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The College of Optometrists has awarded this article 2 CET credits. There are 12 MCQs with a pass mark of 66%.

Radiation effects on the eye
Part 1 - Infrared radiation effects on ocular tissue

August brings the rare occurrence of a total eclipse of the sun, which can be viewed in Cornwall. This article, the first in a series of three, discusses how the heating effect of the sun through infrared rays can be hazardous to ocular tissue, and describes the mechanisms of cataractogenesis resulting from occupational exposure.

Infrared radiation (IR) lies beyond the red end of the visible spectrum, with wavelengths between 780-10000nm (**Figure 1**).
It is divided into three sub-ranges:

- IR-A, or near infrared (from 780 to 1400nm);
- IR-B or far infrared (1400 to 3000nm);
- IR-C (3000 to 10000nm). This band does not normally reach the earth's surface because it is absorbed by the atmosphere, but non-natural sources of IR-C can be a significant hazard.

While most of us enjoy the heating effect of infrared from the sun, industrial sources of infrared are found in workplaces where there are high temperature furnaces, such as in the glass and steel industries. Arc lamps and electric radiant heaters also give off infrared, and various lasers are rich sources, for example the neodymium YAG laser (IR-A) and carbon dioxide laser (IR-C).

When radiation is absorbed by human tissue, it causes significant changes to the cellular material. Some infrared is absorbed by each ocular structure. In general, the cornea absorbs almost all wavelengths greater than 3000nm (IR-C) and most radiation with a wavelength above 1400nm. The crystalline lens absorbs some radiation between 900nm and 1400nm (IR-A) and the retina absorbs most of the remaining infrared with a wavelength less than 1400nm (IR-A) - increasingly as the wavelength reduces (**Figure 2**). The greatest concern is with the heating effects to the lens and the retina.

Cataracts associated with occupational infrared exposure have been known about since 1739 and, historically, it is on this problem that the greatest attention has been focused. The occupations of glass blowing, metal working, chain making and tin plating and forging are those in which infrared levels can be significant. Retinal burns from exposure to industrial sources, such as xenon lamps, infrared lasers and metal arc inert gas welding, have also been a concern for some years. Deliberate gazing at the sun has been shown to produce a solar retinopathy, first described by Agarwal and Malik (1959)[1]. Eclipse blindness, as described by Penner and McNair (1966)[2], is a similar condition, and will be discussed more fully in the next article in this series.

While a number of valuable recent studies on infrared have attempted to establish threshold energy levels and exposure times for damage to the eye, these have mainly been in relation to animal experiments, including those on the monkey eye. The occupational levels typically encountered have only been documented in the past decade and their effects and consequences are still far from being fully understood.

The energy of an infrared photon is, in fact, low, much lower than that of a visible or ultraviolet photon. The tissues most vulnerable in the eye are the cornea and aqueous humour, as the infrared radiation raises the overall temperature of the anterior eye. The lens absorbs only a small proportion of infrared and, for much damage to be sustained here, the overall exposure level would need to be high, or the result of smaller repeated doses. Long wavelength infrared rays also reach the

Figure 1: Electromagnetic spectrum, showing relationship of infrared and ultraviolet band to visible light

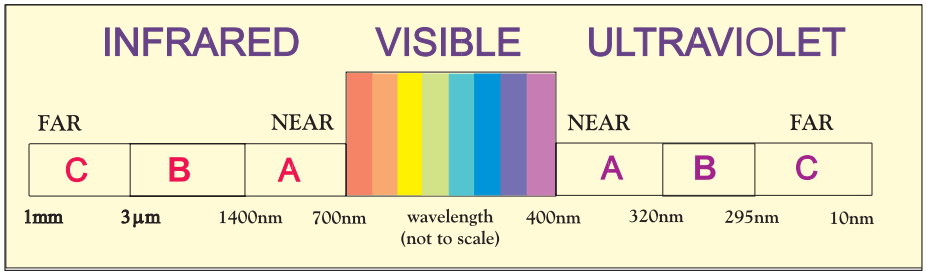
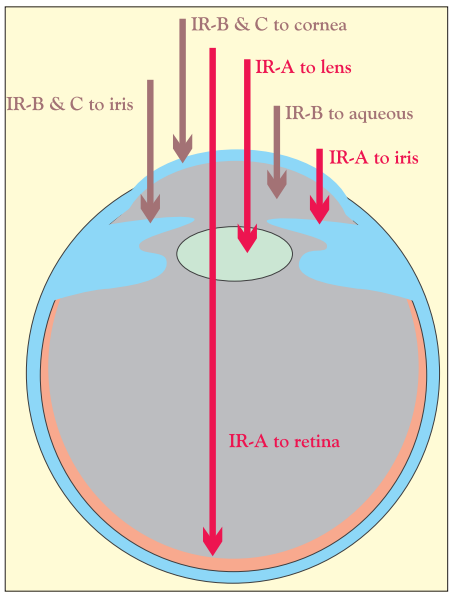


