Sunglasses—an ocular hazard?

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SUMMARY   A suggestion that protective eye gear can pose a threat either to the crystalline lens or to the retina is examined from an epidemiological point of view. It is concluded that it may accentuate a pre-existing high-risk hazard but has little significance for low-risk hazards

The so-called 'blue' hazard[1] has raised the question whether the protection of, say, the retina from potentially harmful radiations in one part of the electromagnetic spectrum may increase hazards to the eye due to those coming from another.[2 3] To be specific, if sunglasses reduce the retinal illumination in the visible part of the spectrum and thereby eliminate glare, but are less absorbent in the ultraviolet, what constraints are there for their ultraviolet absorption coefficient in terms of a possible pupillary dilatation resulting from the operation of the light reflex? Anderson and Gebel[2] occupied themselves with this problem but did not directly relate it to ocular hazards. In particular, all these authors failed to take into account the role of the shape of the crystalline lens. The following is an analysis somewhat more elementary than Anderson and Gebel's, but, as the quantitative conclusions which they reached are perfectly consistent with those obtained below, the epidemiological approach followed here may not be without interest.

Results

The relation between the pupillary area A and the retinal illumination I is sigmoid (see Alexandridis[4] and many others). It follows that for one range of I, and only one such range, the rate of change of A with I is maximal (Fig. 1). This remains true even if the experimental data are plotted on logarithmic scales.

A sigmoid variation is typical of dose-response variations. When the dose and the response are both low, an increase in dosage fails to produce much increase in the response. At the other extreme, if the dose is so high as to maximise or to saturate the response, an increase in dosage will be unaccom-

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panied by one in the response. But between the extremes the increase in response caused by a given increase in dosage will evidently come to a peak before declining towards the saturated response values. If we are dealing with a toxin, this maximum increase is determined by the peak sensitivity of the tissue exposed to the toxin.

In our case the analogue of the toxin is radiation and that of the response of miosis. It may be noted in parentheses that, although miosis implies a diminution, the analogy is rendered more satisfactory if the extent of the iridal area is compared with the response. The maximal rate of change of pupillary area with irradiation striking the eye represents the 'worst case' situation: if the pupillary area A decreased at this rate over the whole gamut of illumination I, then wearing sunglasses would lead to a significant pupillary dilatation, with potential hazards due to an increased presence of ultraviolet radiation within the eye. Let this maximal 'worst case' rate of pupillary dilatation be designated by G, because it represents the maximum gradient in the relation between A and I. In other words, it is defined by

$$G = \frac{d\log A}{d\log I} \quad \dots \dots \dots \dots \dots \dots (1)$$

whence (Fig. 1) $A = K.I^{-G} \quad \dots \dots \dots \dots \dots \dots (2)$

where K is a scaling constant. Since the light reflex operates via the retina, the illumination I is to be weighted by the effective retinal spectral sensitivity S, so that

