PostScript

LETTERS

Are sickness absence frequencies in the study of EU countries underestimates?

The paper by Gimeno *et al* provides a comparison of sickness absence between 15 European Union (EU) countries.¹ According to this study, 14.5% of employees were absent at least one day in the past 12 months by an accident at work, by health problems caused by the work, or by other health problems. For Finnish employees, for instance, this percentage was 24%, the highest among the 15 EU countries; in the UK it was 11.7%.

These figures are much lower than those reported previously. A population based survey of Finnish employed workforce aged 25–64 carried out in 2000 found that 45% of employees took sickness absence during the past six months.² Correspondingly, a population based survey of 5400 British adults aged 15–64 reported that 30% of working adults took time off work in the past year because of their health or feelings.³

Three large cohort studies from Finland and the UK have used absence records instead of self-reports. In 2000, 58% of 77 850 municipal employees participating in the 10 town study⁴ took at least one sickness absence day; the same percentage was obtained in the Hospital Personnel Study⁵ for 30 864 hospital workers aged 15–65. In the Whitehall II study⁶ of over 10 000 British civil servants aged 35–55, 57% of men and 76% of women recorded sick leave 12 months prior to the study entry in 1985–88.

Based on these national studies, we suspect that the figures presented by Gimeno *et al* are underestimates of actual absence frequency in the EU countries. Data on sickness absence were derived from face-to-face interviews that were carried out at the participant's home, a rarely applied assessment strategy for sickness absence. It is possible that the wording of the question led people to report sickness absence only when they believed it to be work related. The authors note that low response rates in some countries and healthy worker effect are potential sources of bias.

We feel that the data presented by Gimeno *et al* are far too preliminary to be the basis of any policy at this stage or of conclusions regarding differences in absence frequency between nations. We fully agree with their recommendation for further research on sickness absence in EU countries.

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Authors' reply

In response to our study,¹ Kivimäki *et al* suggested that reported sickness absence frequencies were underestimates of the total sickness absence burden in European Union (EU) member countries.² This concern about the veracity of these estimates led Kivimäki *et al* to caution policy makers to not use this data to inform policy. While we agree that more research is needed to establish potential biases associated with different approaches to ascertain accurate sickness absence data, we consider the European Survey on Working Conditions (ESWC) to be useful to inform the cross-national policy debate. Country specific studies contribute knowledge to the evidence base, but cross-national studies such as ours help to provide a stronger basis on which to make cross-national inferences. Furthermore, cross-national studies become more relevant as data accumulate and the data collection quality improves. We hope that Kivimäki and colleagues are not suggesting the ESWC be discontinued.

We consider the studies by Kivimäki *et al* to be some of the most relevant epidemiological studies of sickness absence predictors.^{3–5} Although informative, these studies raise several issues in the context of cross-national comparisons. First, epidemiological cohorts in Finland and the United Kingdom represent very homogeneous and specific working populations (that is, municipal employees, hospital workers, and civil servants) with unknown generalisability to the national representative surveys studied in our paper or the ones referenced by Kivimäki and colleagues.^{6,7} Second, a fundamental advantage of national workforce surveys is the ability to capture all workers, whereas registries may lead to an under-representation of marginal work groups typically not included in national registries. Indeed, Kivimäki *et al* are not arguing that the Finnish and British cohorts are representative of the countries' workforces. Even so, labour market inequalities may cause temporary and less protected workers to be under-represented in the type of well designed cohort studies they have referenced.⁸ Temporary and less protected workers are important in the EU economy, and lack of knowledge about their labour market experiences as related to sickness absence could lead to their further marginalisation in the policy debate. Third, Kivimäki

et al criticised the data collection method employed in the ESWC. We are not aware of any cross-national study comparing the reliability, validity, and performance of different sickness absence data collection methods. Concerns have been raised about who is placed on a sickness absence registry. Registered data are very conditioned by the country's social security system criteria for sickness absence, which complicates between-country comparisons.⁹ Therefore, whether registries are the gold standard in sickness absence studies remains a point of debate yet to be closed.

In addition, Kivimäki *et al* compared our results to two survey based studies from Finland and Britain, but differences in sample selection and questionnaire design between these studies may limit comparisons. Our study included people aged 15 years and older who had any paid job during the reference week, or who had a job but were temporarily absent. The recall period for sickness absence was 12 months. The Finnish study was based on employees aged 25–64 using a six month recall period for sickness absence.⁶ The British survey investigated the psychiatric morbidity prevalence among the British adult population. This study sampled workers aged 16–64 years and excluded workers with a psychosis diagnosis. Workers who were currently working or had been working in the last year were asked to report absence days due to their health or feelings.⁷ For these reasons, caution is needed if a direct comparison between these three studies is intended.

Finally, we agree with Kivimäki *et al* that potential bias in the ESWC could be present (see pp. 868–9 in our article). However, we would argue that the best sources of data to inform policy are derived from systematic efforts to collect sickness absence data in a clear and consistent fashion from a representative sample of the labour force within each country. We consider the evidence presented by Kivimäki *et al* to support our argument of the difficulty in establishing between-country comparisons due to the fragmented and insufficient sickness absence data available at the European Union level. We consider our results useful. Although the results are preliminary and may be subjected to scientific scrutiny, the comparative findings may provoke researchers to develop standards for sickness absence studies to facilitate between-country comparisons. In addition, we hope the observed differences will promote further investigation into root causes of between-country differences, especially between northern and southern EU members, as well as within-country gender differences. We certainly welcome cross-national collaborative efforts among the EU sickness absence researchers to address all these issues.

