Correspondence

Allergy to laboratory animals

Sir,—Allergy to laboratory animals has been the subject of several recent studies¹⁻³ and of a symposium.⁴ The results have been surprisingly consistent. Some 20% of those currently working with laboratory animals are allergic to them; the majority have rhinitis, a minority also asthma. Those with rhinitis and asthma are likely to be atopic and to have skin prick test reactions and specific IgE to urines of the animals which provoke asthmatic symptoms; those with rhinitis alone seem no more likely than others comparably exposed (and asymptomatic) to be atopic or to have skin prick test reactions or specific IgE to the urines of animals provoking nasal symptoms.

The first (retrospective) part of the study of Davies *et al*⁵ is made on a volunteer sample. None the less, their findings lend support to those of earlier studies as shown in the table below, extracted from their results:

Laboratory animal allergy	Skin test positive to rat urine	Specific IgE	Total
Rhinitis alone	5 (?)	4	9
Rhinitis and asthma	12	12	13

We were surprised to read in their discussion that there is "no evidence of increased antibody levels of the asthmatic group compared with the rhinitis group" as their results seem to lead to the opposite conclusion. In addition they find "no indication of difference in the incidence of positive skin tests between the two groups." This is difficult to argue as they failed to undertake skin tests in three of the nine with rhinitis, but may in part be attributable to the inherent bias towards those with more severe disease in a volunteer sample.

The findings of the second (prospective) part of the study are potentially of great interest but as has been remarked of statistics, bikini-like: what they reveal is suggestive, what they conceal is vital. In the first year of their employment 22 of 148 individuals developed animal related symptoms. Of these 22, nine (of whom two have asthma) have specific IgE to rat urine and 13 (of whom one has asthma) do not. The interval between starting animal work and the development of asthma may be several years and occurs with or after the onset of rhinitis. Clearly several more cases of asthma are likely to develop in this population, from among those with and without current rhinitis. Specific IgE antibody has developed during this first year in the sera of several. The crucial question is whether those with specific IgE antibody are at particular risk of developing asthma.

The results of cross sectional studies (and of this volunteer study) would suggest that they are. If this is supported by the future results from this prospective study the development of specific IgE antibody to animal urines could become a valuable biological monitor in laboratory animal workers. Similarly, skin prick conversion, from negative to positive reactivity may be a significant marker and a comparison with antibody findings is likely to yield important information for predictive use.

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References

- ¹ Slovak AJM, Hill RN. Laboratory animal allergy: a clinical survey of an exposed population. *Br J Ind Med* 1981;38:38-41.
- ² Cockcroft A, McCarthy P, Edwards J, Andersson N. Allergy in laboratory animal workers. *Lancet* 1981;i:827-30.
- ³ Newman-Taylor AJ, Longbottom JL, Pepys J. Respiratory allergy to urine proteins of rats and mice. *Lancet* 1977;ii:847-9.
- ⁴ Dewdney J. Allergy induced by exposure to animals. *J R Soc Med* 1981;74:928-32.
- ⁵ Davies GE, Thompson AV, Niewola Z. Allergy to laboratory animals: a retrospective and a prospective study. *Br J Ind Med* 1983;40:442-9.

Dr GE Davies and Dr EL Teasdale reply:

Drs Slovak and Newman Taylor appear to question the suggestion that in our retrospective group there was "no evidence of increased antibody levels of the asthmatic group compared with the rhinitic group. . . ." This statement was based on the results given in table 2 of our paper where the mean levels of IgE antibody to rat urine allergen were 19 ± 2.3 (SE) in 12 individuals with chest symptoms and 16 ± 2.5 in five without. This difference is not statistically significant. It should be further noted that all 17 individuals had positive skin prick tests to rat urine. Slovak and Taylor have included the results from the small number in table 3, which includes the eight individuals who had no IgE antibody to rat urine. We did not consider this acceptable for the following reasons: only two individuals (Nos 1 and 5) had more than mild symptoms due to exposure to rats, one with chest symptoms and one without (and if these are included in the total the difference is still

