Letters to the Editor

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Endotoxin, a Possible Agent in the Causation of Parkinson's Disease

To the Editor: Li et al[1] provided an excellent review on pesticides, evaluating their potential in causing Parkinson disease (PD). The original basic tenant, as noted by Li et al, of this association was occurrence of PD in populations living in rural and agricultural environments. As concluded by Li, there is currently insufficient evidence to support a casual association of pesticides with PD. Li et al[1] also mentioned a number of other possible causes for idiopathic PD (eg, infectious agents). It is worth mentioning another agent commonly associated with agriculture not presented in Li's discussion, endotoxin.[2] Previously, it has been proposed[3,4] that endotoxin (lipopolysaccharide [LPS]), which is a component of Gram-negative bacterial cell walls, is a possible agent in the cause of this disease. Endotoxins are commonly found in many environmental[5] and occupational[2] settings, including agriculture.[2] Previous studies[2,6] have reported that endotoxin is associated with a number of occupational (agricultural) diseases.[2] Animal investigations[7] have reported that injection of endotoxin into the brain can result in PD like-effects, supporting the hypothesis.[3] Although epidemiologic studies have not directly evaluated the association between PD and endotoxin,[3,4] such investigations are warranted. The potential of endotoxin as a cause of idiopathic or even an endotoxin-specific PD[3] must be considered, especially in light of reports finding no "sufficient evidence to support a casual association between pesticide exposure and PD" and the suggestion that those in the agricultural industry may be at elevated risk for this disease.[8]

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References

1. Li AA, Mink PJ, McIntosh LJ, Teta MJ, Finley B. Evaluation of epidemiological and animal data associating pesticides with Parkinson's disease. *J Occup Environ Med*. 2005;47:1059–1087.
2. Lange JH. Reduced cancer rates in agricultural workers: a benefit of environmental and occupational endotoxin exposure. *Med Hypotheses*. 2000;55:383–385.
3. Lange JH, Niehaus I, Thomulka KW. Is endotoxin an environmental cause of Parkinson's disease? [Letter] *Neuroepidemiology*. 2003;22:313.
4. Niehaus I, Lange JH. Endotoxin—is it an environmental factor in the cause of Parkinson's disease. [Letter] *Occup Environ Med*. 2003;60:378.
5. Park JH, Szponar B, Larsson L, Gold DR, Milton DK. Characterization of lipopolysaccharides present in settled house dust. *Appl Environ Microbiol*. 2004;70:262–267.
6. Mukherjee AK, Chattopadhyay BP, Bhattacharya SK, Saiyed HN. Airborne endotoxins in its relationship to pulmonary function among workers in an Indian jute mill. *Arch Environ Health*. 2004;59:202–208.
7. Castano A, Herrera AJ, Cano J, Machado A. The degeneration effect of a single intranigral injection of LPS on the dopaminergic system is prevented by dexamethasone and not mimicked by rh-TNF-alpha, IL-1beta and IFN-gamma. *J Neurochem*. 2002;81:150–157.
8. Priyadarshi A, Khuder SA, Schaub EA, Priyadarshi SS. Environmental risk factors and Parkinson's diseases: a meta-analysis. *Environ Res*. 2001;86:122–127.

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Reply: Endotoxin, a Possible Agent in the Causation of Parkinson's Disease

Authors' Response: We thank Lange et al for their comments related to our paper, "Evaluation of Epidemiologic and Animal Data Associating Pesticides With Parkinson's Disease."[1] Lange et al outline the literature indicating that endotoxins may represent an agricultural risk factor for Parkinson's Disease (PD) not covered in our review article. Although this letter raises the important point that future research should explore a variety of agricultural risk factors for PD, we limited the scope of our review to analytical epidemiology studies and whole animal studies using systemic exposure to pesticides. Nonetheless, based on the lack of causal evidence present in the pesticide literature at this time, it seems justified to support research on other possible alternatives, including endotoxins.

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Parkinson's Disease, Macular Degeneration and Cutaneous Signs of Mercury Toxicity.

