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Feasibility study of respiratory questionnaire and peak flow recordings in autobody shop workers exposed to isocyanate-containing spray paint: Observations and limitations

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Diisocyanates, highly reactive monomers which cross-link polyurethane, are the most widely recognized causes of occupational asthma. Many exposed workers are end-users, including autobody spray painters who form a large population at risk. Neither the factors which determine incidence rate nor strategies for control have been adequately studied in this setting. We have conducted a cross-sectional survey of 23 (about one in five) autobody shops in the New Haven area to determine the feasibility of clinical epidemiological studies in this population. Among 102 workers, there was a high rate of airway symptoms consistent with occupational asthma (19.6%). Symptoms were most prevalent among those with the greatest opportunity for exposure (dedicated spray painters) and least among office workers; part-time painters had intermediate rates. Atopy was not associated with risk while smoking seemed to correlate with symptoms. Regular use of air-supplied respirators appeared to be associated with lower risk among workers who painted part- or full-time. We were unable to validate the questionnaire responses with peak expiratory flow record data attempted on a 1/3 sample of the workers. Despite intensive training and effort, subject compliance was limited. Among those who provided adequate data (24 of 38), only two demonstrated unequivocal evidence of labile airways; two others demonstrated lesser changes consistent with an occupational effect on flow rates. There was no clear association between these findings and either questionnaire responses or exposure classification. Overall, the survey suggests that there is a high prevalence of airway symptoms among workers in autobody shops, at least in part due to work-related asthma. However, there is need for both methodological and substantive research in this setting to document rates of occupational asthma and to develop a scientific basis for its effective control.

Key words: Autobody shop; isocyanates; occupational asthma; peak expiratory flow record; spray paint.

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INTRODUCTION

Diisocyanates, such as toluene diisocyanate (TDI), methyl diphenyl diisocyanate (MDI) and many aliphatics, are a family of chemicals used to polymerize polyurethane resins. Typically, the highly reactive molecules are generated in the finishing process of these thermoset plastics. NIOSH has estimated that at least 50,000–100,000 workers in the US are exposed to various isocyanates, the majority in end-user applications such as painting or foaming.[1] This widespread use is paralleled by numerous reports of isocyanate-related diseases, the most common being asthma.

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Several recent studies show that isocyanate asthma currently accounts for 31–54% of all occupational asthma cases diagnosed in tertiary referral centres.[2,3,4]

Several research groups worldwide are trying to uncover the pathophysiological mechanism(s) involved in isocyanate-induced asthma. Possibly involving diverse immunologic mechanisms,[5–8] exposure-host-disease relationships have not been adequately characterized. IgE antibody is observed in only a fraction of isocyanate asthma cases.[6,9,10] Recent clinical studies have suggested a primary role for T lymphocytes.[9,10] This form of asthma is currently grouped in the class of 'low molecular weight asthma'.[11]

Risk and protective factors for isocyanate asthma are poorly understood and only a few studies have addressed exposure characteristics in their relationship to occupational asthma incidence and prevalence. Atopy does not appear to be a risk factor.[10,12] Acquired host factors like smoking, individual work practices and exposure patterns may play a role in susceptibility to the disease process.[10] Excessive short-term isocyanate exposure during work accidents may induce greater risk than continuous low dose exposures.[10,13,14]

Autobody shop workers, one of the largest groups of polyurethane painters in developed countries, are heavily exposed to isocyanates while spray-painting, most commonly to aliphatic isocyanates such as HDI (hexamethylene diisocyanate).[8,15] Routine exposure monitoring in autobody shops is done only in a few US states. Data from routine surveillance by OSHA in Oregon between 1980–1990 showed that two-thirds of all samples exceeded the Oregon permissible exposure level (PEL) for polyisocyanates of 0.5 mg/M^3 (8 hour TWA), itself high relative to the Swedish standard (0.09 mg/M^3) and the recommended standard in the UK (0.07 mg/M^3).[16] Compressed air spray guns were identified as the major generator of paint aerosols.[16] Oregon ventilation standards of 30 air exchanges in a spray booth per hour spray-painting were not met in one third of the facilities.[16,17] Despite these alarming industrial hygiene data, neither the risk for asthma in these shops nor the impact of protective equipment or ventilation in these shops is well studied.

