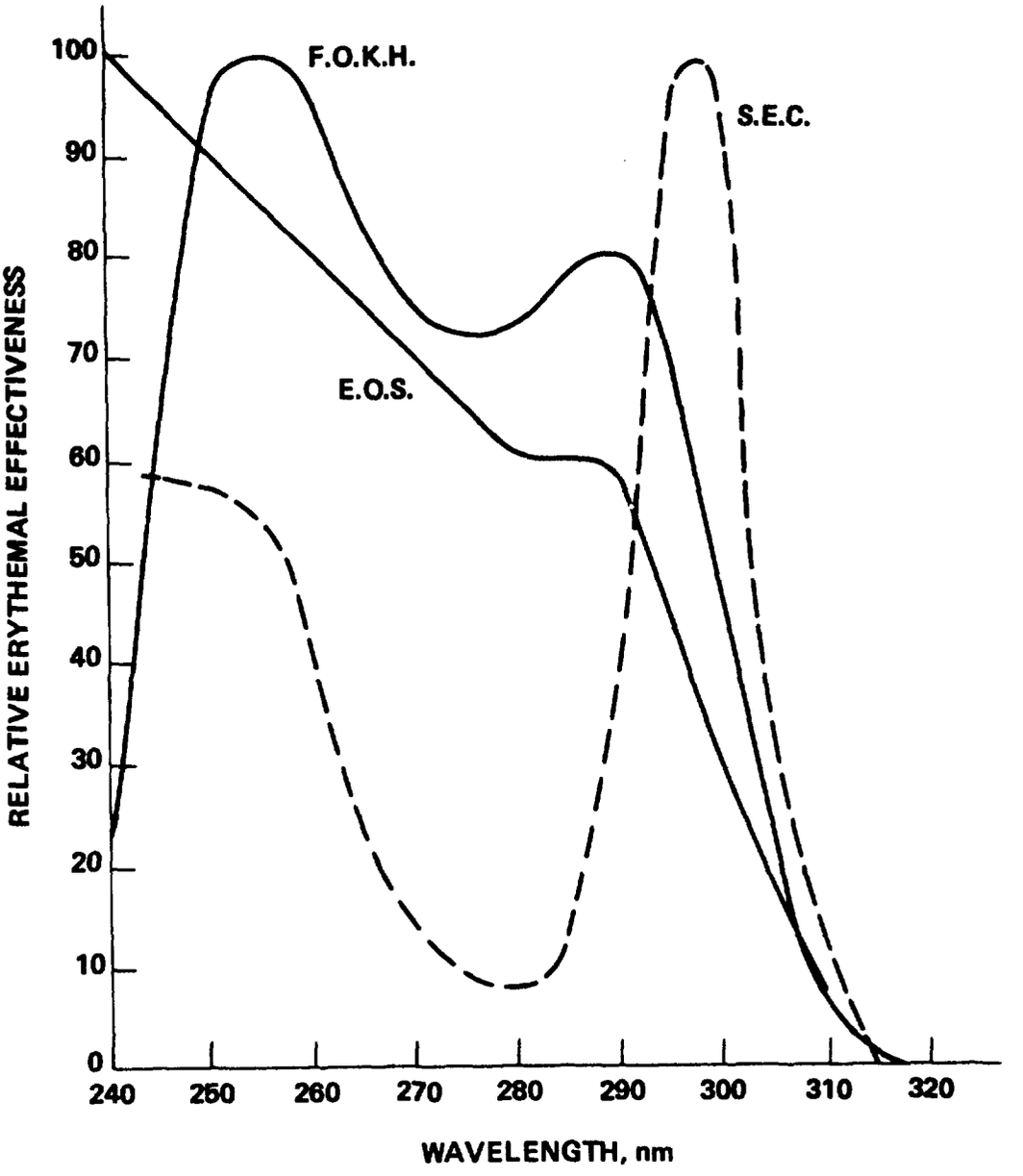

Figure X-3. Comparison of Standard Erythemal Curve (S.E.C.) with relative erythemal effectiveness curves of Everett, Olson, and Sayre (E.O.S.) and Freeman, Ownes, Knox, and Hudson (F.O.K.H.). From Matelsky.[119]

