2. Studies of Cancer in Humans

Many populations have low-grade or infrequent exposure to metallic mercury or mercury compounds. The Working Group restricted their review to studies specific to metallic mercury or mercury compounds and to groups who are known to have considerable exposure.

2.1 Inorganic mercury compounds

2.1.1 *Descriptive studies*

In a study in Poland, mercury was determined in the hair of leukaemia patients and in healthy relatives and unrelated healthy subjects (Janicki *et al.*, 1987). The mean content of total mercury was 1.24 ± 1.93 mg/kg hair from 23 cases of acute leukaemia and 0.49

± 0.41 mg/kg hair from 79 healthy control subjects. In 47 cases of acute leukaemia (chronic granulocytic as well as chronic lymphocytic), the mercury content was 0.92 ± 1.44 mg/kg hair. For 19 leukaemia cases of all groups and their 52 relatives, the corresponding figures were 0.69 ± 0.75 mg/kg and 0.43 ± 0.24 mg/kg, respectively. These differences between cases and control subjects were significant. [The Working Group noted that comparisons of means are inappropriate, as the distributions were highly skewed, and that the distribution of mercury may have been affected by the disease.]

In Washington State, USA, occupational mortality was studied for the period 1950–71 on the basis of death certificates (Milham, 1976). For male dentists, the proportionate mortality ratio (PMR) for all malignant neoplasms was 1.05 (127 cases [95% confidence interval (CI), 0.88–1.25]). When sites with more than five cases were considered, the PMR was 1.53 for pancreatic cancer based on 12 cases [95% CI, 0.79–2.69), 1.32 for prostatic cancer based on 20 cases [95% CI, 0.80–2.03] and 1.45 for neoplasms of the lymphatic and haematopoietic tissues based on 17 cases [95% CI, 0.84–2.33).

Occupational mortality was studied in British Columbia, Canada, by the proportionate mortality method and based on 320 423 deaths for which valid records were available among men over 20 years of age (Gallagher *et al.*, 1985). The occupational codes were those used in conjunction with the censuses of 1951 and 1961. Among dentists, there were four cases of kidney cancer (PMR, 1.94; 95% CI, 0.52–4.96) and five tumours of the brain and central nervous system (PMR, 2.36; 95% CI, 0.76–5.52). There were even fewer cases at other sites, or no more than slightly elevated PMRs.

2.1.2 *Cohort studies* (see Table 12, p. 272)

(a) Nuclear weapons industry workers

A cohort of 2133 white men from Oak Ridge, TN, USA, who were exposed to metallic mercury and an unexposed cohort of 3260 workers from the same plant were studied with regard to mortality in comparison with national rates for white men (Cragle *et al.*, 1984). Exposure to mercury occurred in the context of lithium production in a nuclear weapons plant, which earlier had also produced a fissionable isotope of uranium; anyone in whom mercury had ever been found in the urine, regardless of the concentration, was considered to have been exposed. A mercury monitoring programme was started in mid-1953 and became effective in late 1954. The cohorts were followed-up from 1 January 1953 until 1 January 1979, when vital status was assessable for at least 95.5% of the cohort and death certificates were available for 98% or more. Total mortality was lower than expected for both groups, and there was no excess of any non-cancer death possibly related to mercury exposure (target organs were thought to be liver, lung, brain and other central nervous system, and kidney). The cancer mortality rate was lower than expected for the exposed cohort (standardized mortality ratio [SMR], 0.94 [95% CI, 0.75–1.16]; based on 85 cases) but not for the unexposed (SMR, 1.10 [0.94–1.28]; based on 175 cases). An excess of lung cancer was seen in both cohorts (SMR, 1.34 [0.97–1.81], based on 42 cases among exposed; and 1.34 [1.05–1.69], based on 71 cases among unexposed). For cancers of the brain and central nervous system, the corresponding figures were 1.22 ([0.33–3.12]; based on 4 cases) for the exposed cohort and 2.30 ([1.22–3.94]; based on 13 cases) for the unexposed; for kidney cancer, the SMRs were reported to be 1.65 ([0.45–4.23]; based on 4 cases) for the exposed

cohort and 0.72 ([0.15–2.10]; based on 3 cases) for the unexposed. In subgroups with mercury levels in urine exceeding 0.3 mg/L at least once or with more than one year of exposure, there was also no clear increase in cancer mortality rates. No definite explanation could be given for the excess of lung cancer observed in both cohorts, but life-style factors or some factor other than mercury present in the plant were mentioned.

