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BIOLOGICAL EFFECTS OF INFRARED RADIATION

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ABSTRACT

The continual growth of glassmaking, steel, foundry, welding, and related industries has contributed to a marked increase in human exposure to infrared radiation (IR). New IR sources, such as communication systems and industrial heat sources, have also generated additional occupational concern in that they could result in more workers being exposed to IR.

The purpose of this IR literature review was to determine the current state of scientific knowledge regarding human biological effects resulting from exposure to noncoherent IR sources. The review includes research reported in both domestic and foreign publications, governmental hearings, symposia, conferences, and contract reports. All of the hazards reported in the literature are reviewed and discussed in detail. Ocular hazards received primary coverage, followed by skin hazards and "other" hazards. The report also presents additional information on types of IR sources, modes of biological interaction, existing exposure standards, and control measures.

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INTRODUCTION

During the past 50 years there has been an increase in worker exposure to IR in the glassmaking, metal, and foundry industries, from welding operations and other hot industrial processes. The potential health effects from exposure to newer IR sources, such as industrial heating devices, is particularly of concern because an increasing number of workers may be exposed to broadband IR over long periods of time. While effects on the traditional target organs, the eye and skin, have been fairly well defined, questions persist concerning basic physiological mechanisms through which IR acts on man and its effects on other tissues and organs.

This report reviews the relevant literature to find the current state of scientific knowledge on the biological effects of exposure to broadband, noncoherent IR sources. The report presents and discusses the types and nature of IR sources, modes of interaction, means of detection, existing standards, and control measures.

BACKGROUND

THE ELECTROMAGNETIC SPECTRUM

As commonly used, the word "light" refers to the portion of the electromagnetic spectrum (EMS) that produces a visual effect. The EMS is a classification scheme used to group electromagnetic radiation according to wavelength, frequency, or energy. The radiation is propagated as transverse waves consisting of electric and magnetic fields oscillating in mutually perpendicular planes. The term "radiation" has also been defined as "radiant energy" and is similarly applied to energy that is propagated through space as electromagnetic radiation.

The several types of radiation can be described by either wave or particle characteristics. Both descriptions are necessary to explain certain physical phenomena of radiation. When considered as waves, radiation is characterized by wavelength and frequency of oscillation. The wavelength (λ) is defined as the distance between two points in a periodic wave that has the same phase; it is expressed in units of length, i.e., millimeters (mm), nanometers (nm), etc. The frequency (f) is defined as the number of waves passing a given point in unit time (one second), and is expressed in units of hertz (Hz), which is equal to one cycle per second. Wavelength and frequency are related as:

$$c = \lambda \cdot f \quad (1)$$

where c is the velocity of light.

The wave properties of radiation are evident in such phenomena as diffraction, interference, refraction, and polarization. In some circumstances electromagnetic energy behaves as a collection of particles, energy in discrete bundles, or "quanta." The particle concept of light is used to explain such phenomena as the photoelectric effect, photon emission and absorption, photon-electron interaction, etc. A single quantum of electromagnetic radiation is termed a "photon" and its energy is commonly expressed in units of electron volts (eV).

The frequency range of the EMS is very large. The physical properties of these radiations are the same, but the large frequency range requires separation into more easily manageable regions. The wavelength, energy, or frequency of the radiation is used to separate the EMS into regions such as the radiowave, microwave, IR, visible, ultraviolet (UV), X-ray, and gamma ray regions, as shown in Table 1.

The energy (E) of a photon is directly proportional to the frequency of oscillation of the specific electromagnetic radiation:

$$E = h \cdot f \quad (2)$$

where h is Planck's constant, defined as 6.626×10^{-34} joule.seconds.

If Equation 1 is used, this relationship becomes:

$$E = \frac{hc}{\lambda} \quad (3)$$

Table 1. Characteristics and sources of electromagnetic radiation

Type of radiations	Frequency range*, Hz	Wavelength range*, μm	Photon range*, eV	Energy per Photon range*, eV	Typical industrial source of exposures
Ionizing x-ray -ray	3.0×10^{15}	< 0.1	> 12.0		Electronic tubes, radiography, nuclear power plants, radiation curing, medical uses, uranium mining, sterilization processes.
Nonionizing					
Ultraviolet	7.5×10^{14} to 3.0×10^{15}	0.1 to 0.4	3.0 to 12.0		Lamps, welding arcs, gas discharge tubes.
Visible	4.0×10^{14} to 7.5×10^{14}	0.4 to 0.76	1.6 to 3.0		Lamps, welding arcs hot bodies.
Infrared	3.0×10^{11} to 4.0×10^{14}	0.76 to 1.0×10^3	1.2×10^{-3} to 1.6		Lamps, welding arcs, hot bodies.
Microwaves	3.0×10^8 to 3.0×10^{11}	1.0×10^3 to 1.0×10^6	1.2×10^{-8} to 1.2×10^{-3}		Klystron, magnetron
Radiofrequencies	$< 3.0 \times 10^8$	$> 1.0 \times 10^6$	$< 1.2 \times 10^{-6}$		Plastic sealers, furniture glue.

*The given ranges are only approximations

It is apparent from Equation 3 that the photon energy is inversely proportional to its wavelength. By substituting appropriate values, one can calculate the photon energy in eV:

$$E = \frac{12.4}{\lambda} \quad (4)$$

In this equation, λ must be in units of micrometers (μ m).

Some of the more important properties of electromagnetic radiation are that it

- o travels through empty space and air at a constant velocity (3×10^{10} cm.s⁻¹);
- o is ordinarily propagated in straight lines;
- o is reflected in traveling from one homogeneous medium to another of different density;
- o is refracted or bent in passing from one medium to another of different density;
- o is diffracted when passing an obstacle to form geometrical patterns that are not straight lines;
- o can be polarized, as can all transverse waves;
- o and interacts strongly with charged particles.

NONIONIZING RADIATION

Table 1 also shows the EMS regions that have insufficient photon energy to ionize matter. Any radiation having an energy less than about 12 eV is designated as nonionizing radiation (NIR). The modes by which NIR interacts with matter are either photochemical or thermal. Draper's Law states that no photochemical reaction can occur unless radiant energy is absorbed by matter. Such absorption results in transfer of energy to the absorbing molecules of matter. This may be accomplished by raising atomic energy levels or by increasing either intermolecular or intramolecular translational and rotational vibration modes.

The interaction of NIR with biological systems produces electron excitation and results in (a) dissociation of the molecule, if the outer shell (bonding) electrons are involved; (b) dissipation of excitation energy in the form of luminescence (delayed or immediate emission of visible radiation after exposure to exciting radiation), and/or (c) subsequent dissipation of the energy into vibrational or rotational modes, which produces heat.

The resultant biological effects depend upon the incident photon energy, the transmission characteristics of the tissue exposed, and the ability of specific molecules to be changed chemically when the photon energies are absorbed.

As the frequency of the EMS decreases, a point is reached at which the energy of the photon is insufficient under normal conditions to dislodge the atomic orbital electron and form an ion pair. The minimum photon

energy capable of producing an ion pair in atomic oxygen, hydrogen, and carbon is between 12 and 15 eV which corresponds to wavelengths of approximately 0.1 and 0.08 μm . Since these atoms are the base elements of living biological tissue, Matelsky (1) suggested that 12 eV be considered the lower limit for ionization in biological material. This means that energies below this value may generally be regarded, in terms of biological effects, as nonionizing. Conversely, energies greater than 12 eV can be regarded as biologically ionizing. It has been stated (2), however, that radiation with wavelengths less than 0.2 μm is not biologically significant because these wavelengths are strongly absorbed in air. This further reinforces the conclusion that it is impossible to specify an exact dividing line between the ionizing and nonionizing regions and the value remains arbitrary. The relative effectiveness of different wavelengths in eliciting a specific photochemical or thermal response is referred to as the "action spectrum" or spectral sensitivity for that response (3).

INFRARED RADIATION

Historical Aspects

Man has always been subject to the optical radiation emitted by the sun, fire, and other heat sources. The ability to light a fire by concentrating the sun's rays by means of concave mirrors was known in ancient times. IR was not, however, investigated scientifically until the eighteenth century. At that time the existence and the differentiation of light from heat was unknown. Light and heat were vaguely considered together as "radiant heat." In 1800, Sir William Herschel (4) investigated the thermal effect or "heating power" of each of the colors in the solar spectrum obtained with a glass prism. The prismatic spectrum was projected on a table and a mercury thermometer was used as a heat detector. The temperature was measured at different points within the spectrum and the increase in temperature measured in the solar spectrum relative to the temperature measured in the shade was evaluated. The results showed that the temperature increased from the violet to the red end of the spectrum. Herschel deduced that an "invisible light" existed below the red end of the solar spectrum. After studying the optical characteristic of the "invisible light," he concluded that it is reflected and refracted in a manner similar to visible light.

In the 30 years following Herschel's conclusions, little progress was made in IR measurements beyond establishing that it obeyed the basic laws of optics. This lack of progress was due to the lack of instrumentation more sensitive than the ordinary thermometer.

In 1831, Nobili and Melloni (5) greatly improved IR detection techniques by using the more sensitive thermopile instead of thermometers. They noted that IR consisted of rays of different wavelengths, just as light consists of rays of different colors. They also supported Herschel's observations regarding heat and light effects by suggesting that the same radiation produces different effects, depending on whether it is observed by the eye or a thermopile.

In 1835, Ampere (6) hypothesized that light and IR had similar characteristics. Not all investigators agreed with this claim and the resulting controversy was not settled until 1847 when Fizeau, Foucault, and Knoblauch (7) showed that IR and light exhibit identical physical properties.

After the physical similarities between IR and light were established, adequate methods were sought to measure wavelengths in the IR region, especially the long-wave limit of the IR spectrum. Precision instrumentation was not available and initial measurements made by prismatic or interferometric techniques were unreliable. In 1880, a major advance in measurement technique resulted from Langley's (8) invention of the bolometer to measure radiant heat by registering the change in conductivity. The sensitivity of the bolometer was much greater than that of the thermopile used by Melloni. Rubens and Nichols (6) were able to estimate the long-wave limits of the IR spectrum to be 20 μm and further instrument refinement and development enabled later investigators (9) to extend wavelength measurements.

In this century more sophisticated detection and measurement techniques using photoelectric phenomena have characterized the IR spectrum in more detail. The 20th century also marked the beginning of IR transmission and absorption studies in ocular tissues. World War II and the expanding use of IR in industry stimulated technical developments and health hazard research.

Properties

The IR portion of the EMS extends from the visible red light to the microwave region. All objects, blackbody or otherwise, emit IR as a function of temperature. Although a continuum of IR wavelengths is emitted (Figure 1), the wavelength of maximum intensity is determined by Wien's Displacement Law (10) shown in Equation 5:

$$m = \frac{2898}{T} \quad (5)$$

where T is the temperature in Kelvin and μm is the wavelength in μm . Figure 1 shows that increasing the source temperature causes the peak of the radiation curve to move toward shorter wavelengths and the intensity of the emitted radiation to increase. Physical matter emits IR in accordance to blackbody radiation laws, except for a factor of proportionality called emissivity. The emissivity factor, which is a fraction of unity, equals one for a blackbody.

The properties of IR photons are similar to those of other EMS photons. IR travels in straight lines from the source, obeys the inverse square law, and is propagated in a vacuum as well as in other media. IR is not transferred by thermal convection or conduction in a physical medium and is often incorrectly referred to as heat waves because it generates heat in any absorbing medium. IR can also undergo several interactions, including reflection, absorption, transmission, refraction, and diffraction.

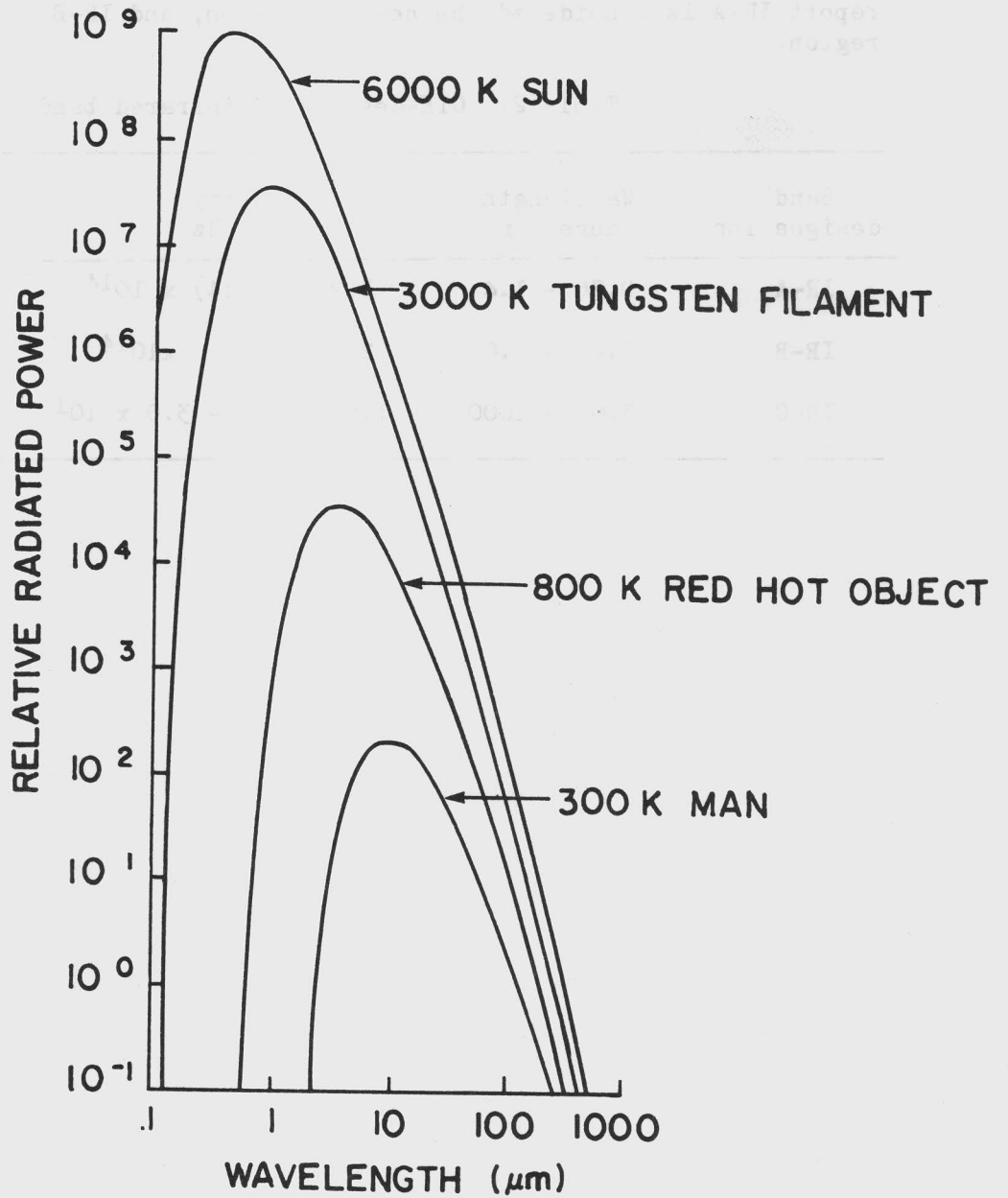


Figure 1. Emission of optical radiation from blackbodies at various temperatures.

The IR region is often divided into specific zones to categorize various biological effects. There is much disagreement among biologists in defining these zones. In an attempt to clarify the situation, the International Commission on Illumination (CIE) (11), has defined three biologically significant bands, that are defined in Table 2. In this report IR-A is considered the near IR region, and IR-B and IR-C the far region.

Table 2. CIE-designated infrared bands

Band designation	Wavelength range, μm	Frequency range, Hz	Energy range, eV
IR-A	0.76 - 1.4	$(3.95 - 2.14) \times 10^{14}$	1.63 - 0.89
IR-B	1.4 - 3.0	$(2.14 - 1.0) \times 10^{14}$	0.89 - 0.41
IR-C	3.0 - 1000	$1.0 \times 10^{14} - 3.0 \times 10^{11}$	0.41-0.001



MEASUREMENT OF INFRARED RADIATION

Quantities and Units

Any discussion of IR hazards requires mention of several physical terms. In specifying exposure levels, one normally uses "radiant exposure" in joules per square centimeter ($J.cm^{-2}$) or "irradiance" in watts per square centimeter ($W.cm^{-2}$) (11). For small wavelength intervals, "spectral irradiance" ($W.cm^{-2}.nm^{-1}$) or "spectral radiant exposure" ($J.cm^{-2}.nm^{-1}$) are used. Retinal exposure for extended IR sources is expressed in terms of "radiance" in watts per square centimeter per steradian ($W.cm^{-2}.sr^{-1}$) or "integrated radiance" in $J.cm^{-2}.sr^{-1}$. A summary of radiometric terms and units has been prepared by Meyer-Arendt (12).

INSTRUMENTATION

When IR impinges upon matter, the portion absorbed increases the energy content of the absorbing material, resulting in a temperature increase. This increase can be measured using secondary effects, such as the variation in physical properties (volume, pressure, refractivity, conductivity, thermoelectricity, pyroelectricity, electron emission) or chemical properties.

The basic function of any IR detector is to convert radiant energy into another form of energy that can be easily measured. The two categories of IR detectors are thermal and photonic. Thermal detectors rely on a rise in temperature in the detecting medium, which causes a change in resistance or capacitance. This change provides a signal proportional to the radiant energy absorbed. These detectors respond to a broad IR spectrum, require no cooling, have low sensitivity, have a response time measured in milliseconds, and are relatively inexpensive. The spectral response of these detectors, e.g., thermistor, bolometer, thermopile, and pyroelectric, depends on the absorption properties of the material used.

Photon detectors are semiconductors with which incident IR photons interact to produce electrons. These detectors usually respond to a narrow range of wavelengths, require cooling, have fast response times, and are more expensive than thermal detectors. The essential difference between the two types is that the photon detector determines the number of quanta per second absorbed, whereas the thermal detector depends on the total power absorbed.

A narrow band spectral filter can be applied to the incident IR energy to selectively transmit to the detector only certain wavelength bands. These filters are made by vacuum depositing thin films of materials that have high transmission properties in the IR region onto suitable transparent substrates.

Various models of spectroradiometers are commercially available that will measure the spectral energy distribution of IR sources, but most of these

devices become expensive when spectral information is required beyond 1.2 μm . When this is the case, radiometers with special filters can be used. Additional information on IR detectors can be found in References 13 and 14.

Considerations of the mechanical, electrical, and thermal variations in the detector; spectral response; aging of components; methods of calibration; atmospheric contaminants; and reflections are very important when making laboratory and field measurements. The investigator must understand the characteristics and limitations of the instrumentation to reliably and accurately assess the biological effects or hazards.

SOURCES AND USES OF INFRARED RADIATION

The many types of IR sources fall into one of two classifications: artificial or natural. Both are found in the work environment.

ARTIFICIAL SOURCES

Artificial sources includes various types of commercial incandescent, fluorescent, and high-intensity-discharge lamps; artificial blackbody sources; metal rod heaters; hot metal; and glass. These broadband sources require filters to limit their output to a specific wavelength band.

NATURAL SOURCES

Natural IR sources are numerous and cannot be readily controlled by man. The most significant natural source is the sun. The total radiant power received from the sun is approximately $135 \text{ mW}\cdot\text{cm}^{-2}$ normal to the earth's surface (15). Approximately one-half of the total radiant power hitting the earth is in the IR region. The sun resembles a blackbody at a temperature of 6000 K and peaks at a wavelength of about $0.55 \mu\text{m}$, although the radiation extends through the UV and into the IR region.

Occupational Exposure

IR normally is considered an occupational rather than an environmental hazard and is regarded as such in this report. In many industries a wide range of IR wavelengths occurs from artificial sources.

The following are some common occupations that have potential for IR exposure:

Bakers and cooks	Glass furnace workers
Blacksmiths	Heat treaters
Braziers	IR laser operators
Chemists	Ironworkers
Cloth inspectors	Kiln operators
Construction workers	Motion picture machine operators
Dryers, lacquer	Plasma torch operators
Electricians	Roofers
Farmers	Skimmers, glass
Firemen, stationary	Solderers
Foundry workers	Steam locomotive, fireman
Furnace workers	Steel mill workers
Gas mantle hardeners	Stokers
Glass blowers	Welders

It should be emphasized that workers in other occupations may be exposed when working conditions dictate that they occupy areas close to IR sources.

POPULATION AT RISK

Little information is available on the types of IR sources and the number of workers exposed. Moss et al. (16) estimated the number of American workers potentially exposed to electromagnetic radiation by occupational group. The conclusions drawn concerning the number of workers potentially exposed to the optical radiation region (UV, visible, and IR) are:

- o The number of workers potentially exposed to optical radiation is estimated to be more than 100 million.
- o The two occupational categories for which exposure is the highest are operative and clerical workers, each of which represents about 17% of the total workforce.
- o Approximately 20% of the workforce receives its major exposure from the sun; most workers are exposed to lamps of some type.

It should be noted that the optical radiation region was analyzed collectively rather than individually because many sources emit UV, visible, and IR radiation simultaneously.

BIOLOGICAL EFFECTS OF INFRARED RADIATION

INTRODUCTION

Generally, IR cannot enter into photochemical reactions in biological systems because of the low photon energy; however, absorption of IR photons increases the kinetic energy of the tissue when the radiant energy is converted to heat. The resulting rise in temperature depends on the wavelength, exposure duration, and total energy absorbed by the tissue. A review of the literature reveals that most research on IR biological effects pertains to ocular effects. Considerably fewer studies have investigated the effect on the skin and other tissues and organs.

IR from all sources constitutes an important component of the microclimate. Traditionally, far IR has been associated with radiant heat such as that found in glassblowing operations, foundries, furnaces, etc. Occupational exposure to high levels of radiant heat in the far IR region may induce a thermal stress condition (17). This effect of industrial IR on thermal stress in the workplace is an important one and normal safety precautions should be taken against such heat stress. For purposes of this review the division between near and far IR regions is assumed to be at 3 μm .

OCULAR EFFECTS

Background

The eye possesses certain protective mechanisms (e.g., blink and pupil reflexes), that are adequate in the natural environment, where IR usually is accompanied by intense visible light. Some industrial IR sources do not emit intense light so these reflexes are not triggered (see Figure 2). Because of its vascularity, the retina can dissipate the heat produced by absorbed radiation fairly well. Other ocular structures do not have this capability, and, therefore, can absorb significant amounts of IR, depending on the wavelength.

An important feature of the eye is its focusing ability. The optic media focuses the incident radiation so that a significant point concentration of energy is produced. The degree of concentration depends on the size of the radiation source and the spread of the incident flux within the eye.

The following sections describe the structure and function of the eye and its optical characteristics.

Anatomy and Physiology

The adult human eyeball is approximately spherical in shape, has a diameter of about 24 mm, and weighs 6 to 8 gm (18). Figure 3 shows the general structure of the human eye. The eyeball is composed of three layers: sclera, choroid, and retina. The sclera and choroid layers each have an anterior and a posterior portion.

