Is smoking cessation stressed enough in the prevention of occupational allergy?

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ABSTRACT
Smoking inconsistently increases the risk of sensitisation to occupational allergens, but is important in some settings, usually when exposure is to high molecular weight agents. The intensity of exposure and atopy may modify the risk of sensitisation in smokers. This article presents findings on the effect of smoking on sensitisation to occupational allergens from four South African occupational allergy studies. Methods: data were reviewed from surveys in a soybean plant (N = 115 workers), a maize processing plant (N = 74), a fish processing plant (N = 513) and a pine sawmill (N = 96). Smoking history was ascertained by questionnaire and atopic status and occupational sensitisation by skin prick tests (positive test ≥ 3 mm > negative control; atopic = positive to at least 1 common aeroallergen). Results: smoking workers were relatively large parts of the workforces (14–22%) and invariably had the highest proportion of sensitised subjects. The population attributable fraction for sensitisation due to smoking ranged from 1.3% (pine) to 42% (fish) and in atopic workers from 6.5% (pine) to 75% (soybean).

Conclusions: the smoking effect was inconsistent but substantial in some settings. Reducing allergen exposure and smoking may prevent sensitisation in these settings.

INTRODUCTION
A simple model of sensitisation to occupational allergens is that some exposed individuals will respond immunologically to the allergens by producing antibodies, typically IgE. These individuals are said to be sensitised and a proportion of them will go on to develop allergic diseases if exposure continues. Secondary agents may act as adjuvant exposures and increase the risk of sensitisation; among others chlorine, diesel exhaust emissions and cigarette smoke have been implicated. Whether a person is sensitised or not can be assessed directly by measuring the levels of specific IgE in serum – for example, a radioallergosorbent test (RAST) – or indirectly by skin prick tests. Skin prick tests involve pricking into the epidermis through a droplet of allergen in solution and observing the skin reaction 10 or 15 minutes later. A positive test is usually one in which the maximum dimension of the skin reaction is at least 3 mm larger than the negative control, a test solution containing an inert material.

Current understanding is that smoking is an inconsistent risk factor for sensitisation to occupational allergens1. For example, workers exposed to snow crab processing2, laboratory animals3, ispagula4, tetrachloropthalic anhydride (TCPA)5 and hexachloroplatinate6 have been shown to be at increased risk. Atopic smokers may be at the highest risk of sensitisation to some agents1.

The strength of the increased risk of sensitisation due to smoking varies by agent. For example, one of the first studies to show a smoking effect was on laboratory animal allergy3. The authors used four measures of laboratory animal allergy (LAA), namely LAA chest symptoms, any LAA symptoms, a positive skin prick test to urine extract and a positive RAST to urine extract. Smokers were 2.6 times more likely than non-smokers to have LAA chest symptoms, but only 1.3 and 1.4 times more likely to have any LAA symptoms or a positive skin prick test respectively. Smokers and non-smokers were equally likely to have a positive RAST. Smoking is more convincingly a risk factor for platinum salt sensitisation. In 1995, the National Centre for Occupational Health (now the National Institute for Occupational Health, NIOH) and others
published a paper on the effects of smoking and exposure intensity in platinum refinery workers. Platinum salts are powerful sensitisers and quite a high proportion of exposed individuals get platinum salt-induced asthma. The group studied was ostensibly non-atopic, since ‘atopics’ had been excluded at pre-employment. Smokers were at 8 times greater risk of sensitisation than non-smokers, when adjusted for exposure intensity. What is also important is that all 16 non-smoking, low exposed workers remained unsensitised.

In this article we examined the associations between smoking and sensitisation in four South African factories to understand better the magnitude of the possible smoking effect in these settings.

**METHODS**

Smoking and sensitisation data from four already completed factory surveys were reviewed. The factories were a fish plant near Cape Town, a maize mill in Johannesburg, a pine sawmill near Sabie and a soybean mill in Potchefstroom. These four workplaces were selected because the studies were done at about the same time and used similar methodology: smoking was ascertained by questionnaire; sensitisation by skin prick tests – using in-house allergens; and atopy was determined using the generally applied criterion of a positive skin prick test to one of a battery of common environmental aeroallergens. In-house allergens means that solutions for skin prick testing were prepared in a local laboratory using material collected from the factory. A positive skin prick test was a reaction 3 mm > negative control and the reactions were read by experienced practitioners.

For data analysis, smoking was considered to be a binary variable (smoker or non-smoker) and all factory workers were considered exposed (i.e. no account was taken of varying exposure intensities).

The effect of smoking on sensitisation was estimated using prevalence ratios (PR) – the prevalence of sensitisation in the smokers divided by the prevalence of sensitisation in the non-smokers – and their 95% confidence intervals. The fraction of all cases (of sensitisation) that would not have occurred if no one smoked was taken to be the population attributable fraction (AFp) which was calculated as AFp = (PR-1) x smoking proportion/(PR-1) x smoking proportion + 1.

**RESULTS**

Table 1 shows the four factories by the number of participating workers, their smoking status and the proportion of smoking atopic workers. Most of the 798 workers were from the fish plant and smoking was particularly common in this workplace at 58%. Atopic smokers were not uncommon, ranging from 14% to 22%.