Few studies exist of isocyanate-asthma in spray-painters. Welinder *et al.*[8] showed in a cross-sectional study of 30 car painters from 21 shops that 33% had occupational asthma symptoms and two workers (6.7%) had documented isocyanate-asthma. Using a cross-sectional design, Seguin *et al.*[18] showed an isocyanate-asthma prevalence of 11.8% among 51 airplane spray painters. Focusing on chronic lung function changes, Tornling showed in a 6-year follow-up of 36 car-painters and 115 controls that smoking painters had an additional loss in lung function compared to smoking controls which correlated well with their peak but not with their mean isocyanate exposures.[14] These studies, using differing strategies for assessment of asthma symptoms and physiological changes, underscored the current deficiencies in available methodologies to study occupational asthma in such a setting.

To address some of the limitations to present knowledge, we developed a preliminary proposal with the local autobody business association to study the problem, with three goals. First, we wanted to test the utility of a questionnaire as an effective screening tool for occupational asthma in this industry. Second, we hoped to utilize peak expiratory flow rate (PEFR) to validate this approach.[19–25] Finally, we hoped to correlate exposure, shop and individual characteristics with asthma risk in order to identify potential strategies for disease control.

METHODS

Autobody shops in New Haven County were chosen using a random number table from among 105 listed in the area's Yellow Pages. When a shop refused or requested additional information, the one listed immediately following was contacted as an alternative until at least 20 agreed.

Using an interviewer-administered questionnaire, shop owners were questioned in person about different characteristics of their autobody shop. The following variables were assessed: shop size, spray paint systems in use, protective devices available to workers, number of employees, availability of health benefits for the employees, productivity measurements (car turnover per week) and annual revenue. Size, paint types, payroll, benefits and turnover were verified in employee interviews.

Industrial hygiene evaluation involved an observational walk-through of each shop and a review of all materials and work practices in that facility. The type(s) of ventilation in use, presence and type of spray paint booth and condition of these controls was recorded.

All currently employed workers at the time of the questionnaire survey (shop floor workers, office workers as well as shop owners) were briefly surveyed during the walk-through visit. Information regarding recent changes in personnel was solicited from the employer and employees. The survey instrument was a modified ATS-questionnaire: we supplemented respiratory symptom questions from the ATS questionnaire with questions regarding the year of symptom onset and the occurrence of symptoms in relationship to work schedules. The instrument was administered by one of four interviewers. The presence of asthma symptoms (cough, wheezing, shortness of breath) as well as their occurrence in a work-related pattern (more frequent at work or better on days off from work) were assessed. A worker was defined as demonstrating 'occupational asthma symptoms' if at least one of these symptoms was reported, stated to occur in a work-related pattern and beginning after the subject had begun work in an autobody shop.

Other parts of the questionnaire obtained demographic and health information. The following variables were queried: age, race, asthma diagnosis by a physician in the past, atopy (defined as self-reported eczema or hayfever), and health problems since in the industry. Smoking was assessed in three categories: non-smoker, ex-smoker or current smoker. Detailed information on the individual's work history (number of years in the industry and in the particular shop) and current work practices (office vs. shop floor work, weekly hours of spray painting, use of personal protective devices when painting, type of protective equipment used) was requested.

A job-exposure matrix was designed to stratify workers according to their presumed potential for isocyanate exposure:

- *office workers:* minimal exposure to the shop area
- *shop floor workers in three different exposure categories:* (1) no spray painting (but exposed to all materials used on the shop floor); (2) < 5 hours/week of spray painting; (3) 5–20 hours/week of spray painting
- *dedicated painters:* > 20 hours spray-painting per week

We tested the questionnaire for its reliability among a subgroup of workers to obtain a summary measurement of questionnaire, interviewer and subject reliability. A different interviewer readministered the same instrument after a time interval of 4–6 months. The questionnaire consistency was determined for a sample of key questions: all symptom questions and their patterns, history of asthma, active car painting and the use of a protective device. Consistency for each question was measured as per cent exact agreement on all categorical questions; per cent exact agreement on year was assessed for symptom duration.

To correlate the symptom questionnaire results with physiological measurements, employees from a subset of cooperating shops were supplied with portable peak flow meters (Min-Wright, Clement Clarke, Columbus, OH) and taught how to do peak expiratory flow rate (PEFR) recordings. Each employee in these shops was asked to keep a peak expiratory flow rate diary for a recording period of 17 consecutive days including two weekends. When possible, we selected shops with more than one symptomatic worker. These symptomatic workers, plus all their shop coworkers, were included as the validation sample. Daily work data were recorded parallel to PEFR measurements; e.g. on or off work day; active spray painting; spray painting by coworkers.