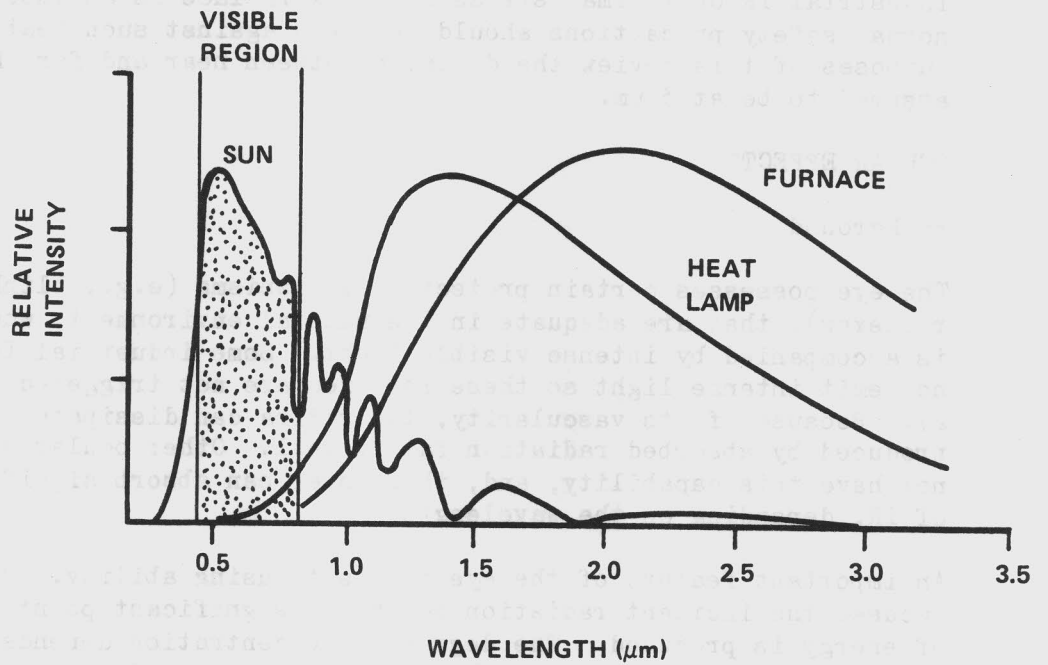


Figure 2. Comparison of IR sources spectral distribution to the visible radiation region.

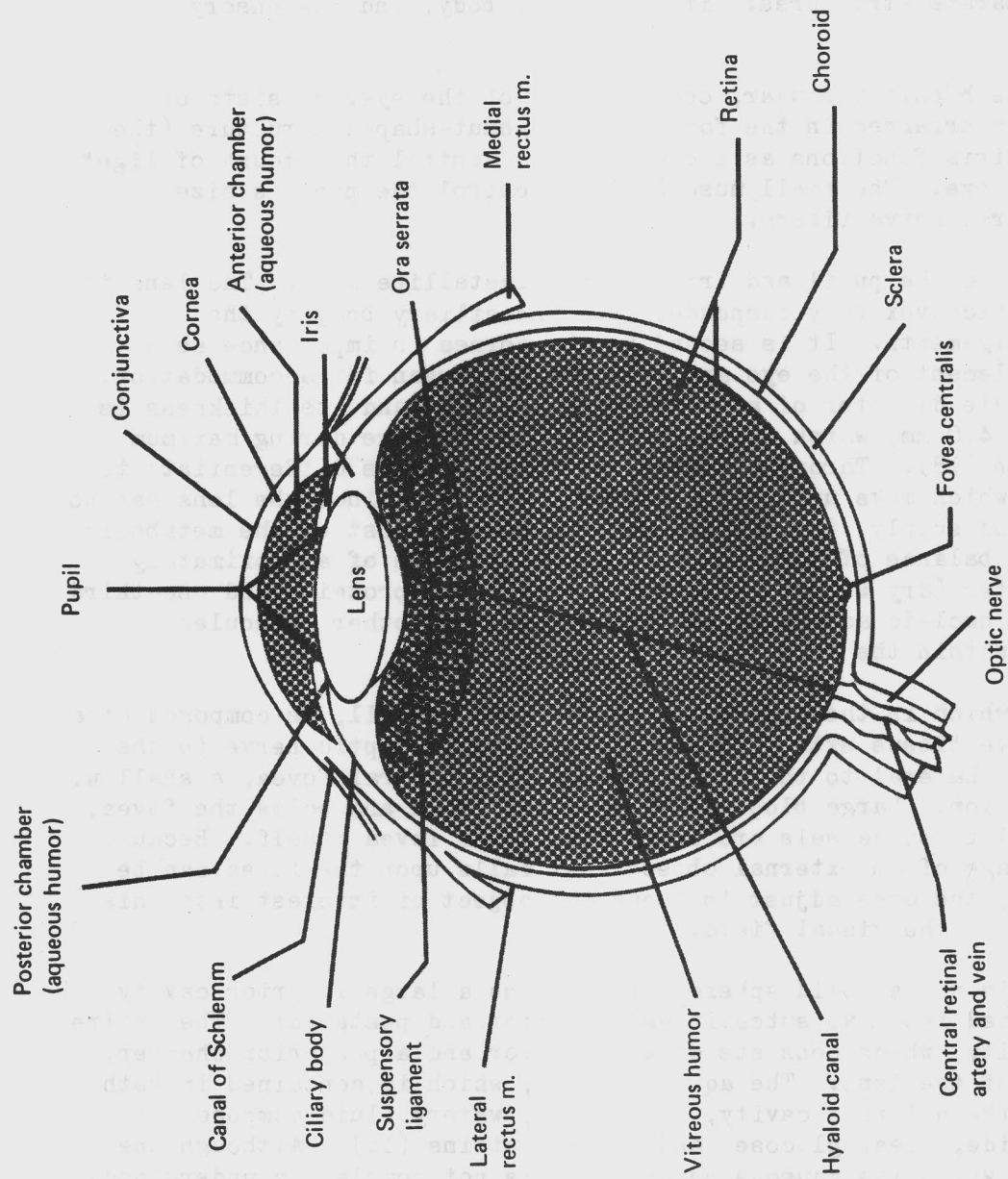


Figure 3. Horizontal section through eyeball.

The sclera is a rigid, white fibrous tissue. Its anterior portion, the cornea, which is a transparent, highly refractive element of the eye, is approximately 0.8 to 0.9 mm thick at the center and about 1 mm thick at the periphery(18). Although the cornea is composed of several layers, the corneal epithelium is thin and extremely sensitive containing numerous nerve endings, but no blood vessels.

The middle or choroid layer of the eye contains many blood vessels and a large amount of pigment. The anterior portion of the choroid is composed of three separate structures: iris, ciliary body, and suspensory ligament.

The iris, the highly vascular, colored part of the eye, consists of muscle fibers arranged in the form of a doughnut-shaped structure (the pupil). The iris functions as a diaphragm to control the amount of light entering the eye. The small muscles that control the pupil's size contain several nerve fibers.

Directly behind the pupil and iris is the crystalline lens. The lens is an elastic, biconvex body suspended from the ciliary body by the suspensory ligaments. It is second to the cornea in importance as a refractive element of the eye and is the major organ for accommodation. The approximate diameter of an adult lens is 10 mm and its thickness is about 3.7 to 4.0 mm, which increases to 4.5 mm or more during maximum accommodation (18). Throughout life, epithelial cells differentiate to form fibers which make up most of the lens tissue. Since the lens has no nerve or blood supply, the aqueous humor maintains most of the metabolic function and balance of the lens. The lens consists of approximately one-third water (dry weight), one-third structural protein, and one-third a mixture of nucleic acids, metabolic enzymes, and other molecules synthesized within the lens itself.

The retina, which is the innermost layer of the eyeball, is composed of a layer of nerve tissue extending anteriorly from the optic nerve to the ora serrata. Lateral to the optic nerve is the central fovea, a shallow, round depression. Large blood vessels circle above and below the fovea, whereas small blood vessels are found within the fovea itself. Because only that image of an external object that falls upon the fovea can be seen sharply, the eyes adjust to focus the object of interest into this central part of the visual field.

The eyeball is not a solid sphere but contains a large interior cavity that is divided into two subcavities, anterior and posterior. The entire anterior cavity, which consists of an anterior and a posterior chamber, is in front of the lens. The aqueous humor, which is contained in both chambers of the anterior cavity, is a clear, watery fluid composed of sodium chloride, urea, glucose, and a few proteins (18). Although the mechanism by which the aqueous humor forms is not completely understood, a constant, replenishing flow enters the posterior chamber from the anterior chamber and drains through the canal of Schlemm. Since this flow is fairly uniform, the amount of aqueous humor remains relatively fixed and thereby maintains the eyeball shape.

The posterior cavity of the eyeball, i.e., the space between the lens and retina, is considerably larger than the anterior cavity. The posterior cavity contains vitreous humor which is a colorless, gelatinous-like mass that is 99% water. The vitreous humor has a "glasslike" transparency and a medium index of refraction (19). This mass also helps to maintain sufficient intraocular pressure to retain the shape of the eyeball. Neither the aqueous nor the vitreous humor contains a nerve or blood supply system.

Accessory Organs

The eyelids and lacrimal glands are two other organs that influence the extent of IR injury to the eye.

Each eyelid is approximately 0.65 mm thick (20) and consists of muscle and skin with a border of thick connective tissue at the free edge. A mucous membrane, the conjunctiva, lines each lid and extends over the exposed surface of the eyeball. The conjunctiva has a system of glands that secrete a fluid which moistens, lubricates, and flushes the surface of the eyeball and the eyelids. The most significant gland in this system is the lacrimal gland, found beneath the conjunctiva. The gland system also consists of lacrimal sacs and lacrimal ducts, which drain the fluid from the lacrimal gland into the conjunctiva producing the fluid film on the cornea.

Optical Properties

The ocular structures reflect, refract, and scatter incident IR. The absorption of specific radiation wavelengths by these structures causes specific biological responses.

The major factor affecting the degree of biological response is the energy absorbed (or dose). In the ocular system however, the amount of IR energy reaching a particular tissue depends on the transmittance of the structures between the source and the tissue. Most researchers have concentrated their investigations on determining the transmission and/or absorption properties of the various ocular structures.

Transmission and Absorption

Early investigations of ocular transmittance assumed that the absorption of IR by the eye was similar to an equal thickness of water. Most reported results were estimates at best. Vogt (21) hypothesized that the lens absorbs more IR than does any other ocular tissue.

Kutscher (22) published the first accurate data on the ocular transmittance, which were later confirmed by Minton (23). The reliability of many studies that were performed before the mid-fifties is questionable, however, because several factors were not considered, e.g., environmental laboratory conditions, species-specific variations, and the effects of enucleation.

Wiesinger et al. (24) compared the transmittance of freshly enucleated rabbit eyes with distilled water and physiological saline solution. Although little difference was found in transmission levels, the use of fresh specimens and consideration of the effects of saline solution on transmission are noteworthy.

In 1960, Geeraets et al. (25) exposed rabbit eyes and human eyes to the 0.35 to 1.5 μ m wavelength region to determine the effect of variation in retinal and choroidal pigmentation on the amount of light absorbed. Wavelengths above 1.1 μ m are much less affected by pigmentation of the retina-choroid layers (see Figures 4 and 5).

Some investigators did not accept the assumption that the difference between transmittance and absorptance in animal and human tissue were minimal despite their anatomical differences. Boettner and Wolter (26) noted the lack of experimental data on the transmittance/absorptance of human eyes. Since most research had been limited to animal eyes, they undertook a comprehensive study of the transmission/absorption characteristics of the separate ocular components in nine human eyes. They measured both direct and total transmittance. Direct transmittance measurement was defined as the radiation passing through the ocular media. Total transmittance included the direct transmittance and scattered radiation. The results are summarized below and shown graphically in Figures 6 and 7.

- o Total corneal transmittance exceeds 85% between 0.5 and 1.3 μ m and reaches the maximum at 1.1 μ m. Beyond 1.3 μ m, two water absorption bands appear at about 1.43 and 1.95 μ m, but corneal transmission remains high outside these bands. The cornea is the only ocular structure that has maximum transmittance as high as 1.0 μ m. Corneal absorption of IR is total beyond 2.0 μ m.
- o The aqueous humor transmits to about 2.4 μ m with water absorption bands at 0.98, 1.2, 1.43, and 1.95 μ m.
- o The lens exhibits high transmission to about 1.4 μ m and has water absorption bands at 0.98 and 1.2 μ m.
- o The transmission of the vitreous humor exceeds 90% and has water absorption bands at 0.98 and 1.2 μ m.

Boettner and Wolter also studied the effects of aging, scattering, and time after enucleation on transmission and absorption for selected ocular structures. Within the IR region, they reported the following:

1. Scattering and age effects in the aqueous humor.
2. A significant decrease in lenticular transmission occurred with age.
3. The amount of scattering through the entire eye (young and old) decreased with increasing wavelength.

Prince (27) evaluated the effects due to delayed post-mortem enucleation, preservation of the eye in saline solution or by refrigeration, and

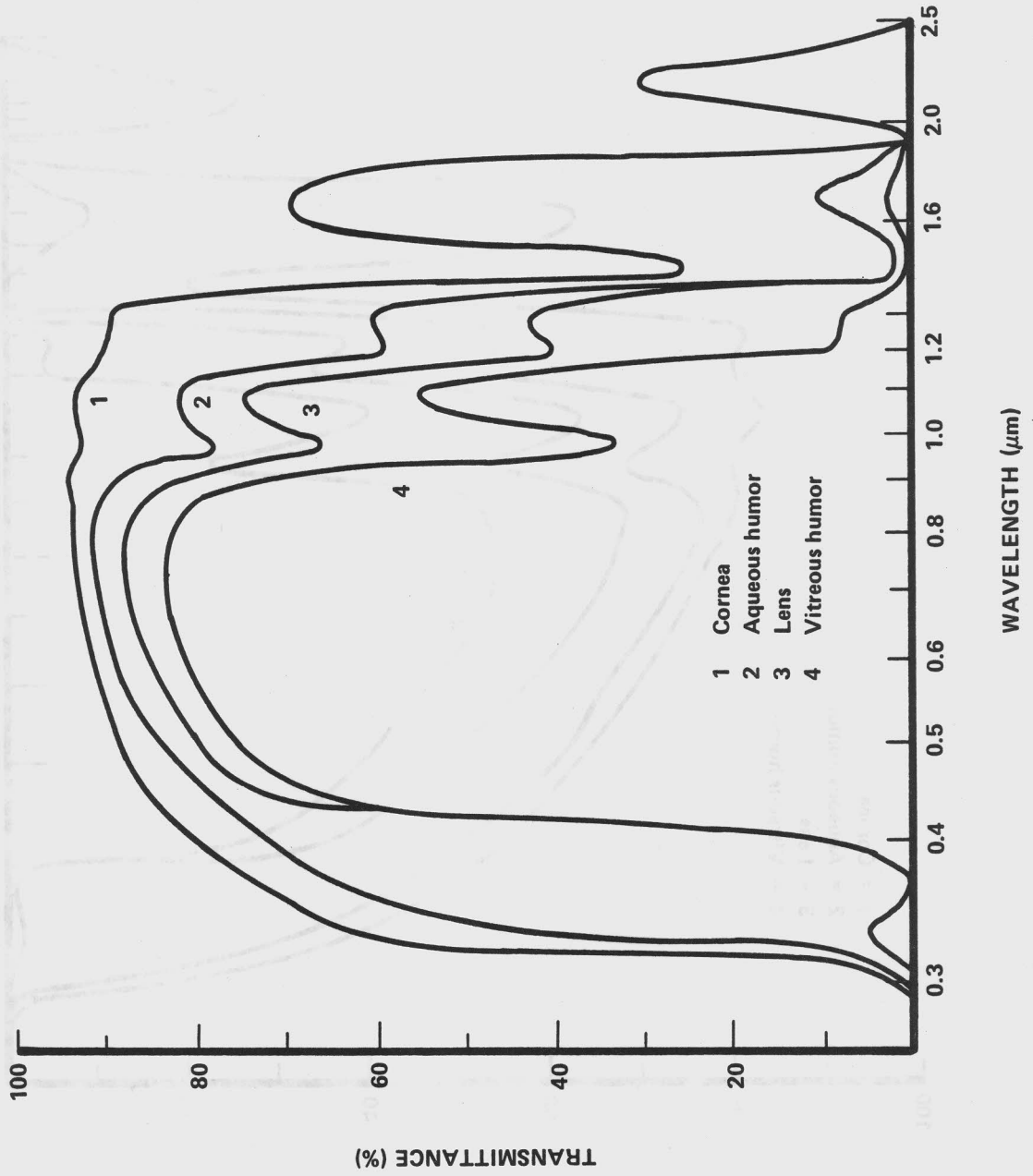


Figure 4. Transmission in the ocular media and retinal-choroidal layer for rabbit eyes. Modified from reference 25.

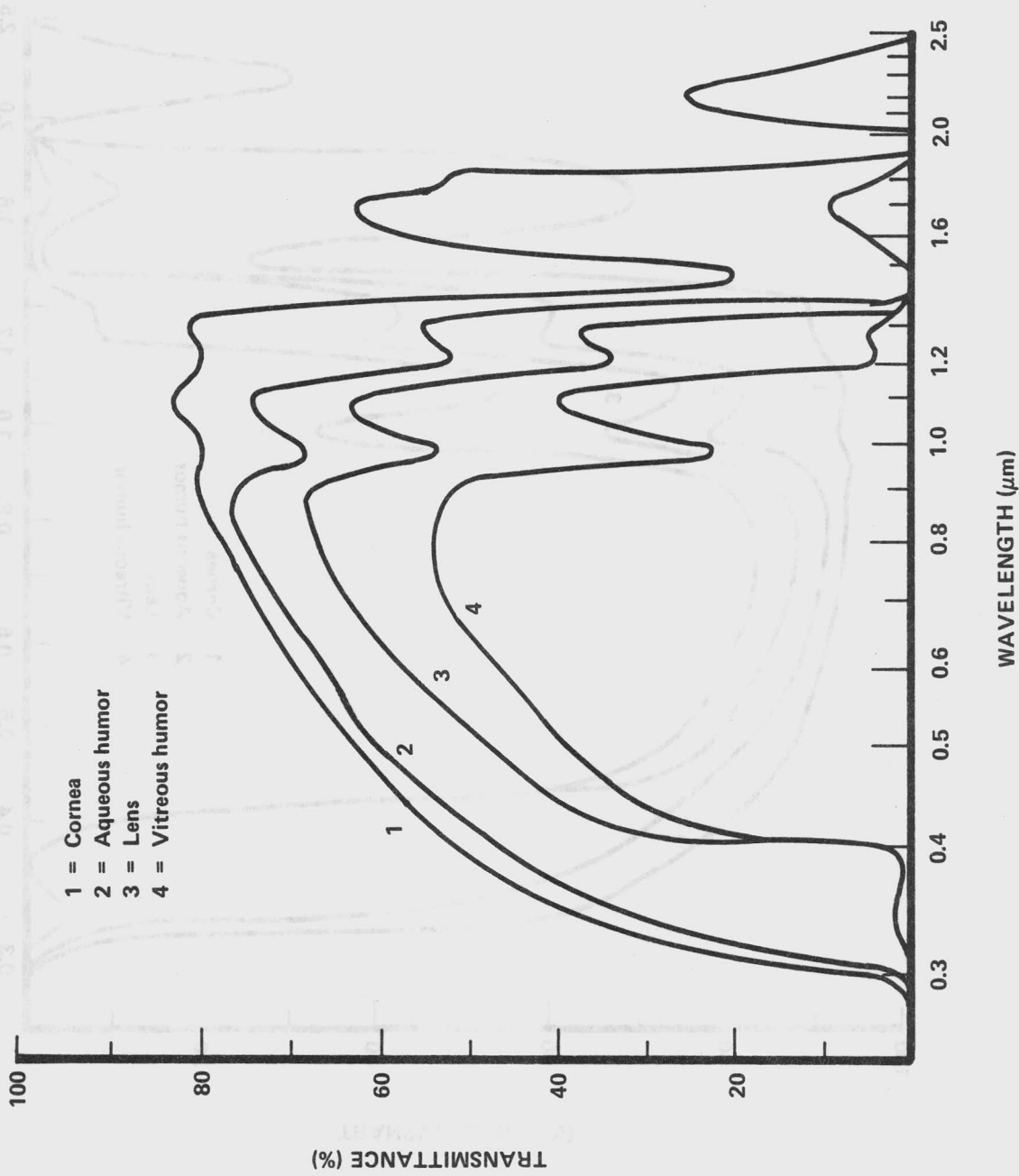


Figure 5. Transmission in the ocular media, retinal-choroidal layer, and entire globe for two human eyes. Modified from reference 25.

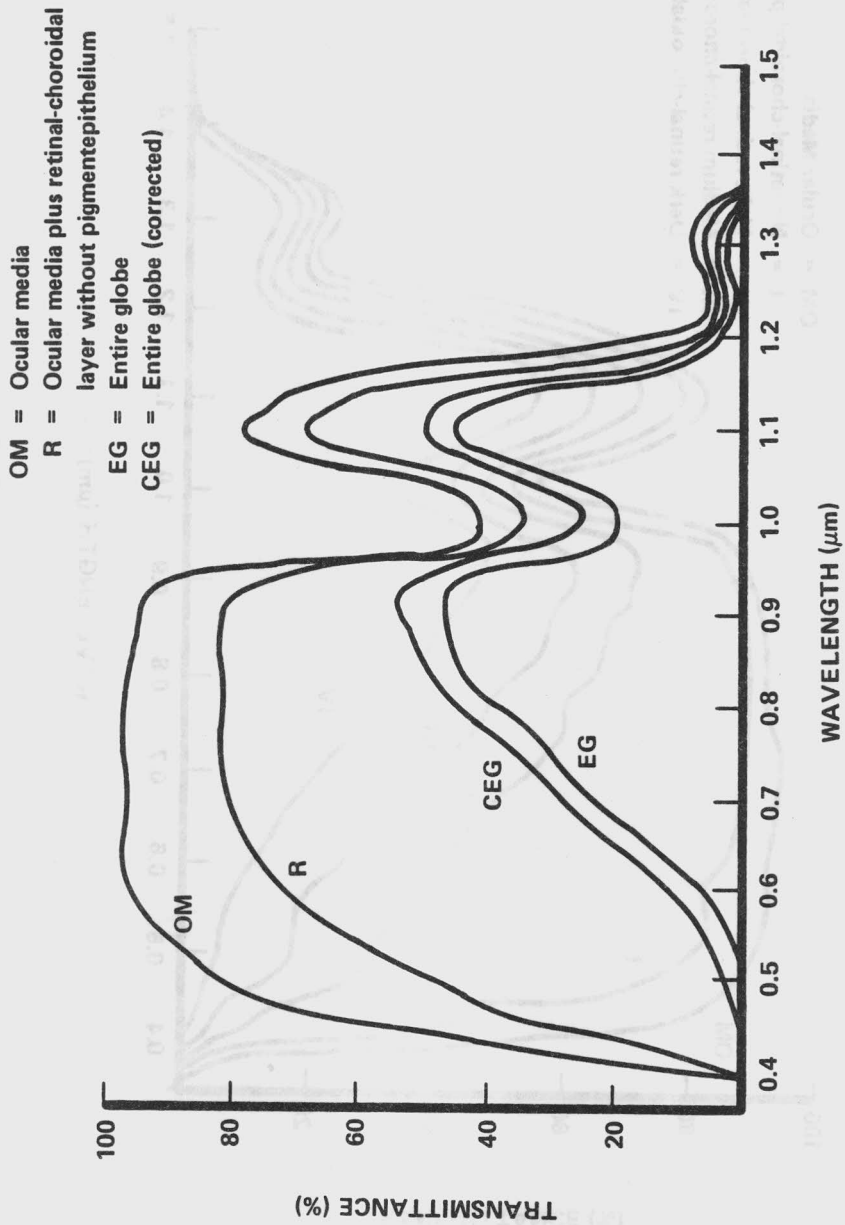


Figure 6. Direct transmittance of the eye.
Modified from reference 26.