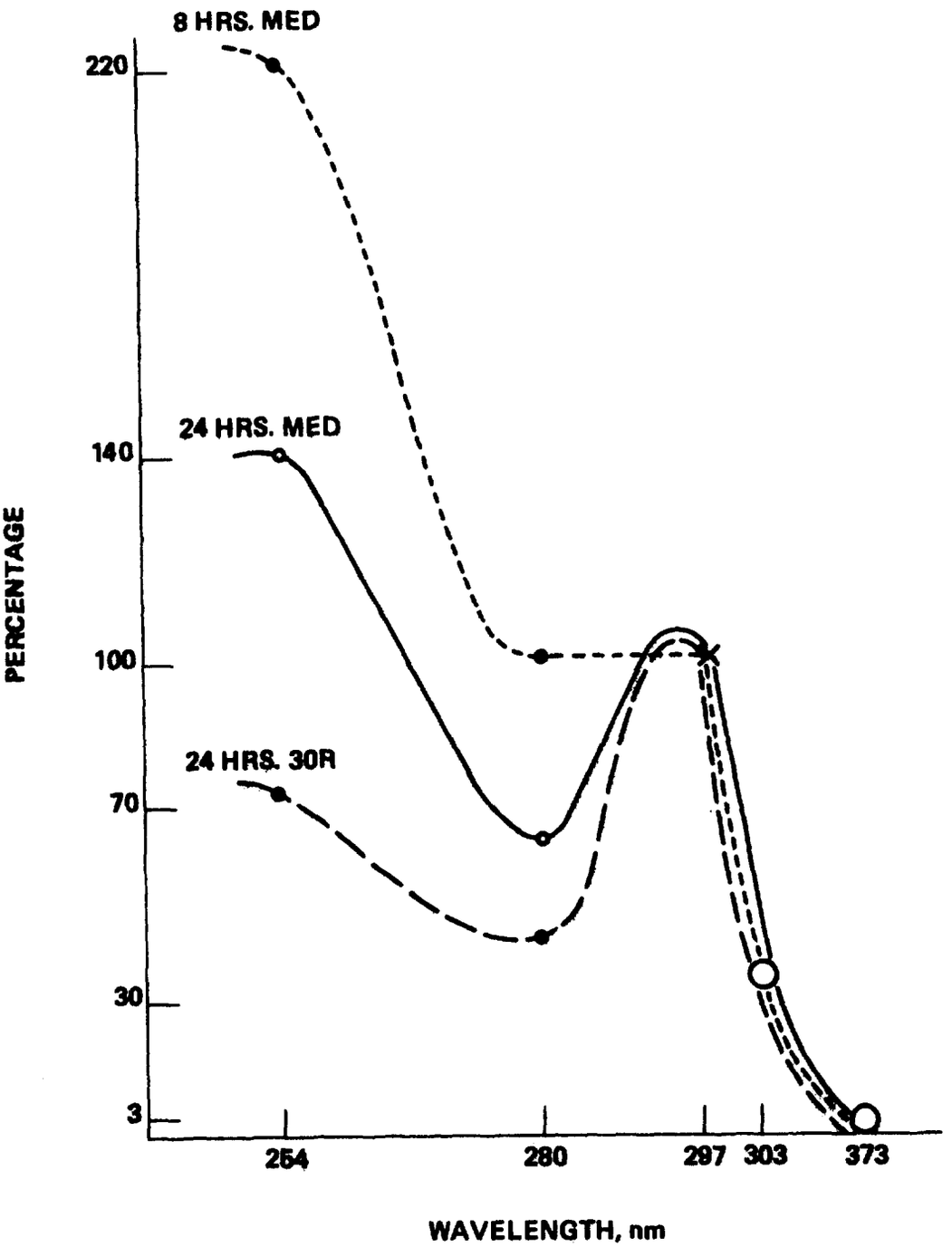

Figure X-4. "Action Spectrum" of Human Skin. Averages of values for five subjects, abdominal skin, second exit slit. Note great similarity for wavelengths from 297 to 313 nm. and marked differences for 8hr. MED, 24 hr. MED and a curve constructed by using values for moderate erythema (Kodak Color Balancing Filter 30 R). From Berger, Urbach, and Davies.[34]