After initial group teaching of the PEFR technique, performance of each worker was observed separately on that day as well as on shop visits on at least two occasions throughout the recording time. For each given day the worker was asked to obtain peak expiratory flow rates at 7 a.m., 11 a.m, 3 p.m and 7 p.m., or within one hour of these time points. At each time point three peak flows were to be obtained and marked in the diary. Anticipating that the evening times might be the most difficult recording times to remember, each worker received a wrist watch alarm programmed to signal at 7 p.m. Signs were posted throughout the shops reminding workers of the peak flow recording times. We tried to enhance weekend recording by regular shop visits on Fridays. Workers were instructed to record missed times as such.

Analysis of the PEFRS and diaries were performed blinded with the reviewer being unaware of the questionnaire results of these subjects. In our analysis we only used the single highest of the three PEFR recordings at each time point. Because of a learning effect in new users of peak flow meters, we decided *a priori* to discard the first two days of the diaries. A diary was judged sufficient for this phase of analysis if three or more peak flows on at least two work days were obtained (after discarding the first two days). We determined the diurnal variability (DV) or variability per cent mean, of each day for which adequate data was available using the following mathematical formula:

$$DV = \frac{(\text{highest PEFR} - \text{lowest PEFR}) \times 100}{\text{mean PEFR of that day}}$$

Maximal diurnal variability was the highest DV on any day. Asthma was defined conservatively as a maximal DV exceeding 20%.

A second level of analysis for the peak expiratory flow records was assessment of variability in relation to work shifts and activity. A diary was considered suitable for this level of analysis only if at least four work plus two off days with at least three (× 3) blows were recorded. The method of analysis was subjective judgement by two of the investigators who were blinded to exposure and symptom classification.

Categorical variables were tested using χ^2 analysis of tables with $p < 0.05$ significance. Fisher's Exact Test was used for tables with cell counts below five. The analysis of $2 \times \kappa$ tables were done with χ^2 test for linear trend. Continuous variables in groups were assessed by analysis of variance.

The study was approved by the Human Investigation Committee of the Yale University School of Medicine.

RESULTS

Shop characteristics

In total 23 autobody shops were selected and visited. The shops had an average of 4.2 ± (SD) 2.6 employees with a range from 1–11 employees. Productivity of the shops ranged from 1/2 to 43 cars per week, with a mean car turnover of seven cars. Annual revenue was from $10,000 to $3 million per year with an average annual revenue of $300,000. These data are summarized in Table 1.

The majority of autobody shops had a spray booth with or without a cross or down-draft ventilation system. Paint was applied to cars by spray guns. Paint mixing was usually performed in a designated area which typically was supplied with a separate ventilation system. Importantly, spray painting often occurred outside paint

Table 1. Autobody shop characteristics ($n = 23$ shops)

	Mean	*SD*	*Range*
Annual revenue (in US $)	300,000	693.400	10,000–3,000,000
Shop size (sq. ft)	5122.5	396.5	500–14,000
Number of employees	4.2	2.6	1–11
Productivity (cars/week)	4	8.86	0.5–43

Figure 1. Part-time painter spraying hub of a truck wheel. Typical of many shops, these 'small' jobs occurred outside the spray painting booths and without the benefit of air-supply respirators. The potential for both indirect aerosol and vapor exposure is apparent despite the use of the cartridge respirator.

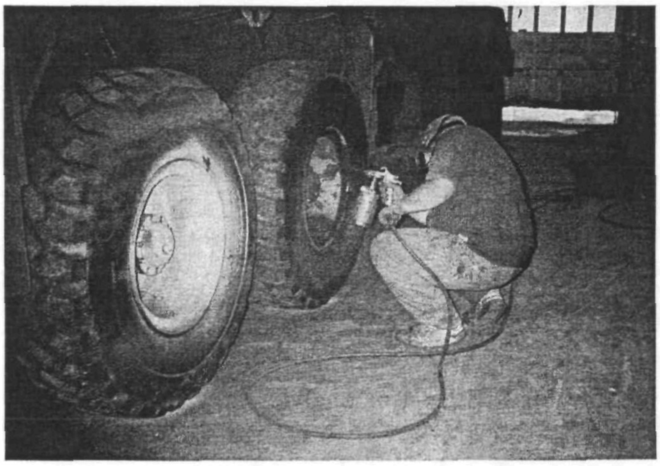


Table 2. Subject characteristics ($n = 102$ subjects)

Characteristic	*Mean*	*Range*	*n*	*%*
Age in years	34	(17–70)		
Years in the shop	4	(0.003–27)		
Years in the industry	10	(0.04–41)		
History of atopy				
Yes			32	31.4%
No			70	68.6%
Race				
Caucasian			89	87.3%
Other			13	12.7%
Health benefits				
Yes			76	74.5%
No			26	25.%%
Smoking status				
Non-smokers			41	40.2%
Ex-smokers			24	23.5%
Current smokers			37	36.2%

booths for conduct of 'small' jobs (Figure 1). Shop and office areas were usually within the same building, allowing for interactions between office and shop personnel.

