

## **Regulating Allergies in the Workplace**

**Given the complexity of the problem and the need for additional research, developing a standard to regulate "sensitizers" in the workplace is a daunting task.**

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OSHA is looking at how it might regulate "sensitizers," those chemicals and biologic workplace agents that produce an allergic response. Part of this effort is examining risk assessment aspects of sensitization. This creates interesting questions and potentially troublesome issues. For example:

- What types of allergies would be covered (e.g., asthma, allergic dermatitis, anaphylaxis)?
- Is there more than one pathologic mechanism through which a substance might create skin, airway or other allergic-like manifestations?
- Can a threshold level be assumed to exist for all allergens? For what substances have thresholds been established?
- What are the dose-response relationships?
- How can we address the problem of sensitive populations?
- Will exacerbation of the symptoms of allergy be part of any regulatory or consensus standard-setting efforts?

### **Common Problem**

Allergies are extremely common in the general population. Allergic rhinitis (hay fever) and other allergy-related conditions, such as sinusitis, affect 20 percent to 40 percent or more of us at some time. Probably the most common allergens (those substances that can produce an allergic response) include dust mites and their excreta, dust, molds, pollens, foods and animal dander. Recent studies have shown that a high percentage of individuals in urban settings, especially children, have antibodies (substances made by the body in response to a specific allergen) to cockroaches and their remnants.

Bronchial asthma is also highly prevalent in the general population, affecting approximately 5 percent of individuals across all ages. A 1990 study reported that as many as 400,000 to 3 million workers may have asthma. Other reports note that work may account for 2 percent to 15 percent of asthma cases.

More distressing is the reported recent increase in the incidence and severity of asthma. Although much speculation has been put forth as to the reasons (e.g., air pollution) for this increase, the underlying etiologic agent(s) have not been clearly elicited. For example, case reports connecting diesel exhaust and asthma, along with studies noting increased mortality during times of increased ambient particulate levels (diesel exhaust is significantly particulate in nature), have fueled concern that reactive airway problems may be related to such exposure. Workplace skin problems, including allergic dermatitis and urticaria (hives), are very common. NIOSH listed it as a "top 10" work-related disease as early as 1982. In 1984, the Bureau of Labor Statistics noted that 34 percent of reported occupational diseases were those affecting the skin. In 1990, there was an increase in reported occupational skin diseases. NIOSH has previously estimated that there are between 1 million and 1.7 million skin disorders in workers annually. It is unclear as to the exact breakdown between simple irritation and allergic dermatitis. According

to one source, 20 percent of all contact dermatitis reactions may be allergy-mediated. It is frequently difficult without appropriate skin testing to differentiate between an irritant response and an allergic skin problem.

There are many agents associated with various allergic disorders. More than 3,000 chemical agents have been associated with the development of allergic dermatitis; however, more than 65,000 chemicals may produce skin irritation.

Comparatively few workplace substances have been noted to cause allergic rhinitis. These include enzyme-containing detergents and trimellitic anhydride. Biologic allergens are associated with rhinitis. Some of these agents include wheat flour dust and its additives, western red cedar, psyllium and laboratory animal exposure.

More than 200 chemicals and biologic agents have been reported to produce allergic sensitization resulting in reactive airway disease (asthma). Some of the more recognized workplace asthma producers are toluene diisocyanate (TDI) and other isocyanates, platinum salts, animal antigens, anhydrides, wood dusts, rubber latex, acrylates and detergent enzymes. Just as in skin disorders, other mechanisms besides allergy may produce reactive airway problems. For example, a brief high exposure to an irritant may result in non-allergically based asthma (i.e., reactive airway dysfunction syndrome).

Skin and respiratory sensitization may be the result of several mechanisms. Probably the best recognized are reaction to immunoglobulins E (IgE) and G for asthma and cellular immune responses for skin dermatitis. Although there is some evidence for cellular response in asthma and urticaria, producing latex exposure has been noted to be mediated by IgE. The precise pathophysiologic mechanisms vary by substance. The influence of other factors, including genetics, will influence the body's response to exposure.

### **Setting an Exposure Limit**

There is little clear evidence as to the precise threshold level necessary to allergically sensitize someone for dermatitis, hives and especially asthma.

In addition to direct skin contact, some studies have also implicated airborne exposure in the production of skin problems. Probably the best known work in this area involves TDI. Exposure at and potentially below 0.02 parts per million (ppm) has been reported as capable of producing TDI-related asthma.

Traditionally, researchers have believed that allergic sensitization was primarily the result of high exposures related to spills or similar events. Other factors may include cumulative exposure, poor working conditions, type of industrial process, atopy, cigarette smoking, genetic influences and geographic location. Weather (climatic factors) may also play a role. Because of all these factors, setting a specific permissible exposure level may be difficult. Research reports would have to be analyzed for types of conditions, processes, and location and worker susceptibility factors to be sure that exposure-response determinations were not unduly influenced by nonwork factors.