Figure 2: Infrared radiation absorbed by the eye



retina and can cause permanent damage to the delicate photoreceptors - for instance, through sun gazing - though the visible part of the spectrum produces the most damage, which is of a photochemical rather than a thermal nature.

OCULAR MEDIA

TRANSMISSION AND ABSORPTION

The cornea transmits the most infrared radiation between 700nm and 1300nm. At 1430nm and 1950nm, the cornea has broad absorption bands for infrared, but between these bands much is transmitted. Beyond 2500nm, absorption occurs. The aqueous humour transmits infrared except in a few selected regions where it absorbs. The lens absorbs a small, but significant amount of near infrared up to 1400nm; an even smaller proportion of IR-A is absorbed by the aqueous and vitreous humours, the remainder passing through to the retina. **Figure 3** summarises the proportions absorbed by each structure as a function of wavelength.

EYELIDS

The effects of infrared radiation on the eyelids range from mild reddening to third degree burns and, eventually, death of the skin. To suffer the more severe effects, the eyelid must be exposed to very high levels of infrared delivered over a short period of time, or to low levels of infrared over a long period. Infrared eyelid damage is not usually found in the industrial context.

CORNEA

The cornea transmits 96% of incident infrared in the range 700nm-1400nm and, as a result, threshold values for damage to occur are quite high, especially in the 750-990nm waveband. The low energy per quantum that these wavelengths deliver also means that the cornea is relatively well protected from them. The radiation effects on the cornea from these wavelengths involve protein coagulation of the front and middle layers, the epithelium and stroma. It is thought that the endothelium layer, the back layer, may in fact sustain most damage because the heating effects have little chance of dissipation here, unlike at the front of the cornea where air and tear fluid have a cooling effect. This is significant since the endothelial layer cannot regenerate once damaged, as can the epithelium tissue. High dose infrared damage to the cornea produces immediate pain and vascularisation. Eventually, loss of transparency and opacification occur, as the cornea responds with a burn that causes ulcers similar to that seen in skin when exposed to infrared.

Pitts et al (1980)[3] examined the efficiency of the cornea in protecting the lens from developing cataract and found that the cornea does not protect the lens for irradiances below about 3.8 Wcm^{-2}, but does give some protection above this value. The cornea protects the lens for exposures above 4 Wcm^{-2}.