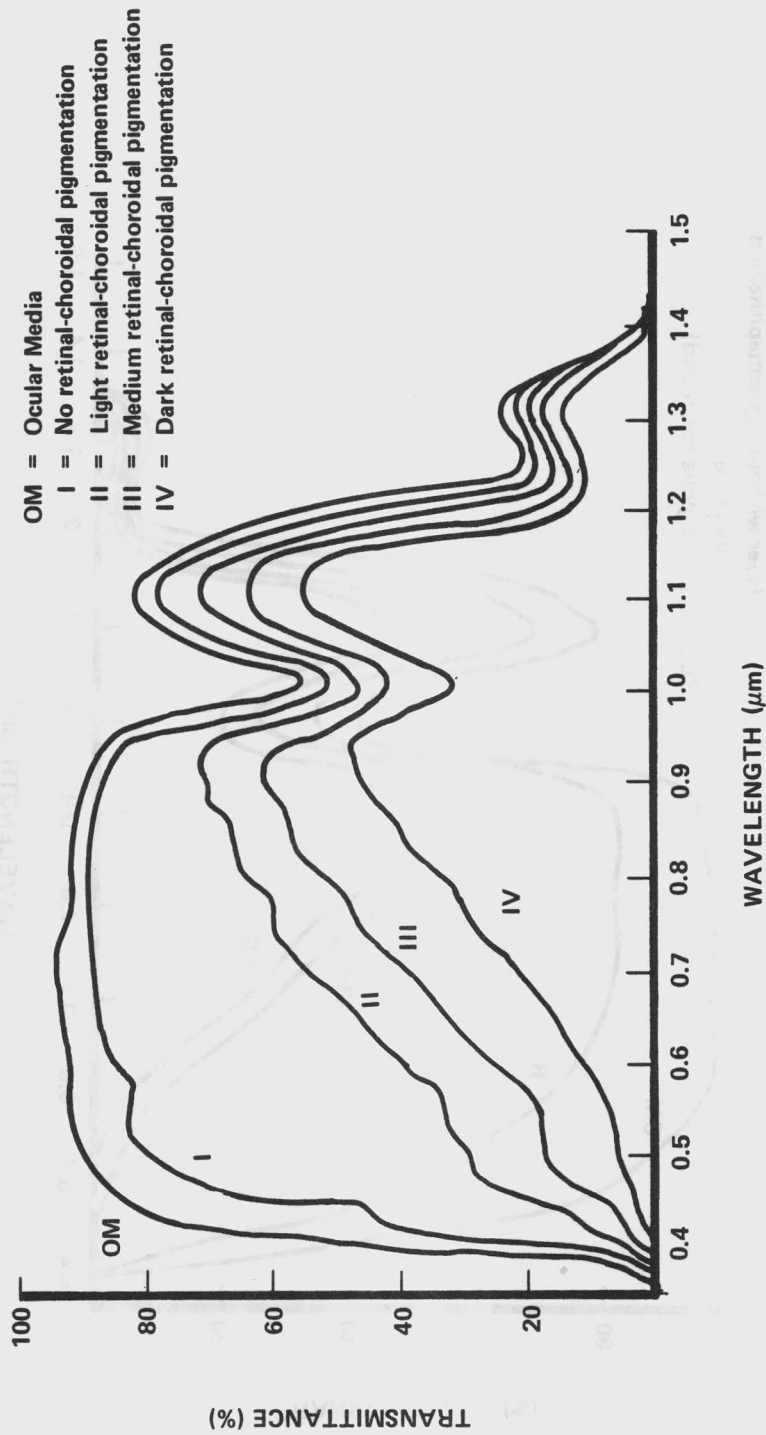


Figure 7. Total transmittance of the eye.
 Modified from reference 26.

variations in the amount of blood retained in the retinal-choroidal vascular system. Based on his rabbit eye studies, he found that dehydration, blood drainage, and preservation in saline solution for an extended period altered specific areas of the absorption curve.

In 1968, Geeraets and Berry (28) examined the transmittance of 28 intact human eyes by using methods similar to those of Boettner and Wolter (26). The data from the two studies are compared in Figure 8. The transmittance difference is substantial and has not been resolved. Sliney (2) hypothesized that the difference may be due to measurement of the transmission of the entire eye versus the transmission of the ocular media separately and the use of correction factors for scatter. In 1979, Barker (29) measured the transmittance of the cornea, aqueous, humor lens, and vitreous humor in nine freshly enucleated pigmented rabbit eyes from 0.2 to 2.5 μm . Infrared transmittance results were similar to the transmittance spectrum of water and agreed with existing literature data.

Ruth et. al. (32) influenced by previous work by Fischer et al. (30) and Franke (31), proposed that the aqueous humor, lens, and vitreous humor be collectively termed the inner eye and that the absorption be viewed compositely, as illustrated in Figure 9. This figure is useful in relating the absorption by the eye to the spectral distribution of various IR sources (Figures 10 and 11). However, Figure 9 does not take into consideration the absorption of optical radiation by the cornea.

Direct and Indirect Effects on Components

At this point, based on the optical characteristics of the eye, it is appropriate to examine the adverse effects and possible damage mechanisms on various ocular components.

Eyelid--

It is often assumed that the eyelid and its associated blink reflex protect the retina from IR burns and replenishes the film of liquid on the exposed surface of the eyeball. Investigations into the extent of IR transmission by the eyelid have not been reported in the literature; however, because the anatomical structure of the eyelid and that of body skin are relatively similar, it is possible to estimate the IR transmission. The eyelid is approximately 0.65 mm thick. Comparison of this value with the average penetration of IR at 1.23 μm (Figure 26) shows that approximately 60% of the incident radiation could penetrate a skin thickness of 0.65 mm. If these statements are accepted, serious doubt is cast on the previously assumed protective effect of the blink reflex. The specific effect of these observations has not been identified, but at least the potential hazard should be at least recognized.

Cornea--

Exposure of the cornea results in a cutaneous burn similar to that which occurs on the skin. In 1916, Verhoeff, Bell, and Walker (33) theorized

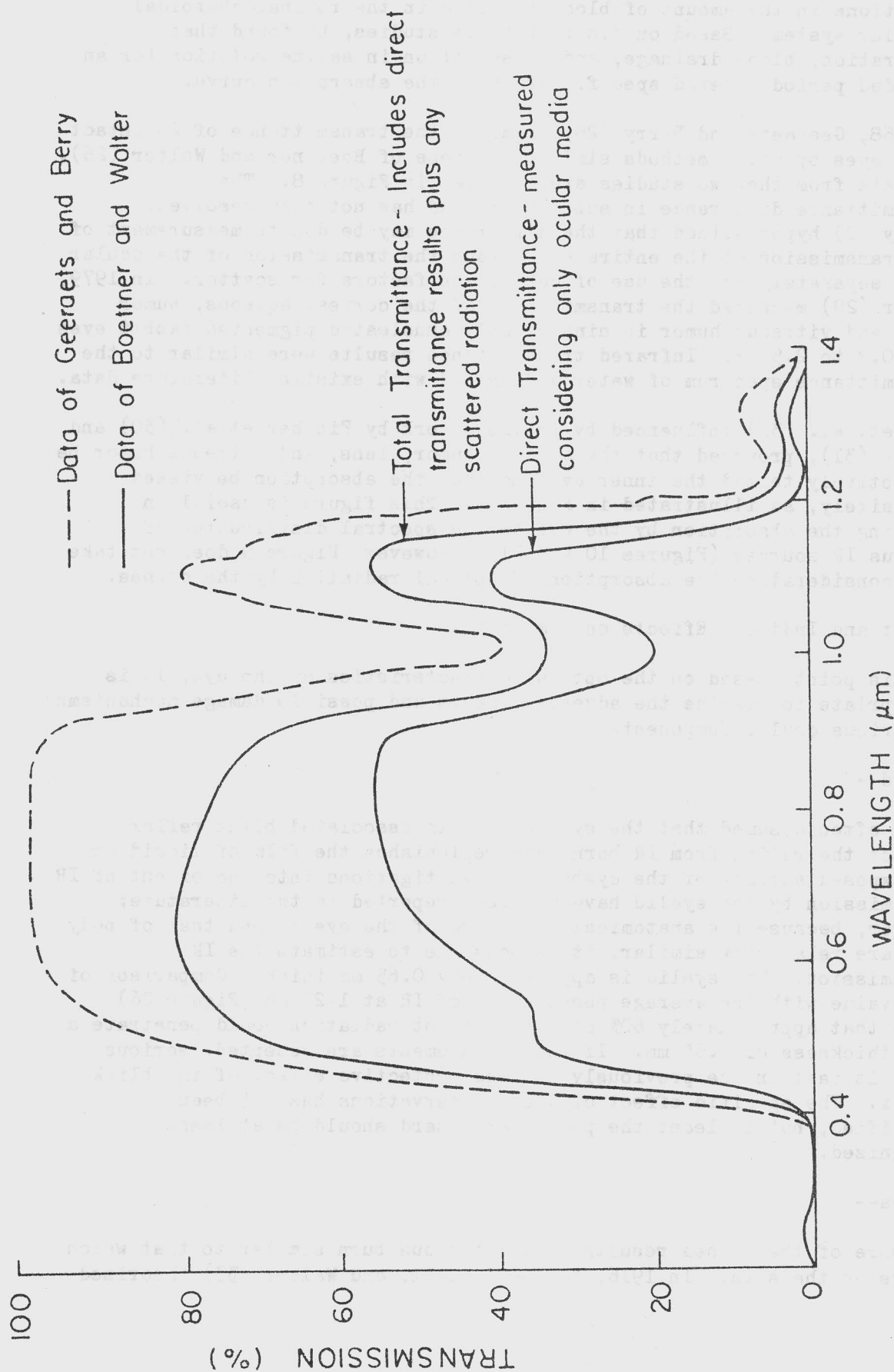


Figure 8. Spectral transmission of the ocular media of the eye. Modified from reference 28.

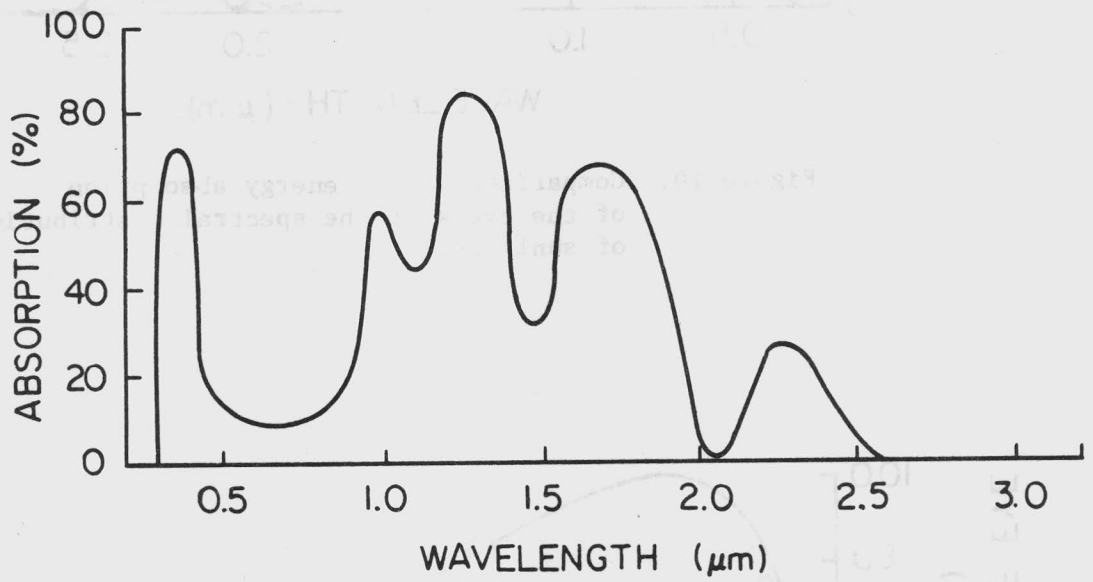


Figure 9. Composite absorption of optical radiation by aqueous humor, lens, and vitreous body.

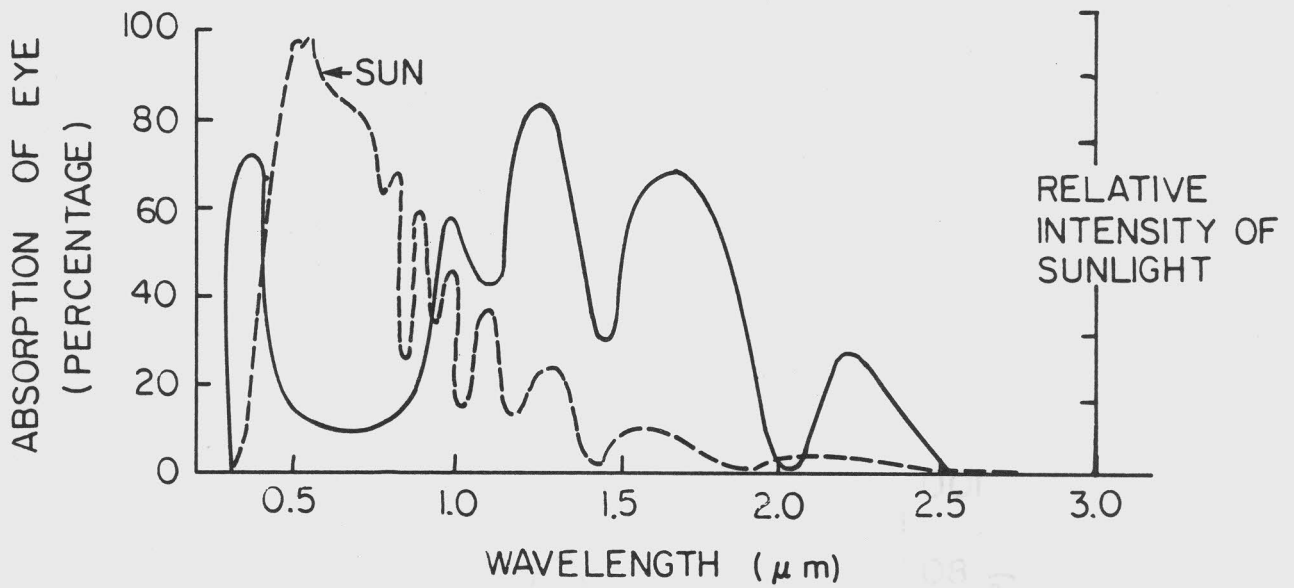


Figure 10. Comparison of the energy absorption of the eye with the spectral distribution of sunlight.

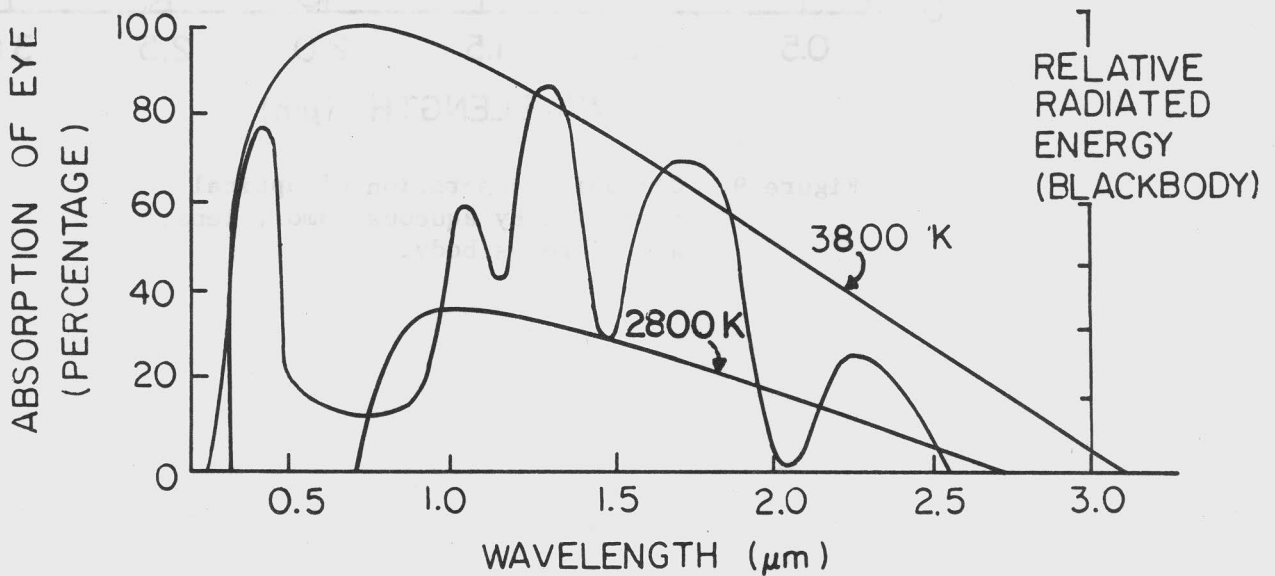


Figure 11. Comparison of the energy absorption of the eye with blackbody temperatures.

that the posterior corneal surface may show more damage than the anterior surface since the latter is cooled by air and lacrimal fluid. Kutscher (22) notes that exposure to high-intensity far IR produces immediate and severe corneal pain that causes reflexive shutting of the eyes and averting of the head. Burn lesions are not common in IR industrial situations (2). The results of such a burn is destruction of the superficial corneal epithelium and denaturation of the underlying proteinaceous stromal layer, which causes corneal opacification (34).

Lele and Weddell (35) suggested that a specific thermal sensation, as well as the sensation of pain, may be produced by the application of IR to the cornea. Their study presents little information on spectral exposure characteristics. Dawson (36) exposed cat corneas to a tungsten filament lamp with a known spectral distribution from 0.4 to 2.6 μm . He found many of the above effects depended on exposure level, but the source used was too intense to ascertain the occurrence of a thermal sensation. Dawson noted that the corneal regeneration capability permits exposure levels exceeding threshold for sensory nerve stimulation without permanent damage. Sliney (2) states that the sensory nerve endings in the human cornea are quite sensitive to a small temperature elevation and a temperature of 47°C elicits a pain response. This suggests that a thermal sensation is activated at a lower temperature than the actual pain stimulus level. Other studies (37, 38) have reported similar results.

In 1941, Krivobok (39) implied that IR irritates the corneal nerve endings. The pain response caused by exposure of the cornea to high-intensity IR seems to indicate this is true, although the meaning of "irritate" is not clear. Further research in this area has not been reported in the literature.

Aqueous Humor--

The aqueous humor, which is located between the cornea, iris, and lens will absorb IR and increase in temperature. This increased temperature could contribute to the temperature rise of other ocular components, most notably the lens; however, little is known about this phenomenon.

Iris--

The effects of IR on the iris have been summarized by Duke-Elder (40). As shown in Figure 12, the melanin pigment in the iris does not strongly absorb IR beyond 0.8 μm . However, since visible radiation is often produced in conjunction with IR, the melanin pigments' higher absorption of visible radiation may produce the thermal damage. Moderate exposure doses result in miosis (constriction of the pupil), hyperemia, and the formation of aqueous flares. More severe exposure may lead to muscle paralysis, congestion with hemorrhage, thrombosis, and stromal inflammation. Necrosis of the iris may occur causing the formation of bleached atrophic areas within a few days. A loss of pigmentation at the edges of the iris occurs within 2 to 4 days after exposure.

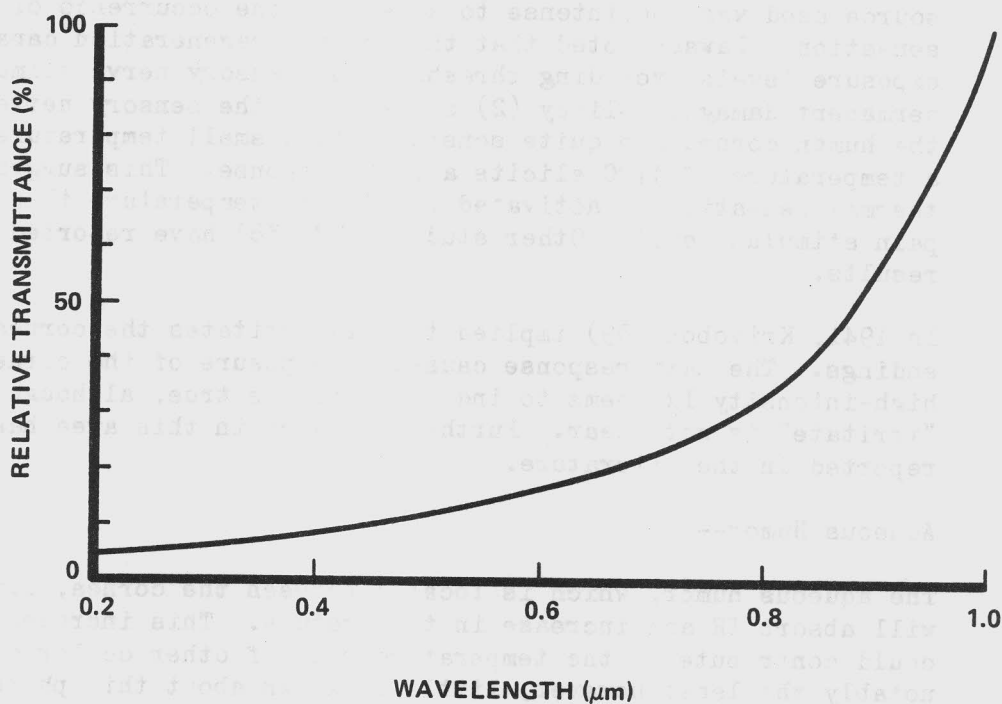


Figure 12. Infrared absorption properties of melanin. (Based on references 41 and 42)

Goldmann (43) proposed that a general heating of the iris occurs as a result of IR absorption. Overheating of the iris may cause adhesions to the lens or cornea, which are not only painful, but could contribute to a quasi-cataract formation. Wolbarsht (44) states that lenticular heating by conduction from the heated iris is not likely to be a significant intermediate step in cataractogenesis that follows chronic IR exposures. A recent communication (45) has indicated the iris may be highly reflective for 0.92 μm wavelength regardless of pigmentation.

Lens--

Lenticular damage by IR is due to several factors. Since the lens is avascular it has a poor dissipating heat capacity. Also, the viewing of "extended" IR sources results in a weak energy concentration in the lens (Figure 13). Furthermore, the lens is a growing and actively metabolizing tissue throughout life. As a result of the continuing metabolic activity, the optical clarity of the lens becomes progressively reduced with age as well as such factors as metabolic disorders, ocular inflammation, and blunt trauma. This reduction in optical clarity is caused by different types of opacities, generally referred to as cataracts, all of which do not, necessarily result in lowered visual acuity.

