gave a very low power, so the lack of statistical significance may have arisen by chance rather than because no differences exist (a so called type II error, or beta error). This paper does not present convincing evidence against the benefits of dietary fibre in the diabetic diet.

M E J LEAN

Diabetic Clinic,
Aberdeen Royal Infirmary,
Aberdeen

B R TENNISON

Department of Community Medicine,
Fulbourn Hospital,
Cambridge

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SIR,—Ms V A Beattie and others asked whether adding fibre to a low energy, high carbohydrate, low fat diet confers any benefit to the management of newly diagnosed overweight type II diabetics (23 April, p 1147). In their abstract and discussion they conclude that neither cereal fibre nor viscous fibre produced benefits beyond those achieved by reduced energy intake and weight reduction. These conclusions are not justified for two reasons.

Firstly, it is always difficult to be confident about a negative result. The three diet groups each consisted of seven to nine patients, and the 95% confidence intervals for both the glycated haemoglobin and fasting plasma glucose concentrations (table II) include values that could have resulted in different conclusions.

Secondly, Ms Beattie and others made no attempt to assess compliance with their dietary advice, perhaps because they felt that many of the methods for doing so are not reliable. Despite the few patients studied, however, their data provide some evidence that their patients may not have altered their diets beyond a reduction of total energy: total cholesterol concentrations were unchanged throughout the study. Reduction of saturated fat to the extent that must have been advised to produce a 60% carbohydrate diet (180 g carbohydrate, 5.0 MJ) should have achieved, if the advice was followed, appreciable reduction in cholesterol concentrations. In view of the fact that the patients seem not to have reduced their intake of fat as directed it seems conceivable that they might also not have followed the advice about fibre, and no conclusions can be drawn beyond the already well established effect of energy restriction and weight reduction on glycaemic control. We urgently need information on the long term effects of dietary fibre in diabetes. Unfortunately this study provides no helpful data.

JIM MANN

Nutrition Department,
University of Otago,
P O Box 56,
Dunedin,
New Zealand

AUTHORS' REPLY.—We concluded that there was little evidence that supplementing a low energy, high carbohydrate diet with fibre confers any therapeutic benefit to type II diabetics on the basis of comparisons of the reductions in body mass index (3 kg/m² on the low fibre diet compared with 2 and 4 kg/m² on the high fibre diets), glycated haemoglobin concentrations, and fasting plasma glucose concentrations. Admittedly, these results were weighted by the reductions occurring during the low fibre run in period. The results from week 4 onwards showed trends in favour of the high fibre diets. The inclusion of greater numbers of patients would certainly have clarified the

situation from a statistical aspect, but even if we had been able to demonstrate a statistical difference in favour of fibre we doubt that it would have been sufficiently large to be relevant therapeutically. We ended the study after two years because we thought that further input would be unlikely to change our conclusions. Perhaps this was not justified.

We do not altogether accept Dr Mann's comments about compliance. The reductions in weight and glycated haemoglobin and fasting plasma glucose concentrations indicate dietary compliance. The average plasma concentrations of cholesterol were also reduced but these results were not statistically significant. The cholesterol concentrations were not abnormally high in most patients at the beginning of the study. It is true that the methods for assessing compliance are unreliable, but we paid particular attention to this aspect of the protocol. The patients were interviewed by the same person at two weekly intervals throughout the study. During these interviews they were asked to recall what they had eaten on the previous day and during the previous week. In addition they were asked to return any guar gum that they had not used. We found no reason from this information to suspect that the patients were not complying with diet requirements.

