Studies of high or moderate quality used for results and conclusions in the present report

First author Pub. Year Reference Country	Design Time to follow-up Setting Performed (years)	Participants Women/men	Occupational factor (-s)	Outcome	Association between occupational factor and cardiovascular disease; least adjusted model	Association between occupational factor and cardiovascular disease; most adjusted model
Ahlman et al 1991 [1] Finland	Cohort study Copper mine 1954–1986	The study cohort consisted of 597 miners first employed between 1954–1973 by a new copper mine and a zinc mine, and employed there for at least 3 years. The period of follow-up was 1954–1986. The number of person-years was 14 782 In addition, the researchers used a separate comparison group of 338 surface workers, at the same mines, who had first	**Several chemicals** Only dust measurements (based on particle counting) were made in the old copper mine. For other exposures, the researchers used estimations based on later measurements, some of which had been made in simulated conditions Silica dust: exposure to dust in the old mine was estimated on the basis of earlier data	In the cohort study, the expected numbers of deaths were calculated from the official statistics on causes of death in 1979 [WHO, 1982] the median year of the deaths in the miners' cohort [Koskela et al., 1987] for the general male population and also for the provincial North Karelian male population	Observed and Expected Numbers of Certain Causes of Death for· the Cohort of Sulphide Orc Miners at the End of 1986 Cardiovascular diseases Observed: 55 Expected Finnish males: 30.8, p<0.001 Expected North Karelian males: 43.0 lschemic heart disease Observed: 44 Expected Finnish males: 22.1, p<0.001 Expected North Karelian males: 31.2, p<0.05 Observed and Expected Numbers of Certain Causes of Death for the Comparison Cohort of Surface Workers at the End of 1986 Cardiovascular diseases Observed: 35 Expected Finnish males: 26.5 Expected North Karelian males: 33.9 lschemic heart disease Observed: 26 Expected Finnish males: 18.8 Expected North Karelian males: 24.1	–

		been employed by the company between 1954–1973	(particle counting) and through interpolation of the results of measurements 1950–1981. The estimated total dust concentration was >50 mg/m^3 during dry Drilling (corresponding respirable silica dust concentration >2 mg/m^3). In the early 1940s, wet drilling decreased the total dust concentration and since 1948, it was estimated to be <1O mg/m^3 Diesel exhaust gases: No diesel machinery had been used in the old copper mine. The only sources of PAH compounds were those possibly contained in oil mist			

			According to measurements made in the beginning of the 1980s, exposure to PAHs, namely benzo(a)pyrene (BaP), in loading work has been fairly low, less than 20 ng/m^3. The mean exposure (8 hours average) to both carbon monoxide (CO) and nitrogen oxides (NO) had been about 5 ppm or less. The highest measured concentrations of CO and NOx were about 25 ppm. Diesel exhaust gases also contain same amounts of nitroarenes, but no analytical results from the mines were available Oil mist: airborne concentrations during drilling were, on the			

			average, 3 mg/m^3 in the 1970s. The range was from 0.1–17 mg/m^3. In the 1950s and 1960s when airleg drilling machines were used, exposure to oil mist was about double that measured in the 1970s Arsenic: the copper and zinc ores contained arsenic only as a trace element, <0.005%			
Andersson et al 2007 [2] Sweden	Cohort study Pulp and paper mills 1952–2001	Participants were employed at 2 Swedish pulp and paper mills. An overall inclusion criterion was >1 year of employment in the mill The median duration of employment was 12 (range 1–60) years. The median age at employment was 24 (range 7–69) years	**Several exposures, eg. dust** The participants were first categorized according to the main pulping process of the mill in which they were employed. Then, the department and job title were defined for each period of employment. The analysis was performed	**Several conditions** The cohort was linked to the national Causes of Death Register, and the causes of death were given according to the 9th revision of the International Classification of Diseases (ICD-9). Acute myocardial infarction (ICD	Standardized mortality ratios by diagnostic group, main mill pulping process, and gender for the Swedish pulp and paper mill workers in 1952–2001. SMR (95% CI) **Acute myocardial infarction** Sulphate mills (all departments) 1.22 (1.12; 1.32) Sulphite mills (all departments) 1.11 (1.02; 1.21) **Ischemic heart disease (410–414)** *Sulphate mills* Women: 0.97 (0.70; 1.30) Men: 1.09 (1.02; 1.16) *Sulphite mills* Women: 1.00 (0.72; 1.36) Men: 0.96 (0.89; 1.02)	–

