Occupational exposures and chronic airflow limitation

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The recent literature was reviewed to evaluate whether chronic airflow limitation is associated with occupational exposures to dusts. Only those studies that controlled for the effects of smoking were included. There is compelling evidence that exposure to inorganic dusts, such as from coal and hardrock mining or asbestos, are associated with the development of chronic airflow limitation, independently of pneumoconiosis. Nonsmoking gold miners are particularly at high risk of airflow obstruction and emphysema. Findings from studies of organic dusts, such as exposures to wood, cotton, grain or other agricultural dusts, or to mixed dust exposures, were less consistent but tended to show positive dose-response associations. In the majority of studies, no statistical interaction was shown between dust exposures and smoking; however, the effects of the dust exposures were often more pronounced. An occupational history should be considered, in addition to a smoking history, as an integral part of an investigation of chronic airflow limitation in a patient.

Key Words: *Chronic airflow limitation, FEV_1, Occupational exposures, Smoking*

Les expositions professionnelles et l'obstruction respiratoire chronique

RÉSUMÉ : On a passé en revue les études récentes pour évaluer si l'obstruction respiratoire chronique a un rapport avec les expositions professionnelles aux poussières. Seules les études avec groupe témoin pour les effets du tabagisme ont été retenues. On peut conclure que les expositions aux poussières inorganiques comme celles provenant du charbon et des roches ou de l'amiante est associée à l'obstruction respiratoire chronique, indépendamment d'une pneumoconiose. Les non-fumeurs qui travaillent dans les mines d'or sont particulièrement exposés au développement d'une obstruction respiratoire et d'un emphysème. Les résultats des études sur les poussières organiques, telles que les expositions aux poussières de bois, de coton, de grain ou d'autres poussières agricoles, ou les expositions à plusieurs types de poussières étaient moins constants mais tendaient à démontrer une association dose-réponse positive. Dans la plupart des études, aucun lien statistique n'a été démontré entre les expositions aux poussières et le tabagisme; toutefois, les effets des expositions aux poussières étaient souvent plus prononcés. Par conséquent, les antécédents professionnels tout comme les antécédents de consommation de tabac devraient faire partie intégrante de l'anamnèse chez un patient présentant une obstruction respiratoire chronique.

Studies have shown that the diseases of dusty occupations, in particular the pneumoconioses, are on the decline in developed countries. In contrast, mortality rates due to the chronic nonmalignant lung diseases including those characterized by airflow obstruction are increasing (1) and these diseases are responsible for high rates of morbidity and extended periods of disablement. It is now generally accepted that occupational exposure to organic dusts such as grain and cotton

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dust may cause asthma and asthma-like disorders. On the other hand, it has not been clearly established whether occupational exposures to either organic or inorganic dusts can lead to chronic airflow limitation. Adding to the complexity of this association is the relative contribution of cigarette smoking, a known determinant of respiratory disease.

Most of the available epidemiological studies have been cross-sectional in design. Although these studies are prone to selection biases, any chronic disease risk shown would be underestimated, in the case where large numbers of affected workers have already quit the industry. The ideal prospective study would follow a cohort of workers from first hiring until death, charting the development of illness in every worker. As time and resources prohibit such a study one must rely on shorter periods of follow-up, and on health measurements that predict rather than document morbidity. The most widely accepted predictor of the eventual development of chronic airflow limitation is an accelerated decline in forced expiratory volume in 1 s (FEV_1) (2). Not only are the longitudinal decline estimates very much dependant upon the age distribution of the population sample, but the estimates of their variance, and therefore of their significance, are vulnerable to the method of analysis (3). Within-study comparisons of exposure groups, whether derived through cross-sectional or longitudinal designs, are relevant provided that differential selection factors are not present and the known confounders of age and smoking are taken into account in the design or analysis.

This review addresses the question, 'can the exposures that workers have encountered in the course of their daily work lead to chronic airflow limitation?' Three types of exposures are reviewed:

- Inorganic dusts: coal, hard rock and asbestos;
- Organic dusts: cotton, grain, wood and agricultural dusts;
- Mixed dusts: including exposure to fumes and gases.