Objective: *The objective of this study was to determined if there was a relationship between Grover's disease and Parkinson disease.* ***Methods:*** *Fourteen patients with Parkinson disease and 14 control patients were randomly selected and examined for cutaneous eruptions and blood mercury levels.* ***Results:*** *Of the 14 patients with Parkinson's disease, 13 had Grover's disease and detectable blood mercury. None of the patients in the control group had a cutaneous eruption and only 2of the 14 had detectable blood mercury.* ***Conclusion:*** *Mercury may play a role in the etiology of Parkinson disease and Grover's disease.*

To the Editor: Two cutaneous signs of mercury toxicity have recently been postulated. One consists of scattered or localized small erythematous spongiotic papules usually seen in people under 50[1] and the other is transient acantholytic dermatitis (Grover's Disease) which consists of small erythematous, often pruritic, scaly or crusted papules, usually seen in people over 50 and often favoring sun damaged skin. Both of these signs have been linked to macular degeneration and may help to identify mercury as the causative agent of that disease.[2] There have been numerous attempts at linking Parkinson disease to environmental factors but to date, nothing has been conclusively determined and the cause of Parkinson disease remains elusive.[3]

Materials and Methods

Fourteen patients with Parkinson disease were randomly selected and examined for signs of mercury toxicity. After obtaining informed consent, all patients had a physical examination, a 2 mm skin biopsy and a blood specimen for mercury which was measured by inductively coupled spectometry. As a control group, 14 patients with no signs of Parkinson disease were randomly selected and examined for signs of mercury toxicity.

Results

None of the patients with Parkinson disease were aware of or complained of any skin problems prior to examination. Of the 14 patients, 13 had 1–2 mm erythematous scaly papules of chest, abdomen, or back (#2–40) which showed characteristic Grover's disease on biopsy and 13 of the 14 had measurable blood mercury levels (mean 5.2 micrograms/Liter). The fact that both skin signs and blood levels can fluctuate means that the one discrepancy in each series could represent a false negative. In the control group, none of the patients had any cutaneous signs of mercury toxicity and only 2 had measurable blood mercury levels (10 and 16 micrograms/Liter).

Comment

These results are strikingly similar to those obtained with macular degeneration and suggest that both diseases may represent part of the spectrum of chronic low levels of mercury poisoning in susceptible individuals. As part of this study, electron microscopy was performed on 3 patients and showed thinning and gaps in the basement membrane of the skin which could possibly represent a portal of entry of mercury into the epidermis and if Grover's disease is proven to be a cutaneous component of Parkinson disease and macular degeneration, explain why some patients are afflicted by these diseases and why there seems to be some genetic susceptibility. Finally, recent studies have shown that mitochondrial dysfunction may play a role in the pathogenesis of Parkinson disease.[3] In Grover's disease, the epidermal cell's inability to manufacture intracellular adhesion proteins also suggests a similar pathogenesis[4] and mercury which has affinity for the sulfide bonds in the mitochondria,[5] could represent the toxin in both diseases.

While these series are relatively small, the evidence suggests that Parkinson disease, macular degeneration and Grover's disease may have a common etiology and further studies on the role of mercury in their etiologies is warranted. If these findings can be confirmed, then Grover's disease might represent an important predictive sign for people at risk to develop Parkinson disease and macular degeneration and these diseases might be greatly reduced or even eradicated through strict and prompt environmental measures.

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References

1. Dantzig PI. A New Cutaneous Sign of Mercury Poisoning? *J Am Acad Dermatol.* 2003;49:1109–1111.
2. Dantzig PI. Age Related Macular Degeneration and Cutaneous Signs of Mercury Toxicity. *Cutan Ocul Toxicol.* 2005;24:1–7.
3. Steece-Collier K, Maries E, Kordower J. Etiology of Parkinson's Disease: Genetics and Environment revisited. *Proc Natl Acad Sci.* 2002;99:13972–19374.
4. Hashimoto K, Fujiwara K, Tada J, Harada M, Setoyama M, Eto H. Desmosomal Dissolution in Grover's Disease, Hailey-Hailey Disease and Darier's Disease. *J Cutan Pathol*. 1979;22:488–501.
5. Shenker BJ, Guo TL, Shapiro IM. Low-level Mercury Exposure Causes Human T-cells to undergo Apoptosis: Evidence of Mitochondrial Dysfunction. *Environ Res*. 1998;77:149–159.