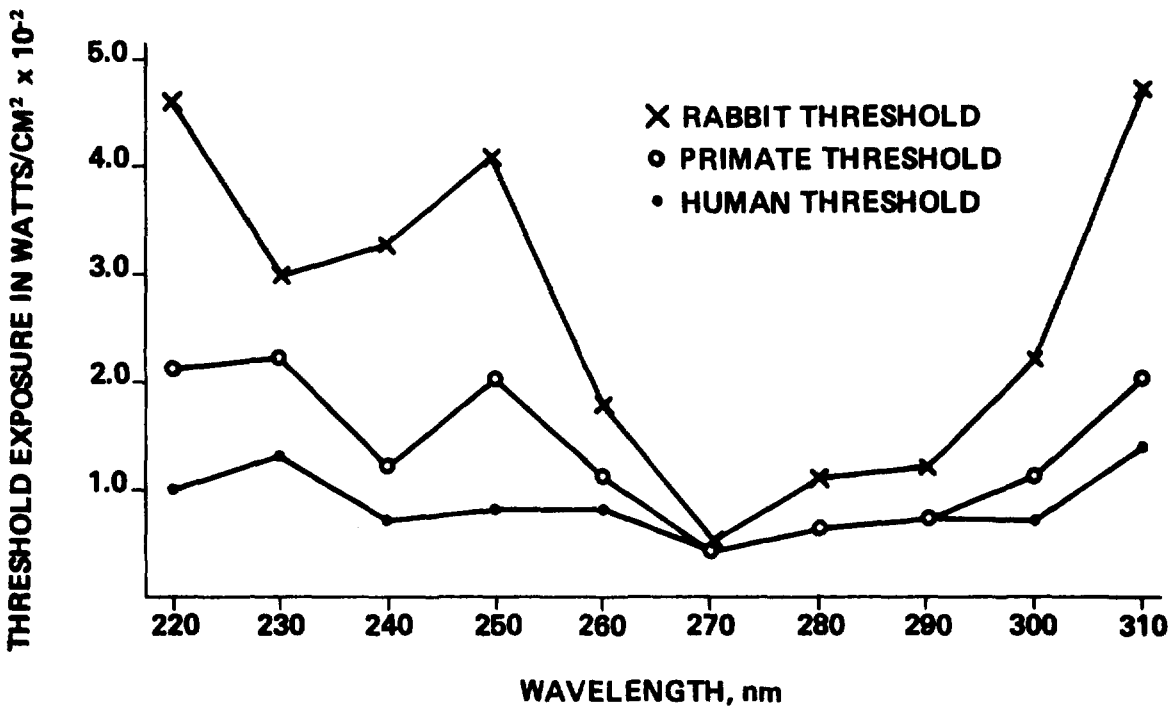

Figure X-5. Comparison of the ultraviolet action spectrum for the rabbit, primate, and human. From Pitts and Gibbons.[9]

