

several authors. The new findings in our study were the relation between body mass index and antibody response, and the interactions between this index and injection site and sex. We do, as suggested by Yassi *et al*, recommend checking antibody concentrations in staff in contamination incidents; this is part of a comprehensive programme of prevention and management of such incidents in our Health Authority.

Yassi *et al* make a good point regarding the need for more information about the rate of fall of antibody concentrations over time. Particularly, what is required is data to allow prediction of duration of persistence of protective antibody concentrations in subjects after immunisation. The studies reporting mean antibody concentrations at intervals after immunisation do not help much in this regard. We are presently analysing data from our immunisation programme, which includes repeat antibody testing at intervals, with the aim of devising a method of predicting the rate of decline of antibody concentrations. Unfortunately, the data presented by Yassi *et al* are inadequate for this purpose; only 60 people were studied and antibody concentrations were measured at varying and unspecified intervals up to six years from primary immunisation. No attempt was made to relate the measured concentrations to the concentrations immediately post immunisation, which were apparently known only in some subjects. The lower proportion with "protective" antibody compared with that in our larger study of concentrations immediately post immunisation is not surprising.

It is a cause of concern to note the apparently low coverage among staff reporting contamination incidents, in the hepatitis B immunisation programme reported by Yassi *et al*. This is despite the fact that 10% of the reported incidents were from known HBsAg positive sources. Achieving a high coverage with hepatitis B immunisation is at least as important as considerations of timing of booster doses.

Role of manmade mineral fibres in the causation of cancer

Sir,—We agree with almost all of the recent editorial by Enterline (1990;47:

145–6). Indeed, the latest update of the American epidemiological studies, of which Enterline is a co-author, make the conclusion of little or no risk in the glass and rock wool production industries even stronger (G Marsh, personal communication). As Enterline points out, any excess of lung cancer is virtually limited to the slag wool production industry more than 20 years ago. Much of the excess mortality in this group occurs among very short term workers (J Hernan, personal communication). Furthermore, the known exposures to asbestos and to other co-exposures in this branch of the production industry are now recognised as being more likely causes of any excess mortality among longer term employees.

Enterline draws the following conclusions:

It is doubtful whether much meaning can be attached to the small excesses among glass wool workers.

There are no excesses in workers in rock wool plants.

Probably fibres do not play the major part in the excess of cancer among slag wool workers so that fibre potency should not be estimated from epidemiological data on workers who produce these fibres.

Given these conclusions, it is surprising to note his final sentence which can be read to imply that exposure to manmade mineral fibres, whether made from rock, slag, or glass, can cause lung cancer. We wonder if certain key words were omitted and suggest that the sentence should read: "There is undoubtedly some evidence of a small cancer hazard attached to the manufacturing process in slag wool plants 20 to 50 years ago, when asbestos was used in some products. Even if glass, rock, or slag wool pose a fibre hazard, it is much less than that of asbestos and perhaps some other kinds of manmade mineral fibres."

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Author's reply:

Rossiter and Douglas suggest that there may be an inconsistency in my editorial. Whereas it is true that in two large epidemiological studies 20 years

from first exposure, respiratory cancer excesses in glass wool plants are small and are non-existent in rock wool plants, I think it is important that the question of whether manmade mineral fibres are capable of causing cancer in man is not dealt with solely on the basis of the existing epidemiological evidence. The usefulness of the data is severely limited because of the extremely low fibre exposures. Because of the likelihood that it is the physical properties of asbestos fibres that cause respiratory cancer, similarities in the physical properties of manmade mineral fibres and asbestos, evidence that manmade mineral fibres are capable of producing cancer in animals, and clear evidence that non-asbestos fibres are capable of producing cancer in man, it would be irresponsible to conclude that ordinary manmade mineral fibres, whether made from rock, slag, or glass, do not carry some risk of cancer.

Rossiter and Douglas also raise the issue as to whether the excess seen in the slag wool plants may have been due to the use of asbestos many years ago. Probably because of the litigation problem in the United States, this has proved to be a difficult area for investigation. Statements from industry have come very slowly and are sometimes contradictory. Recently, however, we have obtained direct evidence of the use of asbestos in one slag wool plant where asbestos was apparently used in a cement mixing operation. In a case-referent study with lung tissue collected from decedents in the United States study we found four of six workers with amosite asbestos in lung tissue and only one in six matched referents.¹ We were also able to obtain lung tissue for five other rock wool or slag wool workers but for only two matched referents. Of the five, three were from a plant in which we have been told asbestos was used and which was originally a rock wool plant. Of these three, two had amosite asbestos present. In only one of the other five specimens (a referent) was any type of asbestos found. In our latest report for the slag wool plant where four of six specimens contained amosite asbestos the respiratory cancer standardised mortality ratio (SMR) 20 years from first exposure was 164.3 (23 deaths) whereas for the rock wool plant where two of three specimens contained amosite asbestos the respiratory cancer SMR was 79.2 (13 deaths).² Asbestos may, of course, have contributed to