Studies that were selected for review all had taken into account the contribution of cigarette smoking to the development of chronic airflow limitation by using one of three strategies: analyzing outcomes for nonsmokers separately; using statistical adjustments for smoking status; or matching according to smoking status. Where relevant, Canadian studies were cited preferentially, as were the more recently published studies concerning the chronic respiratory effects of occupational and environmental exposures. It is recognized that occupational exposures are seldom specific, but to a greater or lesser degree are mixed, either due to concurrent exposures, or as a result of the varying work histories of the subjects (4). For the last category, a dominant exposure has not been identified. Types of dusts have been chosen that have been sufficiently studied to evaluate causality, as defined by Hill's (5) criteria of consistency (demonstrated in most populations at risk); strength of association; dose-response relationship; specificity; coherence of the evidence; and biological plausibility.

INORGANIC DUSTS

Inorganic dust exposure has traditionally been associated with pneumoconiotic changes in the lung parenchyma. However, there has been increasing evidence of associations between inorganic dust exposure and chronic airflow obstruction. For some occupations, eg, coal mining and certain hard rock mining, the evidence is now sufficiently strong for causality to be inferred.

In 1973, Higgins (6) reviewed the published studies of chronic pulmonary disease of men engaged in dusty industries. The majority of studies were cross-sectional in design, comparing dust-exposed and nonexposed workers. In almost all, there were higher prevalence rates of bronchitis and lower ventilatory function in workers exposed to the inorganic dusts, even with adjustment for differences in smoking.

The results of studies published betweeen 1973 and 1985 on coal miners, hardrock miners and quarry workers were reviewed by Becklake (7). Most of the cross-sectional studies showed a higher prevalence of bronchitis and lower mean ventilatory function in exposed compared with nonexposed or less exposed workers. As could be expected, smokers consistently had higher rates of bronchitis and lower average ventilatory function compared with nonsmokers. However, the effects of exposure on the nonsmokers were not evaluated. Longitudinal studies also showed comparable dust and smoking effects on longitudinal decline in FEV_1.

Results of cross-sectional and longitudinal studies (8-12) in which the effects of coal mining dust are considered separately from the effects of smoking are shown in Table 1. Significant airflow obstruction was typically found in 6 to 10% of nonsmoking miners, and a significant dose-response relationship was often shown between cumulative dust exposure and FEV_1 or FEV_1/forced vital capacity (FVC)%. In 1993, Oxman and associates (13) conducted an overview of studies published between 1966 and 1991, concerning the relationship between quantitative estimates of inorganic dust exposure and chronic obstructive lung disease. They concluded that after roughly 35 years of work at a mean coal dust exposure level of 2 mg/m^3, approximately 8% of nonsmoking coal miners (and 6.6% of smoking miners) could be expected to experience a level of FEV_1 less than 80% of predicted, and 1.2% of nonsmoking coal miners (and 2.3% of smoking miners) would have an FEV_1 level of less than 65% predicted. A longitudinal study of American coal miners employed since 1970, when dust exposure levels had been relatively low (12), also showed a significant decrease in both FEV_1 and FEV_1/FVC in relation to dust levels, with an estimated decline in FEV_1 of 5.7 mL for each mg/m^3 year of exposure over their working life.

Studies of gold miners (14-15) suggest an even stronger relationship between dust exposure level and airflow obstruction in hardrock mining compared with coal mining. In the re-analysis by Hnizdo and colleagues (14) of results from a South Africa gold mining cohort the estimated effects of cumulative dust exposure from this mining environment on airflow obstruction was about 10 times greater than that seen in the coal mining cohorts. These effects were even more

TABLE 1
Fibrogenic inorganic dusts: Relationship between exposure and airflow limitation in studies controlling for the effects of smoking