		n=20 454 2 291 women 18 163 men	by department as a proxy for exposure The article presents a table of air sample concentrations of some of the chemical agents in the Swedish database of exposures in the pulp and paper industry. The table includes data on dust (wood, paper, respirable dust, inorganic dust and total dust), sulphur dioxide, calcium oxide, dimethyl sulphide, dimethyl disulphide, dihydrogen sulphide and methyl mercaptan	410) was not distinguished earlier than 1969	**Cerebrovascular disease (430–438)** *Sulphate mills* Women: 1.05 (0.67; 1.56) Men: 1.06 (0.93; 1.20) *Sulphite mills* Women: 0.72 (0.40; 1.19) Men: 0.94 (0.83; 1.07) Standardized mortality ratios by diagnostic group and department for in Swedish pulp and paper mills in 1952–2001. SMR (95% CI) **ischemic heart disease (410–414)** *Women* Paper production: 1.06 (0.73; 1.48) Office: 0.96 (0.66; 1.44) *Men* Wood preparation: 0.96 (0.83; 1.09) Sulphite pulping: 1.03 (0.90; 1.17) Sulphate pulping: 1.17 (1.02; 1.34) Maintenance: 0.96 (0.87; 1.05) Paper production: 1.12 (0.98; 1.27) Office: 0.94 (0.81; 1.07) **Cerebrovascular disease {430–438)** *Women* Paper production: 0.93 (0.52; 1.53) Office: 0.89 (0.45; 1.60) *Men* Wood preparation: 0.82 (0.61; 1.08) Sulphite pulping: 0.81 (0.59; 1.08) Sulphate pulping: 0.95 (0.70; 1.27) Maintenance: 1.01 (0.84; 1.22) Paper production: 1.13 (0.85; 1.46) Office: 0.94 (0.71; 1.22)	

Asp et al 1994 [3] Finland	Prospective cohort study Chemical brushwood control 1955–1989	Participants were workers who had been exposed to chlorophenoxy herbicides for at least 2 weeks during 1955–1971. The cohort was assembled from the personnel records of 4 main Finnish employers involved in chemical brushwood control n=1 909 All participants were men	**Chlorophenoxy herbicides** Since the mid 1950s until the 1970s, chloro-phenoxyagents used for brushwood control were almost exclusively 2,4 D and 2,4,5 T in a 2:1 mixture as emulsified esters and amine salts in water solution. Analysis of five 2, 4, 5 ester preparations that had been used during this period showed that 4 of them contained 0.1–0.95 mg/kg (ppm) TCDD, and in 1 preparation the TCDD content was below the detection limit A questionnaire was mailed 1988 to all living cohort members and to the next-of-kin to the deceased	**Mortality to ischemic heart disease, other heart diseases and hypertension, and cerebro-vascular disease** The cohort members was checked against the National Population Register. For those who had died, the causes of death were obtained from death certificates registered at the Central Statistical Office Expected mortality was based on age-specific mortality rates of the general male population	Cause-participants specific mortality of the Finnish Chlorophenoxy Herbicide Cohort, 1972–1989. SMR (90% CI) *Ischemic heart disease* 0.94 (0.80; 1.10) *Other heart diseases and hypertension* 1.03 (0.63; 1.59) *Cerebrovascular disease* 0.73 (0.48; 1.04)	–