(b) Dentists

Cohorts of 3454 male and 1125 female dentists and 4662 dental nurses identified from the Swedish census in 1960 were followed for cancer development in the period 1961–79 by linkage with cancer register data (Ahlbom *et al.*, 1986). The overall standardized incidence ratio (SIR) was 2.1 (95% CI, 1.3–3.4) for glioblastoma (astrocytoma III–IV) in comparison with national incidence rates, based on 18 cases. The SIRs for the various cohorts were 2.0 for male dentists, 2.5 for female dentists and 2.2 for dental nurses. In the combined cohorts, there were also four gliomas (astrocytoma I–II) (SIR, 1.8; 95% CI, 0.5–4.7) and six meningiomas (SIR, 1.3; 95% CI, 0.5–2.8). There was no excess of all tumours in these cohorts. For comparison, physicians and female nurses were also studied; no indication was found of an excess of glioblastomas. Exposures to amalgam, chloroform and X-radiation were mentioned as possible occupational factors.

In another analysis of this population, occupational risks for intracranial gliomas in Sweden were studied by linking cancer incidence data from the national cancer registry during 1961–79 with census data on occupation from 1960 (McLaughlin *et al.*, 1987). The expected number of cases for each occupational category was calculated on the basis of the general population in the study period, and regional adjustment was applied. There were 3394 gliomas in men and 1035 in women who had been employed in 1960. An excess risk was found for male dentists, with an SIR of 2.1 ($p < 0.05$) based on 12 cases; for female dental assistants, nine cases (SIR, 2.1; $p = 0.09$) were reported. For comparison, it may be noted also that among male physicians there were 14 cases (SIR, 1.4; nonsignificant) and among female physicians, four cases (SIR, 3.7; $p < 0.05$). Male chemists, physicists, veterinary surgeons, agricultural research scientists and pharmacists also had SIRs greater than 2.0. [The Working Group noted that no distinction was made between the various subtypes of glioma.]

Mortality risks by occupation have been studied among veterans who served in the US Armed Forces between 1917 and 1940 (Hrubec *et al.*, 1992). Occupation and smoking status were assessed through questionnaires in 1954 and 1957. Follow-up to 1980 was done using insurance and pension systems (96% complete for First World War veterans). The smoking-adjusted relative risk (RR) for each occupation was estimated by using all other occupations as the standard, and Poisson regression modelling was applied. In a subcohort of 2498 dentists with a total of 1740 deaths, there were 299 cancer deaths (RR, 0.9; 90% CI, 0.80–0.97). The risk for pancreatic cancer was 1.4 (90% CI, 0.98–1.86; 27 deaths). No excess of brain or kidney tumours was detected (RR, 0.9; 90% CI, 0.45–1.74; 6 cases; and RR, 0.8; 90% CI, 0.39–1.50; 6 cases, respectively). For a group of 267 medical and dental technicians, there was an elevated risk for all cancers among 40 nonsmokers (RR, 2.5; 90% CI, 1.36–4.73; 7 deaths). For nonsmokers and smokers in this group, the risk for all cancers was only slightly

elevated (RR, 1.2; 90% CI, 0.87–1.54; 34 deaths), but there was an excess of colon cancer (RR, 1.9; 90% CI, 1.01–3.53; 7 deaths).

(*c*) *Chloralkali workers*

Mortality and cancer incidence were reported for a group of 1190 male Swedish chloralkali workers in whom mercury had been measured in the blood or urine for at least one year between 1946 and 1984 (Barregård *et al.*, 1990). Their mortality and cancer incidence were compared with those of the general male population for the periods 1958–84 and 1958–82, respectively, and the follow-up was complete. The mean level of mercury excreted in the urine had been about 200 μg/L in the 1950s, 150 μg/L in the 1960s and less than 50 μg/L in the 1980s. On the basis of crude estimates, 26% of the cohort was estimated to have had an accumulated urinary mercury dose of 1000 years·μg/L or more, 457 subjects also had some (mostly low-grade) asbestos exposure; exposure to static magnetic fields was reported to have occurred. Mortality from all causes was not significantly increased, the observed to expected mortality being 1.1 (95% CI, 0.9–1.3) based on 147 deaths with 10 or more years of latency. There were 51 incident cases of cancer observed *versus* 42 expected with a latency of 10 years or more, i.e. a rate ratio of 1.2 (95% CI, 0.9–1.6). Lung cancer was the only type of tumour in clear excess, with 10 observed and 4.9 expected with a latency of 10 years or more (rate ratio, 2.0; 95% CI, 1.0–3.8). There were slight excesses of some other cancers with a latency of 10 years or more, namely three brain tumours *versus* 1.1 expected (RR, 2.7; 95% CI, 0.5–7.7), three kidney cancers *versus* 1.9 expected (1.6; 0.3–4.7), five urinary bladder cancers *versus* 2.9 expected (1.7; 0.6–4.1) and 10 prostatic cancers *versus* 8.6 expected (1.2; 0.6–2.1). The excess of lung cancer was thought to be due to exposure to asbestos; one case of mesothelioma was observed. Smoking was considered to explain 10% of the excess of lung cancer, although information on smoking habits was available for only a 7% random sample of the cohort. The authors noted that chloralkali workers have five to 10 times the mercury exposure of dental personnel.