N W READ
V A BEATTIE

Subdepartment of Human Gastrointestinal
Physiology and Nutrition,
Royal Hallamshire Hospital,
Sheffield S10 2JF

Nonoxinol 9 and HIV infection

SIR,—Nonoxinol 9, a non-ionic surfactant, is the active ingredient of several spermicides. Laboratory studies have shown that it inhibits the growth of *Trichomonas pallidum*,¹ *Neisseria gonorrhoeae*,¹ *Chlamydia trachomatis*,^{2,3} herpes simplex virus,^{4,5} and human immunodeficiency virus (HIV) type I.^{6,8} Its use has been recommended, in addition to condoms, as a measure to inactivate human immunodeficiency virus in genital secretions. Preparations contain between 2 and 8% nonoxinol 9 (w/w), and it is incorporated in the lubricant in some condoms. One manufacturer has recently attempted to promote the use of nonoxinol 9 as a lubricant for vaginal and rectal examinations.

If nonoxinol 9 is totally non-toxic to genital tract epithelia and, when appropriate, the rectal mucosa all well and good. If this is not the case can we be certain that it is not increasing the permeability of cells to HIV and thus enhancing the likelihood of infection? Reitmeyer *et al* state that "Nonoxinol 9 containing spermicides are generally considered to have no toxic side effects in penile-vaginal intercourse, but the safety of nonoxinol 9 in rectal use has not been established."⁷ Allergies are known to occur, and stopping the use of nonoxinol 9 is recommended by all manufacturers if penile or vaginal itching develops. Preliminary studies of cytotoxicity reported by Hicks *et al* indicated that 35% of peripheral blood lymphocytes were killed after treatment with 1% nonoxinol 9 and that a 5% solution significantly reduced viability.⁶ Although spermicidal preparations may seem to be acceptably tolerated by those who use them, low grade epithelial damage may remain undetected. If a clinically inapparent inflammatory process is induced the genital tract epithelium or rectal mucosa may be rendered more permeable to HIV. Nonoxinol 9 might also impair local immune defences by damaging cells of the immune system or enhance the risk of infection with HIV by activating Y lymphocytes.

Any putative antiretroviral agent must be assessed for potential benefit and toxicity. Nobody would support introducing a systemic antiretroviral drug without evidence from clinical trials that its benefits outweigh any danger. The same principles should surely be applied to topical preparations such as nonoxinol 9, and recommendations for its use in preventing infection with HIV should be based only on carefully controlled clinical trials.

D J JEFFRIES

Division of Virology,
Department of Medical Microbiology,
Wright Fleming Institute,
St Mary's Hospital Medical School,
London W2 1PG

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Hospital visiting hours

SIR,—Arrowe Park Hospital is one of those referred to by Dr D N W Griffith (7 May, p 1303) which has returned to restricted visiting hours after a trial of open visiting.

This is a new district general hospital which opened in 1982. From the time of commissioning it was decided to have open visiting from 8 am to 8 pm. This proved to be a disaster, primarily because of abuse of the system by visitors. Many would arrive promptly at 8 am and stay all day. They would bring sandwiches and flasks of coffee and camp out by their relative's bed for the day. Others would eat patients' food, ask for extra cups of tea, etc.

The hospital is fortunate in that the wards are modern and divided into four or six bedded bays. During ward rounds it was only necessary to ask visitors to leave one bay at a time. Nevertheless, visitors often refused or left the bay only with ill grace. On at least one occasion there was even a threat of violence from a visitor asked to leave temporarily.

We tried leaving the visitors in during ward rounds but this was even more problematic. Privacy and confidentiality is inevitably limited in a ward. Patients in adjacent beds are bound to hear conversations between doctor and patient. To erode this privacy further by allowing other patients' visitors in at the same time seemed undesirable. Nursing staff became tired of having to ask visitors in the whole bay to leave while an individual patient was on a bed pan or receiving treatment.

Eventually the pressure to change came from the patients themselves, who were exhausted by the constant presence of visitors. We restricted the visiting to six hours (2 pm to 8 pm) which is still fairly liberal by many standards. Even this did not

work for reasons similar to those described above. Once more, after pressure from the patients, we reached agreement with the health authority and the community health councils to have two short visiting spells each day—one in the afternoon and one in the early evening. The precise times and duration depend on the ward concerned.