Typical repair jobs consisted of structural repair, surface preparation and painting. Respiratory protection equipment used during spray-painting ranged from simple paper masks and twin charcoal negative pressure respirators to positive pressure respirators with a fresh air line.

Different brands of paint containing aliphatic isocyanates were in use. The literature describes HDI and partially polymerized HDI derivatives as the major hazards;[15,16] our survey confirmed this. The paint systems were two-compound paints prepared in the autobody shop; a polyol with pigments and solvents had to be mixed with polymeric isocyanates in a solvent. Exposure measurements were not obtained although inspection of the work procedures showed spray painting as the work with the highest exposure to paint aerosols.

Body shop employees

A total of 102 employees from 23 autobody shops were interviewed regarding their biographic data, respiratory symptoms and job characteristics.

The majority of the 102 workers were Caucasian ($n = 89$), the remainder being Hispanic and Afro-American ($n = 13$). On average, they worked in their current shop for four years and in the industry for 10 years. All but two, both office workers, were men. Distribution of atopic status, race, smoking category, and presence of health benefits are summarized in Table 2.

Based on our job-exposure matrix, the 102 workers were distributed fairly evenly with respect to category based on location and spray paint use, as shown in Table 3. Employees involved in spray painting operations used different respiratory protection devices, the device with the best protection factor being a positive pressure (air-supply or air-line) respirator, used by 12 (20.3%). Forty workers (67.8%) used negative pressure dual respirators while the rest used lower levels of protection or none. These data are summarized in Table 4.

Symptoms

Although no subject reported a prior diagnosis of asthma made by a physician, about one-fifth of all workers fulfilled the questionnaire definition for occupational asthma ($n = 20$; 19.6%). The distribution of questionnaire-positive individuals within the five exposure categories is shown in the last column of Table 3. A dose-response correlation from low to high

Table 3. Distribution of workers with occupational asthma symptoms by exposure category

Exposure categories	*Job description*	*Number of workers (% of total)*	*Symptomatic subjects (% of workers in category)*
1	Office employee	15 (14.7%)	1 (6.7%)
2	Work on shop floor—never paints	28 (27.5%)	7 (25.0%)
3	Work on shop floor, paint < 5 hrs/wk	23 (22.5%)	4 (17.4%)
4	Work on shop floor, paint 5–20 hrs/wk	22 (21.6%)	3 (13.6%)
5	Dedicated painter, paint > 20 hrs/wk	14 (13.7%)	5 (35.7%)
Total	All workers	102 (100%)	20 (19.6%)

Table 4. Respirator use among spray-painters

		Occupational asthma symptoms	
Workers with paint exposure (n = 59)	*Total workers n (%)*	*Absent (n = 47)*	*Present (n = 12)*
Workers who did not use positive pressure respirator	47 (79.7%)	36 (76.6%)	11 (23.4%)
Workers using positive pressure respirator	12 (20.3%)	11 (91.7%)	1 (8.3%)

spray painting frequency is suggested when the mild to moderate isocyanate exposure groups (categories 2–4) are grouped together: 6.7% of office-workers, 18.6% of shop floor workers and 35.7% of dedicated painters showed respiratory symptoms of cough, wheeze or shortness of breath in a work-related pattern. For the grouped data, the χ^2 test for linear trend was 2.55 with $p = 0.11$.

Analysis of smoking data showed 50% of the individuals with occupational asthma symptoms to be smokers ($n = 10$); there were more non-smokers (67%; $n = 55$) among the questionnaire-negative individuals. Testing for smoking as a predictor for occupational asthma symptoms (current smokers vs. non-smokers and ex-smokers) suggested smoking to be a modest risk factor (Odds Ratio [OR] = 2.0, $p = 0.15$). Reported atopy, on the other hand, showed no correlation with asthma symptoms.

Occupational asthma symptoms were found three times more frequently among painting shop-floor workers and dedicated painters (exposure categories 3–5) who did not use a positive pressure respirator (23.4%) than among those who used it (8.3%), but the difference was not statistically significant (OR = 3.36, Fisher's Exact p-value 0.42). (See Table 4.)

Questionnaire reliability testing

The average consistency (complete agreement between surveys) measured 66.7%. Responses to questions involving year of onset of wheezing and shortness of breath were much less consistent (wheezing 9%; shortness of breath 25%). The consistency for all other parameters tested varied between 66–90%. An average consistency of 77.4% was achieved when all three time recall questions were excluded.