$$A = K.[I.S]^{-G} \dots \dots \dots \dots \dots \dots (3)$$

However, I is variable owing to the absorbance of the sunglasses, if worn; at a wavelength $\lambda(1)$ they transmit $T(1)\%$ of the incident light. Hence
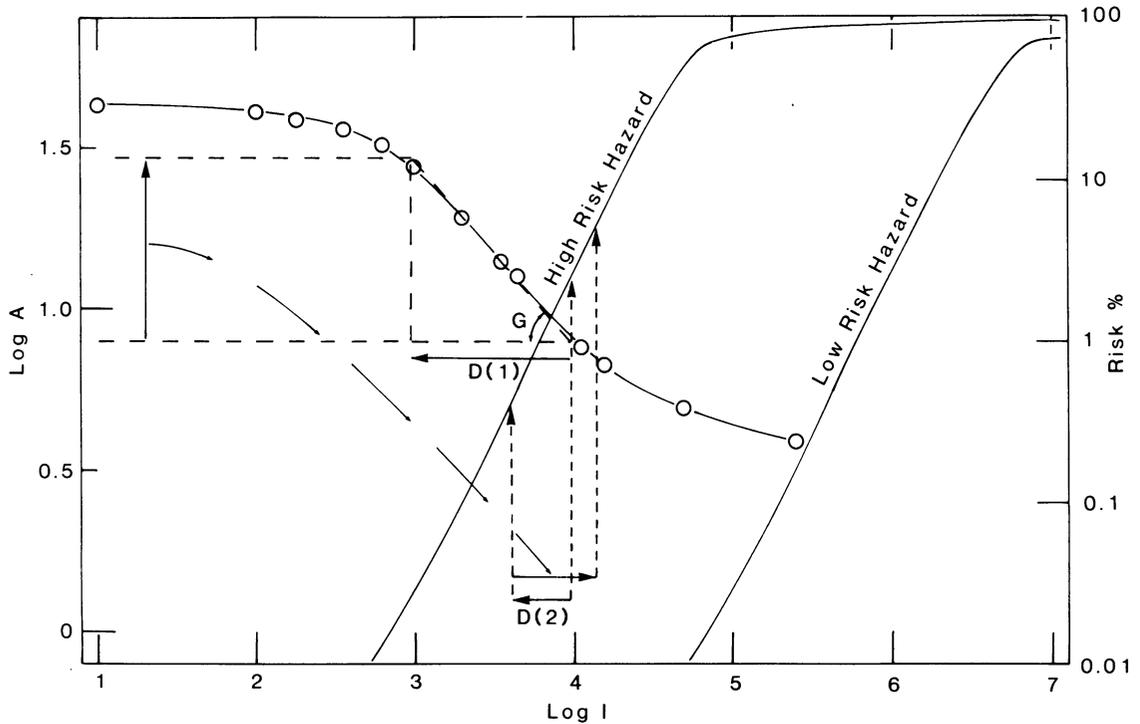
Fig. 1   *Abscissa: log retinal illumination (scot td). Left ordinate: log pupillary area. Right ordinate: risk in percent. Suppose the eye is illuminated with an intensity logI=3·98. At this intensity the risk is 2·5% for a serious hazard but negligible for a low-risk one. When sunglasses with a density D(1)=1 in the visible spectrum are worn, the pupillary area increases maximally by a factor of 3·6 (=antilog of 0·56), as indicated by the arrow on the left. Assume that the density of the glasses for a radiation in the ultraviolet part of the spectrum is D(2). To simplify the presentation, let this also correspond to logI=3·98; the argument does not hinge on this assumption (cf. equation (7)). Consequently the eye will receive from the ultraviolet 3·61 log units, D(2) being chosen as equal to 0·39. However, this reduction is counteracted in this case by the increase in retinal (or lenticular) illumination due to mydriasis, and the risk rises from the original value of 2·5% to 5·4%. If for some reason the pupil had dilated fully after the sunglasses had been put on, it is easy to show that the risk would have climbed to 10·5%. The situation can therefore become grave only if it is serious to begin with: a fourfold rise on the low-risk curve remains what it was originally, namely negligible.*

$$A=K.[I(1).T(1).S(1)]^{-G} \dots\dots\dots\dots\dots (4)$$

At another wavelength $\lambda(2)$, the retinal illumination is given by

$$R(2)=I(2).T(2).A \dots\dots\dots\dots (5)$$

Let us assume that this radiation represents a potential hazard, and we wish to know whether the worst-case assumption of the dependence of A on I(1), as given by equation (3) can lead to an ocular hazard. Substitution of equation (4) in equation (5) yields.