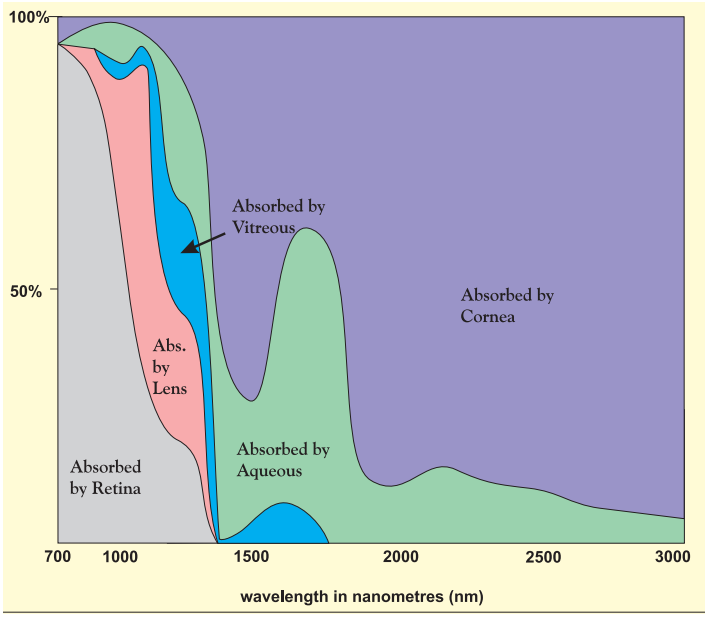


Figure 3: Absorption of infrared radiation by various ocular structures, as a function of wavelength

AQUEOUS HUMOUR

The aqueous and vitreous humours are similar to water in many respects and are not greatly affected by infrared radiation. Any change to these structures is a result of damage to the cornea, lens or retina.

IRIS

The human iris absorbs between 53% and 98% of incident infrared in the 750-900nm range, but varying degrees of pigmentation affect the amount of absorption and, thus, the extent of damage. Overall, the iris is sensitive to infrared, and suffers swelling, cell death, hyperaemia and pupillary miosis. Wavelengths around 900nm cause aqueous flare on account of the leakage of protein from iris vessels into the anterior chamber owing to inflammation of the iris when irradiated by infrared.

Pitts et al (1980)[3] found that the threshold for injury to the iris is about the same as the cornea and the responses of both cornea and iris seen by ophthalmological examination are similar. The leakage of proteins into the aqueous humour causes the aqueous flare.

LENS

Cataract has long been known to be associated with certain types of occupations involving prolonged exposure to infrared, although the exact mechanisms involved have only been fully understood in the last two decades. Meyerhofer (1886)[4], a German, first studied glass workers and identified a typical posterior cortical opacity of the lens which has become accepted as the early stages of infrared-induced cataract.

The crystalline lens transmits most wavelengths up to 1400nm but at selected wavelengths, as for the aqueous humour, absorption occurs. By the time any infrared reaches the vitreous humour, wavelengths between 980nm and 1200nm are absorbed, but there is very little transmittance of IR beyond 1400nm.

The near infrared that passes through the pupil is strongly absorbed by absorption bands above 900nm, while none of the infrared above 1400nm reaches the retina.

Table 1: Summary of typical damage to ocular tissue from IR exposure

OCULAR STRUCTURE	TYPICAL DAMAGE
Cornea	Loss of transparency, opacification, haze, exfoliation, debris
Aqueous humour	Flare
Iris	Swelling, cell death, miosis, hyperaemia/inflammation
Lens	Anterior opacities, sutures more visible
Vitreous Humour	Haze
Retina	Oedema, burns, depigmentation

RETINA AND CHOROID

Any infrared rays that are transmitted through the ocular media to the retina are absorbed by the pigment epithelium of the retina. Injury occurs in the neural layers through indirect heating. The effect of the infrared on the retina and choroid is to cause a rise in temperature, which causes enzymes to denature; in general, temperatures more than 10° above ambient body temperature will produce permanent thermal damage. Many physical factors affect whether damage is done, including pupil size, the optical quality of the retinal IR image, exposure duration, size of the source and of the retinal image, location on the retina, the type and spectral distribution of the source and rate of delivery of the energy. Obviously, exposure duration is a major factor. As the exposure time increases, the radiant power entering the eye necessary to produce retinal structural damage decreases until, at a certain level, duration becomes irrelevant and the damage appears to be determined by the irradiance reaching the retina alone.

Heating effects to the retinal pigment epithelium can occur from infrared exposure durations as low as microseconds. Even shorter durations of laser infrared exposure (picoseconds) cause a different type of damage, that of acoustic or shockwave effects to the melanin pigment.

Many studies have attempted to establish threshold exposure values for the human retina. In addition to the physical factors, such as exposure duration and irradiance levels, eyes vary in their degree of retinal pigmentation, which undoubtedly has an effect on the degree and extent of damage by infrared radiation.