On the other hand, good occupational health practice may require incorporation of such factors into permissible exposure level development. Unfortunately, little data have been presented on the majority of "asthmatigens" or allergic dermatitis-producing agents that would allow

appropriate exposure limits to prevent sensitization. It is important to point out that many studies will show the presence of substance-specific antibodies in many workers. Usually, the majority of these workers do not have evidence of a clinical disorder such as asthma. Thus, setting a standard based on the prevention of antibody production alone does not appear to be an appropriate way to proceed.

To illustrate this point, the American Conference of Governmental Industrial Hygienists set a Threshold Limit Value (TLV) for allergically produced disorders as the result of exposure to flour dust. The TLV documentation shows the basis for the exposure limit to be primarily from one recent study. Also, this fairly restrictive TLV appears to be based on data that does not really address the production of the clinical disorder. Additionally, there was little differentiation between symptoms of irritation and those potentially related to asthma. Lastly, exposure determination has not been clearly demarcated in this area to allow the setting of exposure limits, and researchers in this area have noted problems in using the data to set limits.

### **Irritation vs. Allergy**

There is an important difference between nonspecific irritation of pre-existing problems and the actual production of the disorder. Once reactive airway problems are present, many factors of a nonallergic nature may produce a temporary exacerbation of asthma symptoms such as chest tightness, coughing and wheezing. This may occur at relatively low levels. For example, cold, exercise, dust and even odors can serve as nonspecific irritant triggers of bronchial reactivity. "Irritants" such as heat, hygiene and soaps can also cause a flaring of a dermatologic condition. Thus, documentation of the actual cause of the asthma would be essential in determining whether the exposure is producing the asthma or skin problem, or if it is an irritant response or symptomatic exacerbation.

Another issue along these lines is whether any regulation should set exposure limits once a condition exists. Clearly, employers and occupational safety and health professionals must provide occupational health care to workers who are affected on the job, no matter what the cause. However, "chasing symptoms" is extremely subjective, and standard-setting activities in this area are difficult at best. Moreover, once an individual has been allergically sensitized to a substance, the exposure dose to set off asthmatic symptoms, in many instances, is much lower than the dose necessary to produce the airway reactivity initially. In the case of TDI, exposures in the area of 0.02 ppm may sensitize a worker. Once sensitized, however, airborne concentrations of TDI of 0.005 ppm or less may cause him to react.

### **Susceptible Populations**

A major question in setting exposure limits for allergens is the problem of susceptible populations. Atopy, the enhanced development of allergies, is one of the key factors in the development of certain workplace allergies. Genetic factors may likely explain atopy. Certain genetic influences have been studied and implicated as causing, for instance, increased susceptibility to asthma. Other reports show that atopy may be important in sensitization to certain chemicals such as platinum salts and ethylene diamine, while not important in western red cedar and certain anhydrides.

Cigarette smoking has been reported to predispose workers to allergic sensitization. Potential explanations may include increased IgE levels in smokers and injury to the lining of the airways, allowing enhanced exposure and resulting sensitization.

These susceptibility issues may influence how studies can be used in setting exposure-based regulations. Should workplace regulations be set based on protecting all individuals, even hypersusceptible workers, where the necessary exposure limit to prevent sensitization may be inordinately low? There is probably a diversity of opinion on whether this susceptibility qualifies a worker under the Americans with Disabilities Act and, if so, what reasonable accommodation might be appropriate.

### **Regulatory Approaches**

Would regulation for sensitizers be substance-specific or generic as in the proposed OSHA ergonomics standard? Given the relative paucity of well-performed studies in this area that would permit development of an exposure limit for even a handful of substances, abundant caution should be taken in considering the use of an "injury-based" standard rather than an exposure-based one. A worker may have asthma and be exposed to certain sensitizers and even have antibodies to that workplace allergen. Does the potential exposure on the job actually produce the symptoms, though, or are they due to exposure to ubiquitous dust mites not specific to the workplace and to which the worker is clearly allergic?

Response to regulatory enforcement actions may require medical testing. To truly connect exposure to the respiratory condition, this testing could include methacholine challenges to rule out a psychogenic cough masquerading as asthma, as well as difficult-to-conduct workplace peak flow studies and even expensive bronchoprovocational challenges. Skin testing for allergic dermatitis (patch testing) may also be required to demonstrate causal association and can be expensive.

The range of substances for which such testing is commercially available is limited considering the large number of potential workplace skin allergens. Thus, standard development activities must also take into consideration the resulting expenses and possible morbidity from certain testing to workers that might arise if symptoms and the presence of the allergen are sufficient for regulatory citation purposes.

### **Conclusion**

Allergic diseases can and do occur in the workplace and exact a human and economic toll. Research into skin, pulmonary and other allergic manifestations are needed, especially well-constructed and performed studies to answer questions concerning threshold levels for initial sensitization and post-sensitization, reaction-producing levels.

Any regulatory efforts should be carefully approached to ensure that the appropriate health end points are clearly identified and related to scientifically supported exposure limits. Enforcement parameters must not require employer responses that will pose excessive costs and possible additional health consequences for the workers as well.

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