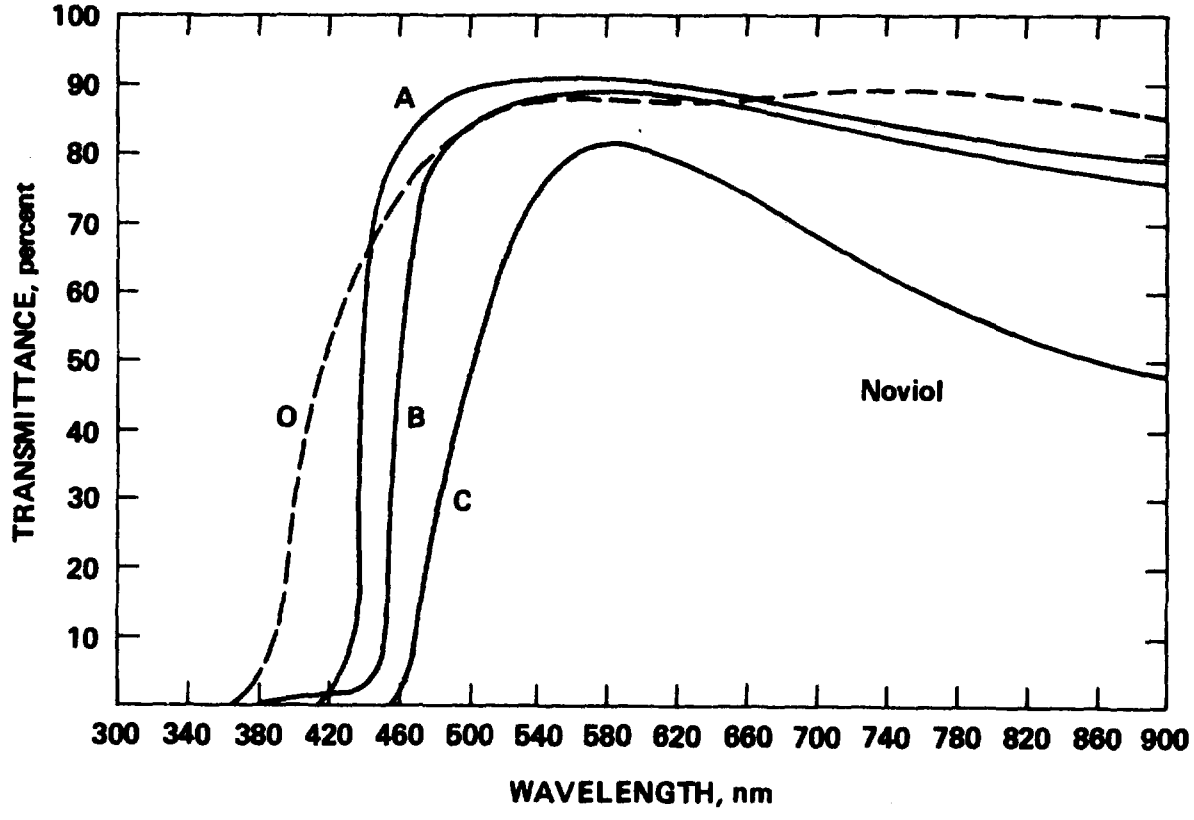

Figure X-6. Spectral transmittance of Noviol glass. Noviol O, thickness 2.63 mm. Noviol A, thickness 1.90 mm. Noviol B, thickness 2.89 mm. Noviol C, thickness, 3.05 mm. From reference 140.

