* Project SENSOR News

Volume 9, No. 3 Summer 1998

WHAT CAN WORK-RELATED ASTHMA TEACH US ABOUT ASTHMA IN GENERAL?

Work-related asthma provides the opportunity to learn more about asthma in general. With work-related asthma one can define the onset of exposure, identify a cohort of exposed individuals, and observe the natural history of the disease once the exposure has ceased. The occurrence of work-related asthma among adults in contrast to the study of asthma among children lends itself to the possibility of studies involving blood or even more invasive tissue samples.

What general principles of work-related asthma are potentially applicable to all patients with asthma?

TIMING OF RESPONSE

The risk of developing work-related asthma is highest within the first two years of exposure.

In the workup of a patient with new onset asthma the first encounter with the potential allergen is most likely to be caused by an environmental exposure that has occurred within the past two years.

EXPOSURE LEVELS

The best predictor of who develops work-related asthma is the level of exposure to an allergen. Workers with the highest exposures have a higher incidence of sensitization, symptoms and diagnosed work-related asthma.

Studies on exposure to dust mites and other allergens present in the home have shown a similar relationship between concentration and disease. Studies in the work setting should allow for a better understanding of the importance of continuous versus intermittent exposures.

CIGARETTES

Cigarette smoking increases the incidence of IgE sensitization and symptoms with exposure to high molecular weight organic substances such as among snow-crab processors and laboratory animal workers. For low molecular weight chemical substances such as the isocyanates where IgE does not appear to play a major role, cigarette smoking does not increase the incidence of work-related asthma.

It has been hypothesized that cigarette smoke disrupts the bronchial epithelium allowing greater access of the allergen to antigen-recognizing cells.

ATOPY

Individuals with positive skin tests to environmental allergens are more likely to develop work-related asthma from high molecular weight organic compounds but not low molecular weight chemical

1997 ANNUAL REPORTS AND NEW WEBSITE

Recently we mailed a cover letter and postage paid postcard to you, announcing the availability of the 1997 Annual Reports on Work-Related Asthma, Silicosis, Occupational Noise-Induced Hearing Loss and Occupational Diseases. We received over 300 requests for copies of the annual reports and have mailed them out.

There were approximately 25 post card requests that we received **without** the requestor's name and address. If you requested a copy of any of the annual reports and have not yet received your copy, please give us a call, toll-free at 1-800-446-7805. We will be happy to mail you a copy.

For your information, the 1997 Annual Reports are also available on our newly-created website: **http://www.chm.msu.edu/oem/index.htm**

The newsletters as well as other information on occupational and environmental health can also be found at our website. As we continue to develop the website, we welcome your comments and suggestions.

compounds. This is consistent with what is known about the mechanism of high molecular weight organic compounds. High molecular weight organic compounds are proteins, glycoproteins or polysaccharides that interact with IgE molecules on mast cells and eosinophils and release and cause the synthesis of various bioactive mediators. In contrast, low molecular weight chemical compounds rarely cause the formation of specific IgE antibodies and alternative cellular mechanisms have been suggested.

Because "intrinsic asthma" has similar pathology, reaction to medication, and physiology as "extrinsic asthma," use of the term has been dropped in recent editions of pulmonary text books. When used now "intrinsic asthma" commonly refers to individuals who are skin test negative to common environmental allergens. Isocyanates and other low molecular weight allergens found in the work place are good models to study "intrinsic asthma."

BRONCHIAL HYPERACTIVITY

Longitudinal studies of workers have shown that individuals who go on to develop work-related asthma typically start out with negative methacholine challenge tests which then become positive with exposure. A certain percentage then go on to develop symptoms. With removal from exposure, response to methacholine decreases and may become negative. Workers with ongoing exposures after symptoms develop are more reactive to methacholine than workers removed from exposure. These findings are true for both IgE mediated and non IgE mediated allergens.