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BOOK REVIEW

Analyses of hazardous substances in biological materials. Volume 9 Special issues: Marker of susceptibility

Edited by J Angerer and M Müller. Weinham: Wiley-VCH, 2004, pp 329. ISBN 3-527-27799-4

This is an unusual edition in a series of books devoted to methods of estimation of chemicals in workplace atmospheres. Protocols for genotyping CYP P450 1A1, 1B1, 2E1, N-acetyltransferase 2, glutathione S-transferase T1, M1 and P1, sulphotransferase 1A1 and 1A2, and phenotyping of glucose-6-phosphate dehydrogenase, N-acetyltransferase 2, and glutathione S-transferase T1 are presented. Each protocol is clearly set out with a discussion of underlying principles, quality control and sources of error. A short section on real time PCR genotyping for a number of polymorphisms is also included. The authors quite sensibly took the decision to repeat essential basics in each protocol (for example, preparation of gels for chromatography) so that each protocol can be read independently. It is inevitable that the methods proposed will become dated but that should not detract from the current value of this edition to bench scientists. A number of the preliminary chapters will be of value to students. Of these I found the section on polymerase chain reaction and background information on polymorphisms which preceded each protocol easy to follow and instructive. The editors do note that the book may contain minor typographical errors. Readers should note the lack of reference numbers in the bibliography for N-acetyltransferase 2 genotyping (although these are listed in numerical order) and the incorrect concentration for the Tris buffer concentrate in the CYP 1A1 genotyping protocol (p. 74). The book is not intended to be a text on molecular epidemiology and the short chapter on evaluation of susceptibility is at most a very basic introduction. Overall, well worth purchasing by academic libraries for use by researchers and students.

J Battershill

Changing trends in US mesothelioma incidence

The paper by Weill and colleagues¹ requires to be read with great circumspection by those concerned with public health policy. It would be premature for them to conclude from it, that with the decline in US mesothelioma incidence, which the authors associated with the earlier decline in use of amphiboles, the asbestos problem has been largely solved. Expert opinion published by WHO, ILO, and IARC is not so sanguine as them about chrysotile, and it is increasingly acknowledged worldwide that its safe use is not reasonably practicable.

There is a danger of understanding from the authors' statement: "Asbestosis and asbestos attributable lung cancer have been found to be linked..." that they are asserting that the evolution of interstitial pulmonary fibrosis is a necessary stage in the carcinogenic process in the bronchus (or for that matter in the pleura or in the peritoneum). In the case of bronchial carcinoma, this was once held to be a self-evident truth supported by unproven mechanisms of varying degrees of ingenuity and vagueness, but it is of note that consensus was reached that the sequence was not necessary, by a group of experts covering the adversarial spectrum.²

The pathologist co-author Churg, having contributed significantly to the literature,³ will be aware of the extensive and persuasive experimental evidence that all species of asbestos are equally potent inducers of bronchial epithelial metaplasia and malignant change in whole animals and in tracheal explants, dissociated from interstitial pulmonary fibrosis. He will also be aware of the tissue culture studies in which from 4 minutes onwards, fibres are seen to interact with

the cell membrane, the cytosolic apparatus, and nuclear spindle, leading to various transformations.

Two statisticians are cited⁴ as authority for the "biological plausibility" of the authors' contention that use of amosite and crocidolite related to temporal trends in US mesothelioma incidence. These statisticians in their review of the literature had relied on an earlier review⁵ in support of the "biological plausibility" of chrysotile being a less powerful tumorigen than the amphiboles, but its author with her limited resources, balking at the task of covering the vast literature, in her turn had relied on earlier "state of the art" reviews. In the adversarial atmosphere pervading attitudes to asbestos, uncritical reliance on such reviews was imprudent. The two statisticians cited, took note of the experimental evidence for the carcinogenic potency of chrysotile being comparable with that of the amphiboles, but discounted it on the basis of it applying only to the brief life of rats, and of it being more rapidly "cleared" than amphibole; the significance of the events observed in the first 4 minutes in cell culture were not discussed.

Weill *et al* attribute the lesser rate of mesothelioma in the USA compared with the UK to amosite being the amphibole used most in the USA, and to its national population being four times larger. Official figures present US amphibole imports in tonnes for the years 1964 and 1965 respectively as: amosite 23 932 and 17 042; and crocidolite 21 163 and 17 042.⁶ (UK imports of amosite in 1965 totalled 22 582 tonnes and crocidolite 3425.) They report the peak US mesothelioma incidence rate to have occurred in the early to mid-1990s, and attribute it to the reduction in amphibole use since its peak import in the USA in the 1960s.

National asbestos import tonnages are no better surrogates for the exposures of the population subsets at risk, than the total populations of the states and metropolitan areas with cancer registers are acceptable as denominators of those occupationally exposed. The derivation and interpretation of trends in mortality and cancer registration rates for malignant mesothelioma from such data for occupational sub-sets present problems.

Between 1900 and 2002, the USA exposed its workforce to a total of some 25 million tonnes of home produced and imported asbestos (all species), and exported a small proportion.⁷ The accelerated flight from asbestos manufacture in the 1980s will be reflected in due course in asbestos worker mortality patterns, but service workers, construction workers, and bystanders will continue to be at risk. The Third Wave of Asbestos Disease⁸ was no Selikoff bugaboo; pace Weill and colleagues' reassurance, public health policy makers require to maintain a watching brief for many years yet

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