In a cohort study of 674 male Norwegian chloralkali workers exposed to inorganic mercury for more than one year prior to 1980, who had a mean cumulative urinary concentration of 740 μg/L, there were 204 deaths *versus* 210.7 expected (SMR, 0.97; 95% CI, 0.84–1.11) and 89 incident cases of cancer *versus* 85.0 expected (SIR, 1.05; 95% CI, 0.84–1.29) (Ellingsen *et al.*, 1993). During the follow-up period (1953–89 for incidence and 1953–88 for mortality), there were 19 incident cases of lung cancer, with 11.5 expected (SIR, 1.66; 95% CI, 1.00–2.59) on the basis of national rates. There was no correlation with cumulative mercury dose, employment or latency; a somewhat increased frequency of smoking and exposure to asbestos (one mesothelioma was found) were considered to explain the excess of lung cancer. Three kidney cancers and two brain tumours were observed *versus* 3.2 and 2.45 expected, respectively. These two sites were considered by the authors to be of primary interest with regard to exposure to mercury.

(*d*) *Mercury miners*

In a cohort study of the relationship between silicosis and mortality from lung cancer in US metal miners, the difference in risk for silicotic miners compared with nonsilicotic white metal miners was greater for mercury miners than for other miners (Amandus & Costello,

Table 12. Cohort studies of populations exposed to inorganic mercury compounds

Study population Period of follow-up	End-point		Site	No. of cases	SMR	95% CI	Reference
Nuclear weapons industry workers							
2133 Mercury exposed,	Mortality	Exposed	Lung	42	1.34	[1.0–1.8]	Cragle *et al.*
3260 unexposed male workers,			Kidney	4	1.65	[0.4–4.2]	(1984)
USA, 1953–79			Brain	4	1.22	[0.3–3.1]	
		Unexposed	Lung	71	1.34	[1.0–1.7]	
			Kidney	3	0.72	[0.1–2.1]	
			Brain	13	2.30	[1.2–3.9]	
Dentists							
9201 Dentists and dental	Incidence		Glioblastoma	18	2.1	1.3–3.4	Ahlbom *et al.*
nurses, Sweden, 1961–79			Glioma	4	1.8	0.5–4.7	(1986)
			Meningioma	6	1.3	0.5–2.8	
2498 Dentists, US veterans,	Mortality		Pancreas	27	1.4	0.96–1.86	Hrubec *et al.*
1954–80			Brain	6	0.9	0.45–1.74	(1992)
			Kidney	6	0.8	0.39–1.50	
267 Medical and dental	Mortality		Colon	7	1.9	1.01–3.53	Hrubec *et al.*
assistants, US veterans,			Brain	1	1.5	NR	(1992)
1954–80			Kidney	2	2.8	NR	
Chloralkali workers							
1190 Males, Sweden,	Incidence		Lung	13	[1.8]	[0.9–3.0]	Barregård *et al.*
1946–82			Kidney	4	[1.3]	[0.4–3.4]	(1990)
			Brain	4	[1.8]	[0.5–4.7]	
674 Males, Norway,	Incidence		Lung	19	1.66	1.00–2.59	Ellingsen *et al.*
1953–89			Kidney	3	0.95	0.2–2.8	(1993)
			Brain	2	0.8	0.1–3.0	
Mercury miners							
274 Males, USA,	Mortality	11 Silicotics	Lung	3	14.0	2.89–41.0	Amandus &
1959/61–75		263 Nonsilicotics	Lung	8	2.66	1.15–5.24	Costello (1991)