Outcome of Subjects Diagnosed with Occupational Asthma and Work-Aggravated Asthma After Removal From Exposure

To the Editor: Work-related asthma can be divided into occupational asthma (OA) and work-aggravated asthma (WAA).[1] OA has been defined as "a disease characterized by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular

TABLE 1
Comparison of the Changes in Functional and Inflammatory Parameters From Diagnosis to the Current Investigation Between Subjects With Occupational Asthma (OA) and Subjects With Work-Aggravated Asthma (WAA)

	OA		WAA	
n	18		10	
Sex	5 F/13 M		3 F/6 M	
Atopy	11		6	
Duration of asthma, y	9.1 ± 7.4		14.3 ± 15.8*	
Duration of exposure, y	14.5 ± 12.6		12.5 ± 12.6	
Duration of exposure after symptoms onset, y	3.8 ± 4.2		2.5 ± 4.8	
Time since diagnosis, mo	32.4 ± 9.4		35.5 ± 13.6	
	Initial Diagnosis	Current Investigation	Initial Diagnosis	Current Investigation
Smoking habits, packs/yr	13.8 ± 12.3	13.8 ± 13.1	8.3 ± 14.4	9.0 ± 15.1
Dose of inhaled corticosteroids, μg/d	430.5 ± 281.9	263.3 ± 330.6	400.0 ± 394.4	336.4 ± 400.5
β_2 agonists, puffs/d	2.1 ± 2.1	1.3 ± 3.3	0.9 ± 1.4	0.7 ± 1.2
Symptoms score	17.3 ± 12.6	4.6 ± 5.2*	14.9 ± 9.4	5.4 ± 3.0*
Asthma quality of life	ND	6.1 ± 0.7	ND	6.0 ± 0.8
Forced expiratory volume in 1 second pred, %	79.7 ± 24.3	84.8 ± 17.0	75.6 ± 18.5	76.5 ± 17.1
PC_{20}, mg/mL	1.7 ± 32.7	3.3 ± 2.6	2.2 ± 8.9	2.1 ± 1.7
Total cell count, 10^6 c/mL	2.3 (3.5)	2.0 (3.0)	2.6 (22.0)	2.5 (6.4)
Eosinophils, %	3.5 (8.4)	0.5 (2.2)	1.0 (7.0)	1.4 (2.3)
Neutrophils, %	33.2 (41.1)	36.6 (42.9)	63.5 (42.6)	49.5 (36.7)

*$P < 0.05$.
ND indicates not done.

occupational environment and not to stimuli encountered outside the workplace."[2] This definition implies that a specific substance induces asthma at the workplace through an immunologic mechanism. WAA is defined as preexisting or concurrent asthma that is exacerbated by workplace exposures,[1,3] which implies that the workplace triggers asthma but does not induce it. Although these two conditions have distinct definitions and underlying physiopathology, they are often very difficult to differentiate in clinical practice. Over the past years, there have been tremendous efforts to improve the understanding of OA. Several guidelines[4,5] have been published to improve the management of this condition. In contrast, the current data available on WAA in the literature are limited. To our knowledge, there are no data describing the functional and inflammatory outcome of subjects with WAA.

WAA is likely to represent an important societal burden due to the disability of the subjects and to the high prevalence of this condition. Indeed, 21% of the asthmatic subjects complain of a worsening of their asthma when they are at work.[6] Therefore, it is important to increase our understanding of WAA to assess the impact of this condition on workers and improve its management.

The aim of this study was to compare the clinical, functional, and occupational outcomes between subjects with OA and subjects with WAA 1 to 4 years after their diagnoses were made.

Materials and Methods

We conducted a cross-sectional study of the subjects previously investigated for work-related asthma in our center within 1 to 4 years after their original diagnosis of OA or WAA. Subjects were considered to have OA if they showed a positive specific inhalation challenge to an occupational agent, whereas subjects were considered as having WAA if they reported a worsening of their asthma symptoms when at work but showed a negative specific inhalation challenge to occupational agents.

