

EDITORIAL

Occupational Asthma: Prevention by Definition

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Appropriately defining an occupational condition is often a critical step in its understanding and prevention. The definition of the condition establishes a framework for investigation and intervention that can assist, restrict, or distract from its control. We are only beginning to be aware of the importance of the perspective taken in determining whether or not a disease is recognized [Levenstein and Tuminaro, 1997].

In this regard we believe that, for practitioners concerned with disease prevention, the condition known as occupational asthma has been made more complex than is desirable. To date, most of the efforts to define occupational asthma have focused on etiology or diagnostic approaches for clinical investigational or medico-legal purposes [Godnic-Cvar, 1996; Chan-Yeung and Malo, 1995; Newman, 1995; Nordman, 1994]. In these settings, occupational asthma usually becomes defined by the specific response to an agent capable of provoking sensitization. For the most part, this way of diagnosing occupational asthma is directed at concerns with treatment and/or compensation of the individual worker-patient.

In a recent issue of this journal, Milton et al. [1998] take a different and creative approach to viewing the work setting and its role in asthma. They start with the experience of the worker patient of an abnormal condition—asthma—and consider the range of ways workers experience asthma.

Workers are generally unaware of the mechanism of their asthma, so Milton et al. move from the standard approach which would otherwise limit their focus to the *onset* of *sensitizer-induced* asthma. Instead, they expand their view to include what they refer to as ‘asthma attributable to occupational exposure.’ By doing so, they add consideration of *irritant-induced* asthma as well as distinguish between 1) onset of new asthma, and 2) onset of a new asthma *attack* in people with previously diagnosed asthma—generally childhood asthma—who have been asthma-free for an extended period. An important consequence of the approach taken by Milton et al. is the discovery that the prevalence of asthma ‘attributed to occupation’ is almost an order of magnitude higher than recognized in occupational lung disease surveillance efforts [Ross et al., 1997; Meredith and Nordman, 1996].

Why is it important to recognize this broader concept of occupational asthma? While sensitizer-induced asthma is the best known cause of occupational asthma, prevention of occupational asthma should not be limited to consideration of initial sensitization alone. Rather, it is essential to see the much larger goal of preventing all acute and chronic asthma-related conditions in all workers potentially at risk. To achieve this objective we must acknowledge that in a workplace with sensitizing exposures, respiratory irritants, or both, there are *two populations* of workers at risk for occupational asthma: those with apparently normal airways and those with hyperresponsive airways from whatever cause. Therefore, a definition of occupational asthma that better serves prevention efforts would include workers who experience any of the following:

1) immunologically mediated asthma resulting from exposure to sensitizers in the workplace;

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2) asthma resulting from exposure to irritants in the workplace (without an immunologic basis);

3) preexisting asthma exacerbated by workplace environmental exposures.

Any of these three asthma variants can lead to increasing disability from chronic pulmonary impairment with fixed airways obstruction if asthma attacks continue.

This broader approach recognizes that occupational asthma is a common endpoint of multiple pathways. Previously healthy individuals (with no clear risk, or with some risk marker such as atopy, or who have had previous asthma and an asthma-free period) exposed in sufficient concentration to agents capable of provoking immunologic change can begin to respond to extremely small concentrations of those same agents. Once asthma is induced by a specific agent, these same individuals may have identical bronchospastic responses to irritants and particulates in workplaces free of the original causative agent. Other previously healthy workers may have asthma resulting from either a single high-level irritant exposure or multiple exposures to lower levels. Again, once they become 'responders' the airways response may be provoked by many more agents than the original causative one. Others with asthma from nonoccupational causes may respond to workplace irritants and particulates with increased frequency or duration of asthma attacks or increased medication use. In reality, these conditions are often clinically indistinguishable and are amenable to a systematic approach to prevention. All should be considered part of the spectrum of occupational asthma.

If we persist in defining occupational asthma exclusively as 'onset of workplace sensitizer-induced asthma,' we sacrifice a significant disease prevention opportunity. Recent strides have been made to move away from this overly-restrictive definition [Chan-Yeung, 1995; Venables and Chan-Yeung, 1997]. However, to exclude by definition 'Pre-existing or concurrent asthma aggravated by irritants or physical stimuli in the workplace...' [Venables and Chan-Yeung, 1997] is to sacrifice an important input to identifying a prevention need. On the contrary, prevention strategies should be directed both toward eliminating new onset asthma from *any* source, as well as preventing new asthma attacks among those with both active or inactive previously diagnosed asthma. There should be dual prevention goals: 1) prevention of all healthy individuals (with or without atopy) from developing hyper-responsive airways or becoming sensitized as a result of workplace exposures, and 2) prevention of chronic disease resulting from workplace exposures in the entire hyper-responsive population.