NR, not reported

1991). The follow-up was from date of examination in 1959–61 to 31 December 1975. For the 11 silicotic mercury miners, the SMR was 14.0 (95% CI, 2.89–41.0) based on three lung cancer deaths, whereas the SMR for the 263 nonsilicotic mercury miners was 2.66 (95% CI, 1.15–5.24) based on eight cases. For other miners (copper, lead–zinc, iron and others), the corresponding figures were 1.39 [95% CI, 0.70–2.49] based on 11 silicotic lung cancer deaths and [1.14; 95% CI, 0.93–1.37] based on 110 deaths from nonsilicotic lung cancer. The reference for calculating the SMRs was death rates in white US males. No explanation was offered for the differences seen between mercury and other miners. [The Working Group noted that the small numbers of silicotic mercury miners may make the estimate unstable.]

2.1.3 *Case–control studies* (see Table 13, p. 274)

In a case–control study of incident cases of lung cancer admitted during 1981–83, 340 male and 36 female cases and 817 male and 75 female hospital controls, all residents of metropolitan Florence, Italy, were drawn from the regional general hospital for the analyses (Buiatti *et al.*, 1985). Occupational histories were collected from each subject directly; six female cases but no control had ever worked as felt-hat makers ($p = 0.01$). Heavy exposure to mercury but also to arsenic and other chemicals was reported to have occurred in the Italian hat-making industry.

In a study described in detail in the monograph on beryllium (pp. 73–74; Carpenter *et al.*, 1988), based on 29 cases identified from information on death certificates as ever exposed to mercury, the odds ratio for cancer of the central nervous system was 1.77 [95% CI, 0.5–5.8] when compared with unexposed cases. The matched analysis by highest rank ever held *versus* rank 0 yielded odds ratios of 2.01, 1.33 and 1.19 for ranks 1, 2 and 3, respectively (all odds ratios had a p value of 0.26 or greater). When risk estimates were calculated with a 10-year latency, the odds ratios were 1.58, 0.77 and 1.57 for ranks 1, 2 and 3, respectively, with a p value of 0.47 or greater. A further analysis based on time spent in ranks 2 and 3, assuming a 10-year latency, yielded odds ratios of 0.00, 0.96, 0.00 and 1.86 for workers with > 1 year and < 3 years, 3–10 years, 11–20 years and 21 years or more in ranks 2 and 3 compared with ranks 0 and 1. The authors concluded that their study does not support the hypothesis that occupational exposures to any of the 26 chemicals studied increase appreciably the risk for cancers of the central nervous system.

The effects of a great number of exposures were considered in a case–control study from Montréal, Canada, involving all major cancer forms and population controls as well as two hospital control series, i.e. cancer cases and other cases (Siemiatycki, 1991). In total, 4576 incident cancer cases were recruited through local informants at the hospitals. Completed questionnaires and interviews on occupational exposures (293 agents were considered) were obtained for 3730 of these (response rate, 81.5%). A total of 740 population controls were drawn from electoral lists or obtained by random-digit dialling. Of these, exposure was successfully assessed for 533 (72.0%). The prevalence of exposure to metallic mercury was 0.6% and that to any mercury compound (including metallic mercury), 2%. For prostatic cancer, 14 of 449 cases were exposed to mercury compounds, resulting in an odds ratio of 1.7 (90% CI, 1.0–3.0); five cases had been exposed to metallic mercury, giving an odds ratio of 6.2 (90% CI, 1.2–33.2). For lung cancer, four of the 857 cases had been exposed to metallic mercury (odds ratio, 4.0; 90% CI, 1.2–13.0). For bladder cancer, 14 of the 484 cases had been

Table 13. Case–control studies of populations exposed to inorganic mercury compounds

Study population	End-point	Exposure	Sex	No. of exposed cases	Odds ratio	95% CI	Reference
Lung cancer							
Hospital-based, Italy	Incidence	Hat makers	F	6		$p = 0.01$	Buiatti *et al.* (1985)
Population-based, Canada	Incidence	Mercury, metallic	M	4	4.0	1.2–13.0[a]	Siemiatycki (1991)
Prostatic cancer							
Population-based, Canada	Incidence	Mercury, metallic	M	5	6.2	1.2–33.2[a]	Siemiatycki (1991)
		Mercury and mercury compounds[b]	M	14	1.7	1.0–3.0	
Bladder cancer							
Population-based, Canada	Incidence	Mercury and mercury compounds[b]	M	14	1.5	0.9–2.6[a]	Siemiatycki (1991)
Brain tumours							
Population-based, USA	Mortality	Nuclear facilities	Central nervous system	29	1.77	[0.5–5.8]	Carpenter *et al.* (1988)
Population-based, Australia	Incidence	Amalgam fillings	Glioma		0.47	0.25–0.91	Ryan *et al.* (1992)
			Meningioma		1.04	0.43–2.47	

