CORRESPONDENCE

Coal mining, emphysema, and compensation revisited

Editor.—We are sorry to see that Morgan, in his Forum article in 1993, has repeated criticisms of our study that are totally without foundation and have already been effectively countered. In our reply to earlier remarks along the same lines made by Lapp and Morgan in our paper,1 we stated that the inclusion of antrachitic workers made no difference to the results, the findings being essentially the same when they were removed from the study group. Our earlier response also noted that the dust exposure effect was evident when various indicators of exposure were used (from a surrogate—years of work underground—to dust exposure estimators that differ little from the others that we questioned). As for the smoking effect—we reported what we found. The results differed little when various analytical approaches were used, and are similar to those reported by other researchers from sectional studies of cohorts of current workers.

Epidemiological studies are hard to do right, and very easy to criticize. The perfect investigation does not exist and may never. In his attempt to further his point of view, Morgan seeks out the inevitable blemishes in studies, while completely ignoring the overall picture. This picture, based on a number of different types of study in several countries, now shows that there is overwhelming evidence1 10 that loss of lung function is related to dust exposure in coal mining.

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Mental retardation and parental occupation: a study on the applicability of job exposure matrices

Editor.—Roeleveld and colleagues identified occupational exposures by the application of job exposure matrices for occupational histories and by asking respondents during their interview to mark their exposures on a checklist. Compared with interviews the method of matrices yielded twice as many exposure categories. This difference can be explained with the inaccuracy of both or one of the methods of data collection. Owing to the rigidity of job exposure matrices, which obscure real associations, authors considered interview as “the gold standard”, although as the time lag between pregnancy and interview was 2-25 years, they were aware of the possibility of underreporting. One of their arguments against exposure data generated by job matrices is that “ORs found by means of the interview cannot be interpreted logically, whereas those for exposures generated by job matrices could hardly be explained.”

Without favouring either of the two methods I wish to call attention to two inaccuracies in their arguments. Firstly, ORs given by matrices move randomly around 1 and all the 95% CIs are below 1 indicating no significant differences at this level. Thus the only justified conclusion is that according to matrices mental retardation in the study group was not associated with parental exposure, a not illogical outcome. The second inconsistency is in the statement that the associations between mental retardation and exposures identified through interviews were in agreement with published information. As far as mercury is concerned this statement was correct but only if exposure had been to methylmercury. It is unlikely, however, or even impossible, that this is the case. The salient point is that among mercurials, methylmercury is the only one for which the adverse effect of pernatal exposure on postnatal development has been proved in epidemiological studies and that is why all the supporting references on mercury given by the authors are on this mercury species.

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Author's reply

Editor.—The opportunity to respond to Magos’ comments is greatly appreciated. It is, indeed, very difficult to compare different methods of occupational exposure assessment and to favor one over the other when the true exposure is unknown. In our study, a personal interview was considered the “gold standard” to which job exposure matrices were compared, in the absence of more accurate exposure information. The results suggested a high degree of classification on exposures generated by job exposure matrices, that is, a result that was reflected in some increased ORs that could hardly be explained. Magos argues that “all ORs move randomly around 1 and thus the only justified conclusion is that mental retardation was not associated with parental occupation.” The fact, however, that ORs that were increased according to the interview (not due to information bias) varied around unity when using the matrices, is indicative of information bias in the exposure assessment. Moreover, job exposure matrices also yield spurious associations that cannot be explained, such as for diesel fuel (OR = 2-0, 90% CI: 1-0-4-1) that had definitely not been used by any of the women in the study population.

After Magos expressed a concern about the association found between mental retardation and exposure to mercury (compounds), which he claims is not in accordance with the scientific literature that exclusively refers to methylmercury. There are, however, a number of publications that report or suggest adverse effects of inorganic mercury compounds and metallic mercury exposure on the central nervous system, as summarised in a review article in this journal. Both maternal exposure to methylmercury, although unlikely, and occupational exposure to other mercury compounds could thus be potential risk factors for mental retardation in offspring.

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*Occup Environ Med* 1994 51: 647
doi: 10.1136/oem.51.9.647

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