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#### Aldehyde disinfectants and health in endoscopy units

EDITOR,—I read with interest this report and would accept that it considers the main issues in an effective and commonsense way. I would, however, question the statement that where an employee develops occupational asthma after exposure to glutaraldehyde and continuing exposure cannot be avoided, that the employee must be made aware of the risks of continuing exposure. The implication is that the worker is left the choice as to whether they can continue being exposed to glutaraldehyde. The employer through its occupational health department has a responsibility to advise on fitness for work. In a case where asthma has been shown to be caused by glutaraldehyde it is not reasonable to leave the decision about continuing exposure with the employee, however well informed. The employer has the responsibility for protecting an employee's health. In these circumstances redeployment and retraining may be the best outcome that an employee can expect. The report drew attention to the need for pre-employment health assessment suggesting enquiry about asthma and other conditions. The authors did not comment on whether subjects with pre-existing asthma should be employed in jobs where exposure to glutaraldehyde may occur. This is a difficult issue that seems to have been avoided.

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#### Reply

EDITOR,—Dr Stevens makes two main points in his letter. Firstly – what is the management of a member of the endoscopy staff who develops glutaraldehyde related asthma?; secondly – what is the recommendation of the

working party regarding employment of people with pre-existing asthma who will be required to work with glutaraldehyde?

We would like to point out that it was not within the remit of the working party to produce a manual detailing how health and safety policies might be implemented. It was assumed that individual hospitals will take steps to comply with COSHH regulations.

With regard to the first point, Dr Stevens has interpreted the report as leaving it up to the subject to decide whether he or she should continue to work with glutaraldehyde. We feel this is an extreme interpretation of what has been said. The report states quite clearly that further exposure to glutaraldehyde should be avoided, but recognises that there may be circumstances when this is not possible or desirable. We have to assume that the development of occupational asthma in a member of staff working in an endoscopy unit will inevitably involve the local occupational health department, which will make an appropriate risk assessment and also inform the hospital management. This would take away the decision from the affected subject and give the responsibility to management.

It is reasonable to assume that any such decision will not be made in a vacuum. If the diagnosis of occupational asthma resulting from glutaraldehyde is definite and exposure to glutaraldehyde will probably continue, the employee should be removed from that working environment. If exposure can be reduced, however, and it is the considered opinion of the medical experts that continuing to work in the environment is not a significant risk to the employee, and there is no suitable alternative workplace such that the subject wishes to continue to work in that environment, having been made aware of the pros and cons of doing so, we believe this is a suitable plan of action. The final decision would have to rest, however, with the manager of that department.

With respect to the issue of employing people with pre-existing asthma in jobs where exposure to glutaraldehyde may occur, we believe it would have been inappropriate for the working party to be more specific than it has been, consistent with its stated intention to formulate recommendations rather than instructions. Once again a risk assessment would have to be carried out to take account of the severity of the asthma and the likelihood of exposure to glutaraldehyde. The final judgement about fitness to work is the responsibility of an occupational physician and we did not think it was the role of the working party to preempt this.

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#### Correlation of PCNA with bromodeoxyuridine

EDITOR,—We noted with interest the paper by Weisgerber *et al* (*Gut* 1993; 34: 1587-92) on proliferation cell nuclear antigen (PCNA) and its correlation with bromodeoxyuridine (BrdU). We would like to express our reservations about their conclusions. The most important one is the authors' decision to only count the 'strongly stained nuclei' in the PCNA assay. This necessarily makes the assay highly subjective because of the diffi-

culty of maintaining an identical standard for every nucleus that is assessed. The assay could only become reproducible in large numbers if either all stained nuclei were counted as positive, or if some form of extremely sophisticated image analysis system that could differentiate stained nuclei on the basis of intensity and colour of their staining was used.

While a significant correlation between the two methods assessed has been shown, the analysis has been performed on only 17 values. The correlation coefficient of 0.6 and Figure 1 shows the rather vague interrelation between PCNA and BrdU in this context. This is more important than the non-significant difference seen between the mean proliferation indices for two reasons. Firstly, there is ongoing debate about which fraction of the replicating population of cells PCNA measures – that is, the growth fraction, as in Ki67 labelling, or the S phase fraction as in tritiated thymidine or BrdU labelling. It may be dependent on the form of tissue fixation.<sup>1-3</sup> This study cannot identify which proliferating component has been labelled with this particular assay. Secondly, if there was a strong numerical relation between the two measured values for a given sample, then some form of paired statistical analysis would be appropriate. No evidence is given that this sort of test has been performed in this study.

We do not feel that this paper does show a close relation between the PCNA and BrdU assays. We believe it is necessary to assess a greater number of biopsy specimens taken from a much greater number of subjects; to score all PCNA stained nuclei; and place more emphasis on the correlation between the assays rather than the actual numerical values measured.

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#### Reply

EDITOR,—We appreciate the critical evaluation of our paper by Wilson and Schofield. We agree with most of their comments, some of which have already been considered in the discussion of our article.

In an attempt at trying to mimic an S phase marker by counting only strongly stained nuclei in the PCNA assay, the evaluation procedure certainly has to be highly standardised and the best way is by use of image analysis. If this standardisation cannot be used, it may be a better alternative to include all labelled cells in the PCNA analysis. The inclusion of all labelled cells in the PCNA assay, however, does not result in a significant correlation between the two markers in our study. We could possibly show such a correlation with a greater number of subjects, as suggested by Wilson and Schofield, and we agree that this correlation is