			persons in order to collect exposure information			
Axelson et al 1978 [4] Sweden	Case control study Copper smelter factory 1960–1976	Male individuals living in parish around copper smelter factory who died at the age 30–74 years Cases were chosen by death cause: cardiovascular diseases, cerebrovascular disease, malignant tumors and cirrhosis of the liver Control cases were all remaining death causes that did not match the exclusion criteria for cases Exclusion criteria were mental deficiency, vague diagnoses, diabetes	**Several types of chemical exposure** Retrospective exposure classification was identified by using employee registers from 1928. Experienced safety engineer estimated the arsenic exposure Exposure categories: (III) persons with more than 36 months exposure above 0.5 mg/m^3 occurring for more than half of the latency period before death (17 years); (II) persons with less than 36 months exposure above 0.5 mg/m^3. In addition, persons	**Cardio-vascular and cerebro-vascular disease** Cardio-vascular diseases (ICD 410–412, 427–428), cerebro-vascular disease (ICD 430–438) Death causes were taken from the national Causes of Death Register	Crude rate ratios by death cause separate for exposure categories for male individuals living in parish around copper smelter factory who died of cardiovascular diseases or cerebrovascular diseases at the age of 30–74 in the years 1960–1976. RR **Cardiovascular diseases (129 cases)** Arsenic exposure, according to categories: I: 0.6 II: 2.9 III: 3.3 **Cerebrovascular diseases (34 cases)** Arsenic exposure total (with or without lead): 1.7 **Cardiovascular diseases (subpopulation ever employed at copper smelter)** Arsenic: 2.0 Sulfur dioxide: 1.2 Lead: 1.1 Copper: 1.3 Nickel: 1.2 Selenium: 0.9 Bismuth: 1.1 Antimony: 2.1 **Cerebrovascular diseases (subpopulation ever employed at copper smelter)** Arsenic: 3.1 Sulfur dioxide: 2.1 Lead: 1.5 Copper: 4.5 Nickel: 1.3 Selenium: 0.5 Bismuth: 1.5	Standardized mortality ratios by death cause separate for exposure categories for male individuals living in parish around copper smelter factory who died of cardiovascular diseases or cerebrovascular diseases at the age of 30–74 in the years 1960–1976. SMR (90% CI) **Cardiovascular diseases (129 cases)** Arsenic exposure, according to exposure category: I: 0.7 II: 3.0 III: 5.8 *Mantel-Haenszel rate ratio (90% CI)* Arsenic exposure (all exposure categories I, II, III): 2.1 (1.2; 3.5) **Cerebrovascular diseases (34 cases)** *Mantel-Haenszel rate ratio (90% CI)* Arsenic exposure total (with or without lead: 1.6 (0.7; 3.4) **Cardiovascular diseases (subpopulation ever employed at copper smelter):** Antimony 1.7 Arsenic: 0.4 Copper: 1.7

		n=325 (74 selected cases, 251 control cases) All participants were men	suffering from arsenic dermatitis; (I) persons never exposed at or above 0.5 mg/m^3 and without arsenic dermatitis. In addition persons with at least 3 months' exposure at any level occurring between 5–17 years before death; (0) Persons without exposure or exposure shorter or more recent than category (I)		Antimony: 1.5	
Barregård et al 1990 [5] Sweden	Cohort study Chloralkali plant 1946–1984	Participants were male employees at 8 chlorine producing factories using mercury cell process, who had been monitored with urinary or blood mercury (U-Hg, B-Hg) for more than 1 year until 1984 according to company registers	**Mercury exposure** Individual yearly mean urinary levels (U-Hg) were calculated and summed up to accumulated mercury dose over years. If only blood Hg was available, it was transformed into U-Hg by multiplying by 2.5, as suggested in	**Ischemic heart disease, cerebro-vascular disease and cardiovascular disease mortality** mortality data were obtained from the National Population Register, and the National Bureau of Statistics	Observed and expected mortality during 1958–1984 in 1 190 men exposed to inorganic mercury at 8 Swedish chloralkali plants. RR (95% CI) **Inorganic mercury** (>10 years exposure latency) Ischaemic heart disease: 1.3 (1.0; 1;6), p<0.05 Cerebrovascular disease: 1.3 (0.7; 2.2) *Cardiovascular mortality by age at death* ≤59 years: 2.0 (1.1; 3.1), p <0.05 60–69 years: 1.0 (0.7; 1.6) 70–74 years: 1.3 (0.8; 1.9) 75–84 years: 1.2 (0.8; 1.8)	–

		n=1190 workers All participants were men	earlier published reports The mercury exposed chlorine factory workers were compared with Swedish male population as reference group (stratified by calendar year, sex, and age) *Accumulated exposure* (U-Hg in years µg/L): <1 000 in 74%; 1 000–2 000 in 16% and >2 000 in 10% of workers Latency time was calculated from the year of first Hg measurement Asbestos exposure: In most cases low, as reported via survey in 457 workers	between 1958–1984 Observed incidence of ischaemic heart disease (ICD 410–412), cerebro-vascular disease (ICD 430–438) and cardiovascular (ICD 390–458*)* were coded according to ICD-8		
Battista et al 1999 [6] Italy	Cohort study Follow-up 27 years	Participants were male workers whose mean age at	**Asbestos** The use of asbestos as an insulating agent	**Circulatory disease mortality**	Cause specific mortality of railway carriage construction workers. SMR (90% CI) Circulatory disease: 73 (58–92)	–

