SENSOR Occupational Lung Disease Bulletin

A project of the Massachusetts Department of Public Health's Occupational Health Surveillance Program,
the Massachusetts Thoracic Society, and the Massachusetts Allergy Society

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Dear Health Care Workers:

Happy New Year! For some of you, this is your first quarterly *Occupational Lung Disease Bulletin*, a project of the Massachusetts Department of Public Health's (MDPH) Occupational Health Surveillance Program (OHSP). We have expanded our mailing list and have added occupational health clinics, occupational health nurses, and a selected group of primary care physicians to the list of pulmonologists and allergists already receiving the *Bulletin.* We hope that you find it informative and that it serves as a reminder to report cases of work-related lung disease.

Because this is the first issue for many readers, we have included a general article on work-related asthma, one of the reportable occupational lung diseases in Massachusetts. In addition, we are enclosing with this *Bulletin*, a list of common asthma causing agents for your reference.

Sincerely,
Catharine Tumpowsky
Project Director, Work-Related Asthma Surveillance Project

Work-Related Asthma

Occupational asthma (OA), strictly defined, is asthma caused by exposure to substances in the workplace. Most OA occurs after sensitization. The dose of an agent required for initial sensitization is not known, but once sensitized, a worker can subsequently develop asthma symptoms from smaller doses. These sensitizers include pharmaceuticals, animal dander, proteins, enzymes, and other low and high molecular weight chemicals. Less common, but increasingly important is workplace asthma developing after exposure to irritants such as the vapors and gases of acids and other compounds. There are now over 200 identified causes of occupational asthma (see insert.) New agents are continually being discovered.

Existing asthma which is aggravated, rather than caused, by exposure to irritants at work is very common, although not considered OA per se. However, it is important to note that for purposes of obtaining Workers' Compensation benefits, pre-existing asthma that is aggravated or exacerbated by workplace exposures may be compensable under the Workers' Compensation laws in many states and is reportable in Massachusetts (see box on page 2). Clearly, all individuals with asthma need to avoid exposures to such irritants both at work and at home.

It is now recognized that individuals with an atopic background (allergic tendency) may become sensitized to any natural product of appropriate size (to reach the lower airways) and allergic potential (antigenicity). An allergic tendency is less important in regard to asthma caused by synthetic products such as isocyanates, although bronchial hyperresponsiveness may increase susceptibility.

The proportion of the working population afflicted with OA tends to be underestimated since workers with a predisposition to asthma tend to leave jobs which cause them to have symptoms. There is, nonetheless, a marked difference in the prevalence of OA in various industries. For example, about 6% of animal handlers and breeders have OA, compared to 5% to 15% of workers exposed to toluene diisocyanate (TDI), a component of paints and polyurethanes. Examples of major industries and occupations in which workers are at risk of developing OA are listed in Table 1.

Timing of Asthmatic Reactions

The timing of asthmatic responses to inhalation of workplace agents can be: immediate (within minutes of exposure), late (after a latent period of 4-8 hours or more), a combination of early and late, or more rarely, sustained or recurrent asthmatic attacks after a single exposure (known as Reactive Airways Dysfunction Syndrome or RADS). There is no symptomatic difference between occupational and non-occupational asthma.

Diagnosis of Occupational Asthma

Essentials for the diagnosis include:

⇒Document Asthma
- •Record Symptoms (wheezing, cough, chest tightness, shortness of breath)
- •Conduct Pulmonary Function Tests:
  - -Peak flows recorded 8 times daily for 2 weeks
  - -Spirometry with and without bronchodialators
  - -Methacholine challenge for strong history and negative pulmonary function tests

⇒Document Work-Relatedness
- •Identify temporal relationship between symptoms and work
- •Obtain work history (duration of exposure, exposures in previous jobs)
- •Assess work exposures (processes, substances used)

REPORT CASES SEEN OCT-DEC 1997

By January 31st, report all occupational lung disease cases seen for the first time during October through December 1997. If you have NOT seen any cases, it is not necessary to return the report form.

- •Are other workers ill with similar symptoms?