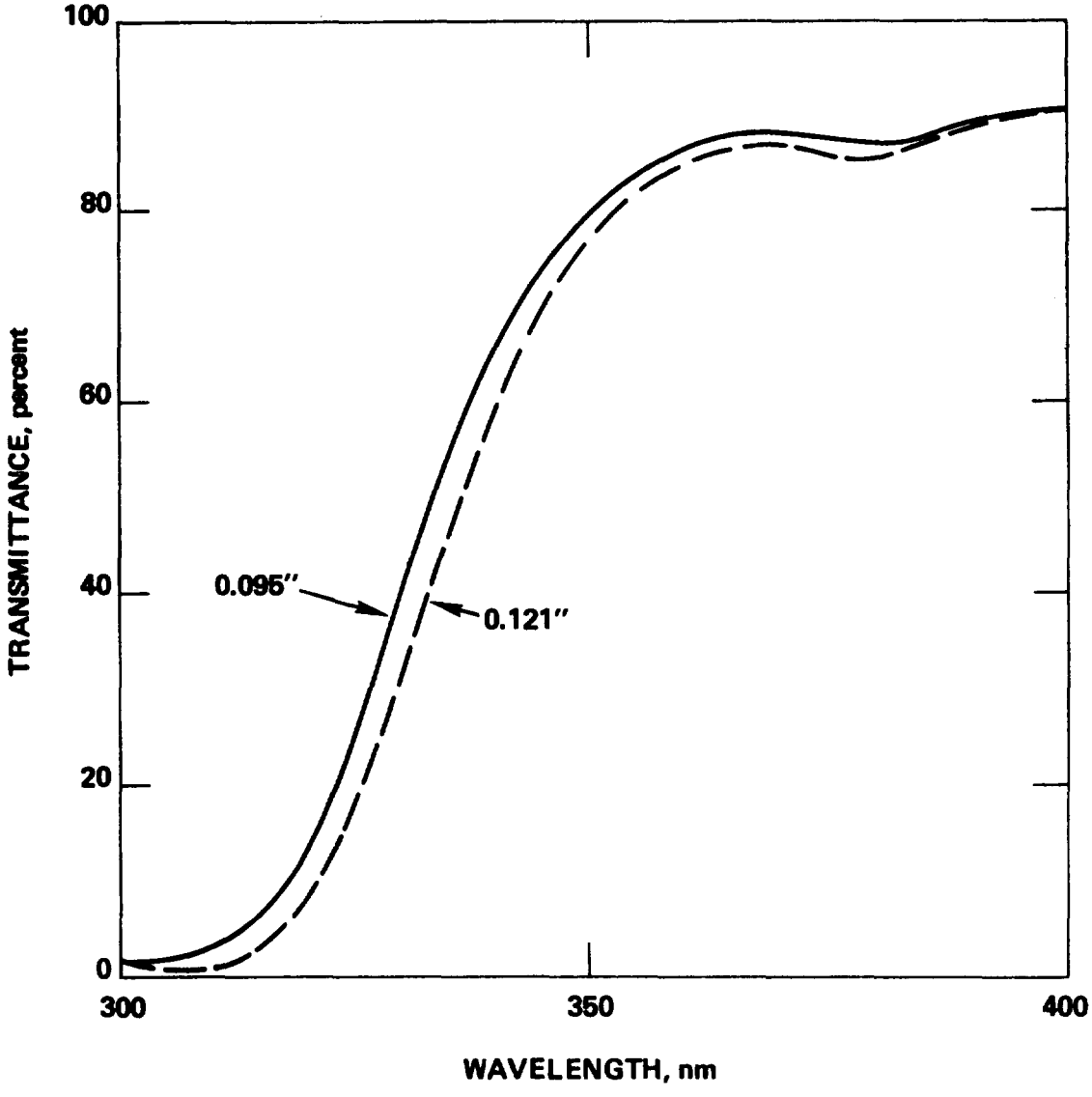

Figure X-7. Transmission for two thicknesses of window glass. From Koller.[141]