$$R(2)=K.I(2).T(2).[I(1).T(1).S(1)]^{-G} \dots\dots (6)$$

This relation is more instructive in its logarithmic form:

$$logR(2)=logI(2)-G.log[I(1).S(1)]+logK-D(2)+G.D(1) \quad (7)$$

where $-log_{10}T=D$, the photometric density of the sunglasses at any particular wavelength. The first three terms on the RHS of equation (7) are constant. Therefore the retinal illumination at the potentially hazardous wavelength $\lambda(2)$ remains constant if

$$D(2)/D(1)=G \dots\dots\dots\dots (8)$$

But D(1) is the density of sunglasses chosen for their perceptible (antiglare) effect; G can equal 0·575.[4] It follows that, on the worst assumption, namely that given by equation (3), the density in a risk, i.e., ultraviolet, region of the spectrum must not be smaller than 60% of that in the yellow-green part of the spectrum, if photic mydriasis due to the wearing of sunglasses is not to increase the risk from ultraviolet radiation. It is in the yellow-green part of the spectrum that the retinal sensitivity S(1) is

maximal. Hence glasses, transmitting 5% in the yellow-green part of the spectrum, should not transmit more than 17% for an increased ultraviolet risk to be avoided. If the 'yellow-green' transmission is 10%, then the upper limit is 25%. Since most materials transmit less at short than at long wavelengths unless special tints are introduced, sunglasses become a hazard only under perverse conditions (Fig. 1). Visually useless but allegedly cosmetic blue tints are therefore prime suspects.

Discussion

It may be noted that, from the point of view of a 'blue' or ultraviolet hazard, the approach followed by Anderson and Gebel[2] and above involves a simplification that tends to lead to a measurable under- or overestimate of the actual hazard, depending on whether we are considering the retina or the lens. The reason for this is the shape of the lens, coupled with the fact that it absorbs more light at its centre than near its equator.[5]

Consider first the retina. As the pupil increases, the flux traversing it rises, but less of this will be absorbed by the lenticular periphery than by an equal but central area. Hence the nominal risk to the retina is increased. Now consider the lens. Like all tissues it can be harmed only by radiations which it absorbs. The same increase in pupillary area will add relatively fewer potentially absorbed and harmful quanta than are present in an equal but central pupillary area. Consequently the overall risk to the lens is reduced. It can be shown that this effect can nullify the rise in hazard illustrated in Fig. 1. It would seem to follow, conversely, that the lens may protect the retina at its own expense. It is clinically significant that van der Hoeve[6] was among the first to emphasise that an eye with a cataract is unlikely to present with senile macular degeneration and vice versa.[7] Thus the above analysis should be used with circumspection. Even if, in equation (7), D(2) <D(1).G, so that there is a rise in the overall potentially noxious energy entering the eye, the risk, though increased mathematically, may still be negligible.

Suppose that the lens is relatively easily damaged by ultraviolet radiation, but the retina only with difficulty. From our point of view it does not matter whether the relative immunity of the retina is

intrinsic or due to some protective filter. Fig. 1 illustrates the risk situation of the two tissues: the curve marked 'High Risk Hazard' refers therefore to the lens, and the other one to the retina. They tell us that much less radiation is needed to damage the lens than the retina. From an epidemiological viewpoint this means that, in a given photic environment, there are likely to be more people with lenticular than with retinal problems attributable to ultraviolet radiation. This is expressed by the risk scale on the right of Fig. 1.

This important aspect helps to put the problem of alleged risks associated with sunglasses into some sort of perspective. The hypothetical mydriasis for the 'worst case' situation indicated in Fig. 1 increases the high risk hazard from approximately 0·4% to 5%, that is from 4 per 1000 persons to 50 per 1000. But, while the fractional increase is analogous on the low risk hazard curve, the number of people affected rises from about 1 per 100 000 to 12 per 100 000, so that we are still dealing with a very small part of the population. These values are merely illustrative. In both types of trauma the real numbers are likely to be appreciably lower. They are determined by the constants in equation (7).

On the face of it this argument seems to be valid for acute exposures. Insufficient information is available to allow a confident assertion that it also holds chronically, when cumulative effects may make themselves felt. But perhaps this analysis helps to point to the minimum number of factors that have to be ascertained if this much more complicated task is to be tackled.

References

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