

PostScript

LETTERS

Respiratory cancer in nickel carbonyl refinery workers

Grimsrud and Peto¹ have usefully compared our recent findings of respiratory cancer risks in nickel carbonyl refinery workers first employed at the Clydach plant in the period 1953–92² with those of earlier year-of-hire cohorts.³ We did highlight the elevated SMR for nasal cancer in our study but epidemiological interpretation is not possible given that it is based on a single death. Our caution is perhaps also justified by the fact that hospital records indicate that this case was a pharyngeal tumour extending into the nasal cavities rather than a nasal primary tumour. Grimsrud and Peto appear to approve of our suggestions that retrospective exposure assessment needs to be improved and that recent studies of other nickel exposed workers need to be pooled. We do not, however, approve of their summary of our own conclusions. We are reported to have concluded that the non-significant lung cancer excess “may well be a chance finding”. In fact we concluded that it “may well be a chance finding, but in light of previous studies some role for nickel exposure cannot be excluded”. We trust your readers can spot the difference.

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References

- 1 Grimsrud TK, Peto J. Persisting risk of nickel related lung cancer and nasal cancer among

Clydach refiners. *Occup Environ Med* 2006;63:365–6.

- 2 Sorahan T, Williams SP. Mortality of workers at a nickel carbonyl refinery, 1958–2000. *Occup Environ Med* 2005;62:80–5.
- 3 Easton DF, Peto J, Morgan LG, et al. Respiratory cancer mortality in Welsh nickel refiners: which nickel compounds are responsible? In: Niebor E, Nrigau J, eds. *Nickel and human health: current perspectives. Advances in environmental sciences and technology*. New York: Wiley & Sons, 1992:603–19.

Authors' reply

The main purpose of our short report¹ was to inform your readers that the excess lung cancer mortality among Clydach nickel refiners employed after 1952—despite a lack of significance in the overall data—carried clear signs of an occupational disease, and that nickel exposure was a likely explanation.

Nickel (Ni) compounds are recognised as human carcinogens. The exposure levels in some process departments (eight-hour time weighted averages) were reported to be as high as 0.57 mg Ni/m³ and 0.37 mg Ni/m³ during the 1980s and 1990s, respectively, based on hundreds of personal measurements.² Under the UK Control of Substances Hazardous to Health Regulations of 1994 (COSHH), the maximum exposure limits were 0.5 mg Ni/m³ for insoluble and 0.1 mg Ni/m³ for water soluble nickel compounds.³

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- 1 Grimsrud TK, Peto J. Persisting risk of nickel related lung cancer and nasal cancer among Clydach refiners. *Occup Environ Med* 2006;63:365–6.
- 2 Sorahan T, Williams SP. Mortality of workers at a nickel carbonyl refinery, 1958–2000. *Occup Environ Med* 2005;62:80–5.
- 3 Health & Safety Executive. Nickel and you. <http://www.hse.gov.uk/pubns/indg351.pdf> (accessed 7 June 2006).

CORRECTIONS

Rempel DM, Krause N, Goldberg R, et al. A randomised controlled trial evaluating the effects of two workstation interventions on upper body pain and incident musculoskeletal disorders among computer operators. *Occup Environ Med* 2006;63:300–306.

Unfortunately the last author was incorrectly listed as G U Goldner. The correct name of the last author is G Urbiel Goldner.

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Langseth H, Kjærheim K. Mortality from non-malignant diseases in a cohort of female pulp and paper workers in Norway. *Occup Environ Med* 2006;63:741–5.

Unfortunately Table 1 of the above paper was set incorrectly. The correct table is reproduced below.

Table 1 Standardised mortality ratios (SMRs) with 95% confidence intervals (95% CIs) for selected causes of death in female pulp and paper workers in Norway by duration of employment

		Duration of employment								
		<3 years			≥3 years			Total		
Cause of death	ICD 9th revision	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All non-malignant causes	000–139, 210–999	100	1.33	1.09–1.62	416	1.10	1.00–1.22	516	1.14	1.05–1.24
All cardiovascular diseases	390–459	54	1.33	1.00–1.74	261	1.14	1.01–1.29	315	1.17	1.05–1.30
Ischaemic heart disease	410–414	32	1.73	1.18–2.44	115	1.12	0.94–1.35	147	1.22	1.03–1.43
Cerebrovascular diseases	430–438	10	0.83	0.40–1.52	85	1.22	0.97–1.51	95	1.16	0.94–1.42
Rheumatic/valve	425–429	6	1.10	0.40–2.40	31	1.03	0.70–1.46	37	1.04	0.73–1.43
Other cardiovascular	420–423	6	1.33	0.49–2.89	30	1.12	0.76–1.60	36	1.15	0.81–1.59
All respiratory diseases	460–519	14	1.79	0.98–3.01	43	1.01	0.73–1.36	57	1.13	0.86–1.47
COPD*	490–494, 496	6	1.92	0.71–4.18	13	0.99	0.53–1.69	19	1.17	0.70–1.82
Pneumonia	480–487	6	1.52	0.56–3.31	27	1.06	0.70–1.54	33	1.12	0.77–1.57
Other respiratory		2	2.66	0.32–9.62	3	0.79	0.16–2.31	5	1.10	0.36–2.56
Diabetes mellitus	250	3	1.85	0.38–5.41	7	0.98	0.39–2.02	10	1.14	0.55–2.10
Urinary system/genitalia	580–594	2	1.34	0.16–4.85	9	1.25	0.57–2.37	11	1.26	0.63–2.26
Digestive system	520–579	6	1.60	0.59–3.49	17	1.00	0.58–1.60	23	1.11	0.70–1.66
Sudden death/violent	E800–E929	12	1.65	0.85–2.86	23	1.01	0.64–1.51	35	1.16	0.81–1.62
Other non-malignant causes		9	0.66	0.30–1.25	56	0.95	0.72–1.23	65	0.89	0.69–1.14

*Chronic obstructive pulmonary disease (asthma included).
Follow up period 1951–2000.