Occupational asthma due to tea dust

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Abstract
Three patients are described who had developed asthma after working as tea packers. In two cases the diagnosis was confirmed by serial monitoring of peak expiratory flow rates, bronchial responsiveness to histamine, and specific inhalation challenges in the laboratory. The third patient experienced isolated changes in bronchial responsiveness to histamine after periods of exposure at work and after specific inhalation challenges in the laboratory without showing spirometric changes. Two of the three subjects were non-atopic; none had an immediate reaction to skin prick testing with a tea solution.

Occupational asthma among workers processing different plants, grains, and beans has been frequently documented. Occupational asthma due to tea dust, a dust released during the processing of tea leaves, has been described and asthma due to tea dust has been documented in a tea packer. We describe three patients who developed occupational asthma as a reaction to tea dust while working in a plant where tea dust was packed into tea bags.

Case reports
Baseline data for all the patients are given in the table. Histamine challenge tests were carried out as described elsewhere.

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (y)</th>
<th>Atopy</th>
<th>Skin prick reaction to tea dust</th>
<th>Smoking habit</th>
<th>Duration of exposure (y)</th>
<th>Duration of symptoms (y)</th>
<th>FEV₁ (mg/ml)</th>
<th>FEV₁/FVC (mg/ml)</th>
<th>PC₂₀ (mg/ml)</th>
<th>Specific IgE to tea dust (cpm)/ND</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43</td>
<td>—</td>
<td>Ex-smoker</td>
<td>Smoking habit</td>
<td>12</td>
<td>4</td>
<td>100</td>
<td>83</td>
<td>125</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>38</td>
<td>—</td>
<td>Ex-smoker</td>
<td>Smoking habit</td>
<td>12</td>
<td>4</td>
<td>100</td>
<td>83</td>
<td>125</td>
<td>ND</td>
</tr>
<tr>
<td>3</td>
<td>40</td>
<td>+</td>
<td>Non-smoker</td>
<td>Smoking habit</td>
<td>12</td>
<td>4</td>
<td>100</td>
<td>83</td>
<td>125</td>
<td>ND</td>
</tr>
</tbody>
</table>

*Mean value of 382 cpm in three non-atopic, non-asthmatic individuals. ND—not done.
occasionally at night. They improved during weekends and holidays. She was treated with oxtriphylline and an inhaled beta₂ adrenergic agent. The diagnosis of asthma was confirmed two years after the start of symptoms, when spirometry showed partially reversible airway obstruction with an FEV₁ and forced vital capacity (FVC) of 1.35 and 2.02 l, improving to 1.74 and 2.68 l after inhaled beta₂ adrenergic agent (predicted values 2.51 and 2.94 l). Her chest symptoms steadily worsened over several years, so she left her job one year before being seen. She was completely symptom free after several months away from work. She underwent specific inhalation challenges as for subject 1. Neither exposure to tea dust for 60 minutes as described above nor inhalation of a tea dust solution for five and 30 minutes via a Wright nebuliser (output 0.14 ml/min) on three consecutive days induced a change in FEV₁ of 8% or more over eight hours (fig 3), though she developed cough and shortness of breath. Her PC₂₀ fell on each occasion; at this time the maximum variability in baseline FEV₁ was 10-9% The diagnosis was not retained because FEV₁ did not change after the challenge test; no explanation was found for the changes in bronchial responsiveness.

Two years later, after a diagnosis of occupational asthma had been confirmed in another worker from the same processing plant (No 3 below), we decided to repeat the tea dust exposure tests at work and in the laboratory for longer periods. The subject was still symptom free and had no bronchial hyperresponsiveness. Returning to work caused her to cough but was not associated with any appreciable changes in peak expiratory flow (PEF). Her PC₂₀ fell after two periods of five days at work (fig 3), but had risen to over 32 mg/ml one week later. Specific inhalation challenges were then repeated in the laboratory. Baseline PC₂₀ was over 16 mg/ml. Exposure to tea dust for four hours resulted in a fall in PC₂₀ from over 32 to 3.8 mg/ml, with progressive recovery over the next six days. The maximum variability in FEV₁ during the day was 7.1%. After exposure to wood dust for four hours the maximum variability in FEV₁, was 8.6% and PC₂₀ was over 16 mg/ml. Repeat exposure to tea dust for four hours did not result in any change of note in FEV₁ (maximum daily variability 4.3%).

There was also no change in lung volumes (residual volume, functional residual capacity, total lung capacity) or maximum flow at 50% of forced vital capacity. There was, however, a fall in PC₂₀ with progressive recovery over the next few days at a time when the maximum variability in baseline FEV₁ before each histamine test was 10.6% (fig 3). The subject had cough and shortness of breath at the time of exposure to tea dust but not with wood dust.