Reference	Population	Exposure measure	Relationship between exposure and outcome
8	British coal miners n=1677	Cumulative dust over 11 years	Significant decline in FEV_1 with increased dust exposure
9	US surface coal miners n=1171	Duration of work (years)	No association between years as a surface miner with FEV_1 No interaction of exposure and smoking
10	British coal miners n=3380	Cumulative dust over 10 years	Significant dose-response between dust and airflow limitation in smokers and nonsmokers. No smoking-exposure interaction
11	US coal miners n=1470	Underground vs face workers	Significant FEV_1 decline (smoker adjusted) for face workers Average ratio of 1.5 for smoking vs exposure effects
12	US coal miners first employed after 1970 n=1885	Cumulative dust	Significant dose-response between dust and FEV_1, adjusted for smoking FEV_1 decline greater in first 5 years of employment
14	South African gold miners n=2209	Cumulative dust	Significant dose-response between dust and FEV_1, similar in smoking categories No smoking-exposure interaction in total group
15	Australian gold miners n=1093	Duration of underground gold mining	Significant dose-response between duration and obstruction measures
21	Canadian nonsmoking chrysotile miners	Asbestos miners vs non-exposed manual workers	Decreased upstream resistance at low volume in miners with increasing severity as extent of alveolitis and asbestosis increases
22	US asbestos insulators n=416	Insulators vs reference population	Significantly decreased FEV_1 and $FEF_{25/75}$ in all smoking categories
23	Canadian asbestos miners n=983	Many exposure-time variables	Airflow limitation associated with exposure, weighted for residence time in the lung An increased effect suggested for earlier exposure
24	US boilermakers n=534	Duration of work in trade	Significantly decreased FEV_1/FVC% associated with more than 20 years in trade, but not with job status (welder, etc)
25	Canadian asbestos insulators over age 50 n=88	Insulators vs reference population	35% had airflow obstruction, associated with diffuse pleural abnormality and history of work in pulp mills
26	US asbestos union members with normal x-rays n=113	Duration of work in trade	Significant decrease in FEV_1/FVC% and $FEF_{25/75}$ with more than 20 years in trade Effect of more than 20 years similar to effect of smoking Effects are additive
27	Italian asbestos cement factory workers n=65	Duration of work in trade; asbestos workers vs controls	Significantly greater FEV_1 and FVC decline in asbestors workers FVC decline increased with more than 15 years in trade
28	US Wollastonite miners n=108	Cumulative exposure; miners vs controls	Significant dose-response of dust with FEV_1/FVC More pronounced in nonsmokers

$FEF_{25/75}$ Maximal midexpiratory flow rate; FEV_1 Forced expiratory volume in 1 s; FVC Forced vital capacity

pronounced among nonsmokers than among smokers. A case-control study of South African gold miners (16) found that 20 years of working in a high dust exposure job was associated with a 13-fold increase in emphysema risk, independent of the effects of smoking. Studies of autopsied lungs (17-20) supported the results of the above epidemiological studies that exposure to mining dusts (coal or silica) were found to increase the risk of developing emphysema.

Airflow obstruction has also been demonstrated in association with asbestos exposure (Table 1) (21-27), and dose-response relationships are apparent (21,24,26). Airflow obstruction tends to be associated with both pleural and parenchymal radiographic abnormalities in asbestos exposed subjects. Wollastonite miners (28) also showed a dose-response relationship of cumulative exposure with decreased lung function; the relationship was more pronounced in nonsmokers.

ORGANIC DUSTS

Many organic dusts have been shown to give rise to occupational asthma. It has also been demonstrated that the majority of patients with occupational asthma do not recover several years after removal from exposure and some patients develop persistent chronic airflow limitation requiring medications for treatment (29).

Cotton dust: Whether cotton dust exposure has chronic effects on the lungs capable of disabling those affected has been the subject of debate for many years and is still not resolved. Neither pathology studies (30,31) nor mortality studi- es (32) concerning cotton dust showed convincing evidence of a relationship between cotton dust exposure and emphysema.

Cross-sectional studies concerning the health effects of cotton dust exposure (33-35) show a dose-response relationship between exposure and the prevalence of chronic bronchitis and/or lung function level. The results of the

TABLE 2
Grain dust exposure: Relationship between exposure and airflow limitation in studies controlling for the effects of smoking

Reference	Population	Exposure measure	Relationship between exposure and outcome
44	New York grain workers n=55	Grain workers vs bakers	Grain workers had significantly lower lung function than bakers Effect of smoking similar to effect of exposure No smoking-exposure interaction
45	Thunder Bay grain workers n=441	Grain workers vs civic controls	Grain workers had significantly lower lung function than civic workers Effect of smoking greater than effect of dust exposure No smoking-exposure interaction
46	St Lawrence grain (n=103) and port workers (n=39)	Grain vs port workers	Grain workers had significantly lower lung function than port workers Effect of exposure greater than effect of smoking No smoking-exposure interaction
47	Vancouver grain workers n=610	Grain workers vs civic controls	Grain workers had significantly lower lung function than civic workers Effect of exposure similar to that of smoking No smoking-exposure interaction
48	Saskatchewan grain workers n=390	Grain workers vs civic controls	Grain workers had significantly lower lung function than civic workers Effect of exposure similar to effect of smoking Smoking and exposure effects additive
49	Alberta grain workers n=63	Grain workers vs civic controls	Grain workers had significantly lower lung function than civic workers Effect of smoking greater than effect of exposure No smoking-exposure interaction