As can be seen in Table 2 the percentage of sensitised workers was high except in the fish plant (6.4%) and in all sites a greater proportion of smokers than non-smokers were sensitised,
although this excess was very small in the pine sawmill and not evident in the table due to rounding-off.

The effect of smoking on sensitisation is shown in Table 3 for all workers and separately for atopic subjects. Smoking was an inconsistent risk factor, being negligible in the pine sawmill and not inconsiderable in the fish plant: 42% of cases of sensitisation might not have occurred had no one smoked in this workplace. Overall, though, in the four factories together the smoking effect was weak and not statistically significant with a PR of 1.1 (95% CI 0.8–1.6). The effect was stronger in the atopic group alone and this was particularly evident in the soybean plant where the population attributable fraction was 75%.

**DISCUSSION**

The inconsistency of the effect of smoking on sensitisation was confirmed by this review in four factories: prevalence ratios varied from 1.0 to 2.3 and the attributable fraction from 1.3% to 42% – in the pine sawmill and fish plant respectively. The association between smoking and sensitisation was, therefore, not convincingly shown in the main. The smoking effect was slightly stronger in atopic workers, and sensitisation may have been prevented to a substantial extent in atopic fish plant workers (31%) and soybean workers (75%). In interpreting these findings, though, it is important to appreciate that the relationships may not be causal.

Of course there are many limitations that should be borne in mind when considering these findings. First, these were cross-sectional surveys with all that this entails. For example, it may be that smoking merely shortens the time to sensitisation rather than actually increasing the proportion of those who become sensitised. If this is true it may not be evident in a cross-sectional survey. Second, smoking was a dichotomous variable and all subjects were considered exposed. But heavily exposed heavy smokers may be at very high risk of sensitisation and the findings of this paper would thus have underestimated the effect in this group. Third, since the smoking effect is agent dependent, the kind of workplaces selected for study will greatly influence the strength of the effect so the data presented here are not necessarily generalisable to other settings.

Nevertheless, these findings are of interest because occupational allergens are found in many workplaces and smoking is common so many occupational health practitioners need to decide what to do about smokers and exposure to allergens at work.

The data presented here may be interpreted to suggest that a reasonable decision is to do nothing except encourage smoking cessation as one would for the unexposed working population. After all, the smoking effect was small overall with a population attributable fraction of only about 5% (and 15% in atopic subjects). This modest effect together with the fact that smoking is notoriously difficult to remedy may make one hesitant to invest a lot of resources and energy in the matter. Added to these considerations is the high proportion of smokers in many communities from which workers are recruited; up to 60% in some areas if the fish plant’s smokers are a reflection of the habit in the community providing its workforce. Excluding smokers from allergen exposed jobs in these settings means excluding the majority, which is problematic both in terms of the recruitment difficulties exclusion would create and the consequent legal and ethical considerations.

But the decision not to act may not be defensible when one considers the strong agent specific effect. In some settings studied in this review, if the relationship is causal, sensitisation may have been prevented in more than 40% of smokers and 75% of atopic smokers. This is important: sensitisation has profound consequences for individuals and for enterprises. It is usual practice for occupational medicine practitioners to advise sensitised workers to avoid further exposure and thus stop the progression to allergic disease, and, of course, if sensitisation is accompanied by allergic disease then allergen avoidance is necessary to manage the condition. In many workplaces avoiding exposure is more often than not a euphemism for retrenchment and unemployment, and even when it is not a change in profession or trade may occur with financial loss. Enterprises lose skills and incur both financial costs and costs associated with adverse industrial relations arising from occupational diseases.
Where occupational allergens are present in the work environment occupational health practitioners need to consider something more intensive than the usual workplace based programmes against smoking. An approach is first to determine the importance of smoking in the specific work environment; and second, if the risk of sensitisation is increased convincingly (e.g. research findings for that particular workplace agent are consistent), to introduce measures to discourage potential new recruits who smoke (e.g. through pre-employment counselling and a no smoking policy at work) and to vigorously support already employed smokers in smoking cessation, including ‘free’ medication (e.g. nicotine patches). This approach is considered to be more positive than simply deciding not to employ smokers or to insist that all currently employed smokers give up by a deadline, as both of these measures may be considered extreme, especially considering the weak and inconsistent findings, and the limitations of cross-sectional studies.

Of course, the best way to avoid sensitisation is to reduce allergen exposure and this strategy should be at the forefront of any programme to preserve worker health.

REFERENCES

### Table 3. The effect of smoking on sensitisation: Prevalence ratios and population attributable fractions in four factories for all workers and for atopic workers separately.

<table>
<thead>
<tr>
<th></th>
<th>All workers</th>
<th>Atopic workers</th>
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<tbody>
<tr>
<td></td>
<td>PR</td>
<td>95%CI</td>
</tr>
<tr>
<td>Pine sawmill</td>
<td>1.0</td>
<td>0.5–2.2</td>
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<tr>
<td>Maize mill</td>
<td>1.7</td>
<td>1.0–3.0</td>
</tr>
<tr>
<td>Soybean plant</td>
<td>1.7</td>
<td>1.0–2.8</td>
</tr>
<tr>
<td>Fish plant</td>
<td>2.3</td>
<td>1.0–4.9</td>
</tr>
<tr>
<td>Total</td>
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<td>0.8–1.6</td>
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