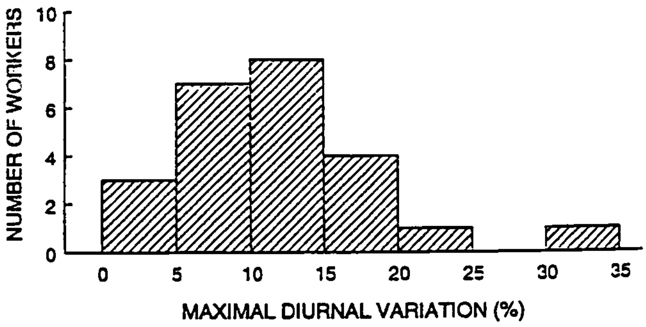
Peak flow validity testing

We selected questionnaire-positive individuals ($n = 10$) plus all their shop coworkers ($n = 28$) for serial PEFR measurements. The ten selected subjects were in shops ($n = 7$) with more than one questionnaire-positive individual ($n = 3$) or from shops within easy reach of the survey team ($n = 4$). A total of 38 workers participated in the PEFR training sessions (37.3% of the study population). Despite intensive training and interim shop visits, only 24 workers (63.4%) of these workers, including all ten with asthma symptoms, obtained measurements sufficient for diurnal variation analysis; 11 failed to produce sufficient peak flows for analysis and three left work before the end of the survey. The subset of 24 workers with adequate PEFR data showed a similar distribution of exposure categories to the total sample, but were on average three years older and were working in the shop and industry about twice as long as the larger group. This group recorded an average of 9.5 days with 7.3 work days and 1.4 off work days, with 0.8 days unspecified.

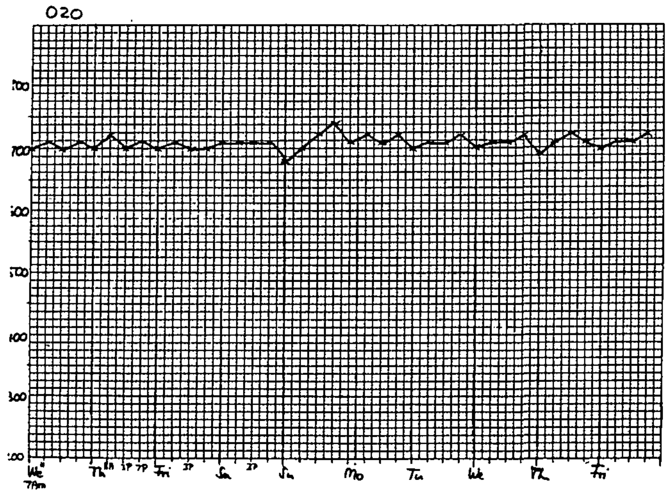
Examining the idea that a 2-day learning time is necessary, we compared the span and absolute values of PEFR from the first two days to the span of all other recorded days. Fourteen workers (53.8%) showed neither a change in PEFR range or absolute minimum and maximum values, but about a quarter of the subjects ($n = 7$; 26%) showed a learning effect.

The highest PEFR variability (maximal per cent diurnal variability) was determined for each worker. The distribution of these results is shown in Figure 2. Two of 24 workers (8.3%) fulfilled the maximal DV > 20% criterion for excessive peak flow variability. Both workers were non-smokers. The diaries of both workers failed to contain the four work plus two off-

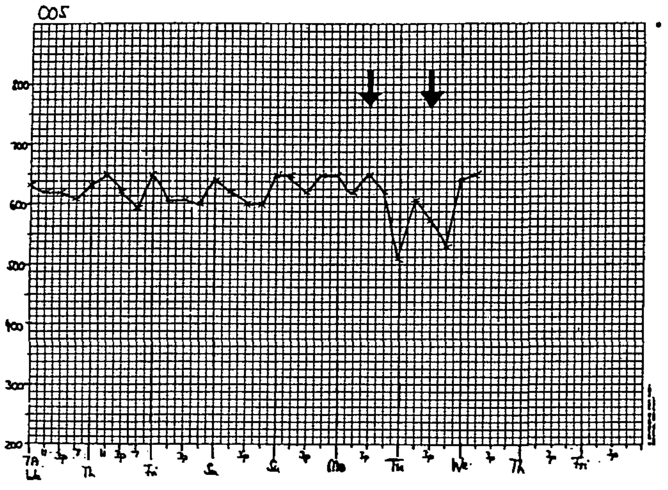
Figure 2. Bar chart of maximal peak flow diurnal variation among the 24 surveyed subjects who provided adequate data for computation. The distribution is roughly normal, without suggestion of an obvious asthmatic subgroup.



