CORRESPONDENCE

Update of the Texaco mortality study 1947-93: part II

EDITOR—It is with a sense of “deja-vu” that I read the paper by Divine *et al*¹ on the update of the Texaco mortality study. I was particularly struck by their conclusions about lung cancer among the maintenance trades. Although large numbers of mesotheliomas were found among these workers, the authors concluded that there was no increased risk of lung cancer because the SMRs were <100. This conclusion is similar to that of Tsai *et al*² who, despite the findings of excess mortality from mesothelioma and increased lung cancer mortality among maintenance workers, in comparison with operators at another Texas refinery, concluded that asbestos exposures were “not sufficient to produce lung cancer”. It is also similar to that of Raabe *et al*³ who stated that, although they had found an increase in lung cancer mortality among maintenance craft workers, the “temporal patterns of these SMRs and findings from other studies of similar cohorts provided evidence that the excesses were most likely to be unrelated to work in maintenance craft jobs”.

Workers in the petroleum refinery industry, when grouped together, have had a lower risk of lung cancer than members of the general population.⁴ This has been found once again in the paper by Divine *et al*. Given this phenomenon, it is appropriate to make internal comparisons—that is, to compare the lung cancer experience of maintenance workers exposed to asbestos with that of operators with minimal or no exposure to asbestos. When this is done, the risk of lung cancer is indeed increased among maintenance workers in the refinery and petrochemical sector.⁵

Table 1 Lung cancer among workers in the Texaco mortality study

Worker group	Observed	Expected	SMR
Unexposed	152	274	56
Maintenance, 0-4 y	219	322	68
Maintenance, 5-19 y	91	121	75
Maintenance, ≥20 y	75	84	89

I have performed some calculations with the data in table 1 of Divine *et al*, and I find that in their study there is a significant trend in risk of lung cancer with duration of employment in the maintenance trades. The table below shows the data. I have subtracted the experience of the maintenance employees from that of the total cohort to produce the SMR for unexposed workers. The SMR for

lung cancer increases with the duration of time employed in the maintenance trades. I used equation number (3.12) in the monograph of Breslow and Day⁶ to compute a test for trend in these SMRs. I used the integers 1 to 4 to rank the exposure levels. The value for χ^2 in the trend test was 12.3 on 1 degree of freedom ($p < 0.001$). I thus conclude that the study of Divine *et al* contributes yet another piece of evidence showing that workers exposed to asbestos in the refinery and petrochemical sector are at increased risk of lung cancer.

MURRAY M FINKELSTEIN

Department of Family Medicine, Mount Sinai Hospital, Toronto, Ontario, Canada, and Program in Occupational Health and Environmental Medicine, McMaster University, Hamilton, Ontario, Canada
email murray.finkelstein@utoronto.ca

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Author's reply—Finkelstein has reviewed the lung cancer results for a select group of maintenance workers in our paper on the update of the Texaco mortality study. In his letter, he suggests that although several papers on petroleum refinery workers have seen no increase in the standardised mortality ratios (SMR) for lung cancer,¹⁻³ if internal comparisons of the SMRs were done, this would show an increased risk of lung cancer among maintenance workers in the refinery and petrochemical sector.

It has been well documented that a valid comparison of two age standardised SMRs requires that the underlying age specific mortality rate within each study group is constant or that the age distributions of person-years between two groups are similar.⁴⁻¹⁰ Finkelstein carried out the assessment of the trends without consideration of the above two conditions although in this case, the results are similar. However, it should be emphasised that the observed numbers of deaths from lung cancer are still less than expected for

each of the maintenance employee groups. Even for the group of maintenance workers potentially exposed >20 years, there is still an 11% deficit of lung cancer (SMR 89, 95% confidence interval (95% CI) 70 to 112).