Many reports in the literature prior to 1930 claim a higher incidence of cataracts among glassblowers and furnace workers than among the non-exposed population. The first lenticular damage associated with optical radiation was reported in 1739 by Heister (46), who suggested a relationship between cataracts and exposure to sunlight. Early researchers, such as Plenk, Wathen, Wenzel, and Beer (47), noted that cataracts seemed more prevalent among workers in certain occupations involving prolonged exposure to heat sources, e.g., hot metal, sun, and glass furnaces. In 1855, MacKenzie (48), suggested that glassblowers, forgemen, cooks, laundryworkers, etc., exhibited this disease more frequently than the general population. In 1886, Meyhofer (49) provided epidemiological data on the number of glassworkers having posterior cortical opacities. In 1907, Legge (50) was instrumental in establishing glassblowers' cataract as a legal occupational disease in Great Britain. Reviews of early literature on occupational heat cataract have been performed by Turner (34), Edbrooke and Edwards (51), and Emarah (47). The beginning of the 20th century brought about intense interest in and debate over the etiology of occupational cataract. In 1915, Robinson (52) stated that IR caused heat induced cataracts. Prior to this time controversy had focused on whether heat, light, or both caused cataracts. According to Verhoeff et al. (33), the cornea and anterior lens both absorb IR; however, the cornea is cooled by air whereas the anterior lens is cooled by heat dissipation into the circulating aqueous humor. These investigations further stated that a heat induced cataract cannot form on the anterior lens surface because prolonged heating of both surfaces does not occur. These investigations also postulated that lack of convective currents in the vitreous humor prevented cooling and caused cataracts to form on the posterior lens. However, they were not totally satisfied with this theory because heat interferes with normal ciliary body function and lens metabolism.

The controversy was further stimulated by Vogt and Goldmann. Vogt (21) maintained that cataracts were due to the direct absorption of the radiant energy by the lens. In studies made between 1930 and 1950, Goldmann (43,53,54) contended that the cataract resulted from indirect heating of the lens by the iris. During the years that followed, numerous investigators added further support to Goldmann's hypothesis, despite some disagreement (34). In a 1960 study undertaken to resolve the issue, Langley et.al. (55) showed that irradiation of the iris only was sufficient to produce a lenticular opacity in the eye of a rabbit. This confirmed Goldmann's view that IR absorbed by the pigment epithelium of the iris converts to heat, which damages the lens and causes the cataract. Zaret (56) noted that the development of adhesions between the iris and lens capsule is delayed. He concluded that IR damages the lens capsule directly by altering its permeability or elasticity and disrupting normal protein metabolism in the adjacent fibers, which is in agreement with Vogt's (21) hypothesis.

In a well-defined study Pitts et al. (57) recently showed that no permanent lenticular opacities could be induced by direct exposure of the lens of pigmented rabbit eyes. Pitts used a filtered 5000-W Xenon, high-pressure lamp to demonstrate that all lens damage is dependent on iris involvement. These findings support the Goldmann hypothesis just discussed. The primary ocular lesion was an anterior, epithelial, subcapsular opacity, which initially appeared as small whitish dots. These dots developed into white patches in the area of the anterior capsule just beneath and in contact with the iris. Ocular damage from IR was related to the rate of delivery of radiation. The following summarizes the findings:

<u>THRESHOLDS (J.cm⁻²)</u>			
<u>IR Irradiances</u>	<u>Cornea</u>	<u>Iris</u>	<u>Lens</u>
4.0 mW.cm ⁻²	5000	3500	3750
4.0 mW.cm ⁻²	1250	1250	2250

A preliminary report by Tengroth (58) indicates that a detailed Swedish epidemiological study of 227 furnace, iron, and steel workers showed no increase in the frequency of lens changes among this group compared with a group of nonexposed persons of the same age.

On the basis of animal experimentation and theoretical analyses, two theories have been advanced to account for the formation of what are now called "infrared cataracts." One theory suggests that cataract formation is due to direct absorption of IR in the lens; the other suggests such formation is secondary to heating of the aqueous humor and iris caused by absorption of IR. Few investigations have considered the role of other types of cataract (e.g., diabetic or senile) in relation to IR cataractogenesis. Investigators have also largely neglected the influence of heredity, race, disease, sunlight, and the immunological and nutritional factors that can affect cataractogenesis. Gehring (50), however, has provided an excellent review of the cataractogenic potential associated with chemical agents.

Retina--

Since absorption by the retina is greatest at short IR wavelengths, it is difficult to distinguish the exposure effects to near IR from those of visible radiation.

Early investigations were concerned mainly with the effects of sunlight on the retina. In 1950, Cogan (60) realized the problem of separating damage of IR from that produced by visible radiation. He noted that far IR probably does not damage the retina because it is absorbed largely by the anterior portions of the eye. But near IR and visible radiation not only reach the retina but are refracted by the ocular media to focus the incoming energy onto the retina. A few years later Duke-Elder (40) proposed that the IR burns or lesions on the retina are caused indiscriminately by IR or visible radiation. He believed that the reaction is unrelated, either quantitatively or qualitatively, to any particular wavelength, but depends only on the concentration of energy incident in the region.

In the establishment of IR threshold values, several factors must be considered. These include pupil size, spectral transmittance of the ocular media, spectral absorptance of the retina, exposure duration, image size, blood flow, source spectral distribution, dose rate, and exposure evaluation criteria. One of the most critical problems has been to define the threshold lesion, which has ranged from the "slightest visible tissue change" to the appearance of an "ophthalmologically detectable lesion."

In 1963, Bredemeyer et al. (61) examined the relative effectiveness of different wavelengths in producing retinal burns. He exposed rabbits to six wavebands in the interval between 0.2 and 3.0 μm while maintaining a constant image size and exposure duration. Bredemeyer's data showed that higher corneal irradiances was required to produce a retinal burn with longer wavelengths.

In 1963 Geeraets and Ridgeway (62) concluded that a thermal mechanism can adequately account for the retinal damage produced by IR. As stated by Ham et al. (63), mathematical model determinations for threshold retinal burn seem to be associated with a critical temperature. Ham found, however, that the time-temperature history is the deciding factor in determining thermal injury. Questions concerning the mechanism(s) leading to retinal injury are still unresolved with regard to the importance of thermal versus photochemical effects. It is difficult to ascertain where thermal injury ceases to play a primary role and thermally enhanced photochemical effects become important. In 1979, Ham et al. (64) proposed a scheme that addressed this dilemma. Figure 14 shows a comparison of the retinal sensitivity for various wavelengths. For extended exposures, about three orders of magnitude greater corneal exposure is required to produce a minimal retinal lesion from infrared radiation (1.064 μm) than from blue light (0.442 μm). Figure 15 shows the increase in retinal temperature above ambient as a function of

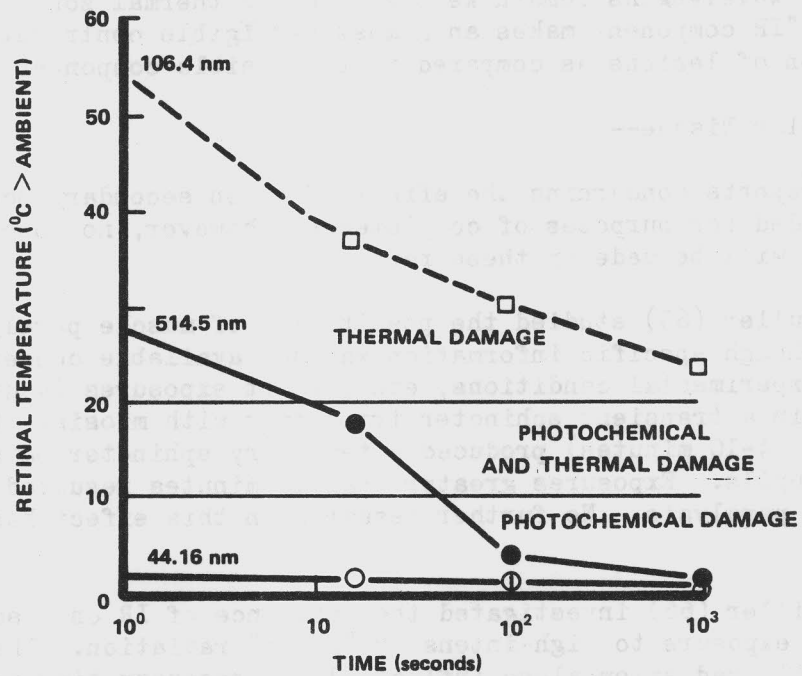


Figure 14. Radiant exposure required to produce a minimal lesion of different exposure times. Modified from reference 64.

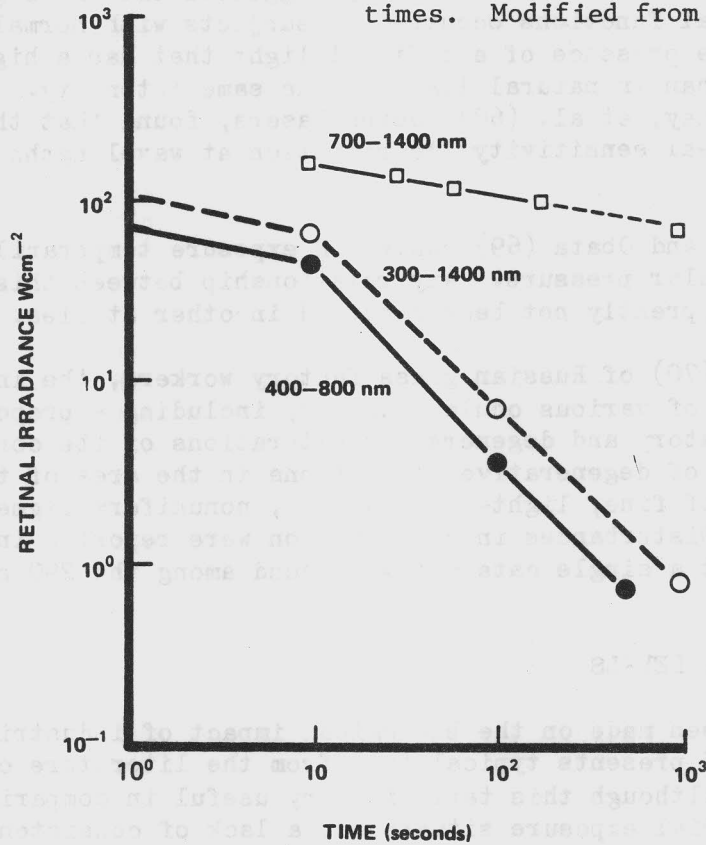


Figure 15. Retinal temperature versus exposure time. Modified from reference 64.

exposure time for several wavelengths. Notice that the infrared radiation wavelengths remain well within the thermal zone. According to Ham, the "IR component makes an almost negligible contribution to the production of lesions as compared to the visible component (Figure 16)."

Other Ocular Tissue--

Several reports concerning the effect of IR on secondary ocular tissues are included for purposes of completeness; however, no conclusive statement will be made on these reports.

In 1924 Muller (65) studied the possibility of muscle paralysis in the iris. Although specific information was not available concerning the source, experimental conditions, etc., short exposures (4 minutes) resulted in a transient sphincter irritation with miosis. Longer exposures (4-10 minutes) produced a temporary sphincter paralysis and dilated pupils. Exposures greater than 10 minutes resulted in permanent sphincter paralysis. No further research on this effect has been reported.

Fry and Miller (66) investigated the influence of IR on visual recovery following exposure to high-intensity "flash" radiation. They reported that visible radiation alone influenced the recovery time for foveal performance and that IR has no effect in prolonging the recovery time. In a related vein, Luckiesh and Moss (67) suggested that no significant difference in visual functions occurred in subjects with normal vision when reading in the presence of artificial light that has a higher percentage of IR than in natural light of the same intensity. Interestingly, Sliney, et al. (68), using lasers, found that the eye could respond (foveal sensitivity) to radiation at wavelengths as far as 1.064 μm .

In a study by Sano and Obata (69) ocular IR exposure temporarily increased intra-ocular pressure. Any relationship between this effect and glaucoma has apparently not been reported in other studies.

In a recent study (70) of Russian glass factory workers, the investigator found a prevalence of various ocular changes, including a preponderance of chronic inflammatory and degenerative alterations of the conjunctiva and the occurrence of degenerative alterations in the area of the macula lutea in the form of fine, light-colored foci, nonuniform pigmentation, and cellularity. Disturbances in color vision were reported in a number of workers, but not a single cataract was found among the 290 subjects and 109 controls.

INDUSTRIAL EXPOSURE LEVELS

Few studies have been made on the biological impact of industrial IR exposures. Table 3 presents typical data from the literature on worker exposure levels. Although this table is very useful in comparing a wide range of IR industrial exposure situations, a lack of consistency is evident in reporting of such parameters as irradiance and exposure duration. This is especially true in earlier studies related to the occurrence of lens damage in the form of "glassblowers' cataracts."

A more recent study (9) of glass... of approximately 1.5 W/cm² for... evidence of contact... number for the contact... will not be exposed... but report... operating in the cap...

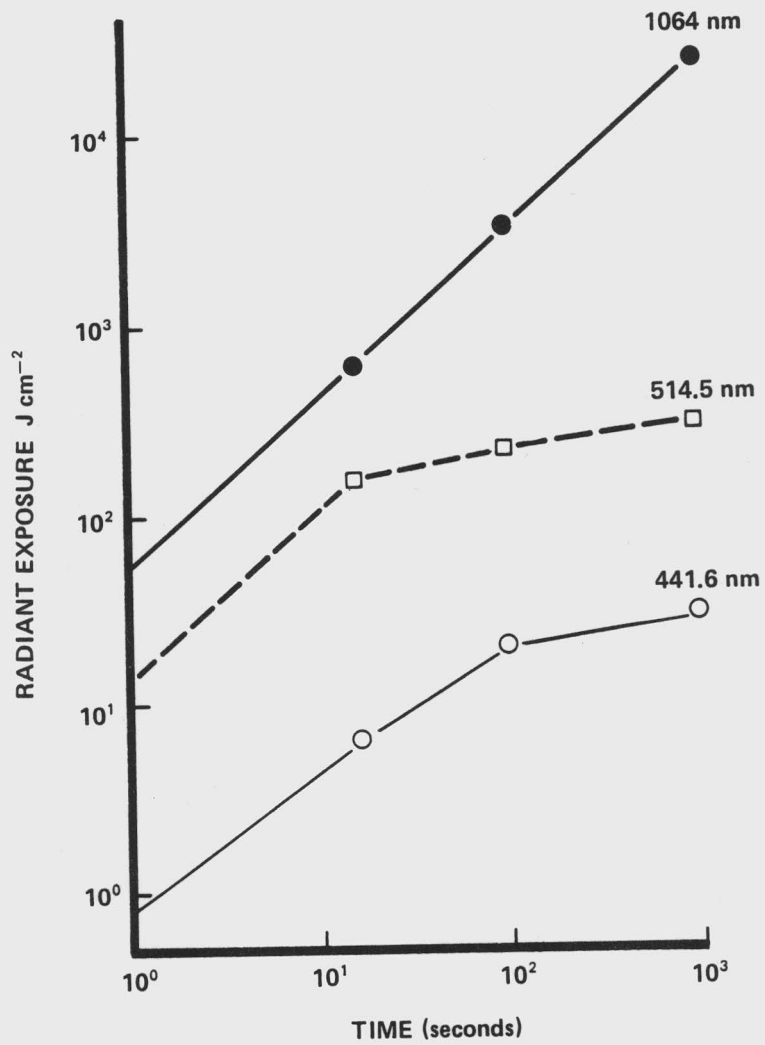


Figure 16. Retinal irradiance necessary to produce a minimal lesion for a given time. Modified from reference 64.

A more recent study (72) of glassblowers exposed to an irradiance level of approximately 0.14 W.cm^{-2} for a number of years has shown no evidence of cataract formation. In addition, Keatinge et al. (73) were unable to find posterior cortical changes in the lens of iron rolling mill workers exposed to irradiance levels of 0.08 to 0.42 W.cm^{-2} . They did report, however, a higher incidence of posterior capsular opacities originating in the capsular plane and extending to the cortex.



Table 3. Industrial data on ocular infrared radiation hazards among workers

Date	Source and time	Effect	Wavelength (μm)	Radiant exposures ($\text{J}\cdot\text{cm}^{-2}$)	Irradiance ($\text{W}\cdot\text{cm}^{-2}$)	Reference
1932-35	Electric furnace 30 sec 231 sec	Aqueous humor temperature increase above 36°C : 3°C 9°C	0.76 - 72.5 Peak at 1.5	4.2 32.2	0.14 0.14	Goldman (43)
1948	Sun Few min.	Retinal burn		301 (at retina)	0.09 (at retina)	Roson cited in (71)
1950	Glass furnace 20 years	None	1.5 (peak)		0.14	Dunn (72)
1955	Iron foundry	Lens: posterior capsular opacities			0.084-0.418	Keatinge et. al. (73)
1960	Sun >2s	Retinal burn		6.3 (at retina)	3.1 (at retina)	Flynn cited in (74)
1968	Furnace	Sensation of corneal warmth			0.08-0.4	Matelsky (1)
1970	Welding arc	None	0.78-2.0		0.003-0.0009	Hubner et. al. (75)

(Continued)

Table 3. (Continued)

Date	Source and time	Effect	Wavelength (μm)	Radiant exposure ($\text{J}\cdot\text{cm}^{-2}$)	Irradiance ($\text{W}\cdot\text{cm}^{-2}$)	Reference
1971	Glass (G) and locomotive (L) furnaces 10 years G L G L G L	Lens cataract	0.8-1.4	305 322 598 617 1074 1116	0.050 0.022 0.098 0.043 0.175 0.076	Hager et.al. (76)
1971	Steel/workers years	Increase in opacities				Wallace et. al. (79)
1973	Occupational exposure 10-15 years	Lens cataract			0.1-0.4	Hall & Crockford cited in (71)
1973	Molten brass several times a day	Aqueous humor and lens temperature increase: 3.9°C 6.4°C 8.5°C (All cases had lenticular opacities but no vision impairment)	0.76-2.5 Peak at 1.8	90 149 198	0.007 0.012 0.016	Glansholm & Tengroth cited in (71)

(Continued)

Table 3. (Continued)

Date	Source and time	Effect	Wavelength (μm)	Radiant exposure ($\text{J}\cdot\text{cm}^{-2}$)	Irradiance ($\text{W}\cdot\text{cm}^{-2}$)	Reference
1973	Osram lamp 135 ms	Retinal burn	0.4-0.8	7.9-12.2		Ham (63)
1975	IR heating lamp	None	0.4-3.2		0.001-0.050	Ruth et. al. (32)
1976	Home range oven 3-4 years	Lens cataract and capsular opacity			0.024-0.097	Zaret et. al. (56)
1976	Welding arc 15 min.	Retinal damage	0.9 (peak)			Ruprecht (77)
1977	Video display terminals months	Opacities	0.76-1.1		Not detected	Moss et. al. (83)
1978	Rotary forge Hours-min.	None	0.76		0.1	Marshall (78)
1978	IR hand-soldering device 2-3 days	Conjunctivitis and decreased lachrymation	0.7-1.4		0.065	Sensintaffar et. al. (80)
1978	Sun min-hours	Solar retinopathy			Less than 0.022-0.092	Rothkoff et. al. (81)

(Continued)

Table 3. (Continued)

Date	Source and time	Effect	Wavelength (μm)	Radiant exposure ($\text{J} \cdot \text{cm}^{-2}$)	Irradiance ($\text{W} \cdot \text{cm}^{-2}$)	Reference
1979	Gas welding Gas brazing Gas cutting	None	0.76-1.1		0.006 <0.001 <0.001	Moss et. al (84)
1979	Furnacemen	Len changes	<3.0		> 1.0 (peak)	Tengroth et. al. (58)
1980	Air carbon arc cutting	None	0.76-1.15		0.014	Marshall et. al. (82)
1980	IR curing min.-hours	Dry eye	0.76-1.1		0.004	Moss (85)
1980	Electric Arc furnace	None	0.76-1.1		> 0.2	Moss et. al. (86)
1980	Various welding processes	None	< 0.8			Marshall et. al. (87)

In 1971, Wallace et al. (78) conducted a large scale epidemiological investigation on 1000 workers in a large steel mill, 900 of whom had been exposed to IR. Exposure levels were classified as high, intermediate, low, and no risk. Different job classifications were scored according to relative exposure. The exposure index was then multiplied by the number of years of risk to arrive at a total exposure estimate in "exposure-years." The investigators defined Type III cataract (no cases were found) as that which grossly disturbs vision and requires surgery. They defined Type II cataracts as posterior, polar subcapsular, saucer-shaped cataracts capable of interfering somewhat with visual acuity. Type I cataracts were defined as not true cataracts, but small nonhomogeneities that did not interfere with visual acuity. Figure 17 shows the percentage of people with Type I bilateral cortical cataracts as a function of population age. As shown in Figure 18, comparison of the percentage of Type I cataracts with the number of "exposure-years" for the whole population, showed that the incidence of Type I cataracts increased only slightly with exposure. In a preliminary report of a study recently conducted in Sweden, Tengroth (58) indicated no increase in frequency of lens changes between exposed and nonexposed iron and steel workers. Whereas, it appears as though the incidence of infrared cataracts caused by the older industrial sources has decreased over the years, recent investigations of exposure to industrial IR sources give rise to some concern. In 1971, Hager et al. (75) found several cases of "fire" cataracts among locomotive firemen exposed to fire temperatures of 1300 to 1500 K with wavelengths in the range of 0.8 to 1.4 μm . Irradiance levels were found to be from 0.05 to 0.18 $\text{W}\cdot\text{cm}^{-2}$. Newer IR sources include welding arcs and industrial heating lamps. Comprehensive studies of different welding processes by Hubner et al. (86), Moss et al. (83, 85), and Marshall et al. (81, 87) have shown that IR irradiance levels can be very high. Despite the high potential exposure hazard, few cataracts have been reported by welders.

The IR lamps used in paint and enamel drying operations are another source of worker exposure. In 1975, Ruth et al. (32) reported on what were designated as eye risk levels of 0.001 to 0.05 $\text{W}\cdot\text{cm}^{-2}$ from various types of heating lamps having spectral distributions in the near IR region. Optical sources growing in applications are fiber optic communication systems, which use light-emitting and laser diodes. These sources, because of the inherent attenuation factors of fiber optics, generally emit radiation in the spectral range from 0.8 to 1.0 μm . Use of IR-LED control units, such as those found in TV sets, are increasing and have stimulated studies on IR ocular effects (88,89). To date, the literature indicates no reported incidence of cataracts from exposure to these newer IR sources.