We reassessed their clinical, functional, inflammatory, and occupational characteristics. Their respiratory symptoms were scored according to a Borg scale from zero (no symptoms) to 10 (worst symptoms ever).[7] Their asthma maintenance treatment and their daily use of short-acting β2-agonists were also recorded. Asthma quality of life was assessed.[8] The workers were asked about their employment status. Spirometry,[9] methacholine challenge,[10] and sputum induction[11] were performed and compared with the results obtained at the time of diagnosis.

Analysis

t test and the Mann-Whitney test were used to compare clinical, functional, and inflammatory parameters between the groups with OA and WAA. The analysis was performed using the SPSS 10.0 statistical package (Chicago, IL).

Results

Eighteen subjects with OA and 10 with WAA were assessed. Their clinical characteristics are summarized in Table 1.

All subjects with OA had to change their work environment, except one worker who had developed

OA due to the exposure to an occupational sensitizer used by a colleague working next to him. No subject with WAA remained at the same workplace after the diagnosis.

Subjects with WAA had asthma for a longer period of time (14.3 ± 15.8 years) than subjects with OA (9.1 ± 7.4 years, $P = 0.02$). Symptoms of asthma occurred before entering their new work setting in four of the 10 subjects with WAA and in one of the 18 subjects with OA. There was a similar and significant improvement in the respiratory symptoms of both groups at the time of this study.

The pulmonary function tests and the sputum cell counts were similar in subjects with OA and WAA (Table 1). Although not statistically significant, we observed a trend toward a greater improvement of PC_{20}, a larger decrease in the dose of inhaled corticosteroids, and in the need of short-acting $beta_2$ agonists in the subjects with OA compared with the subjects with WAA, as shown in Table 1.

Although not statistically significant, subjects with WAA tended to show a decrease of their median neutrophil counts (49.5% [36.7%]) compared with the time of their diagnosis (63.5% [42.6%], $P = 0.1$), whereas subjects with OA tended to have a decrease in their sputum eosinophil counts (0.50% [2.2%]) compared with the time of their diagnosis (3.5% [8.4%], $P = 0.1$).

Discussion

All subjects with WAA and all but one subject with OA were removed from exposure after they underwent investigation for possible OA. Although subjects with WAA did not obtain any financial compensation from the Quebec Worker Compensation Board, they could not be maintained at their original workplace due to their respiratory symptoms. These data are consistent with a previous study in which the socioeconomic outcomes of the subjects with WAA were equivalent to those of the subjects with OA.[12] It is unknown whether an improvement of the measures of prevention such as protective masks, improved ventilation, or increase in asthma medication would have allowed these workers with WAA to remain in their work environment.

We did not find any demographic or functional significant differences between subjects with OA and subjects with WAA a few years after the diagnosis and removal from exposure. The definition of WAA chosen in our study was based on the results of specific inhalation challenges. The occurrence of asthma after entering a new work setting did not exclude the diagnosis of WAA. This contrasts with the epidemiologic study originating from the Sentinel Event Notification Systems for Occupational Risks (SENSOR)[13] in which WAA was identified if the affected individuals experienced asthma symptoms or had treatment for asthma in the 2 years before entering a new work setting and if they experienced an increase in asthma symptoms or increased use of their asthma medications after entering that new exposure setting. The patients referred to our clinic were suspected of having OA and thus, were more likely to have similar clinical characteristics than the subjects with confirmed OA. Furthermore, the subjects with preexisting asthma whose asthma was aggravated by their workplace were less likely to seek medical advice in a tertiary center because they did not attribute the onset of asthma to their workplace and did not seek any financial compensation. Therefore, the population studied in the SENSOR program is likely to differ from ours.

Although not statistically significant probably due to the low number of subjects, there was a trend toward an improvement of asthma as shown by consistent trends toward decreases in airway responsiveness, in the dose of inhaled corticosteroids necessary to control the disease, in the need of short-acting $beta_2$ agonists, and in the sputum eosinophil counts in subjects with OA after removal from exposure. This is consistent with previous studies showing improvement of OA after removal from exposure.[14]

In contrast, we did not find any functional improvement in subjects with WAA, although they had a marked decrease in their respiratory symptoms. It is unknown whether subjects with WAA who would have remained at their workplace would have had a worsening of their asthma.