⇒Document Work Exposures
- •Obtain Material Safety Data Sheets
- •Conduct Worksite Visit

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SENSOR: *Sentinel Event Notification System for Occupational Risk. Massachusetts SENSOR is funded by the National Institute for Occupational Safety and Health.*

- Identify known asthma agents in the workplace or work processes known to cause asthma.
- Identify suspected asthma agents

⇒Control or eliminate worksite exposures and note improvement. Early diagnosis and removal from exposure is critical to improvement. (note: improvement may not be possible in long established cases.)

OHSP staff are available to assist in conducting worksite visits and will work with you to obtain relevant information about a worksite which may be helpful in identifying causes of work-related asthma or in implementing controls to prevent future cases.

Clinical Observations

Clinical confusion exists among health providers with respect to how OA "ought to behave" as a clinical entity. Asthma due to occupational exposures or that of non-occupational etiology is almost identical in clinical patterns and response to treatment. The principles in diagnosis and treatment of both forms of asthma are virtually identical and include: an accurate clinical diagnosis of asthma (vs. chronic bronchitis or other disorders); identification of the causative agent where possible; environmental controls; education of the patient to the mechanisms of asthma; and appropriate pharmacological therapy once the other aspects have been addressed.

In treating patients with OA, removal from the offending exposure may result in only a modest improvement in symptoms. It is a common pattern that leads to the erroneous conclusion that the asthma may not be occupational after all. Since both immunological and non-immunological mechanisms are postulated for asthma, OA patients may be confused by a sudden sensitivity to other agents such as freshly cut grass, ragweed, and dust. These agents may become causes for exacerbations only after increased airway reactivity has been established by the occupational exposure. The treating physician should not be "put off" by this apparent increased sensitivity to environmental elements.

Prevention

Physicians, plant engineers, managers, and workers, all need to become familiar with known sensitizers. Pre-screening of workers is not accurate enough to determine who will develop OA particularly in industries handling synthetic agents. It is therefore, necessary to minimize exposures for all workers, through product substitution or engineering controls (such as exhaust ventilation.) Periodic monitoring is also important. Respirators (gas masks) are not optimal and are used as a last resort of backup control.

The Future

Considerable research is needed to clarify the pathologic mechanisms of OA, predisposing factors, and more effective workplace controls. Only through a concerted effort will this problem be brought under control.

Table 1. Populations at Risk for Work-Related Asthma

Industry/Job	Agent
printers/paper products	vegetable gums, natural glues
grain handlers	grain dust
lumber/wood workers	wood dust
leather workers	formalin, chromium
paint sprayers	diisocyanates, dimet ethanalamine
plastics	epoxy resin, polyurethane
rubber industry	ethylene diamine, diisocyanates
bakers	flour, insect, mite debris
veterinarians	animal dander
laboratory workers	animal dander
vegetable oil production	flax seed, castor bean, cotton seed
beauticians/cosmetologists	diamines, potassium persulfate
health care workers	formaldehyde, medication, latex
pharmaceutical workers	various drugs
solders. electronic fabricators	colophony resin, ethanalamine

This article was adapted from an article written by David Christiani, M.D. which appeared in the Massachusetts SENSOR Update, March 1991 and from an article by Thomas Godar, M.D. which appeared in Occupational Airways (October 1996), a newsletter of the Connecticut Department of Public Health. Additional information came from the March 1996 issue of Occupational Airways.

Reporting Work-Related Asthma in Massachusetts

Physicians are required to report to MDPH all persons with:

a) a physician's diagnosis of asthma; AND

b) an association between symptoms of asthma and work.

Note: Reportable cases include persons newly sensitized by exposures at work; OR with pre-existing asthma exacerbated by exposures at work; OR persons for whom a one-time exposure to a chemical (s) at work resulted in generalized airway hyperactivity (RADS). Both suspected and confirmed cases of work-related asthma are reportable.

Number of Lung Disease Cases Reported to MA SENSOR, March 1992- September 1997

	August 1997	September 1997	Total to Date (3/92-9/97)
Asthma	4	5	529
Silicosis	0	0	12
Asbestosis	1	0	123
Chemical Pneumonitis	0	0	15
Total Number of Lung Disease Reports	5	5	679