	Railway carriage construction 1945–1997	hiring was 29 years. They were employed between 1945–1969 n=734 All participants were men	in railway carriages started in the 1940s, with the application of chrysotile boards in specific parts. Starting from the 1950s, crocidolite-containing mixtures were sprayed on the entire internal surface of the carriage. Insulation workers were employed by specialized companies and wore protective equipment, but they often operated while other workers, with no protective equipment, were engaged in different tasks. Exposure to asbestos could thus be caused both by the work process itself or by ambient air contamination	Mortality was investigated in the time span 1970–1997. Vital status and causes of death were ascertained from the Registrar Offices of the municipalities of residence of cohort members Causes of death were coded according to ICD-8 and ICD-9 The observed mortality was contrasted to expected figures obtained by applying Tuscany Region cause-, sex-, age- and calendar year-specific mortality rates of the cohort's person years		

			No measurements of asbestos fibre concentration are available, because at the time this was not recognized as an occupational risk	Besides mortality analysis based on death certificates, a 'best evidence' approach based on the acquisition of all available clinical and pathological material for every deceased person was undertaken		
Bertke et al 2016 [7] Idaho, USA *Note: same population as in study by Steenland, 1992*	Cohort study Lead smelter plants 1940–2013	Participants in the study cohort were white male hourly workers employed for at least 1 year at an Idaho lead smelter plant with at least 1 day of employment between 1940–1965 were followed until 31/12 2013 Cause-of-death referent rates were available for 119 cause-of-death categories	**Lead** A lead job exposure matrix was based on 143 personal air lead concentrations collected by OSHA, Occupational Safety and Health Administration, between 1973 and 1980; coded by department and operator. For each department, time weighted average lead concentration	**Several conditions** The cohort was linked to the National Death Index (NDI) and NDI Plus for determination of vital status and causes of death through 2013. Deaths were coded according to the revision of the International Classification of Diseases (ICD) in effect at time of death	Rate ratios with reference group of lead smelter cohort exposed to less than 209 mg/m3-days, stratified by cumulative lead exposure. Exposure cut-points were selected so that approximately equal numbers of deaths occurred within each stratum. Poisson regression was performed controlling for age and calendar period with 5 year categories. RR (95% CI) **Cardiovascular disease** *Cumulative lead exposure* <209–757 mg/m^3-days: 1.06 (0.89; 1.26) >757 mg/m^3-days: 1.19 (1.00; 1.42) Trend p=0.04 **Diseases of the heart only** *Cumulative lead exposure* <209–757 mg/m^3-days: 1.08 (0.88; 1.32) >757 mg/m^3-days: 1.20 (0.98; 1.46) **Ischemic heart disease** *Cumulative lead exposure*	–

		based on 1960–2007 for the state of Idaho, which is the smelter's location n=1 990 in the study cohort All participants were men	(TWA, mg/m^3) were calculated by averaging each job title (within each department) and then taking the average of the job title averages over department. Departments that were not sampled were earlier identified as having low lead exposure and were thus assigned 0.06 mg/m^3, i.e. the lowest value measured Work history records from 1975 included beginning and ending dates of employment in 14 lead exposed departments Cumulative lead exposure was calculated for each subject by multiplying the assigned exposure level by duration of		<209–757 mg/m^3-days: 1.02 (0.81; 1.27) >757 mg/m^3-days: 1.16 (0.93; 1.45) **Hypertension with heart disease** *Cumulative lead exposure* <209–757 mg/m^3-days: 2.37 (0.48; 11.6) >757 mg/m^3-days: 2.82 (0.58; 13.8) **Other diseases of the circulatory system** *Cumulative lead exposure* <209–757 mg/m^3-days: 1.00 (0.71; 1.41) >757 mg/m^3-days: 1.19 (0.84; 1.67) **Hypertension without heart disease** *Cumulative lead exposure* <209–757 mg/m^3-days: 0.84 (0.5; 1.42) >757 mg/m^3-days: 0.93 (0.55; 1.58) **Cerebrovascular disease** *Cumulative lead exposure* <209–757 mg/m^3-days: 1.13 (0.73; 1.75) >757 mg/m^3-days: 1.38 (0.90; 2.12) Standardized mortality ratio analyses comparing cohort mortality to referent population in Idaho (1960–2007) using the NIOSH Life Table Analysis System for Windows. Standardized mortality ratios were defined as ratio of observed to expected number of deaths and were indirectly standardized based on age and calendar period. SMR (95% CI) Cardiovascular disease: 1.22 (1.13; 1.31) Diseases of the heart only: 1.16 (1.06; 1.26) Ischemic heart disease: 1.18 (1.07; 1.30) Hypertension with heart disease: 1.00 (0.43; 1.96)	