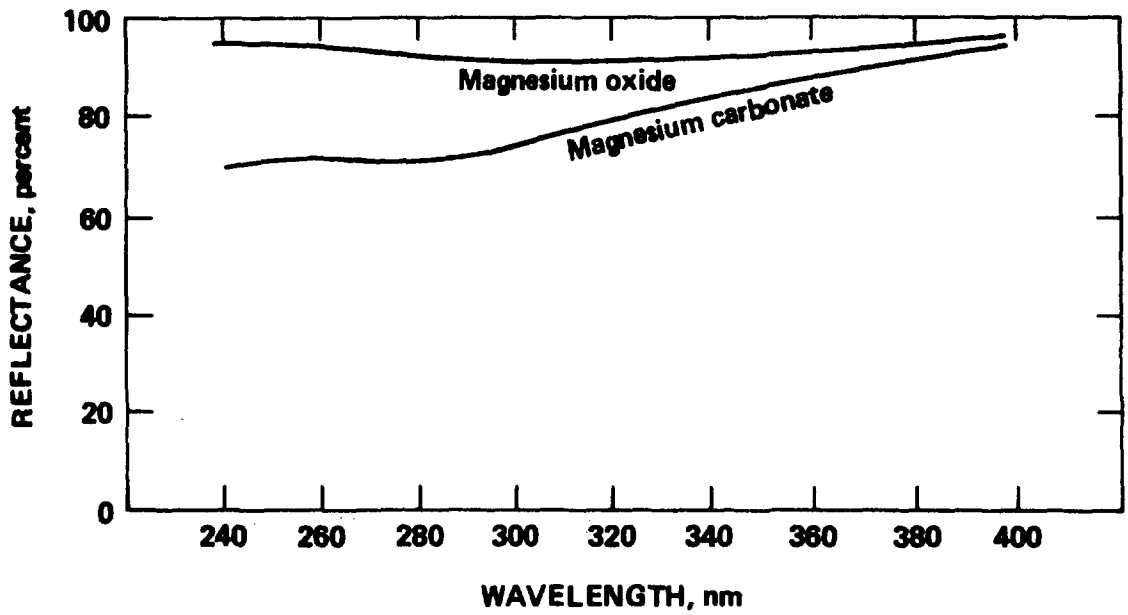

Figure X-8. Reflection from magnesium oxide and magnesium carbonate. From Benford F, Schwartz S, and Lloyd G, J Opt Soc Am 38: 964, 1948.

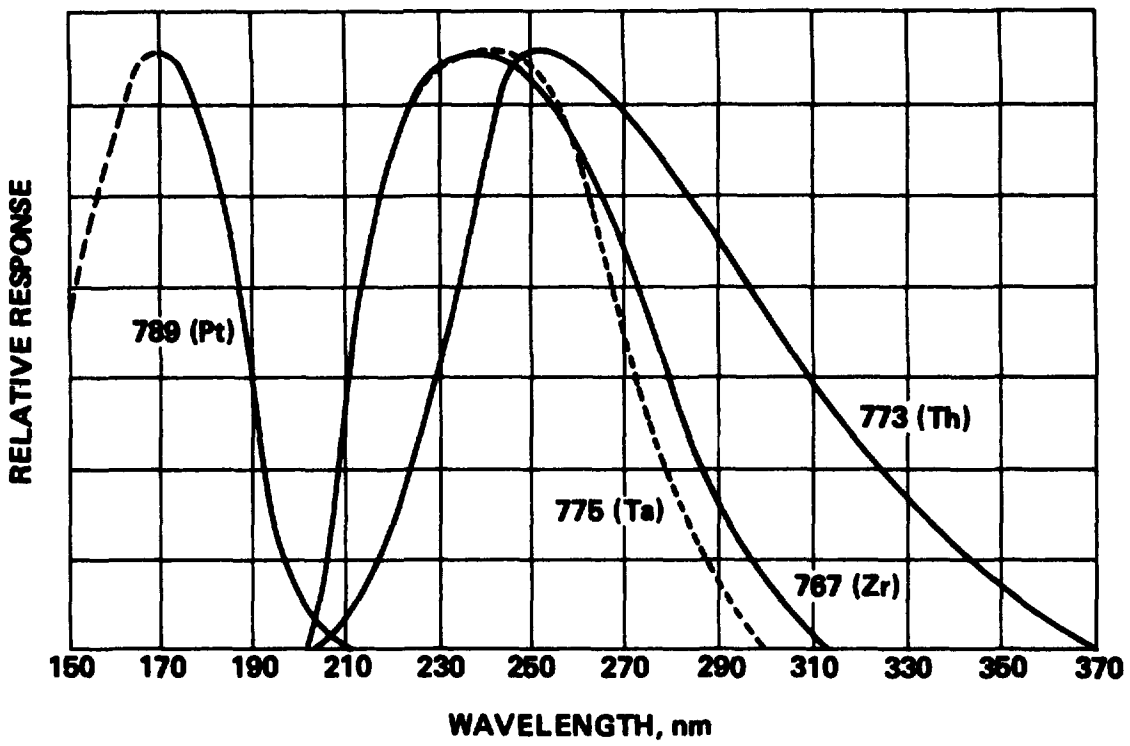

Figure X-9. Response of various phototubes. From Fanney JH, Powell CH: Field measurement of ultraviolet, infrared, and microwave energies, Am Ind Hyg Assoc J 28: 335-42, 1967.