Studies have shown that the longer wavelength infrared radiation requires higher irradiance at the cornea to produce a retinal burn. This is pertinent since at lower wavelengths, much more infrared passes through to the retina.

Work on rhesus monkeys in 1979 by Ham et al[5] found that to produce retinal lesions of 159μm in size (which is the size of the sun's image produced on the retina by sun gazing), radiant exposures on the retina from two spectral wavebands - 400-800nm and 700-1400nm - were required with exposure durations from 1-1000 seconds. The inverse relationship between power and duration was maintained for exposure durations of 10 seconds or longer, with the radiant exposure (product of power and duration) being 400 Jcm^{-2} for the 400-800nm waveband. For the 700-1400nm waveband, a radiant exposure of 70000 Jcm^{-2} was required, with a duration of 1000 seconds only, since they were unable to produce a retinal burn with exposures shorter than 1000 seconds.

Table 1 summarises the typical damage to ocular tissue from infrared exposure. **Tables 2 and 3** detail infrared sources and their effects on the eye.

INFRARED CATARACT

EARLY THEORIES

Cataracts induced by infrared radiation began to attract attention at the beginning of this century when industrial health issues were first considered seriously. In London, Legge (1907)[6] reported to the Home Office on a new legal occupational disease - that of 'glass blower's cataract'. The exact mechanism of cataract formation from infrared has been the subject of considerable dispute for almost 70 years. However, recent evidence has provided a more precise understanding of occupational cataractogenesis.

The original explanation of occupational cataracts came from Vogt (1912)[7] who believed that IR-induced opacities were the result of the direct absorption of the radiant energy by the crystalline lens. However, in his experiments using carbon arc light, the animals produced conjunctivitis in addition to the lenticular opacities. It is now suspected that ultraviolet absorption was responsible for the cataract rather than infrared.

Another early theory suggested that heat exchanges within the anterior eye caused the cataract. Verhoeff and Bell (1916)[8] argued that cataract formed on the posterior surface of the lens because the anterior surface was cooled by circulation of the aqueous humour and so the cornea was air-cooled. Their idea was that the heat from the infrared radiation interfered with the ciliary body functioning which, in turn, affected the metabolism of the crystalline lens. However, anterior lens opacities and corneal damage from infrared are commonly seen, while damage to the ciliary body would involve a change in the aqueous humour, which is not seen.

In the early 1930s in Germany, careful investigations showed that the cataracts were due not to direct absorption of the infrared by the lens, but to the raised temperature induced indirectly through heat absorbed by the iris, where a rich blood supply would be consistent with a high degree of heating (Goldmann[9,10,11]). Indirect heating was thought to cause a secondary denaturation of the lens proteins that led to cataract formation.

In an attempt to evaluate the underlying mechanisms at work, Langley et al (1960)[12] repeated the early experiments of Goldmann and suggested that both visible light and infrared must be absorbed by the iris for cataracts to be produced. They argued that it was not the total heat applied to the eye, but the increase in temperature in a local area that produced cataracts.

More recent occupational observations[13,14] led to the view that both direct absorption by the lens and indirect heating of the lens fibres through the absorption of the iris were responsible for the IR-induced opacities.

In this way, they reconciled the conflicting theories of Vogt[7] and Goldmann[9,10,11]. The experimental work of Pitts et al (1980)[3] on monkey eyes has now provided a clearer understanding of the exact mechanisms at work, and it appears that both indirect and direct heating effects contribute to cataract development.

INDUSTRIAL CATARACT

The experimental work carried out in the early part of the century began to be applied in an industrial context around the time of the Second World War, when occupational injuries were becoming more common. Early in this period, ophthalmologists noted a delayed development of cataract in those exposed to infrared radiation in industry. For example, Kutscher (1946)[15] in the USA reviewed the literature on glass blower's cataract and noted the delayed effect of infrared radiation. He concluded that tin plate and steel workers appeared to develop cataracts after about 15 to 20 years of exposure. At about the same time, Salit (1940)[16] noticed the rise in cataracts one year after a very hot, dry summer in Iowa, highlighting likely environmental causes of cataracts. After the Second World War, interest was renewed in the occupational hazards of the strong and efficient light sources which were becoming available to industry.