longitudinal studies (36-40) are not as consistent. For those studies with dust measurements, a dose-response relationship was found between the annual decline in FEV_1 and the degree of dust exposure. A cross-sectional study of active and retired cotton textile workers in the United States (41) showed a high prevalence of respiratory symptoms among these exposed workers in comparison with controls. A longitudinal study of this cohort (42) showed greater declines in FEV_1 than for controls, even within each smoking group. The average FEV_1 declines were 42 mL/year and 25 mL/year, respectively. It was concluded that chronic lung disease was not only more prevalent among cotton workers, regardless of smoking group, but that the disease may progress after exposure to cotton dust had ended.

Grain dust: The impact of grain dust on the lungs has been reviewed recently in detail (43). Several syndromes have been reported arising from exposure to grain dust: grain dust asthma, asthma-like syndrome, hypersensitivity pneumonitis, and skin and mucous membrane irritation. Although the acute airway diseases to grain dust have been recognized, it is not as clear whether chronic exposure to grain dust leads to chronic airflow limitation with disability. The majority of the cross-sectional studies showed increases in chronic respiratory symptoms and lower lung function in grain workers compared with the unexposed controls (Table 2) (44-49).

The first five cross-sectional surveys of grain elevator workers in the Port of Vancouver showed consistently that, within each smoking group, grain workers have lower lung function than a group of controls studied in the same manner over the years (50). An exposure-response relationship was also shown, which persisted across all smoking categories (51). Longitudinal analysis of workers who took part in the first three years showed that grain workers had a more rapid decline in lung function than the control group; the disparity in FEV_1 decline was greater among nonsmokers (–31 mL/year versus +4 mL/year) than among smokers (–37 mL/year versus –31 mL/year) (52). After 12 years of follow-up, longitudinal changes in lung function were comparable between the two groups, but selection effects were apparent in that dropouts had more symptoms and lower lung function in the initial survey (50). Retired grain workers had significantly lower lung function compared with the controls and a significantly higher proportion with FEV_1 below 80% predicted after adjustment for smoking (53). An exposure-response relationship was also found in this group of retirees. There was no improvement in the lung function of the grain workers after their retirement to indicate that the effect of grain dust exposure is reversible.

Agricultural dust: In agriculture, organic dust particles are formed from microbial cells and spores, abrasion between fragments of feed and bedding, from milling, grinding or chopping feeds, and include animal dander, urine and feces (54).

A review of 14 cross-sectional studies of swine confinement workers by Donham (55) in 1990 found cough and phlegm to be the most common symptoms, ranging from 12 to 55% across the studies.

When unexposed control groups were used, differences in the prevalence of respiratory symptoms, chronic bronchitis and lung function were usually found. For example, dairy farmers were matched to nonfarmer controls by sex, age, height and smoking habits (56). The farmers had a higher prevalence of chronic bronchitis (12% versus 6%) and lower lung function.

Differences in respiratory outcomes were also found, dependent on the type of farming. Swine confinement farmers were found to have more symptoms of chronic bronchitis (28% versus 17%) and lower lung function than grain farmers, even after adjustment for smoking (57). A different study of Canadian farmers found dairy and pig farmers had lower lung function than grain farmers, who were no different from nonfarming controls (58). Exposure-response relationships have been suggested in that the number of hours of work per

TABLE 3
Mixed mineral dust and fume exposure: Relationship between exposure and airflow limitation in studies controlling for the effects of smoking