The functional and occupational outcomes of subjects with OA and WAA after removal from exposure seem to be similar. Although it seems that subjects with OA tend to improve to a greater extent after work removal than subjects with WAA, this needs to be confirmed by studies with a larger sample size.

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References

1. Vandenplas O, Malo JL. Definitions and types of work-related asthma: a nosological approach. *Eur Respir J*. 2003;21: 706–712.
2. Cartier A. Definition and diagnosis of occupational asthma. *Eur Respir J*. 1994; 7:153–160.
3. Wagner GR, Wegman DH. Occupational asthma: prevention by definition. *Am J Ind Med*. 1998;33:427–429.
4. Moscato G, Godnic-Cvar J, Maestrelli P, et al. Statement on self-monitoring of peak expiratory flows in the investigation of occupational asthma. *J Allergy Clin Immunol*. 1995;96:295–301.
5. Tarlo SM, Boulet LP, Cockcroft DW, et al. Directives de la Société canadienne de

thoracologie pour l'asthme professionnel Canadian Thoracic Society guidelines for occupational asthma. *Can Respir J*. 1998; 5:397–410.
6. Saarinen K, Karjalainen A, Martikainen R, et al. Prevalence of work-aggravated symptoms in clinically established asthma. *Eur Respir J*. 2003;22:305–309.
7. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*. 1982;14:377–381.
8. Juniper E, Guyatt G, Ferrie P, et al. Measuring quality of life in asthma. *Am Rev Respir Dis*. 1993;147:832–838.
9. American Thoracic Society. Standardization of spirometry. 1994 update. *Am J Respir Crit Care Med*. 1995;152:1107–1136.
10. Juniper EF, Cockcroft DW, Hargreave FE. *Histamine and Methacholine Inhalation Test: A Laboratory Tidal Breathing Protocol*, 2nd ed. Lund, Sweden: Astra Draco; 1994.
11. Pizzichini E, Pizzichini M, Efthimiadis A, et al. Indices of airway inflammation in induced sputum: reproducibility and validity of cell and fluid-phase measurements. *Am J Respir Crit Care Med*. 1996;154:308–317.
12. Larbanois A, Jamart J, Delwiche JP, et al. Socioeconomic outcome of subjects experiencing asthma symptoms at work. *Eur Respir J*. 2002;19:1107–1113.
13. Goe SK, Henneberger PK, Reilly MJ, et al. A descriptive study of work aggravated asthma. *Occup Environ Med*. 2004; 61:512–517.
14. Perfetti L, Cartier A, Ghezzo H, et al. Follow-up of occupational asthma after removal from or diminution of exposure to the responsible agent: relevance of the length of the interval from cessation of exposure. *Chest*. 1998;114:398–403.

ACOEM Evidenced-Based Statement: Medical Surveillance of Workers Exposed to Crystalline Silica—Some Comments and Suggestions

To the Editor: The recent position statement by the American College of Occupational and Environmental Medicine (ACOEM) published in the *Journal of Occupational and Environmental Medicine (JOEM)*[1] regarding medical surveillance for workers exposed to crystalline silica presents an excellent overview of the health effects related to silica exposure and provides an efficacious guideline for a medical monitoring surveillance program.

Although the position statement outlines an effective medical monitoring surveillance program for silica-exposed workers, the unfortunate fact is that many of the workers with the highest exposure levels are never enrolled in a surveillance program. According to OSHA, there are more than two million workers in this country exposed to silica-containing dust[2]; approximately 100,000 workers participate in high-exposure tasks like abrasive blasting, rock drilling, mining, quarrying, and construction. Based on data from the NIOSH,[3] from 1990 to 1999, silicosis was more frequently cited as a cause of death for construction workers than for workers in any other occupation. Although construction workers are frequently exposed to high levels of silica dust, it is unusual for a construction company to enroll their employees in a long-term medical monitoring surveillance program. This is due to many factors, including the transient nature of the construction projects as well as economic, cultural, and legal factors. The primary contributing factor is the essential temporary nature of construction. Unlike other industries, construction projects and worksites are relatively short-lived. Every project has a completion date. Due to this fact, workers often move from job to job and from employer to employer. Most construction workers will tell you that they are "always working themselves out of a job"; once a job is finished, they move on to the next one. Even if a worker stays with the same employer for an extended period of time, it is unusual for that employer to refer to records regarding long-term exposure histories. Because it normally takes years for silica-related symptoms to become apparent, it is unlikely that any individual employer in a long chain of employers will take on the financial burden of surveillance. In addition, the rugged individualist and macho attitude within the industry does not lend itself to enrolling workers in long-term silica surveillance program. Finally, because there is still no comprehensive OSHA silica standard, employers are not obligated to enroll their employees in a surveillance program. One possibility for establishing silica medical monitoring surveillance programs is that unions representing silica-exposed workers could follow the example of other unions that represent asbestos-exposed workers such as the insulators, pipefitters, and others that have established successful union-supported medical surveillance programs for their membership.