From the mid-1950s, surveys of steel workers were made by various authors, emphasising again the typical posterior lens opacity which had first been observed in the 1880s. It was suspected that some individuals may be more susceptible to radiation cataracts that others. In the UK, a major study by Wallace et al (1971)[17] involving 1,000 steel workers showed only a slight link between infrared exposure and lens opacities, but a higher incidence of cataract possibly also due to ageing. An exposure index was calculated by multiplying the number of years on the job by the exposure risk.

The typical glass blower's cataract after many exposures is a posterior outer cortical opacity in the shape of stars, which can start with a cobweb-like appearance. Fine grey dots that may adhere to the anterior lens capsule have also been typical descriptions of early IR-induced cataract. There follows a clouding of the equator area and movement of the anterior grey dots into the anterior lens cortex, with a gradual appearance of a saucer-shaped posterior opacity developing. Eventually, this becomes U-shaped - the whole process typically following a delay that can be up to 90 days.

LATENT PERIODS

The latent period before the cataract develops has been used to determine whether or not infrared radiation is directly responsible, since cataracts are found in most people over 70 years of age. Investigators in the past, including Goldmann[9-11], described the anterior subcapsular changes as an immediate response to the radiation. The work of Langley et al[12] also mentioned that grey anterior subcapsular dots occurred within 24 hours of the radiant exposure. In the more recent study by Pitts et al[3], the latency for the damage to be seen was between one and a half and 24 hours, but almost always about six hours following irradiation.

The indirect heating of the lens by the iris causes a pattern of lens opacities at the front of the cortex of the lens that Pitts et al[3] found to fade and disappear within six weeks of the acute exposure. It was as if the acute exposure had only a temporary effect. Earlier studies had also shown this reversible effect of early lens changes if only a single acute dose of radiation was involved. If the opacity is induced by infrared through heating, it should appear immediately after the exposure exceeded threshold.

Goldmann[9] described the anterior subcapsular changes as immediate; Vogt[7] had also described them as occurring within the first 24 hours. Langley et al[12] said that the grey dots in the anterior subcapsular region occurred within 24 hours, but in the study of Pitts et al[3] the development was never as long as 24 hours, but closer to six hours.

Since acute exposure to IR-A appears to result in a temporary opacity only, it is possible that cataract production in eyes exposed to infrared may not take place through a thermal mechanism. The

Table 2: Infrared sources and their effects on the eye

WAVELENGTH	LASER SOURCES	OTHER SOURCES	SYMPTOMS	TISSUE AFFECTED
Near IR (IR-A) 780-1400nm	Alexandrite 710-800nm Gallium Arsenide 850-950nm	Sun, furnaces, lamps, glass blowing	Cherry red flash or glow, dull after image Scotoma may be accompanied by pain	Retina (pigment epithelium) Iris (lens, cataract) Blanched retinal lesion or oedema proceeding to vitreous haemorrhage
	Neodymium-Yag 1064nm Helium-Neon 1150nm Iodine 1315nm		Rapid onset of scotoma without light flash (Shock if Q-switched ND-Yag laser) - pain	Deep retinal coagulation, may involve choroid, Possible lens or vitreous involvement Lesions elongated if caused by CW laser
Far IR (IR-B) 1400-3000nm	Erbium 1540nm Holmium 2060nm (pulsed lasers)	Sunlight, furnaces, lamps	Pain Blepharospasm Visual loss	Cornea (opacity) Lens (cataract)
Far IR (IR-C) 3µm-100µm	Deuterium (pulsed) Fluoride 3.8-4µm Carbon dioxide 10.6µm	Furnaces, lamps	White flash, intense pain Blepharospasm Corneal opacity Perforation	Cornea Conjunctiva Skin

other likely cause is inflammation of the iris in response to infrared exposure: Langley et al[12] were the first to notice an acute iris inflammation (uveitis) secondary to IR exposure. In their experimental study on animal eyes (including monkeys), Pitts et al[3] were able to produce acute cataract with and without anterior uveitis.