We agree that the patient's needs are paramount but not the exclusive consideration. One patient's wishes should not be inflicted on other sick patients in neighbouring beds. The caring professions have a duty to tend their patients, but if this is constantly hindered by the excessive and prolonged presence of visitors we have a duty to consider where the priority lies.

BRIAN ALDERMAN

Arrowe Park Hospital,
Upton,
Wirral,
Merseyside L49 5PE

Case-control study of cutaneous malignant melanoma

SIR,—The abstract of the paper by Drs Dallas R English and Bruce K Armstrong on identifying people at high risk of cutaneous malignant melanoma (7 May, p 1285) is misleading in stating that "... time spent outdoors in summer from the age of 10 to 24 ... provided good discrimination between patients [with malignant melanoma] and controls. ...". In fact table I, which shows odds ratios for variables independently predicting risk of cutaneous malignant melanoma, and a small section in the results state that increasing exposure to sunlight between the ages of 10 and 24 was associated with a decreasing incidence of cutaneous malignant melanoma. It would therefore seem that increasing exposure to sunlight between the ages of 10 and 24 years has a protective effect on the development of cutaneous malignant melanoma.

This is an interesting finding, particularly in view of the large amounts of money currently spent by the Australian government on the "Slip, Slop, Slap" campaign aimed at children and discouraging prolonged exposure to sunlight. I was disappointed that Drs English and Armstrong chose not to discuss the implication of this important finding in their paper.

N S BROUGHTON

Department of Orthopaedics,
Luton and Dunstable Hospital,
Luton,
Bedfordshire LU4 0DZ

AUTHORS' REPLY.—The model we developed did provide good discrimination between patients with melanoma and controls. Mr Broughton seems to have inferred that an apparent protective effect of time spent outdoors cannot help distinguish patients from controls. In fact, whether the effect of an "exposure" is to increase or decrease the risk of developing the disease makes no difference to its contribution to the model's ability to discriminate. Time spent outdoors was not the only variable that decreased the risk of melanoma—arrival in Australia after the age of 10 had the same effect. For such factors people in high risk categories are those with the least amount of exposure.

With respect to Mr Broughton's comment about the implications of decreasing the risk of melanoma by increasing the time spent outdoors, we reiterate that the purpose of our paper was to determine whether it was possible to identify a group of people at high risk of melanoma, not to identify risk factors for melanoma. Our finding of a protective effect of increasing time spent outdoors was not new and has been discussed in more detail.¹

Although it seems that a reduction in exposure to the sun by Australians may increase the incidence of melanoma, our study also showed that arrival in Australia after the age of 10 was a strong independent predictor of a reduced risk of melanoma. The prevalence of naevi, which was the strongest risk factor for melanoma, was also reduced in these migrants. As most migrants to Australia come from countries that have less sun it is reasonable to assume that their lower risk is a result of less exposure to the sun early in childhood. Thus the "Slip, Slop, Slap" campaign directed at children should, if successful, reduce the incidence of melanoma.

In addition, the "Slip, Slop, Slap" campaign is directed primarily at the prevention of non-melanocytic skin cancer, which is the commonest cancer in Australia. Because of its high incidence non-melanocytic skin cancer is an important contributor to the use of medical and hospital resources. The indirect evidence from its distribution by age, sex, and site on the body and, to a lesser extent, its association with outdoor occupations would suggest that the incidence of non-melanocytic skin cancer increases with increasing total accumulated exposure to the sun and would be reduced by reducing exposure to the sun.

DALLAS ENGLISH
BRUCE ARMSTRONG

National Health and Medical Research Council,
Unit in Epidemiology and Preventive Medicine,
University Department of Medicine,
Queen Elizabeth II Medical Centre,
Nedlands 6009,
Western Australia

I Holman CDJ, Armstrong BK, Heenan PJ. Relationship of cutaneous malignant melanoma to individual sunlight exposure habits. *JNCI* 1986;76:403-14.