			employment within the department and summing over all jobs worked		Other diseases of the circulatory system 1.40 (1.20; 1.61) Hypertension without heart disease 2.09 (1.00; 3.84) Cerebrovascular disease: 1.32 (1.10; 1.58)	
Bigert et al, 2007 [8] Sweden	Population-based case-control study Subway 1976–1996	The study population consisted of all men 40–69 years of age residing in Stockholm County during 1976–1996 Cases suffered from acute myocardial infarction For the period 1976–1984, the sampling of controls was frequency-matched for gender, age (5-year age groups), and calendar year. 2 controls were selected for each case. For the period 1985–1996, 1 500 controls per age (5-year age groups) and calendar year	**Particles** A participant was classified as a subway driver if he had reported working as such in any census preceding the year of inclusion in the study High levels of airborne particulates have been detected on underground platforms in the subway system of Stockholm (Johansson et al, 2003). The particles originate mainly from brakes, wheels, and rails and contain a high proportion of iron. The particles are mainly in the size range of 1–	**Myocardial infarction** Incident cases of acute myocardial infarction in the study population were identified by using registers of hospital	Relative risk of myocardial infarction for subway drivers in any census and for subway drivers according to the timing or duration of employment. RR (95% CI) adjusted for age group and calendar year **Compared to other manual workers** Subway drivers in any census before inclusion: 0.92 (0.68; 1.25) Job duration ≥5 years before inclusion: 0.84 (0.53; 1.33) Start of employment ≥10 years before inclusion 0.73 (0.48; 1.13) End of employment ≤5 years before inclusion 0.99 (0.59; 1.65) **Compared to others gainfully employed** Subway drivers in any census before inclusion: 1.06 (0.78; 1.43) Job duration ≥5 years before inclusion: 0.96 (0.61; 1.52) Start of employment ≥10 years before inclusion: 0.86 (0.56; 1.32) End of employment ≤5 years before inclusion: 1.10 (0.66; 1.84)	–

		stratum were selected. Persons with a previous history of myocardial infarction were excluded n total=53 807 (22 311 cases and 131 496 controls) n=304 (54 cases and 250 controls had worked as subway drivers) All participants were men	10 µm. The level of particulate matter with an aerodynamic diameter of <10 µm (PM10) in the air of an underground platform in Stockholm was found to be 470 µg/m3 (measured during 2 weeks in the year 2000, average level during weekdays between 07:00–19:00), which is 4–5 times higher than the levels of PM10 found in 1 of the busiest streets in Stockholm (Johansson et al, 2003)			
Bigert et al. 2013 [9] Sweden	Prospective cohort study Restaurants 1987–2005	Participants were cooks and other restaurant workers Restaurant workers were: I) workers identified in the Swedish	**Cooking fumes** Exposure to cooking fumes were approximated by work duration, ≤5 years, ≥5 years and by dividing into subgroups of cook vs.	**First time acute myocardial infarction** (ICD-9 code 410, ICD-10 code I21) during 1987–2005 were identified through the	Hazard ratios for first time acute myocardial infarction are given, separate for women, men, and subgroups. Cox proportional hazards modelling was used adjusting for age, controlled for socioeconomic status. HR (95% CI) Restaurant workers vs reference group *Women* Cooks (n=609): 1.25 (1.14; 1.38) Cold-buffet managers (n=56): 1.11 (0.85; 1.45)	Hazard ratios for first time acute myocardial infarction are given for women, men, and subgroups. Cox proportional hazards modelling was used adjusting for age, hypertension, diabetes, and controlled for socioeconomic status. HR (95% CI) Restaurant workers vs reference group *Women* Cooks (n=609): 1.34 (1.21; 1.48)