The first goal is achieved by keeping sensitizing or irritant exposures below a level which results in increasing airways responsiveness. The second goal is achieved by controlling exposures below the threshold level stimulating reaction in altered or hyper-responsive individuals. This second goal is easily forgotten when the narrower definition

of occupational asthma is followed. However, to preserve the health of the already altered workers, conditions provoking airways response must be eliminated. Furthermore, prevention of asthma attacks appears closely related to prevention of chronic airways hyper-responsiveness and potentially prevention of chronic airways obstruction. In the absence of this second prevention goal, the only health-preserving alternative for the worker with occupational asthma is to terminate work in the implicated environments, with all of its attendant economic consequences.

There has been a tendency to focus asthma investigation and prevention almost exclusively on 'known sensitizers.' If a sensitizer is detected in the work environment, asthma is attributed (correctly or incorrectly) to the presence of that substance. If none is found, the presence of occupational asthma is doubted or denied. Asthma caused by sensitizers not previously identified or by agents which cause or exacerbate asthma by non-immunologic mechanisms may be discounted or overlooked.

In addition, a focus on the concept of the 'sensitive' host has created the impression that environmental surveillance is futile; thus, the potential to prevent workers from *becoming* sensitized is lost. Furthermore, even workplace sensitizer-induced asthma that occurs after an extended disease-free period in people with 'childhood asthma' is too often inappropriately attributed to the childhood diagnosis and therefore does not trigger a prevention-directed focus on the workplace.

It is time to pursue a coherent strategy for preventing occupational asthma by looking broadly at the relationships between work and asthma in order to protect workers with and without hyper-responsive airways. Obtaining a better understanding of the nature of the exposure-response relationships for irritants and for specific sensitizers in the different populations is only one critical piece of knowledge needed to develop effective prevention strategies to meet these goals. Broad conceptualization of occupational asthma assists in developing effective methods for surveillance and intervention for prevention of asthma caused or exacerbated by work.

Effective occupational asthma prevention requires distinguishing between efforts focused on populations from those focused on individuals. There is a tendency to consider the issue of asthma development and sensitization as being a result of some constitutional weakness residing exclusively within the individual worker, in part because of the language we use to talk about asthma. 'Sensitive' individuals are popularly thought to be abnormally responsive to outside stimuli to which 'normal' people do not respond. This may explain why workers with a history of asthma in childhood are treated as already sensitive and are therefore not thought to be at risk of occupational asthma. These views discourage efforts to understand and act on conditions that increase risk in *populations*. The focus on host frailty also implicitly

rejects the concept that the large group of 'normal' workers can be made abnormal, exclusively as a result of an extrinsic exposure in the workplace.

Ultimately, it is necessary to appreciate occupational asthma in the fullness of its diverse etiologies and many manifestations in order to assure its recognition and prevention. While different interventions may be needed to address different combinations of etiologies, host factors, and work environments, prevention must be directed at both sensitizer- and irritant-induced asthma in all workers, without or with preexisting asthma. This will be assisted by defining all asthma caused or exacerbated by workplace exposures—including asthma from sensitizers, irritant-induced asthma, and workplace exposure-induced asthma attacks—as occupational asthma.

REFERENCES

- Chan-Yeung M (1995): Assessment of asthma in the workplace. ACCP Consensus Statement. *Chest* 108:1084–1117.
- Chan-Yeung M, Malo JL (1995): Occupational asthma (Review). *N Engl J Med* 333:107–112.
- Godnic-Cvar J (1996): How to confirm occupational asthma. *Int Arch Occup Environ Health* 67:79–84.
- Levenstein C, Tuminaro DJ (1997): The political economy of occupational disease. In Levenstein C, Wooding J (eds): 'Work, Health, and Environment.' New York: Guildford Press, pp 3–19.
- Meredith S, Nordman H (1996): Occupational asthma: Measures of frequency from four countries. *Thorax* 51:435–440.
- Milton DK, Solomon GM, Rosiello RA, Herrick RF (1998): Risk and incidence of asthma attributable to occupational exposure among HMO members. *Am J Ind Med* 33:1–10.
- Newman LS (1995): Occupational asthma. Diagnosis, management, and prevention. *Clin Chest Med* 16:621–636.
- Nordman H (1994): The diagnosis of occupational asthma (Editorial). *Respir Med* 88:719–721.
- Ross DJ, Keynes HL, McDonald JC (1997): SWORD '96: Surveillance of work-related and occupational respiratory disease in the UK. *Occup Med (Oxf)* 47:377–381.
- Venables KM, Chan-Yeung M (1997): Occupational asthma. *Lancet* 349:1465–1469.