

Letters to the Editor

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Respiratory Protection for Mold Remediation

To the Editor: The Institute for Inspection, Cleaning, and Restoration Certification (IICRC) has published a standard and reference guide for professional mold remediation.¹ Among other laudable goals, the standard seeks to protect remediation workers exposed to mold through appropriate use of personal protective equipment (PPE), citing pertinent standards promulgated by the Occupational Safety and Health Administration (1910.142, 1910.134, 1910.1001). Attached to the end of the standard is Appendix B, which suggests guidance for the selection and use of PPE.² The authors propose an interim substitute for a permissible exposure level (which they term a "subPEL"), based on observed concentrations of mold indoors during remediation, and extrapolations from the National Allergy Bureau's (NAB) criteria for mold spores in outdoor air and associated expected symptoms in allergy sufferers. Using this technique, the authors devise a criterion level against which PPE is to be selected using assigned protection factors. The authors suggest 10,000 spores/m³ is the appropriate level to which workers should be protected. They go on to suggest various levels of respiratory protection, ranging from a filtering facepiece for spore concentrations of 50,000 m³ to supplied air, SCBA, or quantitative fit-test selected devices for concentrations greater than 10,000,000 spores/m³. They propose reducing the limit to a lower level (eg, 2000–6000

spores/m³) if the molds are predominantly toxigenic.

Although this effort is commendable, we are concerned that this proposal is fundamentally misleading on several grounds, including our current knowledge of the health effects of mold and basic industrial hygiene principles. It creates the potential for both over- and underprotection because of erroneous assumptions and inadequate health- and exposure-based data. The authors predicate their logic and their standard on extrapolation from total mold counts in the ambient, outdoor environment and guidance for allergic individuals in the general population, which may not be appropriate for indoor remediation workers in heavily contaminated environments. Indoor exposures can differ significantly from those outdoors in the distribution of species, particle characteristics (proportion of viable and nonviable spores, as well as spore fragments), and biologic characteristics (activated epitopes, secondary metabolites). In addition, there may be important coexposures in moist environments such as endotoxin and bacteria. Thus, to imply comparability of indoor and outdoor concentrations ignores a large body of scientific knowledge. Furthermore, defining the criterion level as a PEL makes the assumption that average 8-hour exposures over a working lifetime of 40-hour weeks will prevent adverse health consequences. We do not know enough at this point to say whether this is true.³

Defining an exposure level without defining sampling frame and characteristics also violates funda-

mental industrial hygiene principles. Area exposures may underestimate personal exposures. Representative activities affect exposure intensity in unpredictable ways. Such a level may therefore represent underprotection if, for example, short-term exposures are more important for certain disease entities. They may also potentially represent substantial overprotection. PELs by definition rely on health-based dose-response data, of which there are simply too few known for molds at this time. However, although these data are developed, it remains important to provide mold remediation workers with adequate respiratory protection. Because of the variability in individual susceptibility, such protection must be coupled with adequate medical input, exposure assessment and control expertise, and program support. PPE represents only one facet of effective exposure control programs.

The authors of the suggested guidance point out that current respiratory protection guidelines for mold-exposed workers (from the New York City Department of Health, the U.S. Environmental Protection Agency, and the ACGIH, as well as OSHA, NIOSH, and ANSI) are generally based on extent of visible mold growth on substrates rather than on calculations of estimated airborne concentrations and resultant respirator penetration. However, the authors fail to indicate how their proposed guidelines represent a significant improvement, because they are not based on health effects, but on a mathematical construct. The construct is based on assumptions for which there is no scientific evidence or published practical experience. In addition, setting a numeric criterion level without detailed biologic discussion sidesteps the current controversy on criteria levels for allergens. Will the same level that prevents sensitization also prevent the exacerbation of existing disease in individuals already sensitized, as is important in isocyanates, platinum, and latex?

Ultimately, PPE should be based on health-based data. Some work is underway, but theoretical arguments argue against the likelihood of a single exposure level providing adequate and appropriate guidance. Existing guidelines for respiratory protection have been in place since at least 1999. The authors' proposed guidelines, although more stringent, are not based on more, newer, or better understanding of health effects, data on exposure, or dose-response relationships than previous guidelines. Current protection strategies have not been critically evaluated and deserve scrutiny before being discarded. At present, remediation jobs require the input of individuals with professional training in several disciplines and an understanding of the health and exposure issues involved.