The subcapsular opacity typically seen as a result of infrared radiation exposure is also exactly the same histologically as that seen as a result of other types of radiation-induced cataract, such as those from X-ray, atomic bomb (from β and γ rays) and cyclotron exposure (neutrons). Cataracts that are experimentally induced by infrared radiation are also similar to those produced by ultraviolet radiation. This inevitably creates problems in identifying the causes of cataracts, since many sources encountered in industry give out infrared and ultraviolet together.

ANTERIOR AND POSTERIOR OPACITIES

The modern theory is that radiation-induced cataracts resulting from exposure to both ionising and non-ionising radiation is initially an anterior opacity of the lens. It would appear from the experimental work of Pitts et al[3] that acute IR-induced opacities occur in the anterior subcapsular or anterior epithelium of the lens. White dots are seen first and, if the exposure has been considerable, these dots then form into a diffuse whitish opacity. Over a period of up to 45 days, the opacities fade and eventually disappear. Earlier studies had also found these reversible early lens changes if only a single acute dose was involved.

Pitts et al[3] found no evidence of infrared exposure (or full spectrum exposure) causing posterior subcapsular opacites immediately after exposure. Thus, they concluded that the IR-induced cataract is an anterior subcapsular opacity while the posterior subcapsular opacity, is largely a delayed process due to normal ageing, which may or may not be accelerated by exposure to infrared. While posterior opacities have a latency of between 60 and 90 days, it is clear that full-blown cataract in the posterior lens seems to be a combination of IR-induced opacities superimposed on the normal age-related cataract seen in most individuals over 60 years of age. The precise effect of the infrared exposure on the development of the age-related cataract is not fully understood.

There are two phases to the lens changes, involving a coagulation process. First, a chemical change (denaturation) occurs, then coagulation. It is possible that the two processes are separated by a period of time. Infrared could be absorbed by the anterior epithelium of the lens and denaturation commences; the white dots of greyish anterior haze that are seen would indicate the denaturation process. Coagulation is completed when the posterior lens material becomes opaque.

ANIMAL THRESHOLD STUDIES

The only clear way to evaluate the potential dangers to human eyes is to subject animals to highly controlled exposures under experimental conditions - a study which the US Air Force has sponsored. This work by Pitts et al[3] has also enabled us to understand more fully the mechanisms at work. They irradiated the unrestrained eyes of monkeys with infrared radiation to determine the threshold level for damage to various ocular tissues.

INDUSTRIAL IMPLICATIONS

It is clear that threshold infrared experiments on monkey eyes need to be related to the typical exposure levels encountered in the occupational context. In an early industrial study, Barthelmess and Borneff (1959)[18] measured the total daily radiation received by glass blowers working near the melting furnaces and found the total radiant exposure to be between 2000 and 3000 Jcm^{-2}, though only approximately 10% of that total was infrared below 1400nm. For comparison, the infrared corneal dose rate from

Table 3: Infrared sources and their detailed effects on the eye

WAVELENGTH	SOURCES	SYMPTOMS	MECHANISMS
400-700nm	Sun, broad band arc lamps, flash and incandescent lamps	Intense light followed by after-image and possibly by scotoma, usually at the macula	Photochemical, thermal
488, 514.5nm	Argon ion laser, continuous wave or mode lock	Intense blue or green flash without pain or shock After-image may be a complementary colour followed by a scotoma - long term insiduous loss of cone function	Photochemical, thermal
530, 586, 648nm	Krypton ion laser, continuous wave or mode lock	Green, yellow or red flash (as above)	Photochemical, thermal
632.8nm	Helium neon laser Continous wave or mode lock	Intense red flash (as above)	Photochemical, thermal
694.3nm	Ruby laser, long pulse or Q-switched	Brilliant red flash perhaps accompanied by a shock waves After-image and scotoma - if vitreous haemorrhage occurs, red apperance, which may proceed to complete loss of vision	Thermo-acoustic
Multiple wavelengths 400-780nm	Dye laser	Colour dependent on wavelength tuned, otherwise as for other ion lasers	Photochemical, thermal

daylight is about 10^{-3} Wcm^{-2}, so that 12 hours of continuous exposure will typically deliver a radiant exposure of about 50 Jcm^{-2}. Sliney and Freasier (1973)[19] noted that glass and steel workers exposed to infrared irradiances as low as 0.04-0.08 Wcm^{-2} daily for 10-15 years develop lenticular cataracts.