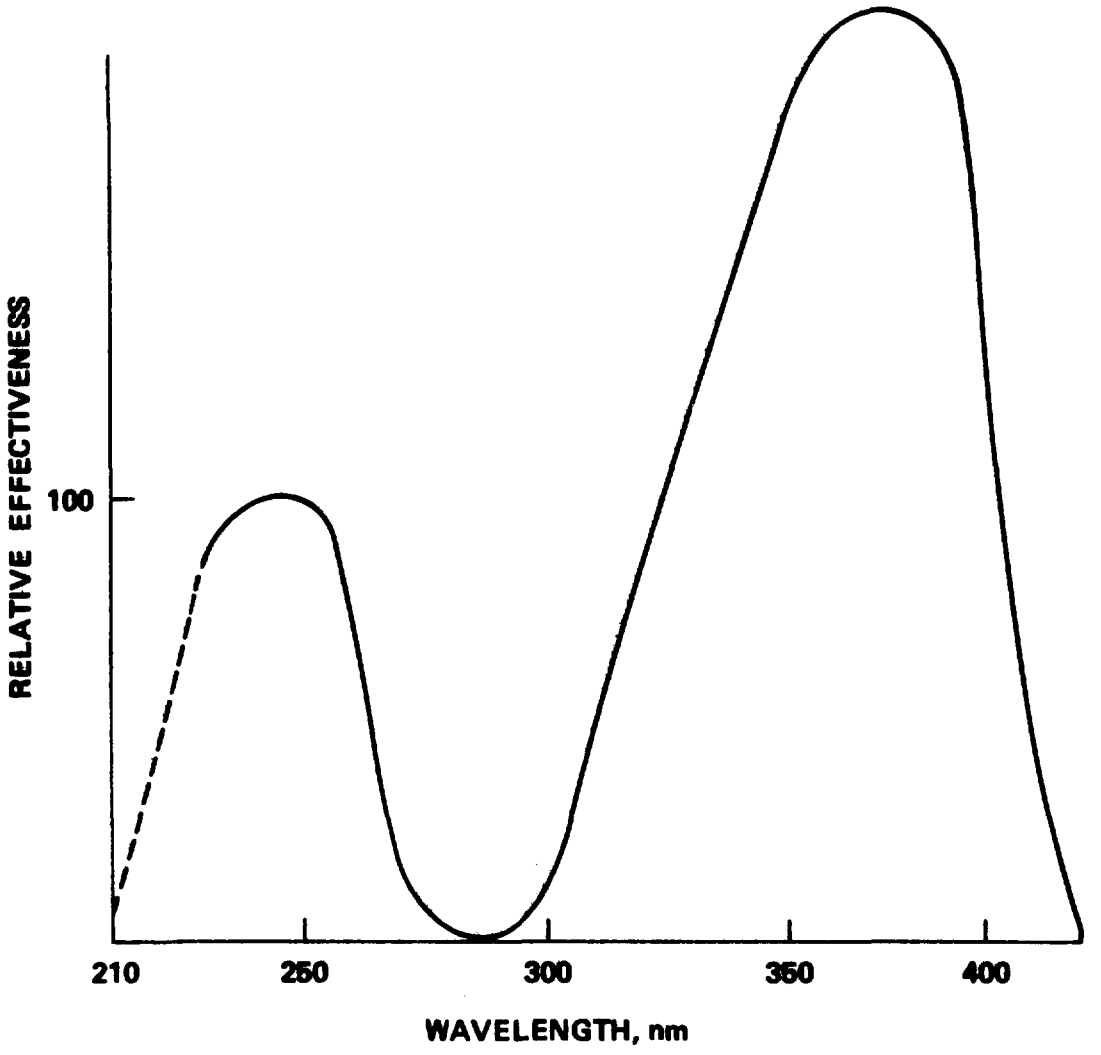

Figure X-10. Relative spectral response of a short-wavelength filter for ultraviolet meter. From Powell, Goldman, and Key.[14]