Figures 3a and 3b.
A. Like many of the workers who complained of some symptoms consistent with occupational asthma, this subject had a completely normal peak flow record, with adequate data for interpretation.



B. One of two symptomatic workers who showed evidence of a drop in flow rate consistent with occupational asthma. Note the daily morning drops (arrows) during the two workdays recorded.



work days sufficient for analysis for 'work-related peak flow variability'. Significantly, neither of these subjects demonstrated asthma symptoms on the questionnaire; in fact, both were currently asymptomatic (see below).

The subjects' peak flow variabilities were compared with their exposure categories. Interestingly, both workers with maximal DV > 20% were current office workers ($\chi^2 = 24$, Fisher's Exact $p = 0.004$); subsequent re-questioning revealed that each had transferred into the office in part *because* of airway symptoms which had occurred while in the shop environment. Results on the remainder were unassociated with exposure category.

Among the ten subjects with occupational asthma symptoms, maximal diurnal variability did not differ from the remainder of the group. In six of these subjects, sufficient data were available for qualitative assessment of work variability. Two tracings showed patterns suggestive of an occupational airway effect (despite maximal DV < 20%, while no pattern was evident in the remainder (see Figure 3A).

DISCUSSION

Unlike many occupational lung diseases, which typically occur in large mining or factory environments, occupational asthma has typically occurred in sporadic form, often in very small workplaces and shops.[26] This is especially true of diisocyanates because of the thermoset nature of the polyurethane resin system, requiring the end user to work directly with unreacted monomers. In our previous clinical experience, sprayers of polyurethane paints, such as workers in small autobody shops, have appeared to represent a very high risk group.[2] This may be in part due to the very high number of exposed workers. Assuming the observed rates are true for all New Haven County, then about 500 persons or 1 per 1,000 in the total county population works in this industry, over half with some direct painting involved. This extrapolates to over 125,000 autobody painters among one quarter million autobody workers in the US population.

Small industries, like autobody shops, pose special challenges to occupational epidemiologists, despite their high prevalence and accessibility. Geographic dispersion and non-uniform work structures, work processes and exposures conflict with the epidemiologic research ideals of large localized populations and precisely delineated exposure classification. Furthermore, economic insecurity on the part of employers and employees limit the extent to which these participants can invest time and resources towards research as is possible in large industrial situations. We have attempted to fit a preliminary research design to these circumstances. This pilot study of 102 autobody shop workers in 23 shops was an attempt to familiarize ourselves with the specifics of the autobody industry and to evaluate the applicability of the methods chosen.

Our approach met with limited success. We were able to document a high rate of respiratory symptoms consistent with occupational asthma and consistent with rates reported among similar workers.[8,14,18] Internal content validity was reinforced by patterns similar to expectation based on clinical experience and the literature: increased risk with increasing opportunity for exposure; absence of strong association with atopy; apparent protection by use of air-supplied respirators and higher risk among current smokers. However, in a subset of workers we were unable to externally validate our survey instrument by the use of peak expiratory flow recordings. Recordings identified only two subjects with maximal DV > 20%, and both were currently asymptomatic, working in office environments, our lowest exposure category. Subsequent questioning revealed that each had transferred into the office from the shop environment because of earlier symptoms, a clear limitation of cross-sectional study design and our exposure classification scheme. We were able to identify two symptomatic individuals with both current exposure and suggestive work associated drops in peak flow (Figure 3B), but the majority of symptomatic workers either failed to provide adequate data for assessment, or failed to demonstrate exposure related variation in peak flow.

Because of the complexity and diversity of the work environments, we categorized workers according to their current job characteristics, classifying isocyanate exposure based on task descriptions. Such ordinal job ranking schemes are attempts to capture important exposure patterns in lieu of direct exposure assessment by environmental or biologic monitoring. Although some direct measurements have been reported from this industry,[14–17] none has been applied directly to classification of individuals or groups for epidemiologic study. As shown in our study, the job-exposure matrix partially explained the risk of symptoms: intrinsic misclassification and selection biases likely blunted this effect. Cross-sectional studies typically miss workers who change jobs or alter work exposures *because* of health problems, one aspect of the 'healthy worker effect.' We assume that shifts within the industry, suggested by our data, are paralleled by moves out of the industry. This is supported by the finding of three younger workers who left their jobs during our peak flow study and suggests a reason why no one with a medical history of asthma nor a marked degree of peak flow variability was identified in the survey.

