Correspondence

Pulmonary function in aluminium smelters

SIR,—In his paper on pulmonary function in aluminium smelters Dr GB Field (October 1984;**39**:743–51) has demonstrated temporary bronchoconstriction in workmen carrying out a specific task—namely, anode changing. Although he did not present formal criteria for diagnosing occupational asthma, few would not accept the lung function changes observed as being diagnostic of that condition. However, a number of other inferences drawn in this paper are more open to dispute.

Some prominence is given to the conclusion that men giving a subjective history of chest tightness (group C) also demonstrated objective evidence of this. This is characterisation of a subpopulation by a dependent variable; the only surprise would be if no correlation existed.

The author is equivocal about whether he has described an irritant or an allergic phenomenon, citing variation in response and atopy as giving contradictory clues. Thus the paper alludes to "a wide range of individual susceptibility ... after a similar exposure ..." as an argument towards accepting an allergic aetiology. Paradoxically, reference is also made to high levels of total particulate causing "a dose-response effect." Such arguments might be taken as mutually exclusive unless they are stated to refer to group data on the one hand and within person findings on the other. If this is so, it is unclear in the text. However, since the hygiene data are not tabulated and no mention is made of the wearing or non-wearing of respiratory protection it is probably safer to discard any conclusions drawn from exposure data. With regard to atopy, the presumption is made that atopy defined by skin prick is a confounding variable, whereas it is as likely or more likely to be entirely independent. This is the situation with other low molecular weight chemicals or fumes causing asthma.

A number of omissions in the paper are curious, especially in comparison with the highly detailed validation of respiratory measurement techniques. For example, there are no detailed data on the four cases of occupational asthma which suggested this study and whose histories are germane to some of the arguments developed in the paper. No occupational history was taken. The Medical Research Council questionnaire is not designed for this purpose. This must be especially important in men recently employed from rural backgrounds, these not being devoid of allergic and irritant potential.

The wider and perhaps main issue is really one of definition or perception. Clues derived from this type of clinical situation will tend to be hard to interpret when applied to mechanisms of mediation. This relates to shortcomings in the concepts we use and our understanding of them. Irritance and allergenicity are not mutually exclusive and in any particular event may occur individually and independently or interact jointly. The effect produced will vary according to the responsiveness of the airways, which is conventionally described as specific (allergenic) or non-specific reactivity. This reactivity in itself may be independently variable with time, treatment, other exposures, etc. The final measured outcome of these interactions we artificially constrain by calling it asthma or non-asthma. However, those of us who see entire populations with occupational exposures will see several cases with symptoms but without objective evidence sufficient to diagnose asthma (that is, non-asthma) for every "classic" asthma case. In this model overt asthma is the top of the iceberg. Looked at in this way, Dr Field's findings in a cross-sectional survey make more coherent sense. I suggest that what he has observed, being acute and frequent (18 out of 31 men), is an irritant response. The presenting tip, the four cases of occupational asthma, may have developed their disease after a latent period because of the gradual development of non-specific airways reactivity.

These hypotheses can be investigated and resolved by conventional immunological, respiratory, and occupational health and hygiene techniques.

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** This letter was sent to Dr Field, who replies below.

SIR,—Dr Slovak's comments are pertinent and perhaps reflect my failure to define the aim of the study in sufficient detail. Its main purpose was to establish the prevalence of asthmatic symptoms in the workforce, to verify the asthmatic nature of the symptoms objectively, and to determine whether pot emissions were inducing acute asthmatic reactions. I do not believe that chest tightness is synonymous with asthma as conventionally defined or that one can accept without question the validity of symptoms in an industrial workforce. The study was directed specifically not at occupational asthma but at asthmatic reactions of any type. However, the high prevalence of asthmatic symptoms, the wide range of individual susceptibility, and the relation between dose and response to pot emissions (not specifically to particulates but perhaps to some associated sensitising agent in the emissions) provide indirect support for an occupational aetiology. An anecdotal description of the four index cases which initiated the study would have contributed little to the scientific merit of the paper and would have been irrelevant to its main purpose. An occupational aetiology can be established only by a prospective study. Such studies are already in progress in four smelters in Australia and New Zealand and will shortly begin in a fifth. In general, I agree with Dr Slovak's comments on respiratory irritation and allergy but is is becoming increasingly clear to those of us who have extensive clinical and epidemiological experience of occupational asthma that conventional allergic mechanisms are not neccessarily involved in occupational asthma even when it presents with the typical clinical features of a hypersensitivity reaction. The data presented in my paper could be explained on the basis of non-specific airway hyperreactivity, but the question that now needs to be examined is why was there such a high prevalence of airway hyperreactivity?

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