		National Census of 1985 by occupational codes. Skilled worker: cooks, cold-buffet manager, and wait staff. Unskilled worker: kitchen and restaurant assistant n=777 496 543 497 women 233 999 men II) Worker subgroup with same occupational code in 1985 and 1990 as a proxy for a work duration of ≥5 years Reference groups were skilled manual workers in the service sector served as reference for cooks, cold-buffet managers, and wait staff in group I) and unskilled	other restaurant workers. Possible exposure levels as reported from other studies are inhalable particles in the range of 0.32–7.51 mg/m^3 during peak hours	nationwide Hospital Discharge Register, recording all in-patient care in Sweden, and through the National Cause of Death Register Risk of myocardial infarction was investigated from 01/01 1987 or immigration, until first of dates: first episode of acute myocardial infarction, death, emigration, or 31/12 2005 Cases of hypertension and diabetes in the study population from registers of hospital discharges and deaths	Wait staff (n=167): 1.15 (0.98; 1.36) Kitchen assistant (n=595): 1.11 (1.02; 1.20) *Women with work duration ≥5 years* Cooks (n=259): 1.20 (1.04; 1.38) Cold-buffet managers (n=23): 1.18 (0.78; 1.78) Wait staff (n=55):1.16 (0.88; 1.53) Kitchen assistants (n=205): 1.08 (0.94; 1.24) *Men* Cooks (n=201): 1.09 (0.94; 1.27) Cold-buffet managers (n=2): 3.27 (0.82; 13.11) Wait staff (n=110): 1.02 (0.84; 1.24) Kitchen assistant (n=86): 1.15 (0.93; 1.42) *Men with work duration ≥5 years* Cooks (n=83): 1.00 (0.79; 1.25) Cold-buffet managers (n=1): 2.42 (0.34; 17.2) Wait staff (n=51): 1.11 (0.84; 1.48) Kitchen assistants (n=25): 1.16 (0.78; 1.71) Risk difference (absolute difference in incidence) of myocardial infarction between cooks and those who never worked as cooks was calculated for men and women, being standardized using the age distribution (in 5 strata) of the entire study base as weights Women: 4.2 (95% CI 0.9; 7.5) per 10 000 person-years Men: 0.5 (95% CI –12.0; 13.0) per 10 000 person-years	Cold-buffet managers (n=56): 1.19 (0.91; 1.55) Wait staff (n=167): 1.25 (1.06; 1.47) Kitchen assistant (n=595): 1.12 (1.03; 1.21) *Women with work duration ≥5 years* Cooks (n=259): 1.31 (1.13; 1.51) Cold-buffet managers (n=23): 1.27 (0.84; 1.93) Wait staff (n=55): 1.28 (0.97; 1.69) Kitchen assistants (n=205): 1.10 (0.96; 1.27) *Men* Cooks (n=201): 1.09 (0.94; 1.27) Cold-buffet managers (n=2): 3.28 (0.82; 13.12) Wait staff (n=110): 1.02 (0.84; 1.24) Kitchen assistant (n=86): 1.14 (0.93; 1.41) *Men with work duration ≥5 years* Cooks (n=83): 1.00 (0.80; 1.26) Cold-buffet managers (n=1): 2.45 (0.35; 17.31) Wait staff (n=51): 1.12 (0.84; 1.49) Kitchen assistants (n=25):1.16 (0.78; 1.72)

		manual workers in the service sector served as reference for kitchen and restaurant assistants in group I) Workers of referent groups who had the same occupational code in 1985 and 1990 served as respective referents for subgroups of group II) Individuals who had worked as a cook, cold-buffet manager, restaurant and kitchen worker, or wait staff (either in 1985 or 1990) were excluded as referents		between 1987 and 2005		
Bjor et al 2010 [10] Sweden	Prospective cohort Iron-ore mines 1923–2001	Participants were employed men at 2 iron-ore mines in Sweden who had been employed for at least 1 year	**Dust** The company has gravimetrically measured the personal exposure to respirable (i.e.	**Myocardial infarction** Mortality was obtained by linking personal identification numbers to	Relative risks (RR) with 95% CI for myocardial infarction mortality in relation to Dust. RR (95% CI) *Total* Not exposed: 1 <35 mg/m^3 x years: 0.98 (0.85; 1.15) >35–100 mg/m^3 x years: 1.21 (1.03; 1.40)	–

