Environmental Tobacco Smoke and Asthma

The article by Frischer et al on maternal smoking and peak flow variability in this issue of Chest (see page 1133) is notable both for its subject matter and its methods. With regard to the methods, there is an increased interest in the use of peak flow in both asthma research and care. Peak flow has certain disadvantages. Peak flow is primarily an index of large airway flow. Its major determinants are large airway size, expiratory respiratory muscle strength, and lung and chest wall recoil. It is more variable and less reproducible than the FEV₁. It lacks sensitivity in detecting small airway obstruction or mild disease. Recently, interest has been focused on using peak flow variability, expressed in a variety of ways, as an index of bronchial responsiveness. Although there is a reasonably good correlation between these two indices, their relationship may be complementary. Additionally, peak flow lacks specificity, identifying only a subset of responsive subjects. Despite these difficulties and uncertainties, the results of Frischer et al are consistent with those of Martinez et al, who found an association between bronchial responsiveness to carbachol and parental smoking in 9-year-old boys. The ability of Frischer and coworkers to demonstrate significant correlations of peak flow variability with maternal smoking is likely to stimulate further research on peak flow monitoring as an important area of asthma investigation.

With regard to results, the association of maternal cigarette smoking with peak flow variability demonstrated by Frischer et al highlights the importance of maternal cigarette smoking in relation to asthma incidence and severity.

It has been well known that maternal cigarette smoking aggravates asthma symptoms and bronchial responsiveness in patients with an established diagnosis of the disease. This increase in clinical and physiologic measures of severity is correlated with an increased use of health services, notably emergency room visits, and a decrease in asthma severity with maternal smoking cessation. Thus, there is no question that maternal cigarette smoking is an important exacerbating factor in established disease.

Three things remain unclear: Does maternal cigarette smoking influence the development of asthma? What is the relative importance of pre- versus postnatal exposure? What is the mechanism by which maternal smoking exerts its effects?

In considering mechanisms and susceptible subgroups, the data are conflicting, at best. At least two studies have linked maternal cigarette smoking to skin-test reactivity in older children. Neither study considered the relative importance of pre- versus postnatal exposure. Other studies have failed to confirm the association of maternal smoking and skin-test reactivity. Frischer and coworkers support a non-atopic mechanism, as their positive association was confined to nonatopic subjects. They note, however, that lack of an association in the atopic subjects might possibly be due to smoking cessation among atopic families. A summary of all existing data suggests that the conflicting results with regard to atopy may be explained by lack of large longitudinal studies that clearly establish the prenatal and postnatal exposure status of the child, measure the relevant covariates, and follow the cohort longitudinally from birth both to eliminate selection away from smoking in the highly susceptible and to allow for a determination of the relative importance of pre- versus postnatal exposure.
Such studies take on added importance because there is a growing body of evidence suggesting that maternal cigarette smoking, either in utero or during the first year of life, is associated with the development of asthma. At least three studies have documented that maternal cigarette smoking is associated with the onset of asthma and infantile eczema during the first year of life.\textsuperscript{17-19} Weitzman and coworkers\textsuperscript{17} performed a cross-sectional analysis in which they examined the relationship of maternal cigarette smoking to asthma incidence during the first year of life using data from a national health interview survey. The odds of developing asthma in infants of smoking mothers was 2.0 relative to infants of nonsmoking mothers. The influence of family history of allergy, lower respiratory tract illness, and diet was not examined. Martinez and coworkers,\textsuperscript{18} utilizing data from the Tucson Epidemiologic Study, found a similar estimate for the effect of maternal cigarette smoke exposure and its influence on asthma during the first 2 years of life. In that study, the effect seemed to predominate in women of low socioeconomic status, as indicated by less than a high school education. Again, the relative importance of pre-versus postnatal exposure and the effect of other putative risk factors, mainly allergy and lower respiratory tract illness, were not examined. In a randomized control trial of allergen avoidance and its influence on allergic disorders of infancy, parental cigarette smoking was found to be a risk factor for the development of allergy, whether only one parent or both parents smoked. The magnitude of the effect was equal to the effect of allergen exposure in this study.\textsuperscript{19} Although this study independently controlled for allergen exposure and diet, the relative importance of pre-versus postnatal exposure to environmental tobacco smoke was not examined.

In summary, there is a growing body of data that suggests that maternal cigarette smoking may be an important factor in the development of childhood asthma. Ecologic data support this hypothesis. The gradual, but significant, increase in childhood asthma prevalence and hospitalization in the United States coincides with a doubling of cigarette smoking rates among women of child-bearing age in this country. Roughly 50 percent of all children under the age of 5 years are exposed to cigarette smoke either pre- or postnatally.\textsuperscript{20} We need to determine the role of maternal smoking in the onset of childhood asthma, and further research is clearly needed to accomplish this goal.

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