The most recent and very extensive onsite work in this area has come from a Swedish study by Lydahl (1984)[20]. He confirmed that a high occupational exposure to infrared radiation considerably increases the risk of cataract, although not usually until after 60 years of age, thus highlighting the link between industrial exposure and ageing. The study attempted to clarify the role of chronic or long-term exposure to infrared in producing cataracts. All the major types of senile cataract were seen in increased frequency in infrared-exposed workers in this investigation. The left eye of glass workers was found to be more affected by cataract than the right, which was considered to be because of the higher infrared doses received by the left eye, because of the position of working.

This major study was the first to detail the types of opacities found in workers in the steel and glass industries, together with radiometric measurements of infrared on site received by workers in these two industries, and link the findings. From the measurements, a dose per unit time was calculated for each job and, by knowing the number of years workers had been exposed, it was possible to calculate the lifetime exposure level. Furthermore, this was the first time that statistical analysis has been applied in comparison with matched controls.

Employees working with heat resistant glass were found to receive almost twice the radiation levels of any other worker in the glass or steel industries. On average, glass workers are exposed to higher doses of infrared radiation than iron and steel workers. A whole variety of tasks at work present the employee in these two main industries with potential damage to eyes from infrared; blacksmiths, chain makers, tin plate rolling mill workers and glass blowers were the high-risk occupations as far as infrared radiation was concerned.

CONCLUSION

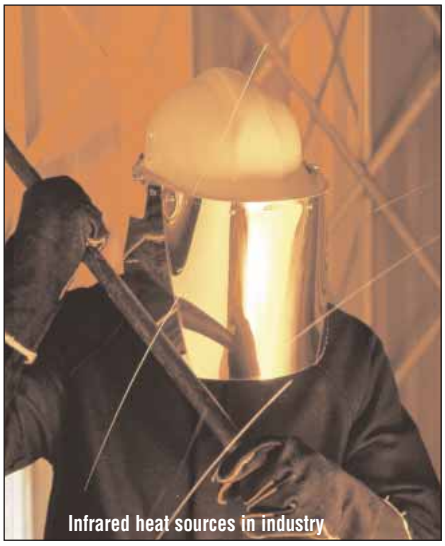
While the original view was that infrared radiation directly interacted with the lens alone to cause cataract, recent evidence supports the view of Goldmann[9] which involves dual mechanisms of indirect heat from the iris acting on the lens through heat transfer from its vascular neighbouring structures. Pitts et al[3] agreed with Goldmann because their experimental work on monkeys could not produce lens opacities by direct infrared exposure to the lens, but only when the iris was irradiated. The zone of the lens affected lay directly beneath the exposed iris region. The development of cataract is, thus, complex. Heat loss is not efficient at the posterior side of the lens, and so it is here that the final opacity occurs in the form of a wedge-shaped area with star-like features. At a later stage, when the cataract develops further, some time after the initial infrared exposure, the opacity is almost indistinguishable from the classic senile cataract.

The critical question this article has addressed concerns whether the star-like posterior cortical opacities of the lens are the result of long-term infrared radiation exposure, or are senile cataracts maturing earlier than usual in people who work with infrared on a long-term basis.

The evidence suggests that accelerated ageing of the lens is the cause of the cataract. This view is supported by our knowledge of ageing, which involves oxidative processes affected by heat and temperature changes. Heating tissue above the normal temperature increases the metabolic rate, which could then lead to premature ageing presumably as a result of accumulation of chemicals. One theory is that accumulation of water-soluble substances may be involved in the loss of transparency of the crystalline lens.

We know too that the development of senile cataract has a strong genetic link or hereditary predisposition. It is possible that individuals who develop cataract as a result of repeated or excessive infrared exposure in fact experience opacification that they were destined to develop in their older years anyway.