As noted, we failed to confirm occupational asthma symptoms by the study of peak flow variability. Several factors may be responsible for this finding. First, occupational asthma symptoms as per our definition, are not disease-specific and some of these symptoms may be found in other diseases like tobacco-related chronic bronchitis or hypersensitivity pneumonitis; we have probably misclassified subjects with these diseases as questionnaire positive. Chronic bronchitis is particularly likely to have confounded our data since the subset of workers that participated in the PEFR survey were older and smoked somewhat more heavily than the group as a whole. Second, the modified ATS questionnaire used has not been studied adequately as a tool to assess occupational asthma. Asthma-specific questionnaires, now being tested may perform better in this setting. The need for a validated occupational asthma questionnaire is obvious.

Third, the 19.6% prevalence rate of respiratory symptoms in a work-related pattern may result from workplace irritants such as paint constituents and solvents, rather than true sensitization. Lee showed in his study of 26 polyurethane foam mixers that 50% of them had symptoms of mucus membrane irritation.[27] The finding that use of positive pressure respirators appears to protect from symptoms is consistent with this interpretation.

Despite initial enthusiasm[28] PEFR measurements themselves have not been established as a completely satisfactory tool for surveillance of occupational asthma. Neither the specific 'statistic' to be measured nor the choice of cut off values has achieved consensus. We used maximal diurnal variability with high cut off (20%) because of its strong correlation with other measures of airway reactivity. Cut-off levels of 16% and of 20% have been suggested.[23,28]

The major limitation of peak flow data in our study was the workers' poor participation and inadequate data collection which had been shown to be a problem in prior studies.[21] Although very inexpensive and useful in the clinical setting to diagnose and monitor a diseased individual, it may not be useful as a surveillance tool for 'healthy', less motivated workers in small scattered work places, despite extensive planning, training, motivational gimmicks, and reminder visits.

From a public health perspective, our preliminary survey supports the published impression that autobody shop workers are at high risk for respiratory symptoms and potentially occupational asthma. A correlation of symptoms with better established diagnostic tests such as specific broncho-provocation or immunologic markers would be needed to distinguish asthma from non-specific or irritative airway conditions. Further studies of the autobody shop industry are needed since our cross-section approach could not give any information on disease incidence and has possibly underestimated rates. Better studies to identify both exposure related and host risk factors for isocyanate asthma will be crucial for disease control given the intrinsic nature of the work organization and tasks. Meanwhile, in the absence of better data, public health efforts need to focus on worker education programmes to alert isocyanate-exposed workers to the possibility of diisocyanate-induced asthma and the potential value of air-line or other positive pressure respirators in the prevention of this disease.

REFERENCES

1. NIOSH. *National Occupational Exposure Survey.* USPHHS. PHS Centers for Disease Control, Publication No. 88–106. 1988.
2. Cullen MR, Cherniack MG. The spectrum of occupational disease in an academic hospital-based referral center in

Connecticut 1979–1987. *Arch Intern Med* 1989; **149**: 1621–1626.