Although silicosis is one the oldest occupational diseases reported, it is unlikely that there will be much change unless unions and occupational healthcare professionals keep silicosis constantly under the spotlight of public awareness. From 1992 to 1995, there were 200 to 300 reported silicosis-associated deaths per year. The current number of silicosis-associated deaths and/or silica-related disease in this country is unknown.[4]

In addition, surprisingly, the ACOEM position paper hardly mentions the role of other skilled occupational health professionals. It has been our clinical experience in an urban-based, heavily attended occupational medicine practice that industrial hygienists (IHs) and occupational nurses can be extremely useful in a team-oriented medical surveillance program. Industrial hygiene is the art and science of anticipating, evaluating, recognizing, and controlling exposures occurring in or from the workplace. IHs can provide skilled technical assistance to the medical staff and the workers regarding risk characterization, site evaluation, exposure assessment, training, and the selection of methods to control/reduce exposure such as engineering controls and/or respiratory protection. IHs can assist the medical staff by reviewing exposure data and employee work history.

Finally, to remain relevant, it is very important that someone on the clinical staff (doctor, occupational nurse, IH) have an understanding of

silica-disturbing tasks as they take place everyday in the workplace.

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References

1. Raymond LW, Wintermeyer S. Medical surveillance of workers exposed to crystalline silica. *J Occup Environ Med.* 2006;48:95–101.
2. Unified Agenda Proposed Rule Stage 1218-AB70-1935. *Occupational Exposure to Crystalline Silica.*
3. *NIOSH Publication No. 2003-111: The Work-Related Lung Disease Surveillance Report.* 2002.
4. *NIOSH Publication No. 2002-129: NIOSH Hazard Review, Health Effects of Occupational Exposure to Respirable Crystalline Silica.*

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Reply: ACOEM Evidence-Based Statement: Medical Surveillance of Workers Exposed to Crystalline Silica—Some Comments and Suggestions

Authors' Response: We appreciate the thoughtful comments of Zuckerman and Szeinuk on the ACOEM position paper on medical surveillance (MS) of silica-exposed workers[1] and the overall status of silica-related disease prevention. It is difficult to overstate the contributions of our health and safety colleagues in preventing occupational and environmental illnesses in general and silica-related conditions in particular. Although strict etymology might hold that MS does not include industrial hygiene and nursing functions, more useful is the appreciation of such expertise as crucial complements to MS. They are *potentially more effective* in limiting the burden of disease. Indeed, had more employers of the 1930s implemented dust control practices recommended by Roy Bonsib (safety), Phillip Drinker (industrial hygiene), and Cary McCord (occupational medicine),[2–4] the bitter harvest of cancer and other diseases due to silica, asbestos, and kindred toxic agents could have been minimized; therefore, today's ongoing need for MS would be accordingly less.

Some 1930s employers realized the role of both physicians and allied health professionals in preventing pneumoconiosis and other occupational diseases. Because of the uncertain validity of the maximum allowable concentrations then recommended for asbestos and silica[2,5]—based as they were on "trial, error and negotiation . . . (having) no scientific basis"—it was considered good practice to carry out medical monitoring on workers who were exposed to such dusts while using multiple methods to control their breathing-zone concentrations.[6] As a senior pulmonologist (Brooks SM, personal communication to Occupational and Environmental Lung Disorders Committee, American College of Occupational and Environmental Medicine, March 23, 2006) currently puts the matter for occupational asthma, "Industrial hygiene and allergen control measures are the cornerstone of a preventive program."