The data on changes in methacholine with exposure reemphasizes the importance of exposure precipitating onset and severity of symptoms. With ubiquitous environmental allergens cessation of exposure is more difficult and it is therefore more difficult to conduct similar studies although methacholine sensitivity has been noted to vary among asthmatics.

NATURAL HISTORY

The majority of workers with work-related asthma continue to have symptoms even after they are removed from exposure. This has been best studied among isocyanate exposed individuals where persistent airway inflammation with eosinophil and lymphocyte infiltration can be seen. The chance of symptoms persisting increases with increased time of symptoms before a diagnosis of asthma and increased length of exposure once asthma is diagnosed.

The persistence of symptoms despite the cessation of exposure explains the difficulty in determining an allergen responsible for initiating the asthma in large numbers of patients. The longer the patient has asthma the more difficult it is to determine what initiated the process because the patient may now have persistent chronic symptoms with little relationship to the original precipitating exposure.

VIRAL ILLNESS

The importance of viral illness precipitating the onset of asthma has been suggested. The long term consequences of an acute high exposure to an irritating substance such as chlorine which causes Reactive Airways Dysfunction Syndrome (RADS) may be a useful model for studying the acute effect of a viral illness.

In summary, work-related asthma can serve as a useful model for both IgE and non IgE mediated asthma. Understanding the pathophysiology of exposure to high and low molecular weight substances in the work place with known dates of onset and cessation of exposure should prove useful in our understanding of asthma in general.

Asthma caused by work exposures appears to be identical to that found in the general population. The natural experiment of work with a defined onset of exposure allows for additional understanding of asthma that should improve management of patients with all causes of asthma.

Selected References

Bernstein DI. Allergic Reactions to Work Place Allergens. JAMA 1997; 278: 1907-1913.

Cullinan P, Newman Taylor AJ. Inferences from Occupational Asthma, in The Rising Trends in Asthma. Chichester: Wiley, Ciba Foundation Symp. 206 1997; 160-172.

Milton DK, Solomon GM, Rosiello RA and Herrick RF. Risk and Incidence of Asthma Attributable to Occupational Exposure Among HMO Members. American Journal of Industrial Medicine 1998; 33:1-10.

A recent study of an HMO in central Massachusetts reported that 21% of asthmatics aged 15 to 55 years who were enrolled in the HMO had onset of their disease attributable to occupational exposure.

The investigators used computerized diagnoses and prescription records of the 79,204 enrolled members to identify asthmatics in the 15 to 55 age group. These individuals were then interviewed and their asthma was attributed to occupation based on review of the interview. The patient charts were also reviewed. Only 15% of the charts documented that the health care provider had asked about work exposures. In 3% of the cases the health care provider documented a positive association with work. This contrasts with the 21% of the cases that the researchers attributed to work based on their interviews. This recent study supports a growing body of literature that work exposures play a significant role in adult onset asthma and that practitioners need to pay more attention to the patient's work place as the source of exposure.

Gassert TH, Hu H, Kelsey KT and Christiani DC. Long-Term Health and Employment Outcomes of Occupational Asthma and Their Determinants. Journal of Occupational and Environmental Medicine 1998; 40:481-491.

Follow up of 55 occupational asthma patients from an occupational health clinic in Boston was conducted 2.5 years after diagnosis. Fifty-four (98%) of the patients still had active asthma of which 26 (47%) were rated severe. Thirty-eight (69%) were unemployed. Women and workers from the industrial sector were more likely to have severe asthma. This study is consistent with previous research that work-related asthma is a condition that persists despite cessation of exposure.

The Project SENSOR News is published quarterly by Michigan State University-College of Human Medicine with funding from the Michigan Department of Consumer and Industry Services and is available at no cost. Suggestions and comments are welcome.

(517)353-1955
MSU-CHM
117 West Fee Hall
East Lansing, MI 48824-1316

Michigan Law Requires the Reporting of Known or Suspected Occupational Diseases

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Project S.E.N.S.O.R. News
MSU - College of Human Medicine
117 West Fee Hall
East Lansing, MI 48824-1316
Phone (517) 353-1955

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