Asthma due to Dust from Redwood 
(Sequoia sempervirens)*

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An atopic patient with adult onset of asthma due to sawdust from redwood (Sequoia sempervirens) is described. Specific late asthmatic reaction developed after challenge with inhalation of his own redwood sawdust. No subjective or objective immediate reactions were detected. Avoidance of exposure resulted in improvement. There were no pulmonary or systemic responses to challenge with inhalation of sawdust from pine wood.

Bronchial, as well as parenchymal, reactions to inhaled wood dusts have been described.1–7 The bronchial reactions have been categorized as (1) immediate in the case of exposure to oak,2 (2) late in exposures to cedar of Lebanon4 and iroko,3 or (3) combined (ie, "dual" immediate and late reactions) following exposure to western red cedar4 and mahogany.8 Asthma due to dust from redwood has not been reported until recently.3

In this report, we describe the case of an atopic man with a late type of asthmatic reaction to sawdust from redwood (Sequoia sempervirens).

CASE REPORT

A 67-year-old retired man had recurrent episodes of tightness in the chest, dyspnea, wheezing, and cough since May 1975. His initial episode, as well as subsequent ones, occurred late in the evenings, at night, or several hours after he had worked with redwood. The symptoms improved after a few days of avoiding exposure or after therapy with small doses of corticosteroids taken for a few days (or both). The patient remained free of symptoms if woodworking with redwood was avoided. Although carpentry had been his hobby for many years, sawdust from walnut, fir, pine, and oak did not induce asthma. In early 1975, the patient started to work with redwood. He had been a very light cigarette smoker until six years ago. There was no previous history of chronic cough, asthma, tightness of the chest, rhinitis, or dermatitis. There was no history of asthma, pulmonary disease, or allergic rhinitis in his family. The patient did not take aspirin. The findings from examination of the cardiovascular system were normal. The patient had a right nasal poly.

Intradermal cutaneous tests with purified protein derivative of tuberculin and histoplasmin were negative. There was a 10-mm reaction to antigen of Candida albicans. Prick tests with a saline solution of redwood sawdust and with common allergens (house dust, mixed insects, cottonseed, feathers, tree pollens, grass pollens, ragweed, Alternaria, Hormoden-

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424 GUILLERMO O. DO PICO

CHEST, 73: 3, MARCH, 1978

drum, dog hair, cat hair, and Aspergillus fumigatus) were negative. Serum precipitating antibodies against saline extracts of cedar, redwood, hickory, and the patient's own redwood dust were not detected by gel immunodiffusion.9 Serum precipitin reactions to Aspergillus fumigatus, A clavatus, A niger, A terreus, Penicillium rubrum and casei, Microspora faeni, Thermoactinomyces vulgaris, house dust, Alternaria, Hormodendrum, Fusarium, Trichoderma, Cephalosporium, Candida albicans, Aerobasidium, Phoma, and pigeon serum were also negative using concentrations of antigen of 10 mg nondialyzable solids per milliliter.10

To confirm the specificity of the etiologic agent identified by history and to evaluate the type and temporal course of the pulmonary reaction to inhalation of redwood dust, provocation tests were performed. The patient was exposed to airborne wood dust produced by tipping and shaking the dust repeatedly from one container to another, placed between 30.5 to 61 cm below his face, for periods of 25 minutes in a small cubicle. On three separate occasions preceded by asymptomatic periods, the patient was exposed to his own redwood sawdust twice and to his own sawdust from pine wood once. Before the challenge and at the regular time periods following challenge, the patient was evaluated by the following: presence of symptoms; auscultation of the lungs; measurement of forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) on a 13.5-L spirometer (Collins) by standard technique; maximal instantaneous forced expiratory flow after 50 percent of the FVC has been exhaled (FEF50%) on a spirometer (wedge); diffusing capacity for carbon monoxide by the single-breath method (Deb); residual volume (RV) and total lung capacity (TLC) calculated from the single-breath helium dilution; arterial oxygen and carbon dioxide tensions (PaO2 and PaCO2); total white blood cell count (WBC), with differential cell analysis; and body temperature. A chest x-ray film and measurement of the third component of the complement system (C3) were obtained before and eight hours after the challenge. The larynx and vocal cords were examined by indirect laryngoscopy.

After the first study using provocation with redwood sawdust, there was no immediate subjective or objective bronchial reaction, but approximately eight hours after the challenge, the patient developed rhinorrhea, cough, dyspnea, and wheezing that lasted four days.

