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Mortality attributable to work

by David Coggon¹

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Assessments of the burden of illness attributable to occupation are needed to plan the allocation of resources for disease prevention and research. In general, chemical, physical, and biological hazards in the workplace are easier to identify and control than those related to diet and life-style. Thus it is worth knowing whether modern work conditions have an important impact on health and, if so, where the major hazards lie. In this issue of the *Scandinavian Journal of Work Environment & Health*, Nurminen & Karjalainen have attempted to estimate the proportion of deaths in Finland that is associated with occupational factors (1). They calculate that some 1800 deaths per year are work-related, most of them among men. This figure represents approximately 4% of all deaths nationally.

Of course, some of the deaths attributed to work may also have been influenced by nonoccupational factors. For example, much of the excess mortality from lung cancer among workers exposed to asbestos could be avoided if the persons concerned did not smoke. Nevertheless, the figure of 4% is surprisingly high, and it is important to look carefully at the methods by which it was derived.

As the authors acknowledge, their investigation inevitably entailed simplifications and a degree of informed guesswork. In some cases, such as the decision to ignore deaths from circulatory diseases after 75 years of age, the effect may have been to underestimate the contribution of occupation to mortality. Of greater concern, however, are biases that could have exaggerated the apparent impact of work. Two in particular deserve mention.

First, the criteria by which occupational exposures were accepted as causes of disease were relatively undemanding as, for example, hydrocarbon solvents for cancers of the oral cavity and cervix, welding fumes for

cancers of the pharynx and colon, styrene for cancer of the rectum, and extremely low-frequency electromagnetic fields for Alzheimer's disease. Decisions to classify diseases as work-related appear to have been based largely on epidemiologic findings with little regard for the relevant biology. Thus there was no attempt to explain how inorganic dusts such as silica would give rise to cancer of the liver or why melanoma should be treated as a hazard of both indoor and outdoor work. Where exposures or associated causes of death are rare, the adoption of more stringent criteria for inclusion would have little impact on the overall calculation. However, for some causes of death, the effect is more important. For example, 10.3% of deaths from stomach cancer among men were attributed to farming and the rearing of livestock, hardly a well-established cause of the disease.

The second problem arises from the dichotomous classification of exposures (present or absent) when, for almost all hazards, risk varies importantly according to the intensity and duration of exposure. For each hazard and disease, the starting point for the calculation of an attributable proportion was an estimate of the prevalence of exposure in the Finnish population and of the risk associated with that exposure. In some cases, risk estimates were derived from cohort studies. This procedure has the advantage that, in comparison with case-referent investigations, cohort studies tend to be less subject to bias and to selective reporting of positive results. However, it is usually the occupational populations with the highest exposures to a known or suspected hazard that are selected for investigation in cohort studies (because they are likely to be at greatest risk). Therefore, their risks cannot necessarily be extrapolated to other workers exposed at lower levels. Nurminen & Karjalainen were aware of this problem and state that they used

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cohort studies in which exposure levels were representative of those in the target populations to which the risk estimates were applied. However, they do not systematically provide the relative risks and exposure prevalences that their calculations assumed, and it is therefore difficult to judge how well this objective was achieved.

An alternative approach, used for many of the hazards, is to derive risk estimates from case-referent studies based on the general population. Then, by definition, exposures should be more representative. On the other hand, risk estimates from case-referent studies may be inflated by biased recall of exposures, and nonpositive results may not find their way into the literature as completely as positive findings. In both cases, the effect will be to overestimate attributable proportions.

For these reasons, the annual number of work-related deaths in Finland is unlikely to be as high as Nurminen & Karjalainen suggest. Nevertheless, their report is a useful starting point for further action. In particular, there is a need to focus more closely on the hazards that they propose carry the highest toll of mortality. These are the risks of lung cancer and mesothelioma from asbestos and of ischemic heart disease from shift work and noise.

With regard to asbestos, the nature of the hazard and the relation of risk to exposure are well established. The challenge is to ensure that controls on exposure are properly applied, especially in the construction industry.

The effects of shift work and noise on coronary heart disease are also potentially important, but further research is needed to confirm the scale of the problem and the underlying pathogenic mechanisms. Whether or not the impact of noise on mortality is as large as suggested, exposures should be controlled to prevent noise-induced hearing loss. However, preventive measures in relation to shift work are more difficult to specify without a better understanding of the precise aspects of shift work that influence risk. To provide a convincing basis for preventive action, controlled intervention studies may be needed.

Reference

1. Nurminen M, Karjalainen A. Epidemiologic estimate of the proportion of fatalities related to occupational factors in Finland. *Scand J Work Environ Health* 2001;27:161–213.