

EDITORIAL

How to assess long-term effects of occupational exposure

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In this issue of the Journal, HUMERFELT *et al.* [1] present the results of a community longitudinal survey of lung function in men, aged 22-54 years. Participants were followed for up to 25 yrs to evaluate the effects of smoking and occupational exposure on the decline of lung function and on the presence of airflow limitation. The authors report that the decline in forced expiratory volume in one second (FEV₁) was associated with personal characteristics, such as age, body-height and smoking, as well as with work-related characteristics, such as exposure to sulphur dioxide gas, metal fumes and an increasing number of specific occupational agents. Further, they found that airflow limitation was present in 9.5% of subjects at follow-up and increased with age and cigarette consumption, but not with exposure to one or more specific occupational agents.

This study, characterized by an unusual length of follow-up, is one of the few published over the last seven years [2-8] that have dealt with respiratory effects of occupational exposure in general population samples. However, only two studies have previously provided information on the longitudinal association between dust exposure and lung function in general populations [2, 9] and only one has indicated a longitudinal relationship of dust exposure with occurrence of respiratory symptoms [3].

This editorial discusses the importance of the assessment of respiratory effects of occupational exposure in general populations, with special focus on the evaluation of long-term effects, so well described by HUMERFELT *et al.* [1].

Why is it important to assess effects of occupational exposure in general population samples

BECKLAKE, in a special supplement of the American Review of Respiratory Disease on the rise in mortality of chronic obstructive pulmonary disease (COPD) [10], mentioned that the official position in the USA, based on the 1984 and 1985 Surgeon General's reports on chronic obstructive lung disease, still considered the causative role of occupational exposure as putative rather than established. Other examples of putative risk factors were atopy, familial factors, past health, airway reactivity and environmental exposure, whereas sex, age, alpha-1-antitrypsin deficiency and tobacco smoke (personal) were regarded as established. However, the feeling of Margaret Becklake, based on the existing studies, was that the evidence now justifies a change in the ranking of occupational exposure as risk factor for COPD.

Other investigators were prompted to assess the effects of occupational exposure on their epidemiological data sets. Cross-sectional surveys carried out in Italy [6], Norway [7] and China [8] obtained similar findings, *i.e.* significant associations of dust exposure with respiratory symptoms and lung function impairment. These results are now extended longitudinally with the important contribution of HUMERFELT *et al.* [1].

The advantage of looking at the effects of occupational exposure in general population samples, rather than in groups of workers, is the avoidance of selection bias that can affect occupational groups: *i.e.* subjects who are less susceptible to effects of certain exposures may remain at work, whereas susceptible subjects may change occupations or leave the work force prematurely.

In addition, since subjects are randomly selected from the community population regardless of occupational status, even possible misclassification of exposure should be random and its effects would be to decrease the extent of any association between occupational exposure and lung function decline.

How to assess occupational exposure

Work related studies offer the advantage of providing adequate exposure prevalence to enable a study of the pathophysiological effects of specific agents. Usually, general community studies have not had sufficient numbers to assess the effects of single exposures and tended to pool the exposures in broad categories, such as dusts and gases or fumes.

However, it was not considered a flaw as American investigators [3] found an independent association of each exposure with the same symptoms, and cumulative dust exposure was highly correlated with cumulative gas or fume exposure. Furthermore, in a Dutch general population sample of elderly men [5], the variable that represented exposure to dusts, fumes, and/or gases contributed more clearly to the multivariate model than did specific exposures. Unfortunately, HUMERFELT *et al.* [1] did not collect information on occupational exposure during the first survey. Thus, unlike KRZYZANOWSKI *et al.* [11], they were not able to divide their population into four groups: those continuously exposed, exposed only during first survey, exposed only during second survey and those continuously unexposed.

However, HUMERFELT *et al.* [1] were able to demonstrate that FEV₁ decline was accelerated in those with more numerous exposures and, in particular, in subjects exposed to sulphur dioxide gas and to metal fumes. They did so using one-way analysis of covariance and multiple linear regression, not a complex model for longitudinal analysis. This could be criticized by statisticians since, in longitudinal analyses, methods to estimate the regression coefficients should be modified to

account for the correlation between repeated measurements on the same subjects [12]. However, even the regression coefficients estimated with complex methods, such as random effect model [13], have been considered [12] to summarize both differences between individual subjects at successive examinations and differences between subjects, therefore giving information that is still "cross-sectional". Conversely, we believe that the design of the study by HUMERFELT *et al.* [1] (only two observations, separated by 25 years), should be minimally affected by the problem of autocorrelation, considering that, even in shorter follow-up studies, cross-sectional and longitudinal models give very similar estimates for the effect of age on pulmonary function level [13]. Furthermore, twenty-five years is far beyond the limit of five years set by DALES *et al.* [14] as the interval between two observations needed to reduce the effects of the intra-interindividual variability that can mask the real decline of lung function.