For the second challenge with redwood sawdust, the patient was hospitalized and acclimatized to the new environment for two days prior to the test. The results are shown in Table 1 and Figure 1. Again, there were no immediate subjective or objective pulmonary or systemic reactions. However, eight hours after the challenge, there was a 22 percent decrease in FEV1, an 18 percent decrease in FVC, and a 29 percent increase in the ratio of RV/TLC from levels before challenge, indicating significant obstruction of airways, which is characteristic of asthma. Simultaneously, the patient developed rhinorrhea, cough, dyspnea, and wheezing. Expiratory rhonchi and wheezes were auscultated bilaterally. The oropharynx was erythematous, but the vocal cords were normal. The hypoxemia present before challenge worsened one hour after exposure. At that point, there were no signs of obstruction of airways. Four hours after challenge, an 11 percent reduction in the Deb and a slight increase in the WBC and the percentage of eosinophils were found. There was no change in body temperature Chest x-ray films and the level of C3 remained normal after challenge. There were no subjective or objective immediate or late changes following exposure to pine sawdust (Fig 1).
Table 1—Results of Provocation Test with Inhalation of Redwood Sawdust

<table>
<thead>
<tr>
<th>Time</th>
<th>FEV&lt;sub&gt;1&lt;/sub&gt;</th>
<th>FVC</th>
<th>FEV&lt;sub&gt;1&lt;/sub&gt;/FVC</th>
<th>FEF50%</th>
<th>RV/TLC</th>
<th>Dsb, ml/min/mm Hg</th>
<th>PaO&lt;sub&gt;2&lt;/sub&gt;, mm Hg</th>
<th>WBC, cells cu mm</th>
<th>Eosinophils, percent</th>
<th>Temperature</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>3,490</td>
<td>5,896</td>
<td>62.4</td>
<td>2.26</td>
<td>36.2</td>
<td>31.0</td>
<td>73</td>
<td>8,700</td>
<td>11</td>
<td>36.3°C</td>
<td>Asymptomatic; lungs clear</td>
</tr>
<tr>
<td>After</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 min</td>
<td>3,400</td>
<td></td>
<td>64.5</td>
<td>2.03*</td>
<td>36.3</td>
<td>31.4</td>
<td>66</td>
<td>9,200</td>
<td>10</td>
<td>36.4°C</td>
<td>No change</td>
</tr>
<tr>
<td>1 hr</td>
<td>3,847</td>
<td>5,837</td>
<td>65.9</td>
<td>2.13</td>
<td>35.0</td>
<td>33.0</td>
<td>80</td>
<td>10,200</td>
<td>13</td>
<td>36.6°C</td>
<td>No change</td>
</tr>
<tr>
<td>4 hr</td>
<td>3,756</td>
<td>5,799</td>
<td>64.8</td>
<td>2.86</td>
<td>36.6</td>
<td>27.7</td>
<td>80</td>
<td>10,200</td>
<td>13</td>
<td>36.6°C</td>
<td>No change</td>
</tr>
<tr>
<td>8 hr</td>
<td>2,734</td>
<td>4,590</td>
<td>59.6</td>
<td>46.7</td>
<td>29.2</td>
<td>79</td>
<td>8,600</td>
<td>11</td>
<td>36.8°C</td>
<td>Symptomatic wheezes</td>
<td></td>
</tr>
<tr>
<td>24 hr</td>
<td>2,624</td>
<td>4,418</td>
<td>59.4</td>
<td>46.4</td>
<td>33.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>36.4°C</td>
<td></td>
</tr>
</tbody>
</table>

*Similar values were obtained at 5 and 30 minutes after challenge.

**DISCUSSION**

The etiologic agents causing nonseasonal asthma of adult onset are usually difficult to identify, particularly when the asthmatic reactions occur several hours after exposure. The patient had no immediate asthmatic reaction following the provocation test with redwood sawdust but did have a severe prolonged late reaction. Two patients recently studied had specific immediate and late asthmatic reactions; like this patient, they showed no cutaneous reactivity to common allergens or redwood extract, as well as no detectable serum precipitating antibodies against redwood.

The lack of elevation of body temperature, the lack of auscultatory or radiographic findings of diffuse interstitial pulmonary disease, and the lack of persistence of the slight, probably not significant changes in the Dsb and WBC do not suggest the presence of alveolitis in response to the redwood sawdust. The hypoxemia found one hour after exposure is difficult to explain. At that point, there were no signs of obstruction of large or small airways; actually, the FEV<sub>1</sub> had risen from the level before challenge, and the FEF50% and Dsb had not changed.

The incidence of pulmonary reactions to redwood sawdust is unknown, but it is probably low, despite the large amounts of redwood handled in this country. Phenolic extractives (eg, sugiresinol, hydroxysugiresinol, and isosequiric acid) in the redwood may be responsible for the respiratory reactions. The mechanism by which redwood dust induces asthma is unknown.

**ACKNOWLEDGMENT:** I am grateful to Drs. D. Flaherty and C. Reed for the immunologic studies and to Mr. J. W. Rowe and Mr. R. C. Koeppen from the US Forest Products Laboratories, Madison, Wis., for their advice and identification of woods.

**REFERENCES**