Dangers of ozone depletion

SIR,—The dangers of ozone depletion in the United Kingdom, particularly with regard to non-melanoma skin cancer, are probably much greater than estimated in the recent article on this topic by Fiona Godlee.¹ The figures given in the article suggest that non-melanoma skin cancer may rise by 8000 cases a year in Britain if there is a 10% reduction in total column ozone. This figure is based on current incidence figures from cancer registries, which are widely accepted as being gross underestimates.²

I recently suggested that the true incidence of non-melanoma skin cancer in England and Wales may be close to 100 000 cases a year.³ If this is true then, using a formula devised at the National Cancer Institute, Bethesda, Maryland,⁴ we might expect something like 35 000 extra non-melanoma skin cancers each year in England and Wales. Furthermore, there would probably be a relative increase in the potentially more dangerous squamous cell carcinoma compared with basal cell carcinoma.⁵ Of course, these figures should not be taken at their face value as it is difficult to give an exact percentage rise in skin cancers because predicting what changes may occur in holiday and clothing habits is impossible.⁶

The article therefore highlights the inadequacy of registration of skin cancer in the United Kingdom. Few attempts have been made to define the extent of the problem, and thus monitoring adequately any changes that may occur in the incidence of skin cancer is going to be impossible. As I have pointed out previously, there is an urgent need to review and improve registration of skin cancer throughout the United Kingdom.³

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Infection acquired from transplant donor

SIR,—Jérôme Babinet and colleagues report the transmission of *Plasmodium falciparum* by cardiac transplantation.¹ The diversity of infections after transplantation is increasing, and we report a new mechanism for transmission of the common cold.

A 37 year old woman with complex cyanotic congenital heart disease received a heart-lung transplant from a 14 year old girl who had died of meningitis of uncertain aetiology. In accordance with our routine practice, bronchial secretions and bronchoalveolar lavage specimens were taken from the donor airways for microbiological culture before transplantation. There was a mild polymorphonuclear cell infiltrate, and culture yielded both *Staphylococcus aureus* and rhinovirus. After an uneventful transplant procedure she was extubated within 24 hours.

On the fifth postoperative day she developed a temperature with coryzal symptoms and signs. There was no evidence of deterioration in chest radiographs or on blood gas analysis, and culture of bronchoalveolar lavage specimens yielded the rhinovirus identified previously. No evidence of

rejection was found on transbronchial biopsy, and her common cold resolved over the next four days with only symptomatic treatment. However, rhinovirus was identified in routine bronchoalveolar lavage samples for up to six weeks after transplantation.

Although infection acquired from the donor is a well recognised problem in pulmonary transplantation,² we believe that this is the first report of a new "exogenous" mechanism of spread of rhinovirus.

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Heart valve records falsified

SIR,—Clare Dyer reported on malfunctioning Björk-Shiley convexoconcave heart valves.¹ Shiley has sent data on the fracture of struts of the valves to specialists but not general practitioners. Prompt diagnosis of fractured struts and treatment may save lives and litigation. As the risk of valve replacement is currently much higher than that of strut fracture,² explanation is not recommended. Concern has been expressed about undue anxiety when asymptomatic patients are notified.

General practitioners have ultimate responsibility in the aftercare of those needing careful counselling. It is often they who have the most contact with the patients and families many years after implantation of the valve. It is vital that they should be equipped with sufficient knowledge to deal with the many questions surrounding this issue.

Tracing recipients of convexoconcave valves will be difficult. General practitioners should check records of patients taking anticoagulants or who had a heart valve implanted between 1979 and 1986. If the make of valve cannot be identified the implant surgeon should be contacted. Medic Alert, with a grant from Shiley, has launched an international implant registry to trace the estimated 4000 recipients in the United Kingdom through the medical profession. Recipients of Shiley valves will be offered free membership and information on future developments.

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Dundee coronary risk-disk

SIR,—Several people who read my article on the Dundee coronary risk-disk have written to me because they have been puzzled by their inability to relate the multiple logistic function formula for the five year risk of coronary heart disease, which always gives a probability below unity, to the Dundee risk score (shown in figure 1 and in the photograph of the disk), which runs from 1 to 50.¹ I

emphasised in the paper that this was a relative risk score, which therefore had no absolute units. Because of this I omitted to state that in the formula used for calculating the Dundee risk score the probability shown in the multiple logistic formula has been multiplied by 100, to the percentage risk over five years, as whole numbers are more manageable than decimals. I apologise for the inadequate explanation.

Some of those concerned wanted to program their own electronic calculators to derive both the Dundee score and the rank. Although the rank follows an S shaped curve (shown in figure 1), it does not trace a simple mathematical formula—it traces the population distribution of the score in 10 000 men and women. Our own IBM compatible computer program has a "look up" table, 100 lines long, to emulate the relation between the two non-linear concentric scales on the disk; this is too much for many calculators. Despite the greater versatility and four figure precision of electronic digital microcomputers a dedicated mechanical analogue calculator such as the disk can still score on simplicity.

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Risk factors in Alzheimer's disease

SIR,—The editorial on risk factors in Alzheimer's disease by L J Whalley¹ outlined the results of a collaborative study on the epidemiology of the disease.² Age, sex, and family history emerged as significant risk factors and were duly noted. The editorial did not, however, mention head trauma. In the same European study a history of head trauma was found in 3.35% of cases.³ Subsequent meta-analysis confirmed head trauma as a significant factor in 2.2% of all cases of Alzheimer's disease.⁴ To place this in context, the autosomal genetic defect is responsible for less than 1% of cases.⁵ Thus the editorial omitted to discuss the risk factor currently identified as causing the largest number of cases of the disease.

Our own investigations indicate that repetitive head trauma as suffered by boxers can lead to a disease process with a molecular pathology indistinguishable from that of Alzheimer's disease.⁶ This also applies to some long term survivors of a single incident of head trauma.⁷ More recently we have shown that about a quarter of patients with severe head trauma show formation of β amyloid plaques (a critical pathological feature of Alzheimer's disease).⁸ This complements reports that mutations within the gene coding for the β amyloid precursor protein may be the cause of some familial cases of Alzheimer's disease.⁹ The critical factor in both the genetic and the head injury studies is the disturbance of the precursor protein metabolism, and it has been proposed that the mismetabolism of this protein may cause the disease.⁸ Experiments on mice expressing engineered gene constructs support this hypothesis.⁹

Thus, rather than the presumed complex pathogenesis discussed in the editorial, the pathogenic event is seductively simple. The over-expression of precursor protein and its subsequent mismetabolism are at the heart of Alzheimer's disease. Preliminary evidence indicates that some vascular disorders may also operate in the same way.¹⁰

If mismetabolism of the precursor protein does prove to be the central pathogenic event underlying Alzheimer's disease, irrespective of the causative