Zaret et al. (56) recently reported two cases of a rapid (3-year) cataract formation and capsular opacification allegedly due to IR exposure from a domestic electric oven and range. Although the cataract observed may not have been entirely caused by IR (patient had previous medical diathermy treatment), it is possible that IR accelerated

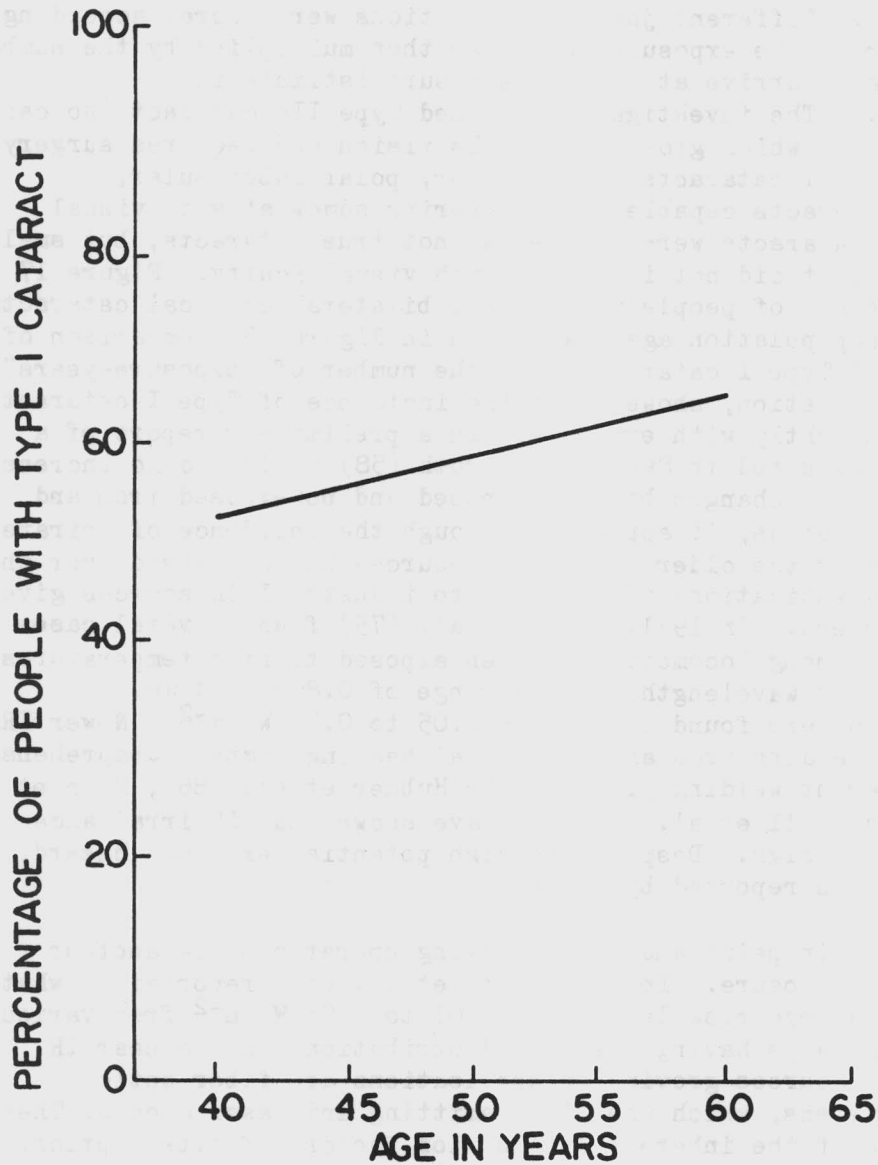


Figure 17. Percentage of people with Type I bilateral cortical cataract as function of age. Modified from reference 78.

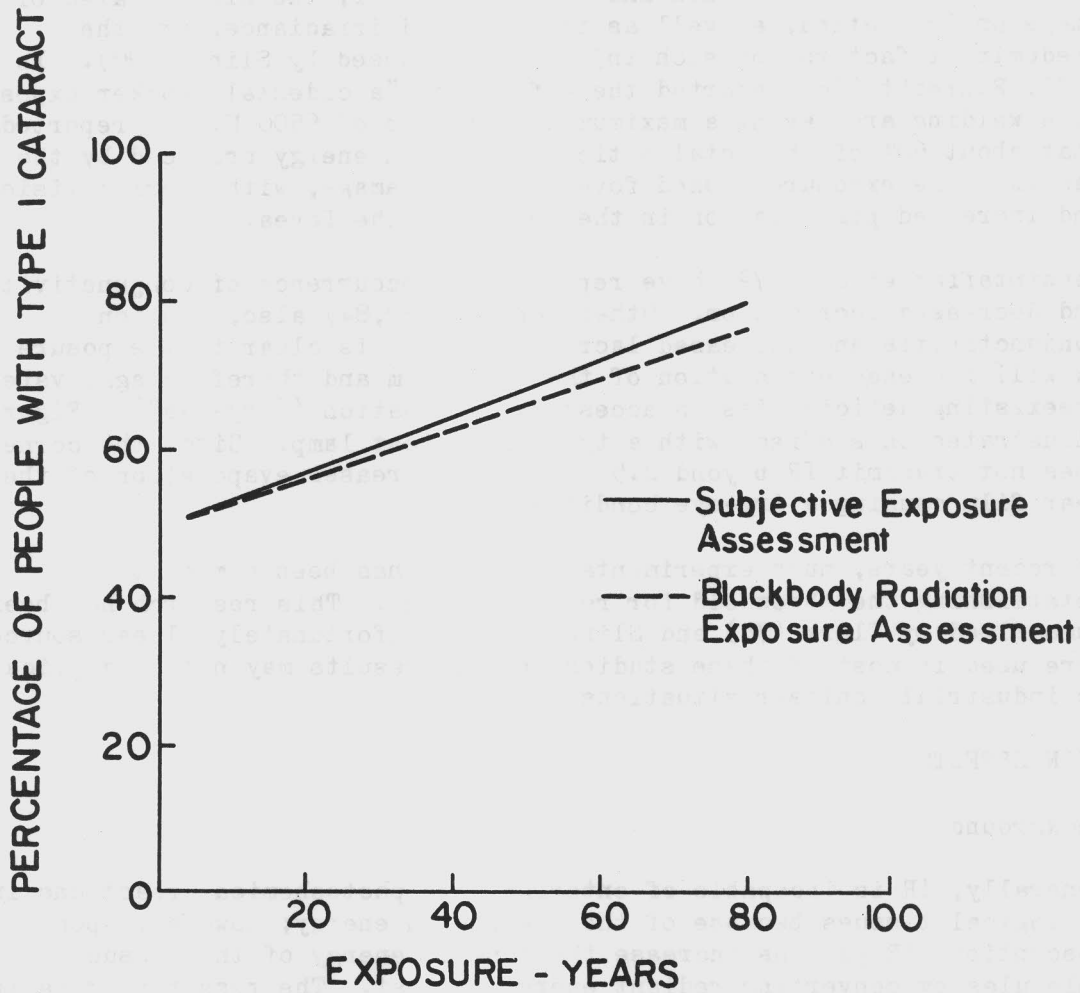


Figure 18. Comparison of percentage of people with Type 1 bilateral cortical cataract as a function of exposure assessment. Modified from reference 78.

cataractogenesis. In another case, Zaret et al. claimed IR was the sole implicating factor. Measurements of the oven ranged from 0.024 to 0.097 W.cm⁻².

As a result of exposure to certain industrial infrared sources (welding arcs, arc lamps, xenon arcs, etc.) retinal injury to workers in the form of burns and/or lesions is possible because of the focusing effect of IR on the retina by the cornea and lens. Moreover, the size or area of the image on the retina, as well as the absorbed irradiance, are the predominant factors for such injury as discussed by Sliney (90). In 1976, Ruprecht (76) reported the effects of "accidental" worker exposure to a welding arc having a maximum temperature of 6500 K. He reported that about 60% of the total optical radiation energy produced by the arc was IR. The exposure caused fovea-macular damage, with blurred vision and increased pigmentation in the center of the fovea.

Sensintaffar et al. (79) have reported the occurrence of conjunctivitis and decreased lacrimation. Other reports (70,84) also, mention conjunctivitis and decreased lacrimation. It is clear that exposure to IR will increase evaporation of the tear film and therefore aggravate preexisting deficiencies in accessory lacrimation ("dry-eye"). Figure 19 illustrates this effect with a typical IR heat lamp. Since the cornea does not transmit IR beyond 2.5 μ m, this increases evaporation of the tear film causing a dry-eye condition.

In recent years, much experimental research has been aimed at establishing the threshold for retinal damage. This research has been summarized by Clark (74) and Sliney (90). Unfortunately, laser sources were used in most of these studies and the results may not be applicable to industrial nonlaser situations.

SKIN EFFECTS

Background

Generally, IR is incapable of entering into photochemical reactions in biological tissues because of the low photon energy; however, upon absorption, IR photons increase the kinetic energy of the tissue molecules by converting radiant energy to heat. The resulting rise in temperature depends on the wavelength, exposure duration, and total energy delivered to the tissue. The degree of damage (direct and/or indirect) can be significantly influenced by the absence or effectiveness of physiological reflexes and responses.

To understand the effects of IR on skin, one must be familiar not only with the optical and thermal properties of skin, but also with its other related characteristics. Because of its high water content (60-70%), skin may be regarded as having absorption properties similar to those of water.

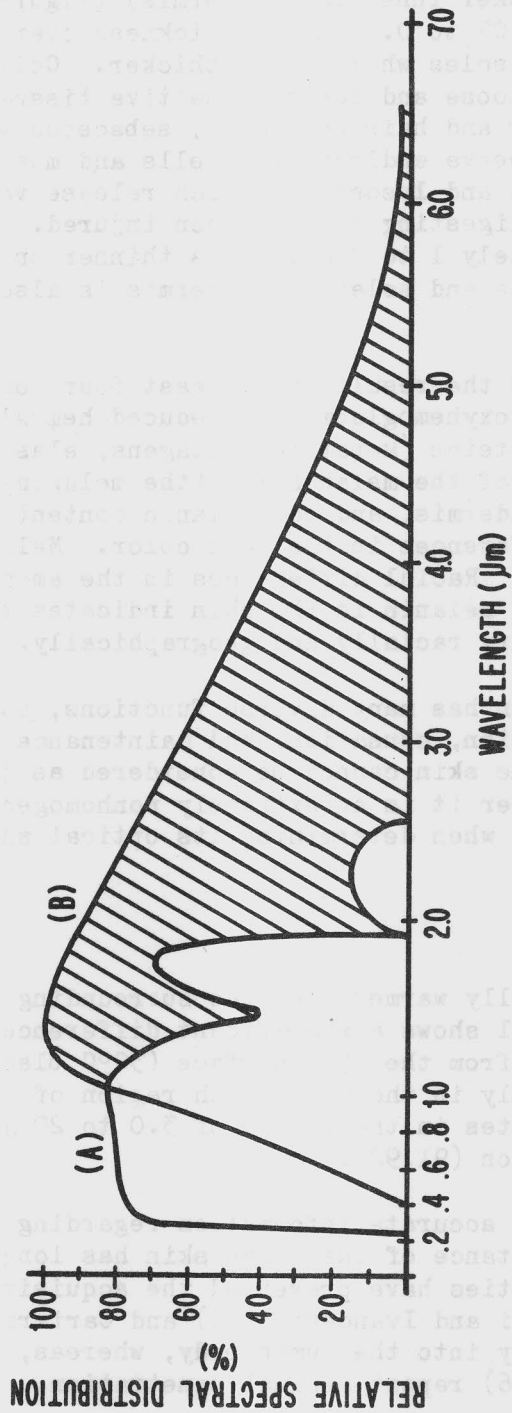


Figure 19. Conceptual explanation of dry eye. Curve (A) is the corneal transmittance and curve (B) is a typical IR lamp spectral distribution. The shaded area represents optical energy that is incident upon the cornea that produces a dry eye condition.

Anatomy and Physiology

The skin is one of the largest of the body's organs, comprising approximately 4% of the body weight and has a surface area of 1.6 to 2.0 m² in adults (20). Thickness varies over different parts of the body generally it is from 1 to 2 mm thick, although some areas may be up to 6 mm thick (18). The skin has two major layers: the thin outer layer (epidermis) and the thicker inner layer (dermis) (Figure 20). The epidermis varies from 0.07 to 0.12 mm in thickness over most of the body, except on the palms and soles where it is thicker. Collectively viewed, the dermis consists of loose and dense connective tissue (collagenous bundles) containing hair and hair follicles, sebaceous and sweat glands, diffuse blood vessels, nerve endings, fat cells and muscle. The dermis also contains mast cells and lysosomes, which release vasodilation substances and protein digesting enzymes when injured. The thickness of the dermis is approximately 1 to 2 mm; it is thinner on the eyelids and much thicker on the palms and soles. The dermis is also thinner in women than on men.

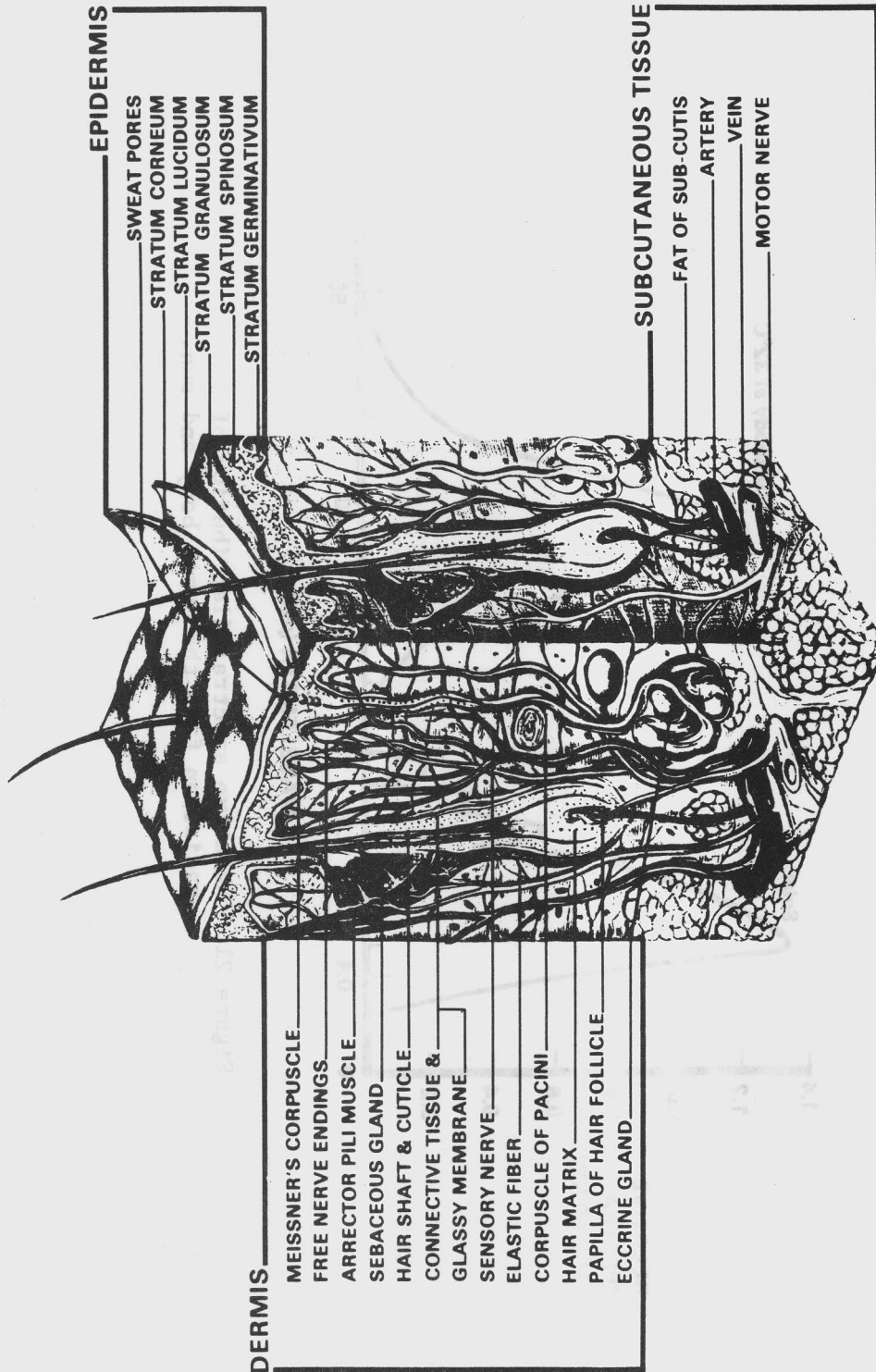
The color of the skin is the result of at least four components: (a) carotenoid pigment, (b) oxyhemoglobin and reduced hemoglobin, (c) melanin, and (d) the proteins (keratin, collagens, elastin, etc.). The number and distribution of the melanosomes (the melanin-containing granules) within the epidermis, and the melanin content of the granules determine the major differences in the skin color. Melanin-free skin is uniformly pinkish white. Racial differences in the amount and distribution patterns of melanin in the skin indicates the skin absorption of IR must vary racially and geographically.

Physiologically, the skin has many diverse functions, such as protection, excretion, thermoregulation, sensation, and maintenance of fluid and electrolyte balance. The skin cannot be considered as just an equivalent thickness of water; rather it is an extremely nonhomogeneous tissue that is important to consider when determining its optical and thermal properties.

Optical Properties

Since the skin is generally warmer than the surrounding environment, it radiates heat. Figure 21 shows a significant difference between the solar spectrum and that from the skin surface (32°C blackbody). The sun radiates predominately in the wavelength region of 0.3 to 2.0 μm , whereas human skin radiates in the region of 3.0 to 20 μm , regardless of the amount of pigmentation (91,92).

The importance of having accurate information regarding spectral reflectance and transmittance of the human skin has long been recognized, but measurement difficulties have prevented the acquisition of meaningful data. For example, Pauli and Ivancevic (93) and Cartwright (94) report that IR penetrates deeply into the human body, whereas, Aldrich (95) and Hardy and Muschenheim (96) report minimal penetration.



EPIDERMIS

- _____ SWEAT PORES
- _____ STRATUM CORNEUM
- _____ STRATUM LUCIDUM
- _____ STRATUM GRANULOSUM
- _____ STRATUM SPINOSUM
- _____ STRATUM GERMINATIVUM

DERMIS

- _____ MEISSNER'S CORPUSCLE
- _____ FREE NERVE ENDINGS
- _____ ARRECTOR PILI MUSCLE
- _____ SEBACEOUS GLAND
- _____ HAIR SHAFT & CUTICLE
- _____ CONNECTIVE TISSUE & GLASSY MEMBRANE
- _____ SENSORY NERVE
- _____ ELASTIC FIBER
- _____ CORPUSCLE OF PACINI
- _____ HAIR MATRIX
- _____ PAPILLA OF HAIR FOLLICLE
- _____ ECCRINE GLAND

SUBCUTANEOUS TISSUE

- _____ FAT OF SUB-CUTIS
- _____ ARTERY
- _____ VEIN
- _____ MOTOR NERVE

Figure 20. Microscopic view of a longitudinal section of the skin.

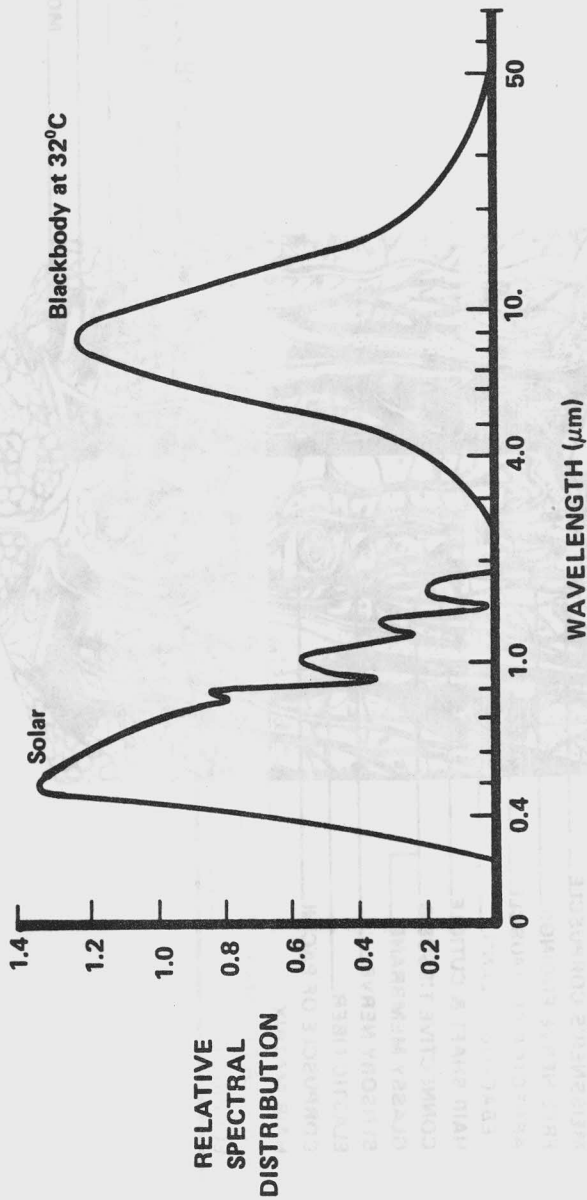


Figure 21. Relative spectral distribution of radiation from the human body and sun.

Although Hardy (91) had previously investigated the subject, Jacquez et al. provided the first reliable and verifiable data concerning the reflectance and transmittance characteristics of Caucasian and black skin (97,98). Figure 22 shows the reflectance curves determined in the above studies. From 3 to 20 μm the reflectivity is approximately 3%. Between 0.4 and 2.0 μm the reflectivity is much higher although it varies with sex pigmentation, skin area and skin blood flow. The maximum reflectivity occurs between 0.8 and 1.2 μm which is comparable to the wavelength distribution of modern IR heating devices (Figure 23). It must be remembered, however, that the different sources have different IR spectral characteristics. It should also be noted that the skin's spectral reflectance is in agreement with the spectral irradiance curve for solar radiation. Thus, the skin reflects most of the solar radiation and still is able to rid the body of heat by radiation. Beyond 1.5 μm , the skin pigmentation does not affect reflectivity.

The second important IR optical characteristic is depth of penetration into the skin surface. The skin, which is a dynamic and heterogeneous tissue, will scatter transmitted radiation as shown in Figures 24 a and b. These figures show the transmission for the indicated wavelengths through excised skin. As Hardy (91) states, if the skin were perfectly diffuse, the transmitted radiation would follow Lambert's Cosine Law for all wavelengths. Figure 24 points up two things. First, short wavelengths are scattered more than long wavelengths; and second, the differences due to wavelengths are minimized as skin thickness increases. Since scattering decreases with longer wavelengths, one would expect a greater percentage of IR transmission. The absorption of IR by skin depends not only upon skin and blood pigments and other substances, but also upon the degree of scattering in the microstructure of the skin. Figure 25 shows the transmission spectrum of excised white human skin. The dips in the spectrum at 1.4, and 1.92 μm are due to water absorption bands. This is important because water is the principal absorber in biological tissues. As illustrated, the skin is essentially opaque beyond 2.6 μm ; however, near IR penetrates the skin surface considerably, the maximum penetration being between 1.1 and 1.2 μm . Figure 25 also suggests that wavelengths in the 0.8 to 1.3 μm region are transmitted more effectively in biological materials. Figure 26 shows the average penetration of near IR below the skin surface in both black and white skin. Although Figure 26 is somewhat schematic because of the variation of skin layers, it indicates that at least 20% of the radiation up to 1.2 μm penetrates to a depth of about 2.0 mm (extrapolated) and hence interacts directly with nerve endings and capillaries. This estimate is lower than that of Forsythe and Christison (100) who reported that 20% of IR at 1.1 μm penetrated the skin to a depth of 5.0 mm. As noted by Hardy (92), the absorption coefficient for skin is not constant because of differences in scattering and absorption within the skin layers. The skin's complexity and variability make an exact calculation of IR penetration difficult. Coblenz (101) has estimated the IR skin penetration range in the spectral region ranging from 0.76 to 1.5 μm as 1 to 10mm, and in the 1.5 to 15.0 μm region as 0.05 to 1.0 mm.