Because Zuckerman and Szeinuk[7] found our mention of industrial hygiene and occupational nursing too stingy, other readers may feel similarly. We remind them of our reference 14, in which (section 5) OSHA describes in some detail the complementary role of these disciplines in monitoring and controlling exposures in its special emphasis program (SEP) for silicosis. Although a specific OSHA Standard for silica is still lacking, this SEP offers valuable guidance. The lack of a truly protective permissible exposure limit for respirable crystalline silica remains a major obstacle to preventing silica-related diseases, however.[8]

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References

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2. Bonsib RS. *Dust Producing Operations in the Production of Petroleum Products*. New York: Standard Oil Co (NJ); July 15, 1937:7–11.
3. Drinker P, Hatch T. *Industrial Dusts.* New York: McGraw Hill Book Co; 1936.
4. McCord CP. *A Blind Hog's Acorns.* Cincinnati: American Conference of Governmental Industrial Hygienists; 1999.
5. Elmes PC. Inorganic dusts. In: Raffle PAB, Lee WR, McCallum RI, Murray R, eds. *Hunter's Diseases of Occupations,* 6th ed. Boston: Little Brown; 1987:652.
6. Raymond LW, Yoder JD, Venable FS, Church FW. Tips for turnaround ventilation. *Petroleum Refiner.* 1961;40:217–240.
7. Friedman-Jimenez G, Beckett WS, Szeinuk J, Petsonk EL. Clinical evaluation, management, and prevention of work-related asthma. *Am J Ind Med.* 2000; 37:121–141.
8. Rosenman KD, Reilly MJ, Kalinowski DJ, Watt FC. Silicosis in the 1990s. *Chest.* 1997;111:779–786.

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A Contingency Plan for Healthcare Worker Protection in the Event of a Flu Pandemic

To the Editor: The growing threat of an avian flu pandemic due to the H5N1 virus is prompting community-and nationwide efforts to plan for patient management.[1,2] The SARS epidemic proved that protection of healthcare workers is crucial to optimal patient management.[3] The World Health Organization and other organizations have engaged in active debate about healthcare worker protection.[4] We described a contingency plan for healthcare worker protection and

training in hospitals that are potentially faced with managing large numbers of patients with influenza.[5] Multidisciplinary discussions were held by occupational physicians, infectiologists, emergency physicians, and managers; and drills were performed. The first component of the plan is identification of workers who have risk factors for acquiring the virus or developing influenza-related complications. These factors may be known in advance by occupational physicians or identified on the day the hospital is requisitioned. The second component of the plan consists of preventing disease caused by exposure to biologic agents. This component requires specific training of healthcare workers, procedures for monitoring healthcare workers who manage patients with influenza, effective protection of healthcare workers, and early medical and administrative management of infectious and noninfectious adverse events. The third component is a communication strategy involving information of managers and other executives in a way that protects patient confidentiality as well as feedback to healthcare workers. This contingency plan must be incorporated into a broad and flexible personnel management policy led by the managers (team distribution, student management, logistics, organization of living areas around the hospital, and so on). In conclusion, the practical vision provided by our approach adds usefully to current nationwide and international efforts to manage the risk of a flu pandemic.

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References

1. Premier Ministre. Plan gouvernamental de prévention et de lutte 'pandémie grippale.' [Government Plan for Prevention and Control of a Flu Pandemic.] 2006.
2. The Writing Committee of the World Health Organization (WHO) Consultation on Human Influenza. Avian influenza A (H5N1) infection in humans. *N Engl J Med.* 2005;353:1374–1385.
3. Li L, Cheng S, Gu J. SARS infection among health care workers in Beijing, China. *JAMA.* 2003;290:2662–2663.
4. World Health Organization. Avian Influenza, Including Influenza A (H5N1), in Humans: WHO Interim Infection Control Guideline for Health Care Facilities. 2006.
5. Descatha A, Salomon J, Dolveck F, et al. A contingency plan for healthcare worker protection in the event of a flu pandemic. 2nd Conference of Hesculaep European project; Seville; May 22–23, 2006.

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