Remediation workers exposed to mold do need appropriate respiratory protection, which should be based on the best available science and prudent efforts to minimize exposures. It is premature, in our opinion, to suggest we know enough to create a single PEL or "subPEL" for mold exposure. In the absence of a PEL, workers and safety and health professionals need to carefully evaluate each mold remediation job and decide on appropriate respiratory protection.

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Occupational Asthma and Rhinitis Caused by Milk Proteins

To the Editor: High-molecular-weight compounds (animal- or plant-derived proteins) and low-molecular-weight compounds (chemicals) known to cause occupational asthma are also able to induce occupational rhinitis. Patients with occupational asthma frequently report work-related rhinitis, which often starts before the onset of asthma. Patients with occupationally induced rhinitis have a high risk of asthma.^{1,2} Allergic sensitization to milk proteins by the oral route is a frequent manifestation in childhood.³ Respiratory symptoms caused by occupational exposure to milk or its constituents are uncommon. Both of our patients had been exposed to milk powder at their work environment. According to the manufacturer, milk powder contains 1% to 26% fat, 26% to 35% proteins, and 39% to 52% lactose. The protein fraction contains 80% caseins, 1% to 1.5% α -lactalbumin, and 2% to 4% β -lactoglobulin.⁴

There is one previous report in the literature regarding occupational asthma and rhinoconjunctivitis caused by α -lactalbumin,⁵ and an occupational asthma case caused by sodium caseinate has been reported.⁶ We describe two cases of sensitization to milk proteins (α -lactalbumin and casein) both with occupational rhinitis and the other also with occupational asthma.

A 38-year-old woman had been working in a bakery for 21 years. During the past 8 years, she had

handled milk powder and rye and wheat flours daily. She had had rhinorrhea, itching of the eyes for 3 years, and cough and shortness of breath for 1 year before investigations. Her respiratory symptoms disappeared or decreased during her vacations and weekends. Symptoms started within a few hours after resuming work. She had no history of atopy and had never smoked. Her sister had hayfever. She did not have other diseases and she did not use any medication. Three years earlier, skin prick tests (SPT) showed negative results to common aeroallergens.

A 40-year-old woman had been working in a dairy for 4 years. She had handled milk and also milk powder daily in her work. She had had nasal blockage, watery rhinorrhea, sneezing, cough, and shortness of breath at work daily for 1 year before referral to our institute. The symptoms worsened, especially when working by a centrifugal cream separator. Her symptoms disappeared during the weekends and holidays when she first presented, but more recently her symptoms had been persistent even on holidays. Earlier she had a cat and a guinea pig at home; specific IgE antibodies were found to both. Giving the pets away had helped somewhat. She had no family history of atopy or atopic symptoms of her own earlier. She had smoked for 20 years, 20 cigarettes per day; she did not have any diseases and was not on any medication. She had had some sinusitis episodes during the last 2 years. Her peak expiratory flow (PEF) follow up at home and at work showed up to 45% diurnal variation both during workdays and also during days off. The first measurements after wakening were the lowest ones; no significant decrease was seen at the workplace.

Inhalation challenge tests were performed according to published guidelines.^{7,8} Specific IgE antibodies to milk proteins were measured by enzyme-linked immunosorbent assay and reported as Phadebas RAST units (PRU)/mL (Pharmacia, Upp-

sala, Sweden). The SPT was done with common environmental inhalant allergens (Allergologisk Laboratorium A/S, ALK, Copenhagen, Denmark), milk powder, whole milk, β -lactoglobulin (Sigma L-8005), casein, and wheat flour as described earlier.⁹

In both cases, the control challenge test with lactose powder did not induce any symptoms or reactions in PEF/forced expiratory volume in 1 second (FEV₁) values and nasal status stayed unchanged. In case no. 1, the challenge test was carried out with milk powder the following day. Immediate nasal and bronchial reaction followed. Nasal obstruction and watery rhinorrhea within 15 minutes from the beginning of the challenge test were seen. At the same time, the PEF value decreased by 41% and wheezing rales were heard. The challenge was stopped. After medication with salbutamol inhalation, the asthmatic symptoms began to resolve, but still after 1 hour, her PEF value was decreased by 25% and FEV₁ by 26%. The obstruction disappeared after 8 hours. The specific allergen in the milk powder was α -lactalbumin according to the specific IgE determinations (RAST for α -lactalbumin was 2.08 kU/L and for milk 4.20 kU/L). β -lactoglobulin and casein RAST were <0.35 kU/L. In SPT, reaction to milk was 28 mm when it was 5 mm for histamine. Regular use of inhalation steroid medication was stressed as well as stopping any further exposure to sensitizing allergens.