[a]90% CI

[b]Including organomercury compounds

exposed to mercury compounds (odds ratio, 1.5; 90% CI, 0.9–2.6). Significant results were not obtained for cancers at other sites. [The Working Group noted that although several potential confounding factors were considered not all possible occupational confounders were addressed.]

A case–control study from Adelaide, Australia, considered incident brain tumours and exposure to amalgam fillings and diagnostic dental X-rays (Ryan *et al.*, 1992). Cases aged 25–74 were notified by neurosurgeons in Adelaide, and there was a further check for cases in cancer and brain tumour registries. Controls were selected from the Australian electoral roll, covering 95% of the adult population. In total, 190 cases of brain tumours were identified, together with 662 controls; of these, 110 glioma cases, 60 meningioma cases and 417 controls were included in the analyses. There was a decreased odds ratio (0.47; 95% CI, 0.25–0.91) for glioma in association with self-reported amalgam fillings (at least one filling) and with diagnostic X-rays (at least one X-ray) (odds ratio, 0.42; 95% CI, 0.24–0.76); the corresponding results for meningioma were 1.04 (95% CI, 0.43–2.47) in relation to fillings, whereas the risk associated with diagnostic X-rays was slightly increased (odds ratio, 1.37; 0.68–2.73). No dose–response pattern was seen for either glioma or meningioma with regard to amalgam fillings. The authors considered a biological protective mechanism unlikely.

2.2 Organomercury compounds

2.2.1 *Descriptive studies*

Direct SMRs for biliary tract cancer in the Japanese prefectures in 1975 were correlated with an environmental pollution index related to use of agricultural chemical products for the years 1962–66 (Yamamoto *et al.*, 1986). In both men and women, only weak, non-significant correlations were found for exposure to mercuric compounds (such as phenylmercury acetate, used as a fungicide in Japan until 1971) converted to the dose of inorganic mercury, whereas positive and significant correlations were obtained, especially for DDT and some phenoxy herbicides.

The mortality pattern was studied in the population of a small area of the city of Minamata, Kumamoto Prefecture, Japan, which consisted mainly of fishermen and their families (Tamashiro *et al.*, 1986) and where 70% of the 1612 confirmed cases (including 527 deaths) of Minamata disease (see pp. 291–292) in the Prefecture through 1983 were known to have occurred. SMRs were computed for different causes of death in 1970–81 by using age-specific rates for the entire city for 1972–78. The total population of the study area in 1975 was 3887 *versus* 36 782 in the city. Some migration took place during the study period, and, in particular, young adults moved out of the area and former residents returned. The SMR for all causes of death was 1.05 (95% CI, 0.95–1.15, based on 412 deaths) and that for all cancers was 1.18 (95% CI, 0.96–1.46, based on 84 deaths). For the various cancers reported, the corresponding figures were: oesophagus, 2.05 (95% CI, 0.67–4.78; 5 cases); stomach, 0.77 (95% CI, 0.42–1.29; 14 cases); liver, 2.07 (95% CI, 1.16–3.42; 15 cases); pancreas, 0.99 (95% CI, 0.20–2.88; 3 cases); trachea–bronchus–lung, 1.52 (95% CI, 0.79–2.65; 12 cases); breast, 2.64 (95% CI, 0.54–7.71; 3 cases); uterus, 0.89 (95% CI, 0.24–2.28; 4 cases); leukaemia, 1.82 (95% CI, 0.50–4.66; 4 cases); and other cancers, 0.98 (95% CI, 0.63–1.46; 24 cases). An elevated SMR was also seen for chronic liver disease and cirrhosis

(2.16; 95% CI, 1.41–3.17; based on 26 cases). There was some evidence that alcohol consumption in the area was above the Japanese average. [The Working Group noted that the increased risk for liver cancer seems consistent with the increased occurrence of chronic liver disease and cirrhosis and with a higher than average alcohol consumption; the latter might also have affected the risk for oesophageal cancer.]