The most recent work demonstrates that there is a clear occupational risk of developing cataract earlier as a result of exposure to infrared radiation, but no study has been able to establish a correlation between the density of opacity and the total infrared dose.


Infrared heat sources in industry

PROTECTION

Finally, it is worthwhile considering methods for protecting eyes against infrared radiation. Spectacles containing reflective metallic coatings and materials that filter out infrared are the ideal means. Aluminium and inconel (and alloy of iron, nickel and chromium) provide excellent infrared reflectance. The main difficulty with using reflective metallic coatings is that they are susceptible to scratching, abrasions and other faults that cause the breakdown of the coating. Hard protective secondary coating over the metallic coating overcomes this problem, or sandwiching the reflecting metal between two optical layers; the layer adjacent to the eye can then be designed to absorb other unwanted radiation such as ultraviolet. A typical combination protective filter is 'Pfund's glass' developed by American Optical, in which a gold layer reflects 96% of infrared while transmitting 75% of visible light. The metal is sandwiched between clear optical crown glass and Crookes A glass, which absorbs 100% of ultraviolet.

The major advantage of reflective coatings is that while protecting the wearer from infrared, they remain cool and so more acceptable. Thus, metallic coatings provide a measure of protection against low level chronic infrared exposure and reduce the total heat load reaching the eye.

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MULTIPLE CHOICE QUESTIONS
RADIATION EFFECTS ON THE EYE - PART 1

Please note there is only ONE correct answer. An answer return form is included in this issue. It should be completed and returned to: RAD 1, Optometry Today, Victoria House, 178-180 Fleet Road, Fleet, Hampshire GU13 8DA, by June 16, 1999.

1. Infrared radiation is absorbed by:.
a. the cornea principally
b. the crystalline lens principally
c. the retina principally
d. all of the ocular structures

2. The crystalline lens absorbs some radiation between:
a. 700-900nm
b. 900-1400nm
c. 1000-1800nm
d. 1400-2000nm

3. The retina absorbs wavelengths:
a. only around 1400nm
b. greater than 1400nm
c. less than 1400nm
d. at all wavelengths except around 1400nm

4. The lens opacity caused by infrared is described as:
a. posterior nuclear
b. inferior cortical
c. anterior cortical
d. posterior cortical

5. Any infrared that reaches the retina is absorbed by:
a. the pigment epithelium
b. the chorocapillaris
c. the ganglion cell layer
d. the rods and cones

6. Infrared damage to the iris causes:
a. the sphincter muscle of the iris to atrophy
b. leakage of protein from the iris vessels, giving rise to the aqueous flare
c. accelerated leakage of aqueous humour into the canal of Schlemn
d. leakage of calcium across the endothelial layer of the cornea

7. Infrared damage to the retina occurs principally as a result of:
a. photochemical action
b. genetic damage
c. indirect heating
d. ionisation of the photopigment molecules

8. Physical factors affecting whether damage is done to the eye by infrared include:
a. density of the retinal photopigment, exposure duration, physical origin of the infrared, spectrum of the infrared
b. pupil size, exposure duration, spectrum of the infrared, rate of delivery of energy
c. total energy absorbed, age of the person, colour of the irides, water content of the lens
d. spectrum of the infrared, rate of delivery of energy, plane of polarisation of the infrared radiation

9. Subcapsular changes on account of infrared exposure occur:
a. over an extended period of up to several years
b. between two and four weeks of exposure
c. within three days of exposure
d. within 24 hours of exposure

10. Infrared cataract is:
a. almost indistinguishable from a classic senile cataract
b. similar to the type of senile cataract found in the very elderly
c. similar to the type of senile cataract caused by oxidising radicals
d. different from all types of senile cataract

11. The correlation between density of opacity and total infrared dose:
a. is measured to be strong and highly statistically significant
b. is measured to be low but statistically significant
c. has been measured but is found to be not statistically significant
d. has never been measured

12. The best lens filters for infrared are those containing:
a. reflective metal coatings
b. suspended particles of cobalt and aluminium
c. a high density of long molecule polymers
d. glass doped with lead and niobium

The next article in this short series will appear in our June 18 issue. It will look at the dangers of viewing the eclipse.