In case no. 2, challenge with milk powder was also performed at day 2. Nasal symptoms were obvious with nasal obstruction measured by acoustic rhinometry and watery rhinorrhea observed by the investigator within 15 minutes from the beginning of the challenge test. There were no significant changes in PEF/FEV₁ measurements and no wheezing on auscultation. She was diagnosed as having occupational rhinitis (α -lactalbumin and casein) but not with occupational asthma. RAST for milk was 4.61 kU/L, 7.68 kU/L for

α -lactalbumin, and 5.79 kU/L for casein. In SPT, the reaction was 7 mm for milk when it was 5 mm for histamine. The findings in her PEF follow up strongly pointed to asthma, but not resulting from occupational allergens, because there was no difference in the PEF values between working days and days off work. Medication with inhaled steroids was started and the clinical picture will be followed. She had already started a retraining program and had stopped work in the dairy. Continuing exposure to allergens (α -lactalbumin and casein) would be a risk factor for her developing occupational asthma later and exacerbating the existing condition.

In conclusion, our two cases show that milk proteins can cause both occupational rhinitis and asthma in bakeries and in dairies. The possibility of this type of protein sensitization should be kept in mind when workers in the food industry are investigated for work-related respiratory symptoms. The exposure should be minimized by using work hygienic measures. Early diagnosis of the disease, when the patient has nasal symptoms only, would prevent occupational asthma and if relocated at this stage to an exposure-free working environment. Because patients with occupational rhinitis are a risk group for asthma,^{1,2} they should be considered as an important target group also for preventive programs.

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Preplacement Nerve Testing for Carpal Tunnel Syndrome

To the Editor: Franzblau et al.¹ concluded that electrodiagnostic screening for carpal tunnel syndrome (CTS) is not cost-effective for postoffer, preplacement evaluation of workers. Although this may be true if the results are used to not hire otherwise qualified workers, there is another strategy that is used by one company that I work with that using the authors' data seems to be cost-effective, at least in the workers' compensation system of Idaho.

For a number of years, this company has had all new hires undergo electrodiagnostic screening for CTS. The results are recorded and shared with the worker but are not used as a criterion for hire. Under

Idaho worker's compensation law, an employee may obtain workers' compensation benefits for aggravation or acceleration of preexisting conditions only if it results from an industrial accident. Gradual onset of CTS symptoms has been found by the Idaho Supreme Court not to meet that criteria.² Using this strategy, the authors would find the company saving \$178,985 per thousand person-years, with added costs only for electrodiagnostic testing of \$42,430, for a net of \$136,555 for the duration of the study, with expected increased savings over time.

In the realm of occupational medicine, the issue of causation is somewhat complicated³ and frequently contentious. Most healthcare providers are unfamiliar with or uninterested in the use of Hill's criteria⁴ to determine causation and are also unaware of the classic definition of fallacy in logic, "post hoc, ergo propter hoc."⁵ Even so, regardless of other factors, it is difficult to argue with the fact that, at least in our universe, a result cannot precede a cause.

More important than unfair costs to employers, incorrect determination of causation can lead to ineffective intervention and unnecessary restriction of activity, job loss, and disability for the worker. These are potential additional benefits for CTS

screening and perhaps the subject for future research.

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Response From Franzblau and Werner

To the Editor: As Dr. Weiss has noted,¹ our study focused on "whether denying employment to otherwise-qualified workers with an abnormal median nerve test result obtained in the postoffer preplacement (POPP) medical examination is cost-effective for employers"; "we did not assess the possible cost-benefit of using POPP nerve tests in

other circumstances."² He describes an interesting alternative strategy for using POPP nerve tests and suggests that this practice is cost-effective for one of his client companies in Idaho. Although this may be true, the description, data, and analyses presented in his letter do not provide an adequate foundation to allow the reader to assess the validity of his conclusion.

POPP nerve testing of one sort or another has been in use for at least 15 years, yet our paper would appear to be the first publication that attempts to address the cost-effectiveness of one version of this practice. We would welcome and encourage Dr. Weiss, and others, to submit more complete descriptions for publication in the peer-reviewed literature so that others may benefit from their experience.

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