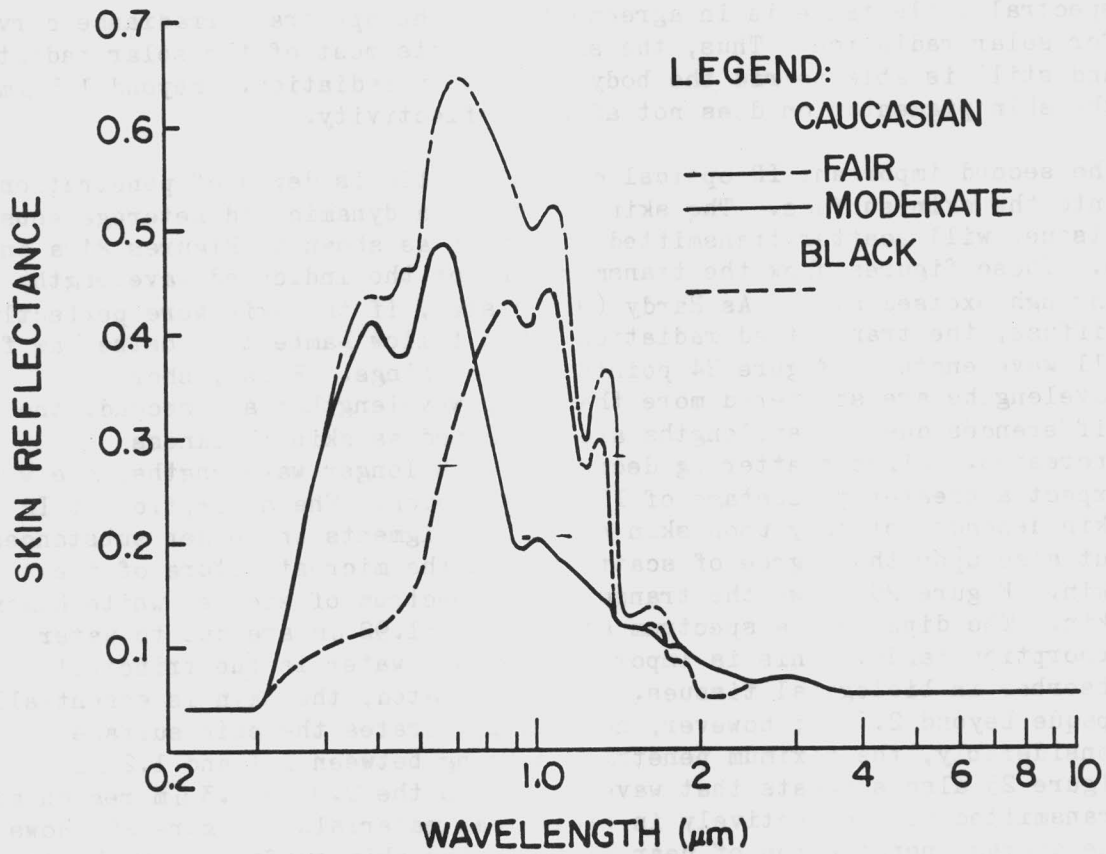


Figure 22. Spectral reflectance of human skin. Modified from references 97 and 98.

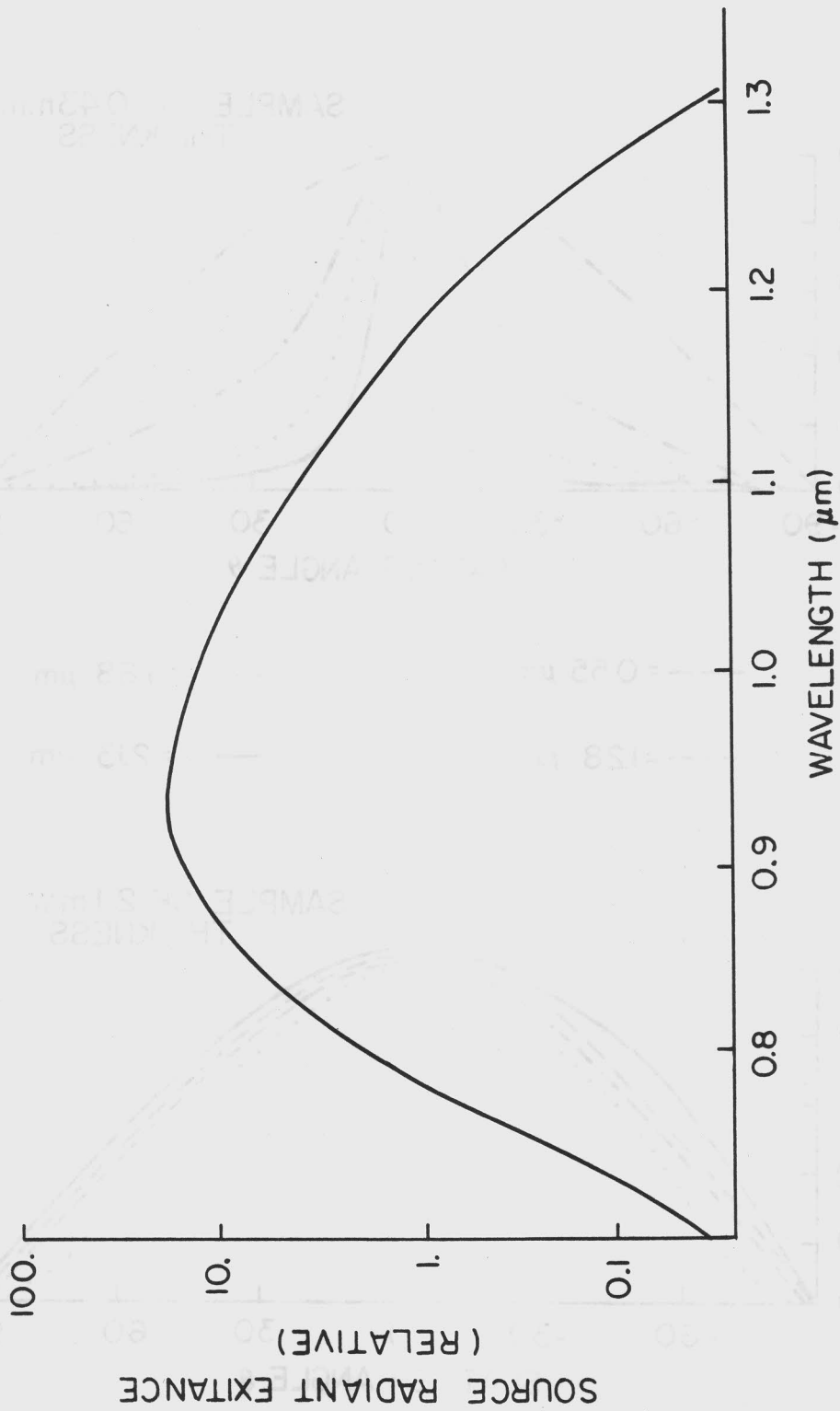


Figure 23. Emission curve for high temperature IR heater.

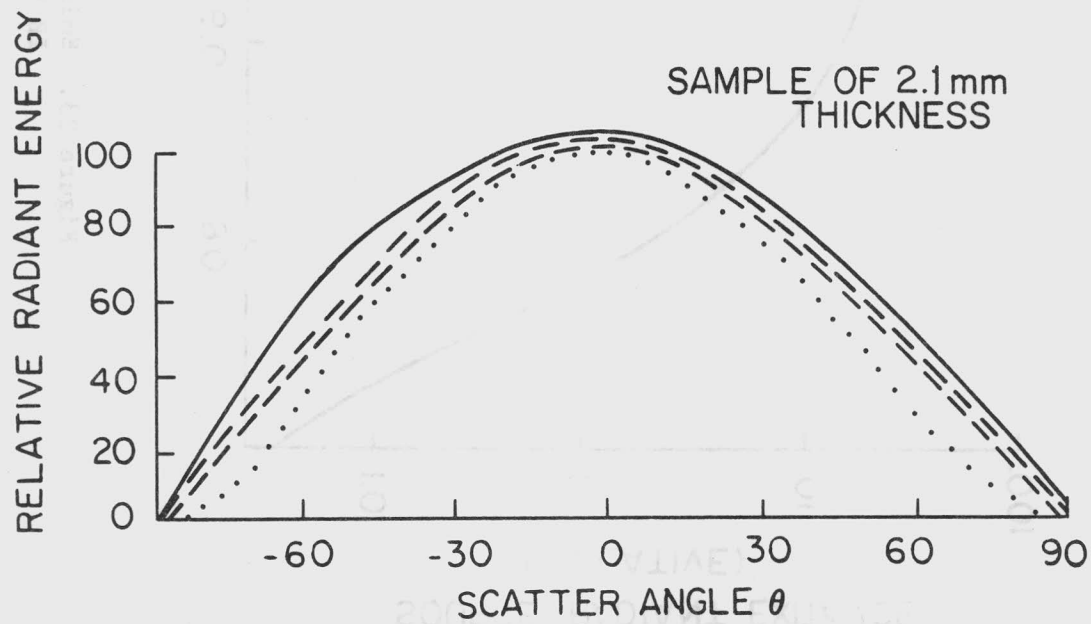
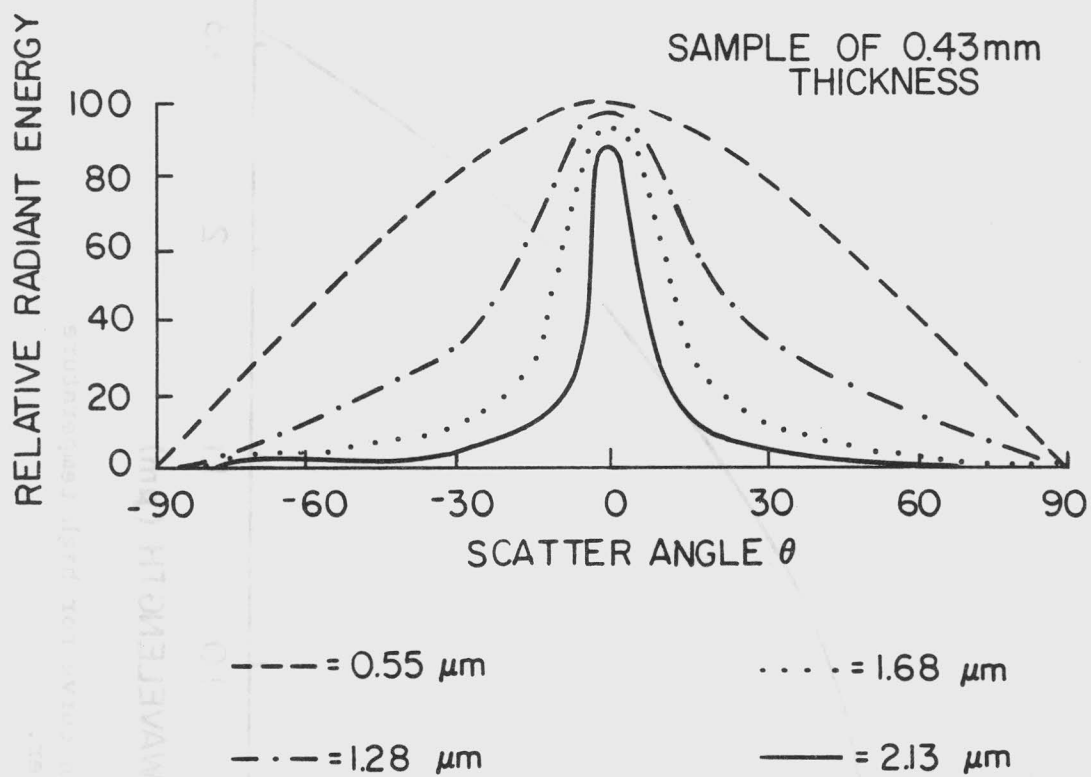


Figure 24. Scattering of transmitted energy by human skin. Modified from reference 91.

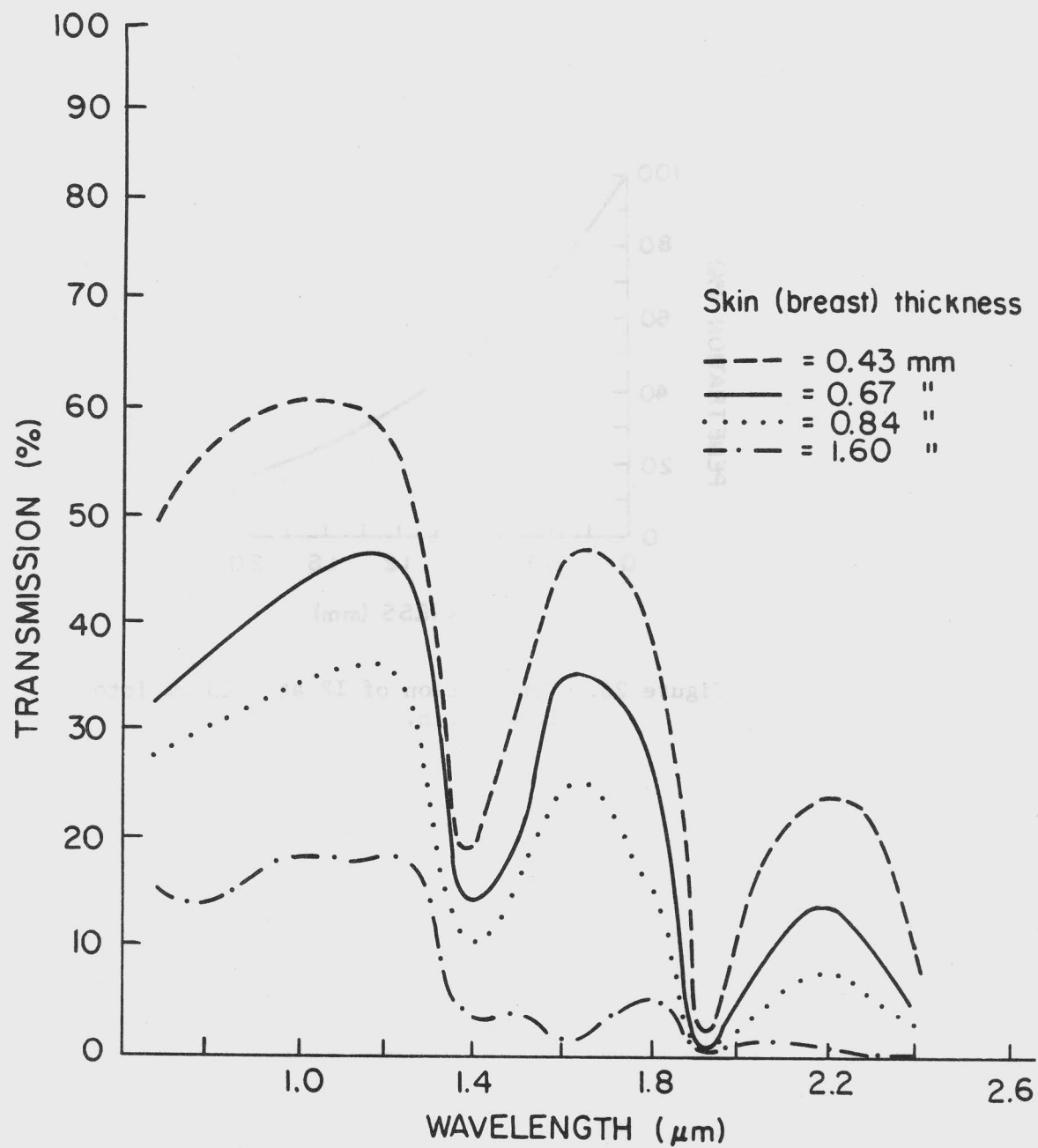


Figure 25. Spectral transmittance of excised white human skin. Modified from reference 91.

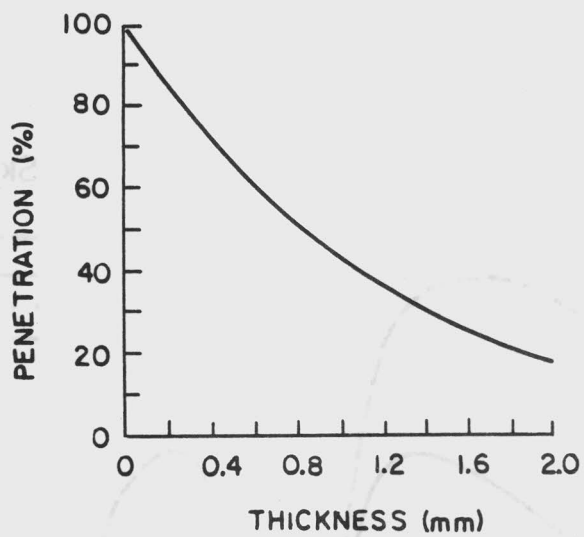


Figure 26. Penetration of IR at 1.23 μm into human skin.

Direct and Indirect Effects

It would appear that the critical wavelength region for skin exposure is between 1.1 and 1.2 μm . This region may be potentially hazardous due to IR photon penetration in the dermal layer of the skin. Caution should be exercised in high-intensity exposure situations, particularly where sources emit in the near IR region. However, non-penetrating IR will cause, for the same intensity, a higher surface temperature.

The effects most often reported include acute skinburn, increased vasodilation of the arterioles, and the gradual increase in pigmentation. The latter may be due to chronic exposure since it persists for an extended time. The development of an erythematous-like appearance among certain occupational groups exposed to high IR intensities may be viewed as a chronic effect (3). Chronic exposure to low level IR has been reported by Matelsky (1) to cause inflammation of the eyelid. Matelsky also reported more severe effects as a result of intense exposure of the eyelid and compared those effects to an ordinary burn with erythema, edema, and blistering. Figure 27 gives a conceptual explanation of these effects.

Damage Mechanisms

The principal mode of damage to tissue depends upon its optical properties and the incident IR wavelengths. For example, far IR is almost totally absorbed at the epidermis resulting in surface heating.

Several factors enter into the heating or thermal effect on tissues and the resulting symptomatology. One factor is the total energy residing in molecular or atomic vibrations which can be transferred to neighboring atoms and molecules. If one area of the skin absorbs the incident IR photons, the energy is quickly dissipated to surrounding tissue. The temperature increase is a measure of the average heat (vibrational energy) of a tissue such as skin. The quantity can be determined by using a mechanical device (thermocouple), mathematic calculations, or subjective responses.

In 1846, Webner (102) proposed that the rate of change in skin temperature was the effective stimulus for sensation (pain). Hendler and Hardy (103) later confirmed this relationship through quantitative measurements. They reported a warmth perception rate threshold of 0.001 to 0.002 $^{\circ}\text{C}$ per second at skin temperatures ranging from 32 $^{\circ}$ to 37 $^{\circ}\text{C}$. How long the skin must be warmed before a temperature sensation is elicited depends on the size of the irradiation area and the density of temperature receptors in that area. While Hendler and Hardy disregarded skin temperature as a factor, Cook (104) indicated that skin temperature alone is the vital factor in heat induced pain.

Henriques (105) proposed a second mechanism (sensation of pain), based on the direct tissue damage (denaturization) that occurred at approximately

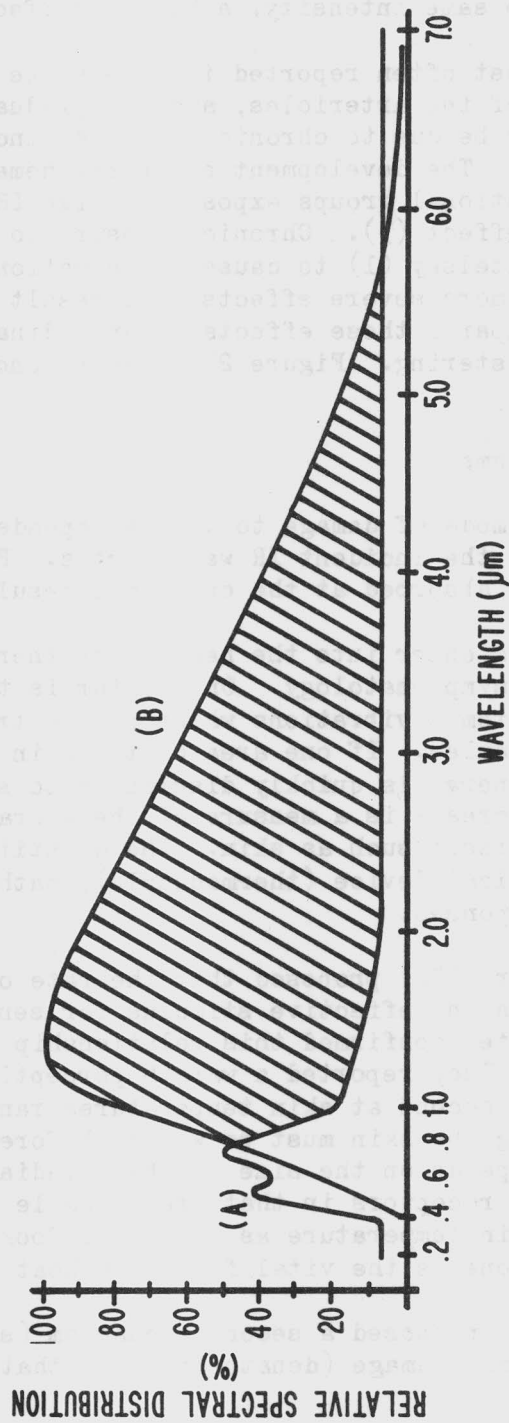


Figure 27. Conceptual explanation of dry skin. Curve A is the spectral reflection of human skin and Curve B is a typical IR lamp spectral distribution. Shaded area represents the optical energy that is not reflected by the skin and hence is absorbed, producing dry skin.

46° to 47°C. The pain threshold, regardless of initial skin temperature, occurs at a mean value of $44.5 \pm 1.3^\circ\text{C}$ (106,107); hence, it depends on skin temperature alone and not upon the rate of skin heating or the rate of change of an internal thermal gradient.

The human body has an extremely sensitive and complex protective mechanism that is stimulated by an increase in skin temperature (warmth sensation). This warmth sensation is perceived when a thermoreceptor is stimulated by heat and sends a nerve impulse to the hypothalamic thermoregulatory center in the brain. Secondary physiological responses (vasodilation) are then initiated to dissipate the absorbed infrared energy (108).

At higher intensities, thermoreceptors may not quickly sense the rapid temperature increase especially in the case of the more penetrating near IR. Effects vary with the temperature of the skin and the exposure duration.

