LETTERS TO THE EDITOR

Re: Preventing baker’s asthma: an alternative strategy

Dear Sir,

We read with interest the recent article by Smith [1] concerning health surveillance in the baking industry and, in particular, the immunological and clinical characterization of bakers with work-related nasal and/or respiratory symptoms.

We are also very much interested in this particular issue, and agree that there is a substantial body of literature to support the fact that many workers exposed to flours and additives in the baking industry develop symptoms due to an ‘irritant’, or non-allergic medicated mechanism.

However, we would like to mention a few specific points in relation to the paper.

If the endpoint in the diagnosis of occupational asthma is chosen as those workers with work-related symptoms and sensitization to (at least one) workplace-encountered allergens, it is clearly important to measure specific IgE to an exhaustive list of known allergens. Whilst most sensitized workers will be sensitized to commonly encountered allergens (i.e. wheat flour and fungal alpha amylase), some may also be sensitized to less common agents (e.g. rye, barley, oats, storage mites or other added enzymes such as cellulase, xylanase or bacterial amylase).

In addition, Smith does not mention the total number of workers with respiratory symptoms who do not have measurable specific IgE antibodies to the workplace allergens tested. In a recent study, our group [2] retrospectively analysed 86 bakers complaining of any respiratory symptoms. Of the 34 workers with work-related respiratory symptoms, only 7 (21%) had such evidence of specific IgE to common bakery allergens.

While it is reasonable to concentrate on the health issues associated with sensitization, it is clearly not sensible to consider those with symptoms and no sensitization as not relevant for two reasons.

First, the presence of specific IgE (assuming this is comprehensively tested) is not a prerequisite in the diagnosis of occupational asthma [3]. It is not adequate merely to class these individuals as suffering from irritant symptoms without a better diagnostic definition of such symptoms. Indeed, there are some data to suggest that work-related symptoms, irrespective of the cause and sensitization, carry a poor prognosis [4].

Second, the remaining workers with work-related symptoms and negative specific IgE are likely to constitute a mix of other respiratory diseases, some of which may be work related. Chronic obstructive pulmonary disease is common in many elderly smokers and there is at least a reasonable epidemiological [5] and clinical evidence [6] to support the role of occupational dust exposure as being causative. Again, from our recent data, of the 27 bakers with work-related respiratory symptoms and negative specific IgE, eight had worked in the baking industry for more than 5 years, and three for more than 20 years. This suggests that these bakers are not just people with short latency irritant symptoms, but may indeed have developed either unrecognized occupational asthma or COPD.

In conclusion, health surveillance schemes are at the heart of preventing harm from significant work-related respiratory disease, but must adopt sound evidence-based diagnostic definitions, and encompass a full immunological assessment when necessary.

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References

to the fall in prevalence of coal workers’ pneumoconiosis (CWP) in the USA over the last 30 years. They then state, ‘Similar trends have been noted in Europe’. We would like to expand on this statement in respect of our experience here in the UK.

In the UK coal mining industry, health surveillance (by chest X-ray) has been provided, over the last four decades, initially at 5 year, and since 1974, 4 year intervals. The prevalence of CWP among employed miners fell from 12% in 1959–1963, to 0.2% (13 cases, all ILO category 1) in 1994–1997 [2]. However, by 2000 it was apparent that an upturn in prevalence had occurred, and that there were some cases of category 2 or greater, including some in relatively young miners.

An investigation by the Health and Safety Executive (HM Inspectorate of Mines) concluded that the upturn was probably attributable mainly to a recent tendency on the part of some miners to work considerable amounts of overtime, thereby increasing their exposure to dust. Contributory factors were thought to be an increase in development work (driving tunnels) in rocky strata and the introduction of more powerful mining machinery, both possibly leading to increased levels of dust production [3].

In conjunction with the occupational hygienist and a medical statistician, we have now completed a comparison of the prevalence of CWP with the available results of dust monitoring over the past 30 years. This reveals, semi-quantitatively, that the increase in prevalence is associated with an increase in average dust concentrations that began approximately 6 years earlier. (A report on this study has been made available to the UK coal mining industry and other interested parties.)

The control of dust in the underground environment must remain a high priority for mining engineers, and health surveillance will be required within the industry for the foreseeable future.

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References

Re: The treatment of cyanide poisoning

Dear Sir,

The in-depth review of ‘The treatment of cyanide poisoning’ [1] describes very succinctly the toxicology of cyanide poisoning and the various cyanide antidotes favoured in different countries. Appropriately, the author makes no mention of solutions A&B, which have no place as an antidote. However, the author does actively refer the reader to a reference text [2] for information regarding ‘a number of other antidotes’. It is important that those who may refer to the latter and otherwise authoritative text are aware of a transcription error relating to an unpublished and ‘limited’ study in rats that gave a falsely favourable impression of solutions A&B. The inappropriateness of solutions A&B and the details of the transcription error are reported in an earlier paper in this journal [3]. That these solutions should not be used is confirmed in a Health and Safety Executive leaflet [4].

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References

doi:10.1093/occmed/kqi006

Three tears for EMAS

Dear Sir,

In 1994, your journal marked the 21st anniversary of the Employment Medical Advisory Service (EMAS) [1]. The celebratory article by Tim Carter, the then HSE Director of Medical Services, appeared to be waving the flag loyally for the organization that Dr Trevor Lloyd Davies built.

Ten years later, the post of Chief Employment Medical Advisor, after having hung on for several years on a part-time basis, has finally expired, the cadre of medical advisors having shrunk below the pre-1970 level for

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doi:10.1093/occmed/kqi003

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