It can be hypothesized that people exposed to sulphur dioxide and metal fumes performed a job with a relevant burden of pollution or that these pollutants are more toxic than others. However, this cannot be ascertained in the absence of a precise quantitative assessment of the exposure. It is interesting that another approach (*i.e.* considering the number of exposures affecting each worker) was successful in showing a relationship with FEV₁ decline. These findings are reinforced by the results achieved in another male population in the same area [15], where a good agreement was observed between self-reported specific occupational exposures and three different exposure-groups, based on occupational title of last job and longest job held.

Conclusion

Thus, we think that questionnaire is a useful and cost-effective tool for information about specific occupational exposures in the community

The contribution by HUMERFELT *et al.* [1] provides further evidence on the adverse health effects of occupational exposure to dusts, gases and fumes in the general population. It is, therefore, time to decide whether occupational exposure can be considered as an established risk factor for COPD as proposed by BECKLAKE [10].

Due to the possible implication of such a statement for public health policy, further contributions are welcome, either from an existing epidemiological data base not yet analyzed in this regard, or from newly designed general population surveys, which might assess occupational exposure more thoroughly by using a job-exposure matrix [16].

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References

1. Humerfelt S, Gulsvik S, Skjaerven R, *et al.* - Decline in FEV₁ and airflow limitation related to occupational exposure in men of an Urban community. *Eur Respir J* 1993; 6, 1095-1103.
2. Kryzanowski M, Jedrychowski W, Wysocki M. - Factors associated with change in ventilatory function and the development of chronic obstructive pulmonary disease in a 13-yr follow-up fo the Cracow study: risk of chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1986; 134: 1011-1019.
3. Korn RJ, Dockery DW, Speizer FE, *et al.* - Occupational exposures and chronic respiratory symptoms: a population-based study. *Am Rev Respir Dis* 1987; 136: 298-304.
4. Kryzanowski M, Kauffmann F. - The relation of respiratory symptoms and ventilatory function to moderate occupational exposure in a general population: results from the French PAARC study of 16,000 adults. *Int J Epidemiol* 1988; 17: 397-406.
5. Heederik D, Pouwels H, Kromhout H, Kromhout D. - Chronic nonspecific lung disease and occupational exposures estimated by means of a job exposure matrix: the Zutphen study. *Int J Epidemiol* 1989; 18: 382-389.
6. Viegi G, Prediletto R, Paoletti P, *et al.* - Respiratory effects off occupational exposure in a general population sample in North Italy. *Am Rev Respir Dis* 1991; 143: 510-515.
7. Bakke P, Eide GE, Hanao R, Gulsvik A. - Occupational dust or gas exposure and prevalences of respiratory symptoms and asthma in a general population. *Eur Respir J* 1991; 4: 273-278.
8. Xu X, Christiani DC, Dockery WD, Wang L. - Exposure-response relationships between occupational exposures and chronic illness: a community-based study. *Am Rev Respir Dis* 1992; 146: 413-418.
9. Lebowitz MD. - The trends in airway obstructive disease morbidity in the Tucson epidemiological study. *Am Rev Respir Dis* 1989; 140 (Suppl. 53-41).
10. Becklake MR. - Occupational exposures: evidence for a causal association with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1989; 140 (Suppl. 85-91).
11. Krzyzanowski M, Jedrychowski W. - Occupational exposures and incidence of chronic respiratory symptoms among resident of Cracow followed for 13 years. *Int Arch Occup Environ Health* 1990; 62: 311-317.
12. Ware JH. - Analysis of longitudinal data: choosing and interpreting regression models. Editorial. *Eur Respir J* 1993; 6: 325-327.
13. Sherrill DL, Lebowitz MD, Knudson RJ, Burrows B. - Longitudinal methods for describing the relationship between pulmonary function, respiratory symptoms and smoking in elderly subjects: the Tucson study. *Eur Respir J* 1993; 6: 342-348.
14. Dales RE, Hanley JA, Ernst P, Becklake MR. - Computer modelling of measurements error in longitudinal lung function data. *J Chron Dis* 1987; 40: 769-773.
15. Bakke P, Baste V, Hanao R, Gulsvik A. - Occupational airborne exposure of the general population of a Norwegian country. *Scand J Work Environ Health* 1992; 18: 44-51.
16. Hsaini M, Kauffmann F, Chavance M, Brochard P. - Personal factors related to the perception of occupational exposure: an application of a job-exposure matrix. *Int J Epidemiol* 1992; 21: 972-980.