It should be noted that the maintenance category that Finkelstein has constructed is a combination of all the different maintenance trades, except for insulators, each with different job responsibilities and exposures. If the trends for lung cancer for the maintenance subgroups from the complete Texaco mortality study report are examined (unpublished data), none of these categories show an increasing trend in the lung cancer SMR with increasing duration of employment in the subgroup (as shown in table 1).

However, in a previous article, Finkelstein again compared the results for lung cancer in maintenance workers who had ≥20 years of potential exposure to asbestos to those for non-maintenance employees who had ≥20 years of employment in refinery or chemical operations.¹¹ Because age was related to cumulative exposure, the maintenance group with ≥20 years of potential exposure to asbestos was older than the corresponding non-maintenance group. In fact, 52% of the person-years in the maintenance group was from workers ≥60 years but only 39% in the non-maintenance group. Although Finkelstein calculated a relative risk, based on the ratio of two SMRs, of 1.24 for the two groups, a more exact age standardised rate ratio for lung cancer is 1.09 (95% CI 0.79 to 1.50) (Tsai SP, Gilstrap EL, Ross CE. Lung cancer among maintenance employees in a refinery and petrochemical plant, unpublished letter).¹² This rate ratio does not support increased lung cancer mortality among maintenance employees when compared with non-maintenance employees.

Also, in their study of maintenance workers potentially exposed to asbestos, Tsai *et al* provided similar SMRs for lung cancer by duration of employment for maintenance workers (a group defined exactly as in our report). The SMRs are 88, 77, and 83 for those employed 1-4 years, 5-19 years, and ≥20 years as maintenance workers, and there is no trend of increasing lung cancer with increasing duration of employment. Thus, the study of Tsai *et al* does not support Finkelstein's allegations that, in general, petroleum industry maintenance workers have an increased risk of lung cancer compared with other workers, which increases with duration of employment as a maintenance worker.¹¹

BARBARA J DIVINE

CHRISTINE M HARTMAN

Texaco, PO Box 1404, Houston, TX 77251, USA

JUDY K WENDT

Equiva Services, PO Box 2463, Houston, TX 77252-2463, USA

Table 1 Lung cancer mortality by duration of employment in specific maintenance crafts

Specific craft	Total		0-4 y		5-19 y		≥20 y		Trend test	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	χ^2	p Value
Insulator	48	90 (66 to 120)	31	77 (52 to 110)	10	133 (63 to 245)	7	128 (51 to 264)	2.25	0.13
Painter	30	76 (51 to 108)	19	69 (41 to 108)	8	100 (43 to 197)	3	87 (14 to 195)	0.03	0.86
Structural steel, pipe fitter, machinist	202	78 (67 to 89)	105	80 (65 to 97)	46	71 (52 to 95)	51	78 (58 to 102)	0.04	0.84
Electrician, instrument	35	78 (53 to 108)	11	89 (44 to 160)	19	97 (58 to 151)	5	36 (12 to 83)	3.40	0.07
Boilermaker, welder, lead burner	52	65 (48 to 85)	23	60 (38 to 90)	19	83 (50 to 130)	10	53 (26 to 98)	0.06	0.80
Equipment operator	59	73 (55 to 94)	42	76 (54 to 103)	9	50 (23 to 98)	8	102 (44 to 202)	0.13	0.72
Cleaner	91	68 (54 to 83)	84	67 (54 to 83)	5	58 (18 to 136)	2	147 (18 to 530)	0.38	0.53
Pipefitter, boilermaker	161	81 (68 to 94)	97	78 (63 to 95)	33	81 (55 to 113)	31	90 (61 to 128)	0.49	0.47
All maintenance	419	74 (67 to 81)	213	76 (66 to 87)	99	64 (52 to 77)	107	82 (67 to 98)	0.20	0.65

- 1 Divine BJ, Hartman CM, Wendt JK. Update of the Texaco mortality study: part II. Analyses of specific causes of death for white men employed in refining, research, and petrochemicals. *Occup Environ Med* 1999;56:174-80.
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Childhood leukaemia, population mixing, and paternal occupation

