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Incidence of lung cancer by histological type among asbestos cement workers in Denmark

Sir,—In their study of asbestos cement workers, Raffin *et al* (1993; 50:85-9) state that their results indicate that the risk for adenocarcinoma of the lung caused by exposure to asbestos is different from the corresponding risk for squamous cell carcinoma and anaplastic cell carcinoma. The conclusion is based on an analysis of the relative risks (RRs). If the absolute risk (AR) is analysed, however, the interpretation may be different. In the table, which is calculated from table 2 in the paper by Raffin *et al*, the absolute risk seems to be similar for adenocarcinoma and squamous cell carcinoma of the lung. The authors do not report the person-years so the absolute risk is expressed in relative terms.

The fact that the interpretation is different depending on the risk measure may seem confusing and there are arguments for using both. In prevention, the absolute risk is a better measure as it gives a direct measure of the number of cases that will be prevented. The relative risk may be a better measure in clinical practice as it can easily be transformed to the aetiological fraction ($EF = (RR-1)/RR$) and thus gives a measure of the chance that the cancer of a certain patient is caused by asbestos.

From a more theoretical epidemiological standpoint the best measure depends on whether the risk due to asbestos is multiplicative or additive compared with the background risk. For a multiplicative risk the incidence

rate (IR) is $IR = IR_0 * f(\text{exposure})$, where f is a function not dependent on the background incidence (IR_0). If the risk is additive an absolute risk is more appropriate as $IR = IR_0 + IR_{\text{asb}}$, where IR_{asb} is the incidence rate caused by exposure. A relative risk may at a first glance be preferred as the risk for lung cancer caused by asbestos is usually expressed as: $SMR = 1 + a * \text{dose}$ where a is a constant. This relation does not seem to fit the data of Raffin *et al*, however, and the only measure of "dose" in their paper is employment time. The relative risk is certainly not related in linear fashion to employment time if all lung cancers are considered (1.9, 1.4, and 1.9 for <1, 1-4, and ≥ 5 years respectively). The group with 1-4 years employment time is small and there are large confidence intervals for the risks especially when stratified according to histological type. Thus there seems to be little justification to restrict the analyses to a multiplicative model. There is an increased risk in the group with <1 year employment time. This raises the question about comparability between the exposed and reference groups. A dose-response model may also consider time since last exposure as some data indicate that the risk of lung cancer decreases some years after the exposure of asbestos has ceased.¹

A different risk according to time from onset of exposure may depend on the different growing rates of the tumours. Anaplastic carcinoma grows faster than squamous cell carcinoma, which grows faster than adenocarcinoma.² The importance of the finding of a higher RR for adenocarcinoma in persons with a long time since onset of exposure compared with persons with other histological types of tumour does not necessarily mean that only adenocarcinoma are caused by exposure to asbestos. My conclusion is that the

data in this paper indicate that asbestos can cause different histological types of lung tumour.

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Authors' reply

Järholm has used our lung cancer data from the Danish asbestos cement industry to illustrate once more the difference between the relative risk and the absolute risk. These two risk measurements are different, and—depending on the purpose—we may prefer sometimes to use the first and sometimes the second.

Järholm mentions that anaplastic carcinomas of the lung may grow faster after exposure to asbestos than adenocarcinomas. We are currently updating the Danish study, and we hope to be able to shed further light on this point in the analysis.

Nasal melanoma

Sir,—The article by Holmstrom *et al* (1991;48:9-11) postulated an association between nasal melanoma and formaldehyde exposure based on three case reports. As part of a population based case-control study of subjects with nasal and nasopharyngeal cancer,^{1,2} we interviewed nine of fourteen cases of nasal and nasopharyngeal melanoma diagnosed in western Washington State between 1979 and the end of 1989. Controls for the study were obtained through random digit dialing and were frequency matched on age at diagnosis and sex.

One subject had lived in a residence with foam insulation (observed/expected = 3.57, 95% confidence interval 0.09-19.8). None, however, reported knowledge of specific occupational exposure to formaldehyde (expected = 0.27) and none reported being employed in industries likely to result in exposure to formaldehyde (expected numbers for wood working = 0.10, furniture

Relative (RR) and absolute (AR) risk of different histological types of lung cancer in the study by Raffin et al (from table 2)

Histological type	RR	AR*	(95% CI†)
Adenocarcinoma	3.31	16.7	(8.1-28.5)
Squamous cell carcinoma	1.67	14.9	(4.0-29.0)
Anaplastic carcinoma	1.58	8.5	(0-20.1)

* Expressed in relative terms (number of cases *person-years), as the number of person-years is not stated in the paper. Number of cases = observed - expected.

† 95% confidence interval of AR calculated by the same method as that used by Raffin *et al* (1993).