Diet and inequalities in health

SIR,—Dr J E Cade and others (14 May, p 1359) shows that, contrary to established wisdom, the fat content of diet in middle age is not related to the death rate from ischaemic heart disease. In fact, if the data from the three towns studied were taken in isolation they would suggest that a high fat diet conferred some protection.

The incidence of ischaemic heart disease in the towns studied rises the further north the town is located, and this supports the suggestion that environmental cold stress may play an important part in explaining the geographical differences in the distribution of ischaemic heart disease, since the degree of cold stress is dependent on absolute temperature, air movement, and dampness.¹

Although the social class distribution was similar in the three towns, the socioeconomic indices were worse in the two northern towns. Social class is usually closely related to socioeconomic indices, and the lower the social class the more likely it is that people are living in cold, damp housing. Low income, more common in the lower social classes, means that house repairs may have to be neglected, clothing may be of inferior quality and quantity, and energy intake may be marginal. Lower social class is also associated with smaller houses, poor insulation, and a higher number of people in each room. These factors increase the risk of damp.^{1,2}

The lower the social class, or the worse the socioeconomic indices, the greater the potential for exposure to cold stress, a relation which matches the known gradation of incidence of ischaemic heart disease through the social classes.^{3,4} One of the physiological responses to exposure to cold stress is a rise in the circulating concentrations of catecholamines,¹ and it has been suggested that these play a part in the aetiology of ischaemic heart disease.⁵ It seems probable that cold stress acts

through catecholamines, and a paper is being prepared which reviews the evidence for the role of cold stress in the aetiology of ischaemic heart disease.

E I LLOYD

Department of Anaesthetics,
Princess Margaret Rose Hospital,
Edinburgh EH10 7ED

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Restraining infants in cars

SIR,—Dr M A Mason (14 May, p 1345) highlights the important problem of mortality and morbidity from road traffic accidents among infant car occupants. Unpublished Scottish Office statistics between 1981 and 1985 show that 288 (just over 7%) of the 3931 children injured while travelling by car were infants. Of these 75% were unrestrained.

We carried out a pilot study in Fife to see if the success of infant car seat loan schemes overseas could be repeated in Britain.¹ Our project was a success with 79% of the babies travelling regularly by car in the intervention population being carried in a rear facing infant car seat compared with only 8% of the control population. In the intervention group 93% of parents said that if they had another baby they would buy or hire an infant car seat. Interestingly, 82% of the mothers given a seat indicated that if one had not been provided they would have carried their baby in their arms or in a carrycot.

We urge health authorities and local government in other areas to give serious consideration to starting infant car seat loan schemes.

ERIC BAIJAL

Fife Health Board,
Glenrothes,
Fife KY7 5PB

1 Baijal E, Carter H, Jones IG, Walkingshaw IW. The role of infant car seats in road safety. *Journal of the Institute of Health Education* (in press).

Clinical carpal scaphoid injuries

SIR,—I thank Mr M Reichl and Dr M A Khan (23 April, p 1198) and Dr J J Langham-Brown (30 April, p 1259) for their comments on my and my colleagues' paper (19 March, p 825).

Traditional management by plaster cast for the clinical scaphoid injury is still practised in many hospitals throughout Britain. Awareness of the potential sequelae of scaphoid fractures has promoted this conservative approach.

Our study showed a low incidence of delayed radiological visualisation of scaphoid fractures (2%). We therefore concluded that the clinical scaphoid syndrome is a soft tissue injury and hence appropriate splintage should be sufficient; it is not the cause of the dangerous sequelae initially thought. As the *BMJ* has a wide distribution among junior and senior doctors we hoped that our article would give guidance to change from a policy of plaster cast management.

We also have reported the use of radionuclide bone imaging¹ and like Mr Reichl and Drs Khan and Langham-Brown would recommend it for diagnosis and management decisions. But not every hospital has an isotope department or immediate access to one. We would suggest that two