The possible infective origin of childhood leukaemia has been the subject of much recent attention.¹ Fear *et al*² have taken up my suggestion that my necessarily limited examination of childhood leukaemia relative to levels of paternal occupational contact in the general population³ be extended and (as in my study) found no evidence of a general increase in risk when fathers have jobs involving contact with many people. However, in reporting my hypothesis they did not bring out that the principal question considered in my study did not concern the general population, but rather whether there was a relation with paternal occupational contacts "in population-mixing situations associated with an excess of childhood leukaemia".³ I investigated this question¹ with data from

previous studies that were carried out to test the view that it is a rare response to some unidentified infection, and that situations of unusual urban-rural population mixing would favour excesses by promoting contacts between infected and susceptible people. This occurs when urban groups are mixed with rural groups that are likely to contain a higher proportion of susceptible people—such as in rural new towns or the North Sea oil industry in northern Scotland. Within the excesses associated with such unusual situations, there was a higher proportion of children with fathers in high contact occupations, as noted in certain established infections.³

Evidence of a relation with occupational levels of contact (although worth checking formally) would not be predicted in data on childhood leukaemia from the whole of England and Wales, dominated as they are by urban areas. In such areas of high population density, even if exposed to population mixing, the prevalence of susceptible people would tend to be lower, and of immune people to be higher, so that the high level of herd immunity would reduce the likelihood of epidemics. It would be unfortunate if infrequent but informative situations were neglected as a result of a misconception that if infection is present, then evidence should be observable generally. In this connection, it may be worth recalling that those who did the classic work on feline leukaemia virus stressed that nothing about the pattern of the usual (sporadic) cases of leukaemia in domestic urban cats suggested infection; only in the special situation of multicat households did the disease show epidemic features.

L J KINLEN

CRC Cancer Epidemiology Group, Department of Public Health, University of Oxford, The Radcliffe Infirmary, Oxford OX2 6HE, UK

- 1 Doll R. The Seascale cluster: a probable explanation. *Br J Cancer* 1999;81:1-3.
- 2 Fear N, Roman E, Reeves G, et al. Are the children of fathers whose jobs involve contact with many people at increased risk of leukaemia? *Occup Environ Med* 1999;56:438-42.
- 3 Kinlen LJ. High-contact paternal occupations, infection and childhood leukaemia: five studies of unusual population-mixing of adults. *Br J Cancer* 1997;76:1539-46.

Fear and Roman reply—In our paper published in an earlier issue of *Occupational and*

Environmental Medicine, we examined the role of father's occupational social contact on the development of childhood leukaemia from data covering the whole of England and Wales.¹ We found no evidence for an association between death during childhood from leukaemia and father's occupational social contact. Kinlen states that because we examined data for the whole of England and Wales rather than for specific "population-mixing situations associated with an excess of childhood leukaemia"² it is unsurprising that we found no association. However, given the worrying nature of the suggestion that parents may transmit a workplace acquired leukaemogenic agent to their offspring,² we think that our result, showing that, in general, children of men who have high levels of occupational social contact are not at an increased risk of leukaemia, is important. Further, the first study published on this topic, based within an area identified as having an excess of childhood leukaemia, found no evidence that parental occupational social contact—either with adults or with children—influenced the risk of leukaemia in their offspring.³

The suggestion that infections may be involved in the aetiology of childhood leukaemia, particularly acute lymphoblastic leukaemia (ALL), is not new. Kinlen's population mixing hypotheses, and those of Greaves,⁴ are currently the subject of much research. To date, however, no specific agents or mechanisms have been identified.⁴

NICOLA T FEAR
EVE ROMAN

Leukaemia Research Fund, Centre for Clinical Epidemiology, University of Leeds, 30 Hyde Terrace, Leeds, LS2 9LN, UK

- 1 Fear NT, Roman E, Reeves G, et al. Are the children of fathers whose jobs involve contact with many people at an increased risk of leukaemia? *Occup Environ Med* 1999;56:438-42.
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