Is atopy and smoking important in the workplace?

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The relationship between the respiratory system and the environment involves a complex dynamic interaction of genetic susceptibility, host defence and toxicity. The chance of an individual developing a respiratory disease is dependant on genetic susceptibility and subsequent hereditary risk factors, life-style risk factors and the amount and nature of the exposure that may be encountered in the working environment. Atopic status is an important pre-existing risk that a worker may bring to the workplace (occupational asthma/rhinitis to high molecular weight agents). Smoking is an avoidable additional risk for certain occupational diseases (occupational asthma/bronchitis/cancer) while it can be protective in other circumstances (allergic alveolitis). More controversially, smoking in some workers may put at increased risk the health of colleagues (passive smoking). This article attempts to clarify the issues surrounding the interaction of atopy, smoking and the workplace.

Key words: Allergens; atopy; environmental tobacco smoke; smoking; workplace.

INTRODUCTION

This article will attempt to summarize the areas in which smoking, atopy and occupational settings may interact. In addition, we will consider the role of the occupational health service in terms of the practical issues surrounding these interactions.

OCCUPATIONAL ASTHMA AND ATOPY

Before interpreting current evidence, we need to define atopy. For the purpose of this discussion `atopic status' will be defined by the presence of an identifiable allergic reaction to one or more of a range of common airborne environmental allergens as can be expressed by either skin-prick testing or a raised specific immunoglobulin E. 'Atopic disease' includes a number of disorders (asthma, rhinitis, conjunctivitis and eczema), which are expressed as a direct response to normal environmental allergen exposure in the presence of a positive atopic status.

It would be surprising if the pre-existent immunological reactivity of an individual had no bearing on his/her development of allergic disease on exposure to a respiratory sensitizing agent at work. Those individuals who have already demonstrated the ability to develop an immunological response to common environmental allergens might be expected to be more likely to develop sensitization to inhaled allergen in any setting. However, the findings of epidemiological studies are not so consistent. The relationship between pre-existing atopic status and occupational asthma is commonly present for high molecular weight compounds. For example, in a study of workers exposed to an enzyme alcalase, Newhouse found 82% of previously atopic individuals developed sensitivity to the occupational agent compared with 37% of non-atopic workers. Similar positive studies for atopic status have been demonstrated with other high molecular weight compounds including laboratory animal allergens, flour antigens and complex platinum salts. The responses to high molecular weight compounds and common environmental allergens are both mediated by the IgE pathway.

Low molecular weight compounds do not demonstrate a consistent relationship with atopic status. For example, no relationship was found for Western Red Cedar-induced occupational asthma, for which the major antigen (plicatic acid) has a molecular weight of 400 Daltons. Similarly, atopic status does not seem to be important in sensitization to isocyanates which are the most common occupational sensitizer in the UK. The immunological processes for these low-molecular weight compounds are more complex and do not normally involve IgE. Most research has concentrated on the presence of atopic status rather than atopic disease. The latter would require prospective evaluation, while atopic status can be measured in cross-sectional populations and is an objective measure. However, it is widely believed that pre-existing atopic disease may increase risk further and, additionally, the development of constitutional asthma.
makes the subsequent diagnosis of occupational asthma more difficult to establish.

OCCUPATIONAL ASThma AND SMOKING

Smoking may, theoretically, predispose the airways to the respiratory effects of inhaled allergens in a number of ways. Pre-existing smoking may directly induce non-specific bronchial hyper-reactivity, and this may increase the risk of developing hyper-reactivity to inhaled allergens and certainly to irritant chemicals. Secondly, smoking influences immune reactions, and some studies have demonstrated an increased propensity to the development of IgE antibodies in smokers. Finally, disruption of the respiratory endothelium and damage to the mucociliary clearance mechanisms may increase the access of the inhaled allergen to the submucosal immune-competent cells.

In platinum workers there is an increased risk of sensitization in workers who are current smokers. The data has been sufficiently convincing for one industry handling platinum salts to exclude current smokers from working in areas with potential exposure to platinum. A similar increased risk associated with smoking has been demonstrated in snow crab processing workers and in at least one of a series of studies on laboratory animal workers. Occupational studies on the development of IgE antibodies have demonstrated a co-effect of smoking in workers exposed to a variety of occupational agents including isphagula and tetrachlorophthalic anhydride. These latter studies suggest that the major means by which smoking increases the likelihood of occupational asthma is through an IgE-mediated mechanism. In Western Red Cedar workers, smoking appears to have a protective effect against sensitization. In 185 individuals with occupational asthma defined by bronchial provocation tests or serial peak flow recordings, only 5% were current smokers and 70% were never smokers.

OCCUPATION, SMOKING AND CHRONIC BRONCHITIS

Smoking is the most important aetiological factor in the development of chronic bronchitis and emphysema. However, smoking and occupational exposures may interact to increase the risk of development of respiratory disease.

