CORRESPONDENCE

Coal mining, emphysema, and compensation revisited

Editor,-We are sorry to see that Morgan, in his Forum article in 1993,1 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 Morgan² on our paper,³ we stated that the inclusion of anthracite 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 exposure estimates derived from data other than that which he questions). 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 found by other researchers for cross sectional studies of cohorts of current workers

Epidemiological studies are hard to do right, and very easy to criticise. 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 over-whelming evidence³⁻¹³ 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.1 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 can 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 could 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 this statement. 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 parenteral exposure, a not illogical outcome. The second inconsistency is in the statement that the associations between mental and exposures identified retardation through interviews were in agreement with published information. As far as mercury is concerned this statement would be correct 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 prenatal 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.1 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 misclassification on exposures generated by job exposure matrices, that among other results 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 bias2) varied around unity when using the matrices, is indicative of non-differential misclassification of exposure resulting in bias towards the null value. Therefore, a justified conclusion pertains not to the absence of associations, but to obscuring of true associations when general job exposure matrices are used as an alternative to personal interviews or other methods of 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.3

After Magos expresses 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 vapour on the fetal central nervous system, as summarised in a review article in this journal.⁴ Both maternal exposure to methylmercury, although unlikely, and occupational exposure to other mercurials could thus be potential risk factors for mental retardation in offspring. NEL ROELEVELD

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