acted using the then-available allergen, mesquite to which 16.2% reacted, and bermuda grass, which produced the largest proportion of reactors (32.2%). Nonasthmatics who reacted to house dust showed 1.9 times more PEF-BR than similar skin test-negative subjects between 15 and 54 years (p<0.05). Similarly, reactors to mesquite (a local allergen) showed more BR (24% vs 37%, p<0.10). Further, analyses show similar trends for dog allergen. They were positive but not significant for cat allergen. Bermuda grass reactivity was not related to BR, even though it is the most frequent positive skin test.

Prevalence rates of asthma, allergy, and BR were related also to environmental tobacco smoke particulate matter (PM) sizes 10 ΜΜ and 2.5 μM (PM10 and PM2.5), nitrogen dioxide (NO2), and formaldehyde. NO2 responses appear independent of the other responses and the formaldehyde response appears related to ETS particles.

Acute pulmonary responses to allergens and pollutants in susceptible and other subjects included PEF-related BR and symptoms. These were related to specific exposures temporally and spatially, especially in asthmatic children. Quantitative relationships between acute responses and several allergens, especially in asthmatics and those with BR, have been demonstrated.

In contrast to characterized “normals,” those classified as “atopics” and “peak flow responsive” (PEFR-BR) subjects showed increased nasal symptom responses with increased pollen (eg, ragweed and mulberry) and some mold concentrations. We think that one can differentiate these effects by assessing individuals who are allergic to specific antigens. Decline in lung function, as measured by the evening PEF, was associated with high concentrations of some pollen types (eg, mulberry), but only for individuals defined as “peak flow responsive.”

Discussion

The quantitative skin test-BR relationships shown in this population are comparable to those determined in collaborative studies in Italy and New Zealand. That is, methacholine BR responses using the provocative concentration causing a fall in FEV1 of 15 or 20%. (PCFEV) are quantitatively related to some, often the same, immunomarkers in nonlinear increasing relationships within the age groups tested (unpublished); PEF-BR had the same relationships to the specific immunomarkers of exposures as methacholine response, including with IgE, but PEF-BR relations plateau within the range tested (ie, has a different nonlinear relationship), especially with IgE. IgE usually disappears in multiple regression models relating BR to skin tests and relating acute respiratory responses to allergens and air pollutants. We are quite surprised that analyses to date show that acute allergen-related responses do not appear to interact with irritants in the induction of greater respiratory responses. Acute responses to air pollutants (eg, PM, ozone [O3], NO2) and allergens continue to be evaluated for further understanding of the respiratory response.

The process that starts with exposure to an allergen and its inhalation leads to immunological changes that are measured by the immunomarkers. This response is based on the interactions of the inhaled allergen and the host immune system characteristics. The health outcome can be acute, even reversible, or lead to chronic changes and frank clinical disease, based on the characteristics of the exposure and the host.1-3 Even the immunologic status and BR themselves become pathologic conditions affecting long-term function.12

In summary, immunologic sensitization, as indicated by skin tests as immunomarkers, is important in the acute and chronic pulmonary responses to allergens and to pollutants.

References


Wood Smoke Exposure and Risk for Obstructive Airways Disease Among Women*

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Exposure to firewood, the most common biomass fuel used for cooking and heating in the developing world, was investigated as a potential risk factor for obstructive

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airways disease (OAD) among women in Bogota, Colombia, in whom cigarette smoking and other known risk factors may not be the most frequent factors encountered.

A hospital-based case-control study was conducted to identify risk factors for OAD among women in Bogota. An interview was conducted using a modified questionnaire recommended by the American Thoracic Society for epidemiologic studies. We compared 104 OAD cases with 104 control subjects matched by hospital (three) and frequency matched by age. The odds ratio (OR) was used as the basic statistic to evaluate risk. Multivariate analysis was conducted by Mantel-Haenszel stratification procedures and by logistic regression analysis.