Studies of coal workers and the development of chronic bronchitis have now resulted in the acceptance of an effect of the combination of both smoking and dust exposure. More recently, epidemiological evidence has shown additive effects of occupational exposure and smoking on the development of chronic bronchitis as determined by respiratory symptoms and the presence of chronic airflow obstruction in workers exposed to cotton dust and in welders, although a study of foundry workers failed to show a significant additional effect of occupational exposure.

SMOKING, ATOPY AND OTHER RESPIRATORY DISORDERS

Smoking has been studied in terms of the increased risk of development of lung cancer for a number of occupational carcinogens. Unsurprisingly, there is an additional risk from combined exposure. The evidence is most clear for the interaction of asbestos and smoking on the development of lung cancer. In a study by Hammond et al., the mortality ratio for lung cancer increases to 53 times that of controls for those with asbestos exposure who also smoke compared with an 11-fold increase in smokers and a fivefold increase in non-smoking exposed workers.

Byssinosis is an unusual disease occurring in workers exposed to cotton, flax or hemp dust and is characterized by the development of respiratory symptoms experienced at the start of the working week, in association with bronchial hyperreactivity and a degree of airway obstruction. Many years of exposure are required before the disease develops. In early epidemiological studies, smoking appeared to be a significant risk factor, although more recent prospective studies have failed to show a significant role of smoking in the development of the disease. This may be due to a lessening of a synergistic response with a reduction of occupational exposure to the cotton dust, or it may be that the studies have more clearly separated the diagnosis of byssinosis from chronic bronchitis.

However, smokers in occupational settings have a reduced risk of allergic alveolitis because smoking appears to offer an element of protection from the disease. The mechanism is thought to be due to an immune modulation.

PASSIVE SMOKING AND THE WORKPLACE

The role of passive smoking in the workplace has come into the spotlight following test cases in the UK which attempted to gain compensation for workers with asthma and lung cancer on the basis of occupational exposures to environmental tobacco smoke (ETS). The arguments are complex and can only be summarized here. There is evidence that in children, early exposure to ETS increases the risk of the development of asthma and bronchial reactivity. There is no such convincing data to support an inducing effect of ETS on asthma in adults. Despite a lack of epidemiological evidence supporting a role for ETS exposure in the induction of asthma or in the prevalence of lung cancer, a limited
number of awards appear to have been made. Judging from this background, it would seem unwise for current employers to allow continued exposure of employees to ETS, wherever practicable. In the USA, the state of California has taken these concerns to their limit by banning all smoking in bars and restaurants to protect staff from being exposed to ETS by the customers.

OCCUPATIONAL HEALTH, SMOKING AND ATOPY

There is indisputable evidence that smoking increases the risk of developing a number of occupational respiratory disorders. Primarily for this reason, but also for the general health of workers and subsequent economic gain obtained through smoking cessation, occupational health is a perfect vehicle for health education aimed at smokers. Occupational health should also ensure that a suitable no-smoking policy is instituted in the workplace with provision of specific smoking areas where deemed appropriate. Failure to prevent passive exposure to ETS in the workplace is a high risk strategy, which is likely to prove expensive in the long term. In certain circumstances, occupational health physicians may be asked to consider whether atopic or smoking individuals should be recommended as suitable for working in a particular environment, where there is an increased risk of the development of an occupational disease. There are major ethical issues involved in preventing atopic individuals from working with respiratory sensitizers. Analysis of data from one study of laboratory animal workers suggested that the exclusion of atopic workers from the workplace would have prevented seven cases of occupational asthma, but excluded 36 individuals from working who would not have been affected. It is therefore unreasonable to recommend this approach, but rather to reduce occupational exposures and introduce a careful health surveillance programme with early intervention in those who do develop respiratory disease. In addition, individual workers need to be made aware of the potential increased risk. Controversially, a 'no smoker' policy has succeeded with the cooperation of unions, management and occupational health in one group of platinum workers. However, platinum is the agent with the most convincing evidence of the effect of smoking on the subsequent development of occupational asthma and extrapolation of this policy to all respiratory sensitizers would be inappropriate. To exclude all atotics (~ 40% of the general population) and/or current smokers (up to 60% in some social class 3 populations from which employees may be drawn), would exclude the majority of the working population from working with respiratory sensitizers.

Atopic disease presents additional difficulties because of the added problem of making the diagnosis of occupational sensitization in an individual with pre-existing respiratory disease. It is probably prudent to preclude asthmatic individuals at pre-employment screening from working with potent respiratory sensitizers, and they should be carefully monitored where there is potential exposure to respiratory irritants.

In summary, smoking and atopy are important in the workplace in many situations. The role of the occupational health team should be to determine the importance of these factors in specific work environments.

REFERENCES