A skin temperature lower than 44 to 45°C does not generally produce a burn (3). At or slightly above this level, reddening of the skin or erythema can occur, causing increased pain due to release of bradykinin. As the skin temperature increases to values much above 50°C most enzyme systems will be destroyed. Numerous factors affect the degree of skin responses to IR including individual variability (physical and physiological), environmental conditions (humidity), clothing, and state of physical factors.

Exposure Thresholds

Several investigations have sought to establish the level at which a pain response and simultaneous skin burn threshold occurs. Practically speaking, few skin burns result from high-intensity IR exposures. Thermal burns, which do occur in industry, usually result from contact with hot objects rather than exposure to IR sources. The level of far IR exposure upon the skin (whole body irradiance) that causes the sensation of warmth, is less than about $0.01 \text{ W}\cdot\text{cm}^{-2}$. Thus the sensation of warmth and pain serve as an inherent protection for the prevention of thermal skin injury.

OTHER REPORTED EFFECTS FROM INFRARED RADIATION

A number of reports have described specific effects of IR on man, animals, and isolated cells. Although in some cases these reports may not have been confirmed by independent workers, they are included here for completeness.

Studies by Krivobok (39) indicated that IR produced a limited level of organ and tissue degradation in areas remote from the eye. He reported changes such as vascular congestion in the spleen and kidneys. Zelentsova (109,111) reported a long-term decrease in the immunological reactivity (i.e., phagocyte count, phagocytic index, and bactericidal properties of the skin) of foundry workers exposed to IR at irradiance levels from 0.07 to 0.02 W.cm⁻². The latter report indicated that, at lower intensity levels, IR stimulated the body's protective mechanisms.

Lehmann et al. (111) demonstrated that the application of IR to the ulnar nerve area at the elbow produced a distal analgesic effect in the area supplied by this nerve. Lehmann states that this finding is in agreement with previous experimental evidence that nerve conduction can be temporarily blocked by application of IR.

A study by Broneff and Blumlein (112) indicated that the upper respiratory passages of iron foundry workers were damaged by many years of exposure to intensive near IR. Chronic rhinitis (in most instances with polyps and hyperphasia of the mucous membranes), chronic laryngitis and sinus troubles were prevalent in almost 50% of the exposed workers. According to the study, these disorders were 5 to 10 times more frequent in the exposed group than in the control group.

Episodes of apnea (a transient suspension of respiration) have been reported in infants exposed to radiant warmers (113). The relationship between changes in irradiance levels and the incidence of apnea is not known. Radiant warmers for infants are used to make up for loss of body heat. These warmers normally produce far IR, rather than near IR, radiation so as to maximize heating and minimize potential skin and eye damage. Knish (114) reported that exposure of teenage children to IR from a Solux lamp in doses of 0.5 to 0.6 cal.cm⁻² caused reaction of the sympathetic adrenergic systems and produced phasic changes in the excretion of epinephrine and neopinephrine in the urine.

Tissues located near the body surface and sensitive to thermal insult are the thyroid and testicle. The principal biological effect encountered from thermal insult to the thyroid is variance in hormone production; in the testicle, the effect is a temporary aspermia. Little information has been reported on these potential interactions, but they should not be overlooked.

The safety of overhead IR heating lamps have been questioned because of their widespread use in neonatal operating rooms. Arima and Fonkalsrud (115) studied the incidence of postoperative intestinal adhesions and

microscopic intestinal injury resulting from the use of these overhead lamps. In the study the intestines of 45 rabbits were exposed to IR for 2 hours or longer. The incidence and severity of adhesions correlated directly with the length of exposure to IR. No histological evidence of intestinal injury was apparent in exposed intestines under the study conditions. Although the results of this study may not be definitely applicable to the human infant, it suggests that "precautions" be taken when viscera are exposed to external IR sources.

Gordon and Surrey (116) used rat liver mitochondria to study the genetic effects of IR. They postulate that the sites of adenosine triphosphate production were the primary target of IR and the occurrence of chromosome aberrations was a secondary phenomenon. In 1971, Gordon et al. (117) again demonstrated such effects by exposing pig kidney cells to near IR. They observed significant increases in chromatid breaks and exchanges. Summarizing the work of other investigators in the field, Krell et al. (118) stated that near IR inhibits repair of spontaneous aberrations by interfering with chromosome hydrogen bonding, whether in base pairing in the double stranded DNA; in the complex structure of histone, enzymes, and DNA; or in both. The average energy required to dissociate hydrogen bonds is 0.06 eV, which is within the capability of IR. If IR can cause these chromosome effects, perhaps mutagenesis can occur; however, hereditary changes due to IR have not been demonstrated to date. The significant number of reports in the literature on genetic effects produced by IR exposure of insects, plants, and animal cells creates some concern that IR may have the potential to affect human cells.

Although there is no evidence IR per se can cause cancer, it may be implicated in carcinogenesis induced primarily by other agents (119-121). Henry (122), for instance, reports an increased incidence of skin cancer in various workers occupationally exposed to heat. Peterkin (123) reported that exposure to heat has also been associated with an increased incidence of squamous cell carcinoma of lower extremities in Irish women. These women spent much time sitting in front of peat fires and, as a result, developed erythema ab igne, a condition that is presumably related to chronic exposure to hot fires. Because peat fires can emit ultraviolet radiation and chemical carcinogens then the carcinomas reported by Henry and Peterkin may have resulted from chronic exposure to ultraviolet radiation and other chemicals rather than IR.

In a related vein, Lawrence (124) observed a rare occurrence of squamous cell carcinoma in old burn scars (Marjolin ulcers). Whether or not a casual relationship exist between the two is still unknown. Of interest is the evidence by Owens (125), which shows that mice irradiated with UV-B develop more skin tumors at a faster rate when they are kept in a heated environment (38 to 39°C) than at normal room temperature.

Similar conclusions may be made regarding possible synergistic effects between IR and chemical carcinogens. Hahn and Strand (126) have shown that hyperthermia (43°C) increases mammalian cell membrane permeability and enhances the effects of adriamycin and other cytotoxic agents. This

suggests that hyperthermia--and by implication, exposure to IR--might also potentiate the effects of chemical carcinogens (127).

Beyond this indirect evidence, little information exists that links exposure to IR with carcinogenesis. Because of the serious consequences of underestimating cancer risk, however, further investigations should be performed.

INFRARED STANDARDS

There are currently no official standards covering exposure of skin or eyes to non-coherent IR "extended" sources. The only standard available for evaluating such sources is the American National Standards Institute (ANSI) Z-136.1 standard (128) which was developed only for laser sources.

Several investigators have suggested safe IR exposure levels for the eye. In 1968, Matelsky (1) stated that acute ocular damage from incandescent "hot" bodies can occur with a radiant exposure on the cornea of 4 to 8 J.cm⁻²; however, this single radiant exposure concept ignores exposure duration. He further recommended that a maximum permissible radiant exposure of 0.4 to 0.8 J.cm⁻² would probably prevent the occurrence of chronic effects on intraocular tissues.

While Matelsky's proposal was based on industrial source measurements, Sliney (2) used experimental laser criteria to recommend that a chronic exposure to industrial IR sources be limited to an average ocular irradiance of approximately 0.01 W.cm⁻² to prevent ocular effects. Exposures up to 0.1 W.cm⁻² would be permitted for several minutes.

The American Conference of Governmental Industrial Hygienists (ACGIH) has issued a notice of intent to establish a TLV for ocular exposure to near IR from noncoherent sources (129). The TLV would apply to ocular exposure in an 8-hour workday and require knowledge of the spectral radiance and total irradiance of the source as measured at the eye of the worker.

It must be noted that the use of a laser standard to limit exposure to broadband IR sources can only be regarded as a temporary measure. The lack of data on biological effects of occupational IR exposure impedes the setting of rational occupational standards for acute and chronic exposures.

INFRARED PROTECTION AND CONTROL MEASURES

The primary goal in limiting exposure to IR sources, especially in the near IR region, is protection of the skin and eyes. This is best accomplished by engineering means, e.g., controlling the emission from the source and erecting barriers. Although such measures are not extremely sophisticated, if used properly, they can be reasonably effective. The high surface temperature of hot equipment may be reduced by insulation. However, this method is usually practical only for equipment with low surface temperatures due to thickness of material needed for effective insulation.

Barriers can be placed between the source and the worker to reflect or absorb IR. Aluminum is widely used to control IR in the industrial environment because of its high reflectance. If used, its surface must be well polished and clean for maximum effectiveness. Other types of shields, such as glass, heat exchangers and aluminum cloth, absorb IR and give up heat by convection. Cooling is accomplished by special ventilation or water circulation. For convenience purposes, shields should be portable in case they have to be removed for emergency repair or maintenance of machinery. Baffled lamp housings are often used to control laboratory arc sources such as spectroscopic equipment and optical calibration sources. Reflective booths and curtains are used in welding operations to protect passersby. Although conventional welding curtains (canvas) are effective in absorbing optical radiation, recent investigations of transparent curtains demonstrate IR transmittance levels up to 80% (130). The spectral transmission properties of such curtains should be examined before they are used for protection against IR exposure (131). In paint and enamel drying operations that use arrays of intense heating lamps, enclosures should be interlocked with glass or metal doors. Posting of warning signs or labels is also advisable. Pre-placement medical examinations with particular attention to eye and skin lesions can be an important means of avoiding misassignment of susceptible workers to jobs associated with intensive IR.

In cases where engineering controls are nonexistent or inadequate, personal protective devices can be used. Lightweight cotton clothing is recommended for skin protection. Additional protection is afforded by reflective aluminized aprons or coats, and when radiant heat loads are excessive, ventilated suits may be necessary. The transmittance values specified for filters in welding goggles and furnace inspection goggles for glass workers, steel and foundry workers, and welders were originally developed empirically. Optical transmission characteristics are now standardized as shades numbers and are specific for particular applications.

Although maximum transmittance for IR are specified for each shade, the visual transmittance (T_v) or visual optical density (D_v) defines the shade number ($S\#$) as follows:

$$S\# = 7/3 (D_v) + 1 \quad (5)$$

where $D_v = -\log_{10} T_v$. For example, a filter with a visual

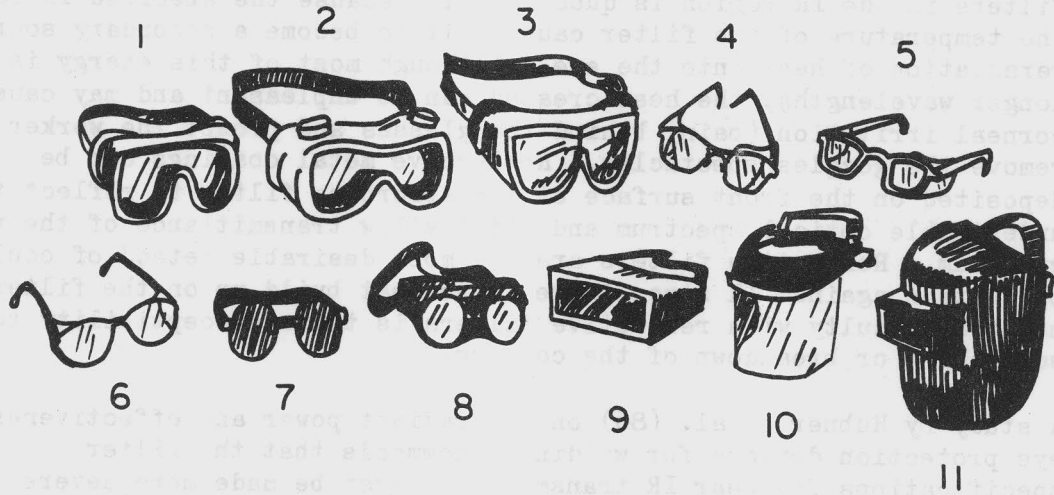
transmittance of 1000 (e.g., $D_V = 3$) has a shade number of 8. The standards for shade number were developed in the late 1920s at the National Bureau of Standards (132) and have since been adapted, with slight modification, for transmission tolerances as a Federal Standard (133) and as ANSI Standard Z87.1 (134). Various types of goggles and spectacles with proper filters can be worn (Figure 28).

In a recent study, Campbell (135) found that only one of 55 shade models of welding filter plates failed to meet the ANSI specifications for IR transmittance up to $2.6 \mu\text{m}$. The overall effectiveness of absorptive filters in the IR region is questionable because the absorbed IR raises the temperature of the filter causing it to become a secondary source for reradiation of heat into the eye. Although most of this energy is at longer wavelengths, the heat created can be unpleasant and may cause corneal irritation (pain) behind the glasses and prompt the worker to remove the goggles/spectacles. Reflective metal coatings can be deposited on the front surface of the absorbing filter to reflect the undesirable optical spectrum and still allow transmittance of the visible spectrum. Reflective filters are the most desirable method of ocular protection against IR since there is no heat build up on the filter. The major difficulty with reflective filters is their susceptibility to scratching or breakdown of the coating.

A study by Hubner et al. (86) on the radiant power and effectiveness of eye protection devices for welding recommends that the filter specifications for near IR transmittance must be made more severe, whereas they may be less stringent for the far IR region (except for high temperature welding). The study also recommends that the eye protection specifications for furnace inspection goggles be made much more stringent.

Probably the most important factor in the design of eye-protection devices is comfort. No matter how effective the filter may be, goggles that are not reasonably comfortable will not be worn. A compromise must be reached between comfort and safety considerations. In the future, however, safety may have to receive more emphasis because of the rapid increase in new, near IR sources and in the number of potentially exposed workers. Increased responsibility will be imparted to the industrial hygienist to recognize potential IR hazards and to administer the proper control measures.

SELECTION CHART OF RECOMMENDED EYE PROTECTORS



1. GOGGLES, Flexible Fitting, Regular Ventilation
2. GOGGLES, Flexible Fitting, Hooded Ventilation
3. GOGGLES, Cushioned Fitting, Rigid Body
4. SPECTACLES, Metal Frame, with Sideshields
5. SPECTACLES, Plastic Frame, with Sideshields
6. SPECTACLES, Metal-Plastic Frame, with Sideshields
7. WELDING GOGGLES, Eyecup Type, Tinted Lenses
8. WELDING GOGGLES, Coverspec Type, Tinted Lenses
9. WELDING GOGGLES, Coverspec Type, Tinted Plate Lens
10. FACE SHIELD (available with plastic or mesh window)
11. WELDING HELMETS

Figure 28. Selection chart of eye protectors.

SUMMARY

In general the early literature dealing with the biological effects of IR is of questionable value because available instrumentation lacked sophistication and sensitivity. Because IR is primarily an occupational hazard, more research is needed on actual industrial exposure to determine threshold limits and irradiance values and to study the epidemiology of IR-induced damage. Whereas the IR emitted by older occupational sources (i.e., glass and metal furnaces) has been predominantly in the far region; that emitted by newer and more intense sources (i.e., home and industrial heating devices) is mainly in the near region. As a result, the risk of ocular and skin damage could increase.

The eye is a complex organ consisting of several interdependent, yet optically and biologically differing, ocular structures. The absence of an extensive heat-dissipation mechanism and the strong focusing ability make the eye the primary critical organ for damage by IR radiation. Transmission and absorption studies indicate that the cornea, iris, lens, and retina are the ocular structures most likely to be damaged. The action spectrum of eye hazards seems to be confined predominately to the near IR region. Peak transmittance is about $1.1 \mu\text{m}$ for the cornea. A strong absorption of IR energy by the aqueous humor, iris, lens, and vitreous humor occurs at wavelength bands of 0.9 to $1.1 \mu\text{m}$ and 1.2 to $1.4 \mu\text{m}$. The cornea is essentially opaque beyond $2 \mu\text{m}$. The effects of IR on secondary ocular tissues have not been confirmed. Figure 29 shows the potential IR biological effects as a function of wavelength.

In general, near IR is potentially far more hazardous than IR. The direct relationship between exposure to IR and the development of lenticular opacities as definitive for cataract development seems valid. The primary cause-effect presently remains unresolved. Most of the literature favors Goldman's hypothesis which states that results from IR damage to the lens (cataract) results from absorption of the radiation by the iris and subsequent heat transfer to the lens. Recent investigations, however, support the "combined" theory which involves both heat transfer from the iris and direct absorption by the lens. The acute effect on the retina is thermal with lesion development. The effect on the eyelid and cornea is essentially that of a cutaneous burn (3).

The ability of significant amounts of IR to penetrate the eyelid to the cornea increases the probability of internal ocular damage. The iris is very susceptible to damage by absorption of IR, apparently because of its heavy pigmentation. Except for general heating, no other effects have been reported. The literature contains no references to investigations dealing with the transmittance or absorptance properties of the iris. There is a lack of quantitative occupational exposure data on low-level, chronic effects in these ocular structures.

The definition of what is a cataract has received considerable attention in the literature. A central question is the difference between a temporary opacity and a permanent cataract. Apparently, many researchers have noted lenticular opacities, but whether or not these opacities are made visible by the increased heat associated with IR exposure or by

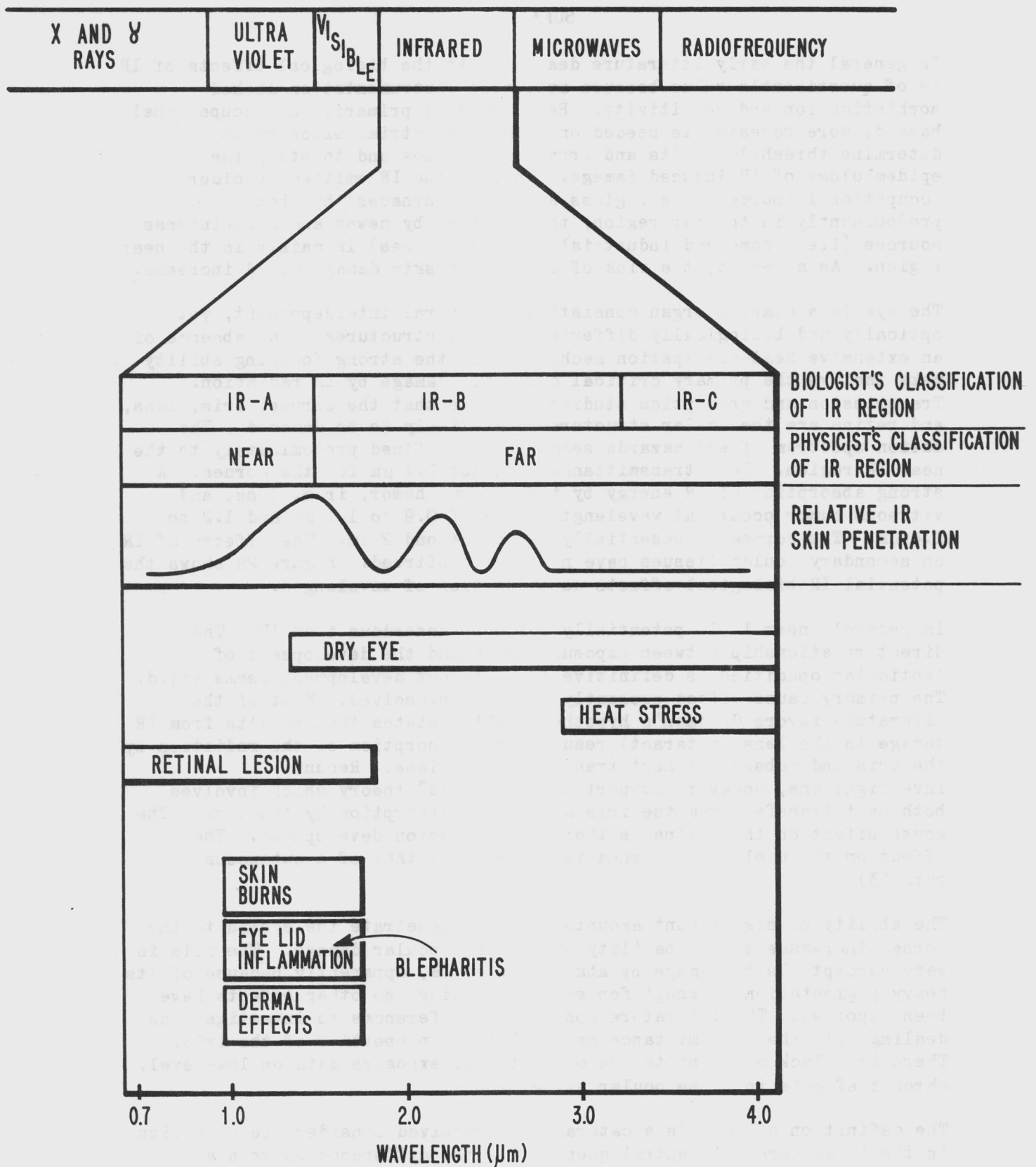


Figure 29. Classification scheme for IR.

direct photonic involvement is not clear. Other questions concern the location of opacities and their duration. One thing is very apparent, however, regardless of the etiology (if any) of opacities, it is probably safe to state that this potential hazard has largely been eliminated by use of appropriate filters, control measures, and automated techniques. No epidemics of IR cataracts have been or are being currently reported anywhere.

The skin is a fairly complex nonhomogeneous structure with well-defined and generally accepted optical properties. The action spectrum of skin hazards seems to be confined essentially to the near IR region with a peak around 1.1 to 1.2 μm . The near region is potentially more hazardous than the far region because of its ability to penetrate into the dermis. Reported damaging effects are acute skin burn, vasodilation of capillary beds, and long-lasting pigmentation increase. Caution should, therefore, be exercised in cases of possible exposure to high-intensity occupational sources, i.e., newer sources that emit in the near region. Such sources are IR space heaters that are being used more and more by consumers and industries to reduce home and office heating costs. Such devices warm up any solid object in front of it almost instantly with little energy being expended on heating air. These devices could conceivably emit high IR irradiance levels at close distances and produce adverse effects on portions of unsuspecting populations, i.e., infants and elderly. Extensive research is lacking on the effects of low intensity chronic exposure from near IR sources. Available data indicate only the occurrence of an erythematous-like appearance of the skin and some eyelid inflammation in certain occupations.

The skin has an inherent, natural protective mechanism to sense the thermal effect of IR before the pain or burn threshold is reached. In addition, the body has a fairly effective heat-dissipating mechanism. Future injuries may occur from excessive exposure to high-intensity, near IR sources, but thus far such cases have been infrequent. The effect of far IR as a cause of heat stress may decrease, because newer sources emit predominantly in the near IR region. The other biological effects attributed to IR are only reported observations and further investigation is necessary before any conclusions can be made. No human mutagenic or carcinogenic effects from exposure to IR have been reported to date.

It therefore appears that the greatest biological concern associated with IR, based on all the available information, would either be a dry eye/skin condition or retinal damage - rather than the historical cataract involvement. This is not to imply that cataract production is impossible for broad-band IR exposure, but rather to suggest to Industrial Hygienists that a far more realistic and controllable effect exists. The recent studies by Pitts and Tengroth on cataractogenesis have helped to make this risk interpretation. With the rapid rise of new IR sources, both near and far, it is recognized that more investigations need to be performed on the role of IR synergism with ultraviolet and/or visible radiation as well as microwave radiation.

