Subclinical impairment of colour vision among workers exposed to styrene

Sir,—Does chronic exposure to styrene impair colour vision? Fallas et al (1992;49:679-82) found subclinical impairment of colour vision among workers exposed to styrene applying the Farnsworth 100 hue test during working hours in daylight. In daylight, however, neither colour temperature nor illumination are constant and good results depend on the use of standard lighting conditions,2 for instance standard illuminant C or D65.3

The authors do not state if those wearing glasses used their own. In our experience most glasses are coloured at least slightly and it is imperative that colour testing should not be performed on subjects wearing coloured glasses or coloured contact lenses.4 In such cases we use clear glasses with different refraction taking into account the fact that we cannot correct astigmatism.

The Farnsworth 100 hue test was designed to test hue discrimination among subjects with normal colour vision and to evaluate chromatic discrimination loss in those with congenital defects of colour vision.3 Subsequently it was applied to test acquired defects. The prevalence of congenital dyschromatopsia is about 8% among men.5 Fallas et al apparently did not distinguish between those with congenital and acquired colour vision defects when calculating the error scores and the ranges. We guess that the results are influenced by congenital defects in colour vision. Furthermore the term “range” was not defined by the authors.

Acquired dyschromatopsias can be caused by many systemic and ocular diseases. Therefore a complete ophthalmological examination is desirable, but probably not feasible in many epidemiological studies. For screening at least the visus should be examined, however. The mean of the error score of the controls given by the authors is high compared with data published by others.5; we think that this discrepancy could be caused by extraprofessional and by congenital dyschromatopsias.

The subjects were examined during the shift so that they were actually exposed to styrene before testing. Ethanol, another organic solvent, is known to cause an acute and transient impairment of colour vision.7 8 To our knowledge, comparable studies on the effect of styrene have not been published. It is an obvious supposition that styrene can cause an acute and transient impairment of colour vision, too, if there are effects caused by a chronic exposure.9 If the colour vision is examined during a shift, it is impossible to differentiate between acute and chronic effects.

In conclusion we think that the paper does not give any evidence of an impairment of colour vision caused by styrene.

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Authors’ reply
We are not the first to find evidence of impairment of colour vision in workers exposed to styrene. A short time previously, Gobba et al1 independently published similar findings. Mergler et al9 had observed evidence of colour vision impairment in workers exposed to mixtures of organic solvents. Perhaps this vision defect can occur with exposure to various solvents.

Our study, like others, cannot distinguish between long lasting and transient effects of exposure to styrene. In our paper we present no hypothesis in this respect. What is suggested is a subclinical impairment of colour vision in workers exposed to styrene and that alone.

Acquired dyschromatopsia is difficult to distinguish from congenital dyschromatopsia but it is hard to understand why workers exposed to styrene should be more often affected by congenital defects than those of a control population living in the same area and matched for age, sex and ethnic origin. In our trials each subject was examined every year by an occupational physician who was familiar with their medical history. In both the exposed and the control groups we have been able to discard people affected by other causes of impairment of colour vision such as alcoholism or diabetes mellitus.

In our study, psychometric examinations were carried out during the shift. Examinations of colour vision were performed independently, also during working hours, because the procedure in each case was too long for a single session. We apologise for using the word “daylight” which could be confusing. The Farnsworth-Munsell procedural guidelines indicate that “sunlight” is irrelevant and that “daylight” together with fluorescent lighting is more appropriate. We have therefore applied the Farnsworth-Munsell procedure. “Range” refers to circuferential errors. Finally, we had no subjects in our sample populations who wore either coloured glasses or coloured contact lenses.


Incidence of lung cancer by histological type among asbestos cement workers in Denmark

Sir,—In their study of asbestos cement workers, Raffin et al (1993; 50:85–9) state that their results indicate that the risk for adenocarcinoma of the lung caused by exposure to asbestos is different from the corresponding risk for squamous cell carcinoma and anaplastic cell carcinoma. The conclusion is based on an analysis of the relative risks (RRs). If the absolute risk (AR) is analysed, however, the interpretation may be different. In the table, which is calculated from table 2 in the paper by Raffin et al, the absolute risk seems to be similar for adenocarcinoma and squamous cell carcinoma of the lung. The authors do not report the person-years so the absolute risk is expressed in relative terms.

The fact that the interpretation is different depending on the risk measure may seem confusing and there are arguments for both. In prevention, the absolute risk is a better measure as it gives a direct measure of the number of cases that will be prevented. The relative risk may be a better measure in clinical practice as it can easily be transformed to the aetiological fraction (EF = (RR-1)/RR) and thus gives a measure of the chance that the cancer of a certain patient is caused by asbestos.

From a more theoretical epidemiological standpoint the best measure depends on whether the risk due to asbestos is multiplicative or additive compared with the background risk. For a multiplicative risk the incidence rate (IR) is IR = IRo* f(exposure), where f is a function not dependent on the background incidence (IRo). If the risk is additive an absolute risk is more appropriate as AR = IRo + IRab where IRab is the incidence rate caused by exposure. A relative risk may at first glance be preferred as the risk for lung cancer caused by asbestos is usually expressed as: SMR = 1 + a * dose where a is a constant. This relation does not seem to fit the data of Raffin et al, however, and the only measure of “dose” in their paper is employment time. The relative risk is certainly not related in linear fashion to employment time if all lung cancers are considered (1.9, 1.4, 1.9 for <1, 1–4, and ≥5 years respectively). The group with 1–4 years employment time is small and there are large confidence intervals for the risks especially when stratified according to histological type. Thus there seems to be little justification to restrict the analyses to a multiplicative model. There is an increased risk in the group with <1 year employment time. This raises the question about comparability between the exposed and reference groups. A dose-response model may also consider time since last exposure as some data indicate that the risk of lung cancer decreases some years after the exposure of asbestos has ceased.1

A different risk according to time from onset of exposure may depend on the different growing rates of the tumours. Anaplastic carcinoma grows faster than squamous cell carcinoma, which grows faster than adenocarcinoma.2 The importance of the finding of a higher RR for adenocarcinoma in persons with a long time since onset of exposure compared with persons with other histological types of tumour does not necessarily mean that only adenocarcinoma are caused by exposure to asbestos. My conclusion is that the

Correspondence


Relative (RR) and absolute (AR) risk of different histological types of lung cancer in the study by Raffin et al (from table 2)

<table>
<thead>
<tr>
<th>Histological type</th>
<th>RR</th>
<th>AR*</th>
<th>(95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma</td>
<td>3.31</td>
<td>16.7</td>
<td>(8.1–28.5)</td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
<td>1.67</td>
<td>14.9</td>
<td>(4.0–29.0)</td>
</tr>
<tr>
<td>Anaplastic carcinoma</td>
<td>1.58</td>
<td>8.5</td>
<td>(0.2–10.1)</td>
</tr>
</tbody>
</table>

* Expressed in relative terms (number of cases *person-years), as the number of person-years is not stated in the paper. Number of cases = observed – expected.
† 95% confidence interval of AR calculated by the same method as that used by Raffin et al (1993).