insignificant); one individual was allergic to mice and one to guinea pigs and IgE antibodies to allergens from these species were not tested; two had skin reactions only (one very mild) and two had mild nasal symptoms only. We would also emphasise that this retrospective group was chosen from individuals who in a previous survey had reported allergic symptoms and our comments about increased antibody levels referred only to this group and should not be taken to indicate that the statement is necessarily true of a larger, controlled population. Even with this proviso our results do not support the statements made by Newman Talor *et al* in their abstract that "these findings suggest that the immunological mechanisms responsible for asthmatic reactions to laboratory animals are different from those involved in rhinitis and urticaria."¹

We fail to see the point of the last paragraph of their letter. There was no intention to conceal anything. The prospective group is, as stated, a clearly defined population of individuals during their first year of employment. We agree that it is crucial to know if those with specific IgE are at particular risk of developing asthma, and this is one of the reasons for the continuing study referred to in our paper. Slovak and Newman Taylor seem to be reading more into our results than is justified from the defined nature of the study. We also agree that the development of specific IgE could become a valuable monitor and that skin prick test conversion may be a significant marker. We are seeking scientific evidence for these assertions.

We should further point out that although our observations were "made on a volunteer sample" there was a 100% response; all individuals who began employment during the study period agreed to take part.

References

- ¹ Newman-Taylor AJ, Myers JR, Longbottom JL, Spackman D, Slovak AJM. Immunological differences between asthma and other allergic reactions in laboratory workers. *Thorax* 1981;**36**:229.

Book reviews

IARC monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol 31: *Some Food Additives, Feed Additives and Naturally Occurring Substances*. (Pp 314; \$30.00.) Vol 32: *Polynuclear Aromatic Hydrocarbons, part 1, Chemical, Environmental and Experimental Data*. (Pp 477; \$40.00.) Vol 33: *Polynuclear Aromatic Hydrocar-*

bons, part 2, Carbon Blacks, Mineral Oils and some Nitroarenes. (Pp 245; \$25.00.) Lyon, International Agency for Research on Cancer, (distributed by the World Health Organisation), 1983.

The IARC monographs will need no introductory description for anyone with an interest in chemical carcinogenesis. It would be superfluous to undertake an evaluative review of publications of this type, which summarise the conclusions of expert evaluation panels. Nevertheless, since these works constitute one of the most important information resources on carcinogenesis, the appearance of these titles will be noted alongside other reviews, with a brief indication of the subjects covered.

Volume 31 covers a diverse range of additives and other substances that may be present by design or accident in food for human or animal consumption. Apart from various antibiotics, preservatives, and similar materials, there are several interesting natural compounds in the list, including intrinsic components: cholesterol and flavonoids such as quercetin, for example. Pyrolysis products, and fungal metabolites such as T₂ trichothecene, were also considered. In addition to their possible importance as dietary ingredients and occupational agents for the food industry, readers of this journal might care to speculate whether such chemicals have any interactions with toxic or carcinogenic substances of occupational origin.

Volumes 32 and 33 deal with the polycyclic aromatic hydrocarbons: as befits a group of compounds which include some of the earliest known and most extensively studied chemical carcinogens, a substantial body of information is provided. Forty two hydrocarbons and six heterocyclic compounds present in the environment are reviewed, to add to a previous series considered by the IARC in 1973. The subjects of volume 33 include several materials of more complex composition, such as oils and carbon black, that may be of interest in an occupational context. Some nitroarenes are also considered; many combustion processes seem to generate both these and the polycyclic aromatic hydrocarbons.

A G SALMON

Short and thin mineral fibres Identification, exposure, and health effects. Proceedings from a symposium. EJ Chatfield, PC Elmes, H Muhle, F Pott, and FD Pooley. (No price given.) Solna; National Board of Occupational Safety and Health Research Department, 1983.

These four invited lectures were given by recognised