The lack of pertinent industrial exposure data has resulted in the

virtual nonexistence of occupational standards for broadband, noncoherent IR sources. A great need exists for research in industrial situations to establish exposure standards; to determine irradiance levels; and to evaluate the role that length of exposure plays in damaging the eyes and skin. Data collected should include not only the area and spectral distribution of the source, but also the working conditions, the protective measures, and the distance of workers from the source so more precise estimates can be made of the radiation dose, and the dose rate incident on the eye and skin.

REFERENCES

1. Matelsky, I. 1968. The Non-Ionizing Radiations. In: Industrial Hygiene Highlights. Industrial Hygiene Foundation of America, Volume 1 p. 140. Pittsburgh.
2. Sliney, D.H. 1972. Nonionizing Radiation. In: Industrial Environmental Health. L.V. Cralley (Editor), Volume 1, pp. 171-241. Academic Press, Inc. New York.
3. Michaelson, S.M. 1972. Human Exposure to Non-Ionizing Radiant Energy Potential Hazards and Safety Standards. Proceedings of the IEEE. 60(4): 389-421.
4. Barr, E.S. 1961. The Infra-red Pioneers: I. Sir William Herschel. Infrared Phys. 1:1.
5. Barr, E.S. 1962. The Infra-red Pioneers: II. M. Melloni. Infrared Phys. 2:67.
6. Vasko, A. 1968. Infra-red Radiation. The Chemical Rubber Company Press, Cleveland.
7. Smith, R.A., F.E. Jones, and R.P. Chasmar. 1968. The Detection and Measurement of Infra-red Radiation. Oxford University Press, London.
8. Barr, E.S. 1963. The Infra-red Pioneers: III. S.P. Langley. Infrared Phys. 3:195.
9. Cited in Reference 6
10. Vanzetti, R. 1972. Practical Application of Infrared Techniques. John Wiley and Sons, Inc., New York.
11. Commission Internationale de L'Eclairage (International Commission on Illumination). 1970. International Lighting Vocabulary, 3rd Edition, Publ. CIE No 17 (E-1.1), CIE, Paris.
12. Meyer-Arendt, J.R. 1968. Radiometry and Photometry: Units and Conversion Factors. Appl. Opt. 7:2081.
13. Keyes, R.J. (Editor). 1977. Optical and Infrared Detectors. Volume 19 of Topics in Applied Physics. Springer-Verlag, Berlin.
14. Wolfe, W., and G.J. Zissis (Editors). 1979. The Infrared Handbook. Available from ERIM, P.O. Box 618, Ann Arbor, Michigan 48107.
15. Thekaekara, M.P. 1974. Data on Incident Solar Energy. In: The Energy Crisis and Energy From the Sun. Institute of Environmental Sciences, p. 21. Mt. Pleasant, Illinois.

16. Moss, C.E., W.E. Murray, D.L. Conover, and D. Kuhre. May 1977
Estimated Number of United States Workers Potentially Exposed to
Electromagnetic Radiation. Paper presented to AIHC, New Orleans.
17. Criteria for a Recommended Standard...Occupational Exposure to Hot
Environments. 1972. NIOSH Publication No. (HSM 72-10269).
18. Bloom, W., and D.W. Fawcett. 1968. A Textbook of Histology. W.B.
Saunders Company, Philadelphia, Pa.
19. Anthony, C.P., and N.J. Kolthoff. 1971. Textbook of Anatomy and
Physiology. The C.V. Mosby Company, St. Louis.
20. Report of the Task Group on Reference Man. 1975. International
Commissions on Radiological Protection Publication No. 23, Pergamon
Press, New York.
21. Vogt, A. 1932. Fundamental Investigations of the Biology of
Infrared. Klin. Monatsbl f. Augenh. 89:251-263.
22. Kutscher, C.F. 1946. Ocular Effects of Radiant Energy. Ind. Med.
15:311.
23. Minton, J.M. 1949. Occupational Eye Diseases and Injuries.
William Heineman Books, Ltd., London.
24. Wiesinger, H. et al. 1956. The Transmission of Light Through the
Ocular Media of the Rabbit Eye. Am. J. Ophth. 42:907.
25. Geeraets, W.J. et al. 1960. The Loss of Light Energy in Retina and
Choroid. Arch. Ophth. 64:606.
26. Boettner, E.A., and J.R. Wolter. 1962. Transmission of the Ocular
Media. Invest. Ophth. 1(6):766-783.
27. Prince, J.H. 1962. Spectral Absorption of the Retina and Choroid
from 340-1700 nm. Publication 14, Institute for Research in Vision,
Ohio State University.
28. Geeraets, W.J., and E.R. Berry. 1968. Ocular Spectral
Characteristics as Related to Hazards from Lasers and Other Light
Sources. Amer. J. Ophth. 66:15-20.
29. Barker, F.M. 1979. The Transmittance of the Electromagnetic
Spectrum From 200 to 2500 nm Through the Optical Tissues of the Eye
of the Pigmented Rabbit. Doctoral thesis, College of Optometry,
University of Houston.
30. Fischer, W., B. Vermeulen, and W. Eymers. 1936. Arch.
Augenheilkunst 109:462.

31. Franke, W., 1958. Archiv, Fur Gewerbepathologie und Gewerbehygiene 16:1539.
32. Ruth, W., M. Levin, and B. Knave. 1976. Occupational Hygiene Evaluation of Infrared Emitters for Drying Automobile Enamel. Study Report AMMF 104/76, Board of Occupational Safety, Stockholm.
33. Verhoeff, F.H., L. Bell, and C.B. Walker. 1916. The Pathological Effects of Radiant Energy on the Eye: An Experimental Investigation with a Systematic Review of the Literature. Proc. Am Acad. Sci. 51:630-818.
34. Turner, H.S. 1970. The Interaction of Infrared Radiation with the Eye: A Review of the Literature. The Ohio State Research Foundation, Columbus, Ohio.
35. Lele, P.P., and G. Weddell. 1956. The Relationship Between Neurohistology and Corneal Sensibility. Brain. 79:119.
36. Dawson, W.W. 1963. Experimental Ocular Pathology Related to Corneal Transmittance and Infrared Psychophysics. J. Appl. Phys. 18:1013.
37. Fine, B.S., S. Fine, L. Fiegen, and D. MacKeen. 1968. Corneal Injury Threshold to Carbon Dioxide Laser Irradiation. Amer. J. Ophthalmol. 66:1.
38. Liebowitz, H.M., and G.R. Peacock. 1969. Corneal Injury Produced by Carbon Dioxide Laser Radiation. Arch. Ophthalmol. 81:713.
39. Krivobok, V.T. 1941. L'Action des Rayon Infra-Rouges Sur L'Oeil (The Action of Infra-Red Rays on the Eye). Eksper. Med. 8(2): 53-55.
40. Duke-Elder, W.W. 1954. Textbook of Ophthalmology: VI Injuries. The C.V. Mosby Co., St. Louis.
41. Blois, M.S. 1969. Biological Free Radicals and the Melanins. In: Solid State Biophysics (S.J. Wyard, ed). McGraw-Hill, New York, 243-262.
42. Hayes, J.R., and M.L. Wolbarsht. 1968. A Thermal Model for Retinal Damage Induced by Pulsed Lasers. Aerospace Med. 39:474-480.
43. Goldmann, H. 1933. Genesis of Heat Cataract. Arch. Ophthalmol. (New Series). 9:314-316.
44. Wolbarsht, M.L. 1978. Safe Ocular Levels for IR Occupational Exposures. Final report, NIOSH Grant OH 0053-04. November.
45. Glansholm, A. 1977. Personal communication.

46. Heister. 1739. Cited in reference 40.
47. Emarah. M.H.M. 1973. Occupational Cataract Review of Literature Report of A Case Thermal Cataract In The Printing Industry. Bull. Ophth. Soc. Egypt. 66:159-165.
48. MacKenzie, W.A. 1855. A Practical Treatise on Diseases of the Eye. Blanchard and Lea, Philadelphia.
49. Meyhofer, W. 1886. Cited in Reference 34.
50. Legge. 1907. Cited in Parson, J.H. 1910. Some Effects of Bright Light Upon the Eyes. J. Am. Med. Assoc. 55:2027-30.
51. Edbrooke, C.M., and C. Edwards. 1967. Industrial Radiation Cataract: The Hazards and the Protective Measures. Ann. Occup. Hyg 10:293.
52. Robinson, W.D. 1915. Glass-Workers' Cataract. Ophth. 13:538-554.
53. Goldmann, H. 1935. The Origin of Glassblowers' Cataract. Ann d' Ocul. 172:13-41.
54. Goldmann, H., H. Koenig, and F. Maeder. 1958. The Permiability of the Eye Lens to Infrared. Ophthalmologica. 120:198-205.
55. Langley, R.K., C.B. Mortimer, and C. McCulloch. 1960. The Experimental Production of Cataracts by Exposure to Heat and Light. Arch. Ophth. 63:473.
56. Zaret, M.M., W. Z. Snyder, and L. Birenbaum. 1976. Cataract After Exposure To Non-Ionizing Radiant Energy. Brit J. Ophthal. 60:632.
57. Pitts, D G., A.P. Cullen, and P. Dayhaw-Barker. 1980. Infrared Cataract Ocular Threshold Levels for Infrared Radiation Cataractogenesis. DHHS (NIOSH) Publication No. 80-121, June.
58. Tengroth, B.M., E. Lydahl, and B.T. Philipson. 1979. Infrared Cataract In Furnacemen. Paper presented to ACGIH Non-Ionizing Radiation Symposium, Washington, D.C.
59. Gehring, P.J. 1971. The Cataractogenic Activity of Chemical Agents. CRC Critical Reviews in Toxicology. September: 93-118.
60. Cogan, D.G. 1950. Lesions of the Eye From Radiant Energy. J. Am. Med. Assoc. 142:145.
61. Bredemeyer, H.G., O.A. Wiegmann, A. Bredemeyer, and H.R. Blackwell. 1963. Radiation Thresholds for Chorioretinal Burns. Technical Documentry Report No. AMRL-TRL-63-71, Wright-Patterson AFB, Ohio.

62. Geeraets, W.J., and D. Ridgeway. 1963. Retinal Damage From High Intensity Light. *Acta. Opth. Suppl.* 76, 73-77:109.
63. Ham, W.T. Jr., H.A. Mueller, R.C. Williams, and W.J. Geeraets. 1973. Ocular Hazards From Viewing the Sun Unprotected and Through Various Windows and Filters. *Applied Optics.* 12:2122.
64. Ham, W.T. Jr., H.A. Mueller, J.J. Ruffold, and A.M. Clarke. 1979. Sensitivity of the Retina to Radiation Damage as a Function of Wavelength. *P. Chemistry and P. Biology.* 29:735-743.
65. Mueller, H. 1924. Cited in reference 34.
66. Fry, G.A., and N.D. Miller. 1964. Visual Recovery From Brief Exposures To Very High Luminance Levels. Technical Document Report No. SAM-TDR-64-36. USAF School of Aerospace Medicine, Brooks AFB, Texas.
67. Luckiesh, M., and F.K. Moss. 1937. Infrared Radiation and Visual Function. *J. Opt. Soc. Amer.* 27(2):69-71.
68. Sliney, D.H., R.T. Wangemann, J.F. Franks, and M.L. Wolbarsht. 1976. Visual Sensitivity of the Eye to Infrared Laser Radiation. *J. Opt. Soc. Amer.* 66(4): 339-341.
69. Sand and Obata. 1957. Cited in reference 34.
70. Medvedovskaya, T.P. 1970. Data on the Condition of the Eye in Workers at a Glass Factory. *Gigiena i Sanitariya.* 35: 105-106.
71. Ruth, W. 1975. A Method to Evaluate Occupational Hazards From Infrared Radiation. Master's Thesis, Loughborough University, Loughborough, England.
72. Dunn, K.L. 1950. Cataract From Infrared Rays (Glass-Workers Cataract). *Arch. Ind. Hyg. Occ. Med.* 1: 166-180.
73. Keatinge, G.F., J. Pearson, J.P. Simons, and E.E. White. 1955. Radiation Cataracts in Industry. *Arch. Ind. Hlth.* 11: 305-314.
74. Clark, B.A.J. 1968. Welding Filters and Thermal Damage to the Retina. *Australian J. Optom.* SI(4): 91-98.
75. Hubner, H.J., E. Sutter, and K. Wicke. 1970. Measurement of Radiant Power at Welding Processes and Consequences for Eye Protection Against IR Radiation. *Optik.* 31(5): 462-476.
76. Hager, G., S. Pagel, and D. Broschmann. 1971. Fire Cataracts Among Locomotive Firemen. *Verk. Med.* 18(10): 443-449.
77. Ruprecht, K.W. 1976. Fovea-Maculopathy Resulting From Arc Welding. *Zentralblatt Arbeitsmid.* 26: 200.

78. Marshall, W.J. 1978. Nonionizing Radiation Protection Special Study No. 42-0360-78, Infrared Radiation Hazard Evaluation of the Rotary Forge. U.S. Army Environmental Hygiene Agency, Aberdeen Proving Grounds, Maryland.
79. Wallace, J., P.M. Sweetnam, C.G. Warner, P.A. Graham, and A.L. Cochrane. 1971. An Epidemiological Study of Lens Opacities Among Steel Workers. *Brit. J. Industr. Med.* 28: 265-271.
80. Sensintaffar, E.L., D.H. Sliney, and W.H. Parr. 1978. An Analysis of a Reported Occupational Exposure to Infrared Radiation. *Am. Ind. Hyg. Assoc. J.* 39(1):63-69.
81. Rothkoff, L., A. Kushelevsky, and M. Blumenthal. 1978. Solar Retinopathy: Visual Prognosis in 20 Cases. *Israel J. Med. Sci.* 14(2): 238-243.
82. Marshall, W.J., D.H. Sliney, M. Hoikkala, and C.E. Moss. 1980. Optical Radiation Levels Produced by Arc Carbon Arc Cutting Processes. *Welding Journal.* 59(3): 44-46.
83. Moss, C.E., W.E. Murray, W.H. Parr, J. Messite, and G.J. Karches. 1977. A Report on Electromagnetic Radiation Surveys of Video Display Terminals. DHEW (NIOSH) Publication No. 78-129. December.
84. Moss, C.E., and W.E. Murray. 1979. Optical Radiation Levels Produced in Gas Welding, Brazing, and Cutting Processes. *Welding Journal.* 58(9): 37-46.
85. Moss, C.E., and W. Chrostek. October 1980. NIOSH Health Hazard Evaluation HE 79-149-758.
86. Moss, C.E. and W. E. Murray. 1981. Optical Radiation Levels Produced By Single-Phase Direct Arc Furnaces. *AIHA Journal.* 35(4) 212-215.
87. Marshall, W.J. et al., 1977. Non-Ionizing Radiation Protection Special Study No. 42-0312-77, U. S. Army Environmental Hygiene Agency, Aberdeen Proving Grounds, Maryland (NTIS No. ADA 043023).
88. Is Infrared Light Harmful to the Eye? 1979. *Siemens Review XLVI* (3), 21-22.
89. Infrared Health Hazard? 1979. *Electro-Optical Systems Design* 11(9), 24-25
90. Sliney, D.H. 1972. Non-ionizing Radiation. In: Cralky, L.V., Ed. *Industrial Environmental Health.* New York, Academic Press, Vol. 1 pp. 171-241.
91. Hardy, J.D. 1939. The Radiation Power of the Human Skin in the Infrared. *Am. Jour. Physiol.* 172: 454.

92. Hardy, J.D. 1962. Physiological Effects of High Intensity Infrared Heating. ASHRAE Journal, November.
93. Pauli and Ivancevic. 1927. Cited in Hardy, J.D., H.T. Hammel, and D. Murgatroyd. 1956. Spectral Transmittance and Reflectance of Excised Human Skin. Jour. Appl. Physiol. 9: 257.
94. Cartwright. 1930. Cited in Hardy, J.D., H.T. Hammel, and D. Murgatroyd. 1956. Spectral Transmittance and Reflectance of Human Skin. Jour. Appl. Physiol. 9: 257.
95. Aldrich. 1932. Cited in Hardy, J.D., H.T. Hammel, and D. Murgatroyd. 1956. Spectral Transmittance and Reflectance of Excised Human Skin. Jour. Appl. Physiol. 9: 257.
96. Hardy and Muschenheim. 1934. Cited in Hardy, J.D., H.T. Hammel, and D. Murgatroyd. 1956. Spectral Transmittance and Reflectance of Excised Human Skin. Jour. Appl. Physiol. 9: 257.
97. Jacquez, J.A. and H.F. Kuppenheim. 1955. Spectral Reflectance of Human Skin in the Region 235-1000 nm. Jour. Appl. Physiol. 7: 523.
98. Jacquez, J.A., J. Huss, W. McKeenan, J.M. Dimitroff, and H.F. Kuppenheim. 1955. Spectral Reflectance of Human Skin in the Region 0.7-2.6 μ m. Jour. Appl. Physiol. 8:297-299.
99. Hardy, J.D., H.T. Hammel, and D. Murgatroyd. 1956. Spectral Transmittance and Reflectance of Excised Human Skin. Jour. Appl. Physiol. 9:257-264.
100. Forsythe, W.E., and F. Christison. 1930. The Absorption of Radiation from Different Sources by Water and Body Tissue. J. Opt. Soc. Amer. 20: 693.
101. Coblentz, W.W. 1946. Penetration of Radiation into the Human Skin. J. Am. Med. Assoc. 123: 378.
102. Webner. 1846. Cited in Hendler, E. 1968. Cutaneous Receptor Response to Microwave Radiation. In: Thermal Problems in Aerospace Medicine. J.D. Hardy ed. p. 159. Surrey, England.
103. Hendler, E., and J.D. Hardy. 1960. Infrared and Microwave Effects on Skin Heating and Temperature Sensation. IRE Trans. Med. Electron. ME-7: 143.
104. Cook, H.F. 1952. The Pain Threshold for Microwave and Infrared Radiations. J. Physiol. 118: 1.
105. Henriques, F.C. Jr. 1947. Studies of Thermal Injury: V. Predictability and Significance of Thermally Induced Rate Processes Leading to Invisible Thermal Injury. Arch. Pathol. 43: 489-502.

106. Hardy, J.D., H. Goodell, and H.G. Wolff. 1951. Influence of Skin Temperature upon Pain Threshold as Evoked by Thermal Radiation. *Science*. 114: 149.
107. Hardy, J.D. 1958. Thermal Radiation, Pain and Injury. In: *Therapeutic Heat*. S. Kicht (editor) Vol. 2, p. 157, New Haven, Connecticut.
108. Rothman, S. 1954. *Physiology and Biochemistry of the Skin*. The University of Chicago Press, Chicago.
109. Zelentsova, S.P. 1970. The Effect of Intermittent Infrared Radiation on the Status of Natural Immunological Reactivity of Workers. *Gig. Tr. Proff. Zabol.* 14: 22.
110. Zelentsova, S.P. 1968. Immunological Reactivity of the Organism Under the Effects of Interrupted Infrared Radiation. *Vrach. Delo.* 12: 88-91.
111. Lehmann, J.F., G.D. Brunner, and R.W. Stow. 1958. Pain Threshold Measurements after Therapeutic Application of Ultrasound, Microwaves, and Infrared. *Arch. Phys. Med. and Rehab.* 39: 560.
112. Borneff, J., and H. Blumlein. 1960. Damages of the Respiratory Passages in Occupational Exposure to Heat. *Medizinische Klinik.* 13:494-497.
113. Infrared Radiant Warmers. 1973. *Health Devices*. p.4, November.
114. Knish, Y.P. 1973. The Effect of Small Doses of IR on the Sympathetic Adrenergic System of Children. *Akush-Ginekol.* 6: 23-25.
115. Arima, E., and E. W. Fonkalsrud. 1975. The Relationship of Intestinal Adhesions to Infrared Heating Lamp Exposure. *J. Ped. Surg.* 10: 231.
116. Gordon, S. A., and K. Surrey. 1960. Red and Far-red Action on Oxidative Phosphorylation. *Rad. Res.* 12:325-339.
117. Gordon, S.A., A.N. Stroud, and C.H. Chen. 1971. The Induction of Chromosomal Aberrations in Pig Kidney Cells by Far-red Light. *Rad. Res.* 45:274-287.
118. Krell. K., E.D. Jacobson, T.J. Withrow, K. Selby, and R.O. Martin. 1977. Genetic Effects of Red, Far-red, and Infra-red Radiant Energy. *Symposium on Biological Effects and Measurement of Light Sources*. DHEW (FDA) Publication No. 77-8002: 247-254.
119. Cunningham-Dunlop, S. and B.H. Kleinstein. 1977. A Current Literature Report on the Carcinogenic Properties of Ionizing and Non-ionizing Radiation. Part I. Optical Radiation. DHEW (NIOSH) Publication No. 78-122, December.

120. Bang, F. 1925. Bull du. Cancer. 14:203
121. Geschickter, C.F., and H. P. Koehler. 1935 Am. J. Cancer 23:804
122. Henry, S.K., 1946. Cancer of the Scrotum in Relation to Occupational Health, Oxford University Press.
123. Peterkin, G.A., 1955, Malignant Change in Erythema Ab Igne. Brit. Med. J. 2:1599.
124. Lawrence, E.A., 1952, Carcinoma Arising in Scars of Thermal Burns with Special Reference to Influence of Age at Burn on Length of Induction Period. Surgery, Gynecology, and Obstetrics. 95:579.
125. Owens, D.W., 1977, The Influence of Heat, Wind, and Humidity of UV Injury (abstract), International Conference on Ultraviolet Carcinogenesis, Warrenton, Virginia 21-23 March.
126. Hahn, G.M. and Strand, D.P., 1976, Cytotoxic Effects of Hyperthermia and Adriamycin on Chinese Hamster Cells. J. National Cancer Inst. 57:1063.
127. Mulay, D.M., 1963, Skin Cancer in India. First International Conference on the Biology of Cutaneous Cancer, NCI Monograph No. 10, 215.
128. American National Standards Institute (ANSI). 1976. Safe Use of Lasers. ANSI Z-136.1.
129. American Conference of Governmental Industrial Hygienists (ACGIH). 1980. Threshold Limit Value (TLV) for Chemical Substances and Physical Agents in the Workroom Environment, Cincinnati, Ohio.
130. Moss, C.E., and M.C. Gawenda. 1978. Optical Radiation Transmission Levels Through Transparent Welding Curtains. DHEW (NIOSH) Publication No. 78-176, July.
131. Sliney, D.H., Moss, C.E., Miller, C.G., and Stephens, J.B. 1981. Semitransparent Curtains for Control of Optical Radiation Hazards. Applied Optics. 20:2352-2366.
132. Coblentz, W.W., and R. Stair. 1930. Correlation of the Shade Numbers and Densities of Eye-Protective Glasses. J. Opt. Soc. Amer. 20: 624.
133. U.S. Department of Labor. 1976. Code of Federal Regulations, Title 29, Chapter XVII, Part 1910.252, Welding, Cutting, and Brazing.
134. American National Standards Institute (ANSI). 1968. American National Standard Practice for Occupational and Educational Eye and Face Protection, ANSI Z87.1 and subsequent revisions.
135. Campbell, D.L. 1976. Report on Tests of Welding Filter Plates. DHEW (NIOSH) Publication No. 76-198, July.

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