Univariate analysis showed that tobacco use (OR: 2.2.2, p<0.01), wood use for cooking (OR: 3.43, p<0.001), passive smoking (OR: 2.05, p<0.01), and gasoline use for cooking (OR: 0.52, p<0.02) were associated with OAD. Trends for years of tobacco use and years of wood cooking were present (p<0.05). With multivariate analysis, variables remained significant except gasoline use.

This study showed that among elderly women of low socioeconomic status in Bogota, wood smoke exposure was associated with the development of OAD, and the population attributable risk may be about 50% of all OAD cases. The role of passive smoking remains to be clarified. This work may set the basis for interventional studies in similar settings.

Occupational Asthma in a Community-Based Survey of Adult Asthma*

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There are few studies providing systematic estimates of the contribution of occupational factors to the prevalence of asthma among adults. Most investigations of asthma are work site or industry specific, identifying particular risk factors for occupational asthma (OA) without providing an overall measure of the population-attributable risk across a full spectrum of exposures. In an earlier secondary analysis of data from a national population survey of persons at risk of disability, we estimated that up to 15% of adults with asthma may have an occupational component to their disease.

We wished to utilize an ongoing study of disability as another approach to estimating the prevalence of OA. Because the data we analyzed are derived from a community-based panel of persons with physician-diagnosed asthma not selected for etiology, this study provides the opportunity to examine potential work-related asthma across a variety of exposures and occupations.

We analyzed cross-sectional data from an ongoing patient panel study, the focus of which is the identification of risk factors for disability among adults with asthma. As such, the panel study is not an investigation of OA per se, nor do all of the subjects in the panel have a history of labor force participation.

The design of the panel was as follows: 18- to 50-year-old adults with a diagnosis of asthma were recruited from patient visit logs (follow-up or new patient visits) maintained by pulmonologists (n=56) or allergist internists (n=18) sampled at random from among all such specialists practicing in northern California. Study eligibility was not limited on the basis of asthma etiology, age of asthma onset, severity of condition, or work history. We studied 601 patient subjects by structured telephone interview, ascertaining demographics, symptoms, and medical and employment histories. Included in the interview schedule were a series of questions identifying specific job-related exposures selected a priori because of their association with OA in previous studies. Subjects were not asked whether they attributed the cause of their asthma to their work.

We identified all subjects with first onset of any asthma in adulthood (age ≥18 years) who were employed at the time of onset. We then defined probable OA by two principal approaches. The first was based on reported job exposures. We took as the most conservative measure of OA risk, reported exposure to at least one of the following specific substances known as sensitizing agents for classical OA: grain or flour dust; epoxies or urethane; cotton dust; wood dust; animal fur or dander; or pharmaceuticals. We also used an expanded list that would also take into account exposures associated with irritant-induced asthma, such as irritant gases, paints, and sealants, or fire smoke. This definition of probable OA is consistent with the surveillance definition of OA employed by the National Institute for Occupational Safety and Health in its SENSOR program.

Our second approach in defining OA was not based on subject report of exposure, but rather on an asthma-specific job exposure matrix for occupations at risk of OA. This scoring matrix was originally developed for the California Department of Health Services Occupational Health Program. We used the matrix to identify those persons at highest job risk among those working at the time of adult asthma onset. In the matrix, lower score reflects higher job-related asthma sensitization risk, with scores ranging from 1.5 to 4.

Of the 601 subjects in the panel, 551 (92%) had a history of labor force participation. Of these, 288 (52%) had adult-onset asthma and 255 (46%) were employed at that time. There were 53 persons who reported high-sensitizer-risk exposures. Of the entire group of 601, persons meeting this conservative OA definition accounted for 8.8% (95% confidence interval [CI] 6.5 to 11.1%). When the exposure list was expanded to include irritants, the number of subjects with probable OA increased to 79, comprising 13.1% of the study population (95% CI, 10.4 to 15.8%). The job exposure matrix score was highly skewed (median=3.7; interquartile range, 2.4 to 3.8), demonstrating a distinct bimodal distribution with a clear high-risk job score group (n=85). These persons accounted for 14.1% of the entire study group of 601 (95% CI, 11.3 to 16.9). Limiting the definition of OA by re-

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