3. Malo JL, Ghezzo H, L'Archeveque J, Lagier F, Perrin B, Cartier A. Is the clinical history a satisfactory means of diagnosing occupational asthma? *Am Rev Respir Dis* 1991; **143**: 528–532.
4. Moscato G, Dellabianca A, Vinci G, Candura SM, Bossi MC. Toluene diisocyanate-induced asthma: clinical findings and bronchial responsiveness studies in 113 exposed subjects with work-related respiratory symptoms. *J Occup Med* 1991; **33**: 720–725.
5. Butcher BT, Karr RM, O'Neil CE, Wilson MR, Dharmarajan V, Salvaggio JE, Weill H. Inhalation challenge and pharmacologic studies of toluene diisocyanate (TDI)-sensitive workers. *J Allergy Clin Immunol* 1979; **64**: 146–152.
6. Cartier A, Grammer L, Malo JL, Lagler F, Ghezzo H, Harris K, Patterson R. Specific serum antibodies against isocyanates: association with occupational asthma. *J Allergy Clin Immunol* 1989; **84**: 507–514.
7. Gallagher JS, Ted Tse CS, Brooks SM, Bernstein IL. Diverse profiles of immunoreactivity in Toluene Diisocyanate (TDI) asthma. *J Occup Med* 1981; **23**: 610–616.
8. Welinder H, Nielsen J, Bensryd I, Skerfving S. IgG antibodies against polyisocyanates in car painters. *Clinical Allergy* 1988; **8**: 85–93.
9. Fabbri LM, Maestrelli P, Saetta M, Mapp CM. Mechanisms of occupational asthma. *Clin Exper* 1994; **24**: 628–635.
10. Vanderplas O, Malo JL, Saetta M, Mapp CE, Fabbri LM. Occupational asthma and extrinsic allergic alveolitis due to isocyanates: Current status and perspectives. *Brit J Indus Med* 1993; **50**: 213–228.
11. Chan-Yeung M, Malo ML. Aetiological agents in occupational asthma. *Eur Respir J* 1994; **7**: 346–371.
12. Butcher, BT, Jones RN, O'Neill CE, *et al.* Longitudinal study of workers employed in the manufacture of toluene diisocyanate. *Am Rev Respir Dis* 1977; **16**: 411.
13. Mapp CE, Corona PC, DeMarzo N, Fabbri L. Persistent asthma due to isocyanates. A follow-up study of subjects with occupational asthma due to toluene diisocyanate (TDI). *Am Rev Respir Dis* 1988; **137**: 1326–1329.
14. Tornling G, Alexandersson R, Hedenstierna G, Plato N. Decreased lung function and exposure to diisocyanates (HDI and HDI-BT) in car repair painters: Observations on re-examination 6 years after initial study. *Am J Industr Med* 1990; **17**: 299–310.
15. Alexandersson R, Plato N, Kolomodin-Hedman B, Hedenstierna G. Exposure, lung function, and symptoms in car painters exposed to hexamethylendiisocyanate and biuret modified hexamethylendiisocyanate. *Arch Environ Health* 1987; **42**: 367–373.
16. Janko M, McCarthy K, Fajer M, van Raalte J. Occupational exposure to 1,6-Hexamethylene diisocyanate-based polyisocyanates in the State of Oregon, 1980–1990. *Am Ind Hyg Assoc J* 1992; **53**: 331–338.
17. Lesage J, Goyer N, Desjardins F, Vincent JY, Perrault G. Workers' exposure to isocyanates. *Am Ind Hyg Assoc J* 1992; **53**: 146–153.
18. Seguin P, Allard A, Cartier A, Malo JL. Prevalence of occupational asthma in spray painters exposed to several types of isocyanates, including polymethylene polyphenylisocyanate. *J Occup Med* 1987; **24**: 340–344.
19. Berube D, Cartier A, L'Archeveque J, Ghezzeo H, Malo JL. Comparison of peak expiratory flow rate and FEV_1 in assessing bronchomotor tone after challenges with occupational sensitizers. *Chest* 1991; **99**: 831–836.
20. Cote J, Kennedy SM, Chan-Yeung M. Objective versus qualitative analysis of PEFR in the diagnosis of occupational asthma. *Am Rev Respir Dis* 1990; **141(Suppl)**: A82.
21. Henneberger PK, Stanbury MJ, Trimbath LS, Kipen HM. The use of portable peak flowmeters in the surveillance of occupational asthma. *Chest* 1991; **100**: 1515–1521.
22. Hetzel MR, Clark TJH. Comparison of normal and asthmatic circadian rhythms in peak expiratory flow rate. *Thorax* 1980; **35**: 732–738.
23. Quackenboss JJ, Lebowitz MD, Krzyzanowski M. The normal range of diurnal changes in peak expiratory flow rates. Relationship to symptoms and respiratory disease. *Am Rev Respir Dis* 1991; **143**: 323–330.
24. Bellia V, Cibella F, Visconti A, Peralta G, Cuttitta G, Trizzino A, Giannone G, Bonsignore G. Peak flow records in asthma: Evaluation of an algorithm for interpretation of patterns. *Eur Respir J* 1989; **(Suppl 6)**: 532s–535s.
25. Clark NM, Evans D, Mellins RB. Patient use of peak flow monitoring. *Am Rev Respir Dis* 1992; **145**: 722–725.
26. Cullen MR. Clinical surveillance and management of occupational asthma. Teriary prevention by the primary practitioner. *Chest* 1990; **98(Suppl)**: 196S–201S.
27. Lee HS, Phoon WH. Diurnal variation in peak expiratory flow rate among workers exposed to toluene diisocyanate in the polyurethane foam manufacturing industry. *Brit J Indus Med* 1992; **49**: 423–427.
28. Burge PS, O'Brien IM, Harries MG. Peak flow rate records in the diagnosis of occupational asthma due to isocyanates. *Thorax* 1979; **34**: 317–323.
29. Ghio AJ, Castellan RM, Kingley KB, Hankinson JL. Changes in forced expiratory volume in one and peak expiratory flow rate across a work shift among unexposed blue collar workers. *Am Rev Resp Dis* 1991; **143**: 1231–1234.