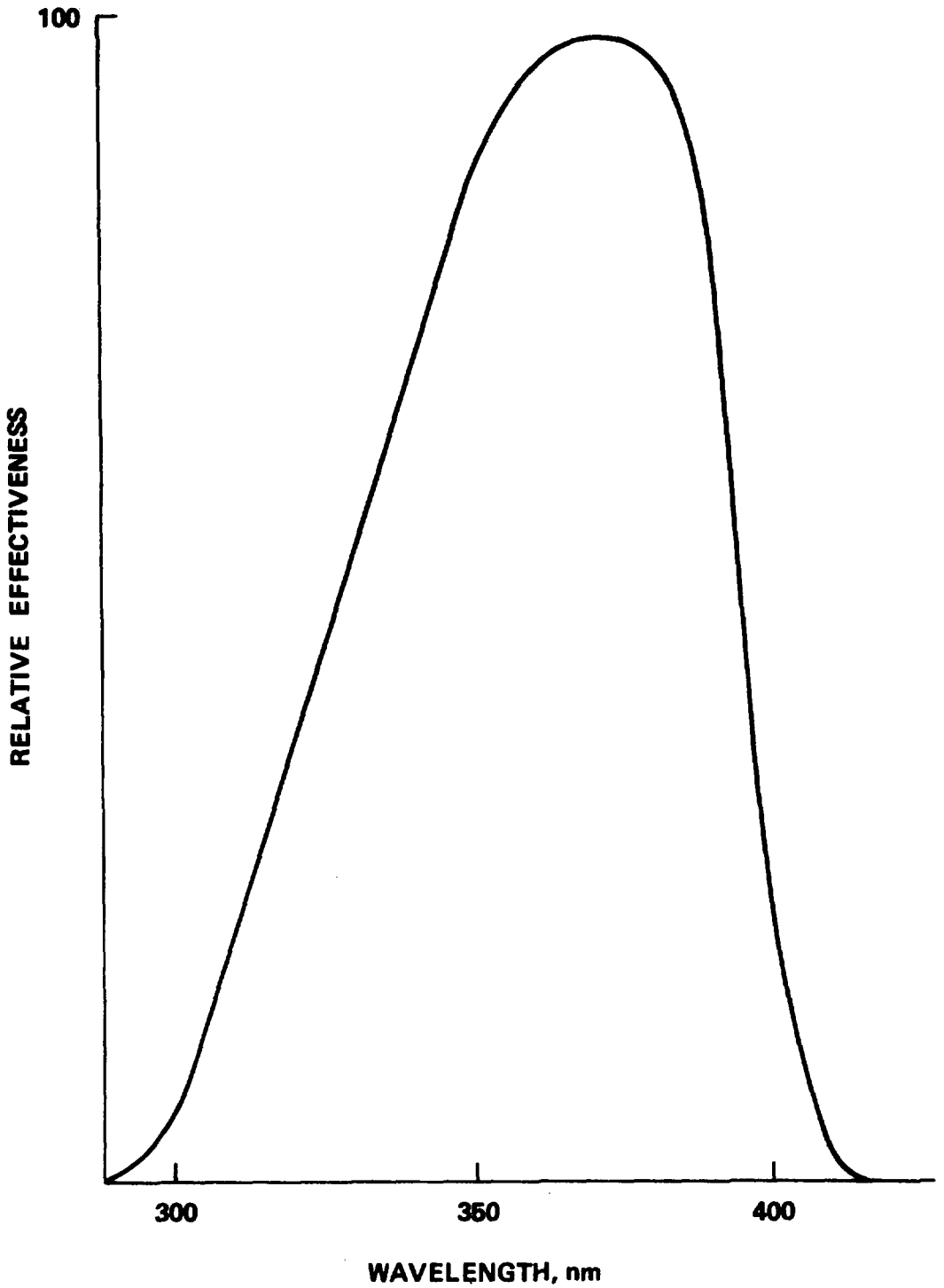

Figure X-11. Relative spectral response of a long-wavelength filter for ultraviolet. From Powell, Goldman, and Key.[14]

*U.S. GOVERNMENT PRINTING OFFICE: 1992-648-004/60050