Reference	Population	Exposure measure	Relationship between outcome and exposure
65	Canadian miners and smelter workers n=241	Miners, smelter workers vs population controls	Significant reduction in airflow rates in exposed smokers, most prominent among smelter workers Decreased airflow rates in association with increased duration of employment (miners only) Positive smoking-exposure interaction
66	British engineering factory welders n=258	Welders vs controls	No difference in airflow rates between welders and controls for the total group A subset of smoking welders had small reductions in flow rates at low lung volumes
67	British shipyard welders and caulkers/burners n=607	Average total welding fume exposure	Significantly reduced airflow rates associated with average exposure in smokers and ex-smokers Positive interaction between welding exposure and age effects on airflow
68	British shipyard welders and caulkers/burners n=488	Welders, caulkers/burners vs other trade	Annual decline in FEV_1 2 times greater in welders or caulker/burners than in other trades Significant positive interaction between smoking and exposure
69,70	US rubber manufacturing workers (curing, n=121; production, n=65)	Job type vs controls, duration of exposure	Significantly decreased FEV_1/FVC associated with production work Significantly decreased FEV_1 associated with increasing years of exposure to dust Steeper FEV_1 slope associated with increased years of employment in curing department

FEV_1 Forced expiratory volume in 1 s; FVC Forced vital capacity

day in confinement buildings was negatively correlated to FVC (57) in a Canadian study, while a 12 mL reduction in FEV_1 was associated with each year of hog farming in a Danish study (59).

Wood dust: Exposure to many of the exotic woods has been shown to give rise to occupational asthma. Exposure to wood dust has also been found to give rise to chronic respiratory symptoms and lower lung function in surveys of woodworkers compared with unexposed controls.

A greater prevalence of chronic cough and phlegm and lower lung function was found among Canadian sawmill workers exposed to Western red cedar as opposed to other types of wood dust such as hemlock and fir (60). Compared with office workers with no exposure to wood dust, cedar sawmill workers had higher odds ratios of cough and phlegm and lower lung function after adjusting for differences in smoking habits (61). The decrease in lung function was not due to the increased prevalence of asthma in cedar mills since exclusion of these subjects from analysis failed to influence the results. The annual decline in lung function in cedar workers was also significantly greater than in the control group (62) (–29 mL/year versus 2 mL/year in nonsmokers), suggesting that workers exposed to Western red cedar are at risk for developing chronic airflow obstruction.

A study was conducted on workers exposed to hardwood dust (mostly maple) and varying levels of soft wood dust (pine) (63) who did not have exposure to other industrial agents such as adhesives and finishing agents. Workers in the high exposure category to both hard wood and pine dust had two to four times the prevalence of low expiratory flow rates compared with those exposed to lower levels of dust, irrespective of their smoking habits. Cabinet workers exposed to different types of wood (64) reported more cough, phlegm and wheeze but their mean lung function was not significantly different from that of the controls.

MIXED DUST, FUMES AND GAS EXPOSURE

Studies of chronic airflow obstruction in groups exposed to mixtures of dust, fumes and gases have often found a significant excess of chronic airflow limitation in these workers (65-70), although there are exceptions (66) (Table 3). Such exposures are found in mineral processing (steelmaking, foundries, smelters) and in a wide variety of other industries ranging from rubber manufacturing to firefighting. Temperature extremes are also generally found in these work environments.

Mixed or nonspecific exposures have also been examined in several population-based cross-sectional (71-74) and longitudinal studies (75-76) (Table 4). In these studies, exposure measurements are seldom available; rather, exposure is assigned based on responses to questions regarding general work exposures, job and industry titles, and employment duration. As such, it is important to bear in mind that the inevitable misclassification of exposure will result in a diminution of effect. The populations studied have included rural and urban locations from the United States, Norway, France, China and Poland. The studies generally found increased prevalence rates for cough and phlegm and measures of airflow obstruction for those with mixed dust exposures, after controlling for the effects of smoking, and dose-response relationships were often seen (77). The effect of temperature extremes and gas or fume exposure in addition to dust appears to be additive in most studies. In a longitudinal study of Paris area workers, Kauffmann (75) found that the rate of longitudinal decline in FEV_1 in men with no change in smoking habit increased with exposure combinations as follows:

TABLE 4
Population based studies: Relationship between exposure and airflow limitation in studies controlling for the effects of smoking