Figure 1 Combined monitoring of peak expiratory flow (PEF) and responsiveness to histamine (PC₂₀) in subject 1. Significant changes in PEF were documented in the second week of exposure, coinciding with a need for an inhaled beta₂ adrenergic agent (S). FEV₁ values at the time of PC₂₀ assessment are given.

An asthmatic subject with a PC₂₀ of 1.4 mg/ml was exposed to tea dust for two hours in the laboratory as a control; the exposure, which was similar to the one for the other subjects, did not induce any changes in FEV₁ (<10%) or in PC₂₀ (2.1 mg/ml at the end of the day).

Figure 2 Results of specific inhalation challenges with wood dust (two hours, O) and tea dust (30 minutes, S) in subject 1. S—inhalation of a beta₂, adrenergic agent. Specific inhalation challenges were performed during 22 August–2 September.

SUBJECT 3
This 40 year old woman reported cough with chest tightness and wheezing that was more pronounced in the afternoon when she was at work. She also reported waking at night with asthmatic symptoms. There was improvement during weekends and holidays. She was atopic (positive skin prick test responses to ragweed pollen and Dermatophagoides farinae) but reported no history of hay fever. The PC₂₀ was 7.4 mg/ml (baseline FEV₁, 2.74 l) after two months away from work. After returning to work for two weeks her PC₂₀ had fallen to 1.5 mg/ml (baseline FEV₁, 2.56 l). PEF monitoring showed greater fluctuations when she was at work than during a weekend and a period before exposure at work (fig 4). Specific inhala-
Discussion

Two cases of occupational asthma due to tea dust have been already documented. Although in the first case the worker was employed in the primary industry, in the second case the worker was a tea packer like our subjects. In the second case the diagnosis was confirmed by serial monitoring of PEF and specific inhalation challenges, whereas in the first case the diagnosis was a clinical one. In our two workers the diagnosis of occupational asthma was confirmed by monitoring peak expiratory flow. As this does not exclude the possibility of an irritant reaction, we combined monitoring with serial assessment of bronchial responsiveness. Substantial changes in PC20 were documented in both subjects and were prolonged in the first subject. Finally, specific inhalation challenges confirmed the diagnosis of occupational asthma as they induced a late reaction in one subject and an atypical immediate or early late reaction in the other. Although the exposure level at the time of specific inhalation challenges was not monitored and could have been high at this time, this pattern of reaction excludes a non-specific irritant mechanism; asthmatic subjects exposed to high levels of particles such as sawdust do not generally show changes in spirometric values and bronchial responsiveness after exposure. Furthermore, a control asthmatic subject showed no changes in FEV1 or PC20 after a similar exposure to tea.

The second subject illustrates an interesting point. Although exposure to tea dust caused little change in FEV1 or PEF, there was a change in PC20 of up to four doubling doses, from normal to within the asthmatic range. Such changes cannot be attributed to an irritant reaction; the subject did not show bronchial hyperresponsiveness at the start of the challenges on three separate occasions and recovery of PC20 took several days. Finally, changes in PC20 were not documented in a control subject who had bronchial hyperresponsiveness. The effect of exposure to an environmental asthma inducing agent may at times be detected more readily from change in bronchial responsiveness than from changes in FEV1 or FVC. The lack of changes in FEV1 and/or FVC after exposure to tea dust might be due to the fact that the subject had been away from work for a long interval when the tests were carried out. She is likely to have lost some sensitisation. It is difficult to label this case occupational asthma, though she

![Figure 3](image1.png)

Figure 3  Results of specific inhalation challenges in subject 2. No significant changes in FEV1 were documented. The duration of exposure to each agent at work and in the laboratory is shown.

![Figure 4](image2.png)

Figure 4  Monitoring of peak expiratory flow (PEF) at work and away from work in subject 3. S—inhaled of a beta2 adrenergic agent.

![Figure 5](image3.png)

Figure 5  Results of specific inhalation challenges with wood dust (●) and tea dust (○) in subject 3. S—inhaled of a beta2 adrenergic agent.
definitely had asthma when she was working at the tea plant, as documented by her reversible airway obstruction. There is a similar case of a snow crab worker in whom the first specific challenge, done after several months away from work, gave negative results but who developed asthma again several weeks after returning to work; challenges at that time gave positive results. This has also been reported in a worker exposed to isocyanate. For us to prove the point definitively our subject would have had to return to work for several weeks or months.

That two of the three workers started having symptoms after stopping smoking is interesting. Smoking is inconsistently related to occupational asthma, but asthma that occurs or recurs after cessation of smoking has been described; the mechanism is unknown.

The mechanism of this type of occupational asthma is not understood. We and others were unable to detect specific IgE, and skin tests failed to elicit an immediate reaction. The causative agent could be the tea plant itself or a microbial contaminant, though immediate skin reactions were found in the patient in the original report. The prevalence of occupational asthma among workers exposed to tea dust remains to be explored.

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