The effects on life expectancy of elevated exposure to methylmercury compounds were studied in five coastal towns of southern Japan in comparison with a surrounding control area (Tamashiro *et al.*, 1987). The average hair concentrations of mercury were reported to be three to six times higher in the exposed area than in the control area. The study period was from 1969 through to 1982. The crude RR for death from malignant neoplasms was [1.05].

2.2.2 *Cohort study*

It was reported in letter to the Editor that 1657 people with a licence for seed disinfection using organomercury compounds and other agents, issued between 1965 and 1976, were followed through the Swedish Cancer Registry from the date of licencing until death or December 1982 (Wiklund *et al.*, 1988). The mean follow-up time was 14.7 years, resulting in 24 429 person-years of observation. Five tumours of the nervous system were observed *versus* 4.98 expected (SIR, 1.0; 95% CI, 0.33–2.34); rates of tumours at other sites were not reported. The authors noted that the use of alkylmercury compounds was banned in Sweden in the mid-1960s, and limitations were placed on mercury disinfection.

2.2.3 *Case–control studies* (see Table 14, p. 277)

Three similarly designed studies on soft-tissue sarcomas in different parts of Sweden, mainly focusing on exposure to phenoxyacetic acid herbicides and chlorophenols, also provide data on exposure to organomercury seed dressings and other pesticides (Eriksson *et al.*, 1981; Hardell & Eriksson, 1988; Eriksson *et al.*, 1990). The first study encompassed 110 cases and 219 population controls in the five southernmost counties. The second study involved 55 cases and 220 living and 110 dead controls and a third group of 190 other cancer controls in the three northernmost counties. In the third study, there were 237 cases and 237 controls from the seven central counties, matched on vital status. Information on exposure was obtained from questionnaires to the subjects or their next-of-kin, supplemented with telephone interviews. Exposure to mercury seed dressings was reported for 8.2% of cases and 4.6% of controls in the first study; for 1.9% of cases and 3.5% of living and 2.8% of dead controls in the second study; and for 4.6% of cases and 5.2% of controls in the third study. The resulting odds ratio in the first study [not given] was said to have a 90% CI that included unity. [A calculation results in a crude odds ratio of 1.9 (95% CI, 0.65–5.3) for the first study and, for the second study, 0.52 (95% CI, 0.01–4.3) with regard to living controls and 0.66 (95% CI, 0.08–5.74) using dead controls.] The odds ratio in the third study was given as 0.89 (95% CI, 0.40–1.96).

In a study from northern Sweden on malignant lymphomas, which mainly considered exposure to organic solvents, chlorophenols and phenoxyacetic acid herbicides, exposure frequencies to organomercury seed dressings were also reported (Hardell *et al.*, 1981). The study included 169 cases (60 Hodgkin's lymphomas, 109 non-Hodgkin lymphomas) and 338

(335 used in the calculation) population controls. Information on exposure was obtained through questionnaires. For the cases and controls, 5.3 and 3.0%, respectively, exposure to mercury seed dressings co-varied with exposure to phenoxyacetic acid herbicides, whereas asbestos and glass fibre exposure co-varied with chlorophenol exposure. Exposure to phenoxyacetic acid herbicides as well as to chlorophenols appeared to be strong risk factors for lymphomas, but after exclusion of subjects with exposure to phenoxy herbicides, 128 cases and 311 controls remained, with exposure frequencies to mercury seed dressings of 4.7 and 2.9%, respectively; for DDT, the corresponding figures were 5.5 and 3.5%. [For the restricted material, a calculation results in a crude odds ratio of 1.78 (95% CI, 0.62–5.11) for mercury seed dressings and 1.6 (95% CI, 0.51–4.6) for DDT.]

Table 14. Population-based case–control studies of populations of men exposed to organomercury seed dressings in Sweden

No. of exposed cases	Odds ratio	95% CI	Reference
Soft-tissue sarcomas			
[9]	[1.9]	[0.65–5.3]	Eriksson *et al.* (1981)
[1]	[0.52][a] [0.66][b]	[0.01–4.3] [0.08–5.7]	Hardell & Eriksson (1988)
[10]	0.89	0.40–1.96	Eriksson *et al.* (1990)
Lymphomas			
[6]	1.78	[0.62–5.1]	Hardell *et al.* (1981)

[a] Living controls

[b] Dead controls