Reference	Population	Exposure measure	Relationship between outcome and exposure
71	Sample of residents from 6 US cities n=8515	Questionnaire: job with potential exposure to dust, gas/fumes	Increased relative odds for airflow limitation associated with dust alone (OR=1.7) and with dust and fumes combined (OR=1.6) No smoking-exposure interaction
72	Stratified sample of residents of Hordaland county, Norway n=714	Questionnaire: job with potential 'airborne exposure' (3 levels)	Increased relative odds for obstruction associated with high level exposure jobs (OR=3.6) Effect of exposure more pronounced in older workers No smoking-exposure interaction
73	Residents from 24 areas of 7 French cities (excluding households headed by a 'manual worker') n=12,182	Questionnaire: exposed to dust, gases or chemical fumes in any job	FEV_1/FVC significantly reduced in exposed group Effect of exposure most pronounced in older subjects. Exposure effect about half the smoking effect
74	Sample of residents of Beijing not using coal for heat n=1094	Questionnaire: cumulative exposure (3 levels) to dust or to gas/fumes	Significant reduction in FEV_1 and FEF_{25-75} associated with exposure to dust (compared to no exposure) Significant dose-response trend for FEV_1 related to increasing gas/fumes exposure among exposed subjects Signicant interaction between smoking and dust exposure on FEV_1/FVC
75	Workers from 11 Paris factories n=556	Technical survey: exposure to dust (5 levels), gases (2 levels), heat (3 levels)	More rapid decline in FEV_1 associated with dust alone, heat alone, and gas in combination with dust and/or heat Steeper slopes among unskilled workers Workers in 30-39 age group had higher initial FEV_1 but equally steep slopes as for older workers
76	Random sample of residents of Cracow n=1679	Questionnaire: history of exposure to dusts, variable temperature, chemicals	More rapid decline in FEV_1 associated with prolonged exposure to variable temperature, and acute exposure to irritating gases Exposure regression coefficients larger than that for smoking; no smoking-exposure interaction

$FEF_{25/75}$ Maximal midexpiratory flow rate; FEV_1 Forced expiratory volume in 1 s; FVC Forced vital capacity; OR Odds ratio

no exposure or exposure only to slight dust or only to gases (44 mL/year); exposure to noticeable dust (50 mL/year); exposure to heat (59 mL/year); exposure to noticeable dust and heat (56 mL/year); and exposure to noticeable dust, high level of gases, and heat (67 mL/year). Similar combinations of the effect of variable temperature and chemicals were also seen in a 13-year follow-up study of Cracow residents (76,77). A threefold increase in the risk for emphysema has also been shown to be related to exposure in a population-based case-control study (78) in Norway.

CONCLUSIONS

Both epidemiological and autopsy findings support the suggestion that exposure to inorganic dust is causally associated with chronic airflow obstruction because they satisfy most of Hill's criteria for causality. In particular, the demonstration of an exposure-response relationship according to the degree of dust exposure provides a strong basis for concluding that there is a causal relationship, particularly where cigarette smoking has been taken into account in the design or analysis of the studies.

The results of studies of organic dusts are not nearly as uniform. Studies of cotton and grain dust tend to show cross-sectional differences in lung function between exposed and unexposed groups; the findings for longitudinal studies were less consistent. The studies of retired grain workers provide further evidence of the deleterious effects of exposure to grain dust, which confirms the previous findings of chronic airflow limitation. Farmers tend to show increased respiratory symptoms and lower lung function, compatible with chronic airflow limitation. The degree of limitation appears to be dependent on the type of farming, such as animal confinement, but cumulative exposure analyses to determine dose-response relationships have not yet been investigated. Exposures to different types of wood dust were found to be associated with chronic respiratory symptoms and some impairment of lung function; however, further studies are necessary on woodworkers with specific exposure to individual species of wood in order to delineate the respiratory effects of exposure.

There is sufficient evidence that occupational exposures to dust can result in chronic airflow limitation. In most instances no statistical interaction was shown between smoking and dust exposure, ie, they were separate additive effects. In many studies, the effects of dust exposure were found to be more pronounced than what was found for smoking. Not all dust exposures are implicated, nor all levels of exposure, nor all circumstances under which an exposure might occur. However, the evidence is strong enough, particularly in relation to inorganic dust and grain dust exposure, that an occupational history should be considered as integral a part of the investigation of a patient with chronic airflow limitation, as is the smoking history. Further study of the chronic respiratory effects of organic dust exposure is warranted, especially where quantitative estimates of both dust exposures and smoking are considered.

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