Passive Smoking
Its Relationship to Respiratory Symptoms, Pulmonary Function and Nonspecific Bronchial Responsiveness

Considerable research interest has been focused on the health effects of involuntary smoking, particularly upon children. The majority of studies that have investigated the relationship between respiratory symptoms and illness in children and maternal or parental smoking have shown a positive association. This association seems strongest the younger the age of the children. Symptoms and illnesses in children related to passive smoking include chronic cough,1,2 wheezing,3 particularly episodic of pneumonia, bronchitis,4 or unspecified lower respiratory illness.5 6

The issue with regard to the effect of involuntary smoking on pulmonary function is less clear. Two cross-sectional studies have shown that maternal cigarette smoking has a negative effect upon the level of the child’s FEV1,7 and MMEF.2,8 However, another cross-sectional investigation failed to find any such relationship.9

The study of Ekwo and co-workers in this issue of Chest (see page 662), has demonstrated a relationship between parental smoking and hospitalization for respiratory illness before the age of two years and the occurrence of cough with colds. In the case of this latter association, no data are provided concerning the chronologic relationship between the age of the children and the excess symptom frequency. The authors’ failure to reference the occurrence of symptoms to specific ages of the children may have further diluted any potential associations. Studies in England10,11 suggest that the major effect of parental smoking on wheezing symptoms occurs during the first two years of life.

While Ekwo and colleagues found no relationship between status of pulmonary function and parental smoking, they conclude that there is a relationship between parental cigarette smoking and bronchial responsiveness as measured by isoproterenol inhalation. However, the actual differences in responsiveness observed between children of smoking and nonsmoking parents are very small, and when expressed as a percentage of the prebronchodilator value, are not statistically significant. Thus, it appears that this investigation does not support an effect of parental smoking on either level of lung function or bronchial responsiveness.

However, the interrelationships between lower respiratory illness, parental cigarette smoking and the atopic state are exceedingly complex. It is quite possible that a cross-sectional study of children in this age range, using bronchodilatation rather than bronchoconstriction as the index of airways responsiveness, is not sensitive enough to detect a true effect of parental smoking on bronchial responsiveness. Another possibility is that the effect of parental smoking on responsiveness is mediated through lower respiratory illness or atopy. Only prospective studies of very young children could detect this, if it were truly present.

It appears that parental cigarette smoking may contribute to respiratory symptom and illness morbidity in young children. The precise magnitude of this health risk on the severity and overall number of respiratory illnesses in children is unknown. Further studies in this area should be directed toward providing quantitative estimates of the effect of parental smoking on the incidence of respiratory morbidity in children.

There are other possible health consequences of passive smoke exposure in early life. One potential effect is for childhood events (of which passive smoking is only one) to be related to the occurrence of adult obstructive lung disease. The effect of passive smoking experienced in childhood on maximally attained lung function in early adulthood is likely to be small and difficult to detect in cross-sectional investigations. The importance of identifying and quantitating this effect lies in the possibility that even a modest reduction in pulmonary function may be an indicator of who is at risk for developing obstructive airways disease when exposed more directly to personal (eg, cigarette smoke) or environmental agents in adult life. This hypothesis is under active investigation at the present time.

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REFERENCES
1 Colley JRT, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. Lancet 1974; 2:1031-34

Table 1—Serum IgE Levels in Smokers and Nonsmokers

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Serum IgE (IU/ml)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Smokers</td>
<td>Nonsmokers</td>
</tr>
<tr>
<td>Gerrard et al</td>
<td>1980</td>
<td>72</td>
<td>44</td>
</tr>
<tr>
<td>Burrows et al</td>
<td>1981</td>
<td>25.5</td>
<td>15.9</td>
</tr>
<tr>
<td>Zetterström et al</td>
<td>1981</td>
<td>20.8</td>
<td>13.2</td>
</tr>
<tr>
<td>Leitch et al</td>
<td>1981</td>
<td>1.54</td>
<td>1.53</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.03*</td>
<td>0.02*</td>
</tr>
<tr>
<td>Warren et al</td>
<td>1982</td>
<td>34.4 (men)</td>
<td>19.6 (men)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>26.3 (women)</td>
<td>17.3 (women)</td>
</tr>
<tr>
<td>Bahna et al</td>
<td>1983</td>
<td>41.7</td>
<td>19.3</td>
</tr>
</tbody>
</table>

*Sputum/serum ratio

Interaction of Immunoglobulin E and Cigarette Smoke
Predisposition to Symptomatic Lung Disease?

The mention of immunoglobulin E (IgE) in association with lung disease usually brings to mind extrinsic asthma, bronchopulmonary aspergillosis or parasitic infestations. In these diseases, moderate or even markedly elevated serum IgE levels are usual. Several investigators over recent years have reported elevated serum IgE levels in smokers.1,4 In most of these studies, serum IgE levels in smokers are approximately 7 to 28 IU higher than in nonsmokers, although the absolute values of both groups are within normal range (Table 1). These differences are statistically significant and have led investigators to study other aspects of hypersensitivity in chronic respiratory diseases, especially obstructive airway disease. The most recent analysis of IgE patterns in cigarette smokers has shown a striking rise in serum IgE levels associated with light smoking and a remarkable drop in heavy smokers, changes which are reversible after cessation of smoking.8

In this issue of Chest, Burrows and coworkers (see page 657) have attempted to correlate immediate-type hypersensitivity parameters (IgE levels and prick tests) to parameters of airway obstruction (FEV1). The authors studied 1,182 subjects. Careful statistical analysis of the data showed that positive allergy prick tests are associated with higher rather than lower FEV1. Also, elevation of serum IgE is related to low FEV1 (less than 80 percent of predicted) along with symptomatic asthma or chronic bronchitis. This serum IgE cannot be related to usual aeroallergens, yet appears to be making a significant contribution to FEV1 impairment. Thus, in addition to confirming previous observations about elevated serum IgE in smokers, this article suggests a clinical correlation between elevation of serum IgE and the development of symptomatic lung disease. It is possible that smokers who develop pulmonary symptoms are in fact expressing a hypersensitivity reaction to a "mystery antigen" —perhaps a component of tobacco or smoke. Salvaggio and coworkers were unable to detect skin reactivity against tobacco leaf and smoke antigen. Clearly, further characterization and purification of cigarette antigen(s) is needed.

Burrows et al also raise the possibility of an IgE response to microorganisms inhabiting the respiratory tract of patients with chronic bronchitis and asthma. IgE levels in patients with far-advanced tuberculosis have been shown to be 29 IU, not significantly different from control subjects (18 IU).8 At least in tuberculosis, chronic bacterial infection does not raise serum IgE levels. This is in contrast to bronchopulmonary aspergillosis, in which the presence of Aspergillus fumigatus in the bronchial tree stimulates a tremendous IgE response of 5,000-20,000 ng/ml.8 Thus, the observed elevation of serum IgE levels in smokers is unexplained on the basis of chronic infection alone.

Perhaps interaction of the T and B cell immune systems is somehow responsible for the change in IgE levels in smokers. Hallgren et al demonstrated elevated IgE levels in male smokers with bronchial carcinoma. These authors postulated that smoking induces impaired cellular immunity which is reflected by enhanced IgE synthesis and a depressed resistance.

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