		from 1923–1996 The cohort was defined based on employee records at a Swedish mining company. The records contain detailed job histories. Almost 1 200 occupation codes were defined and used n=13 621 All participants were men	dust with aerodynamic diameter ≤5 µm) dust since 1968 The content of crystalline silica dioxide in the respirable fraction dust was estimated to 2.5% Safety engineers from the mines and an occupational hygienist constructed a job-exposure matrix for dust exposure based on occupation. Estimated exposure in mg/m^3 was multiplied by the number of years each worker was employed to achieve a personal cumulative exposure (mg/m^3 x years)	the national cause of. Death register (1952–2001). International Classifications of Diseases (ICD-6–ICD-10) were used for MI classification and the underlying cause of death in MI was used for analysis	>100 mg/m^3 x years: 1.31 (1.13; 1.52) Dust>0: 1.15 (1.02; 1.31) *Attained age ≤60 years* Not exposed: 1 <35 mg/m^3 x years: 0.93 (0.71; 1.23) 35–100mg/m^3 x years: 1.36 (1.01; 1.84) >100 mg/m^3 x years: 1.82 (1.33; 2.49) Dust>0: 1.21 (0.95; 1.53) *Attained age >60 years* Not exposed: 1 <35 mg/m^{3}x years: 1.04 (0.87; 1.25) 35–100mg/m^3 x years: 1.12 (0.84; 1.34) >100 mg/m^3 x years: 1.16 (0.98; 1.37) Dust>0: 1.11 (0.96; 1.29)	
Boers et al 2012 [11]	Retrospective cohort	Participants were workers from 2 factories involved in the	**TCDD** Exposure assessment was based on a	**Several diseases of the circulatory**	Hazard ratios for lagged TCDD plasma levels and selected causes of death. HR (95% CI) adjusted for age	–

The Netherlands	About half of the participants had worked more than 5 years at the factories Pesticide factories 1955–1986	manufacturing of chloro-phenoxy herbicides Other pesticides were also produced in the factories, e.g. including 2.5-dichlorophenol (2.5-DCP), 2,4,5-TCP, dichlobenil, tetradifon1 lindane1 MCPA and MCPP 1 factory had an accident with TCDD released into the production hall after an explosion n=2 056 All participants were men	predictive model for TCDD plasma levels at the time of assumed last exposure that was derived by 2-stage regression modelling A description of the sampling, blood collection and exposure modelling is provided in the article The levels of TCDD was set to the following: Reference: ≤0.4 ppt Low: 0.4–1.9 ppt Medium 1.9–9.9 ppt High: ≥9.9 ppt	**system, mortality** Information on vital status was obtained from municipal records Cause-specific mortality for workers was obtained by linkage to death certificates at the national Bureau of Statistics	**Exposure lagged 1 year** *Total cohort* Diseases, circulatory system: 1.07 (0.98; 1.16} Ischaemic heart disease: 1.19 (1.08; 1.32) Other heart diseases: 0.77 {0.56; 1.05) Cerebrovascular diseases: 0.9B (0.83; 1.16) *Workers at factory exposed to TCDD* Diseases, circulatory system: 1.04 (0.94; 1.16) Ischaemic heart disease: 1.24 (1.09; 1.43) Other heart diseases: 0.67 (0.42; 1.04) Cerebrovascular diseases: 0.90 (0.73; 1.11) *Empirical model*, based on occupational history enrolling periods of employment in different departments. Total cohort by tertiles of (lagged) TCDD plasma levels; compared with workers with background TCDD levels *Ischaemic heart disease (120–125)* Background (reference):1.00 Low: 1.17 (0.65; 2.09) Medium: 1.00 (0.54; 1.85) High: 2.60 (1.57; 4.31) *A priori model*, based on a priori assumed exposure status. Total cohort by textiles of (lagged) TCDD plasma levels; compared with workers with background TCDD levels *Ischaemic heart disease (120–125)* Background (reference): 1.0 Low: 1.02 (0.60; 1.76) Medium: 1.25 (0.72; 2.18) High: 2.78 (1.57; 4.91)	
Bofetta et al 1988 [12] USA	Prospective cohort General population	Participants were men aged 40–79 years with known smoking habit who	**Diesel exhaust** Subjects filled out a 4-page confidential questionnaire	**Several conditions** Ischemic heart disease ICD: 410–414	Association of diesel exposure with cerebrovascular disease mortality. Relative risk for death, RR (95% CI) *Duration of diesel exposure* 1–15 years: 1.43 (0.89; 2.29)	–

	The 2-year mortality 1982–1988	volunteered to participate in the study n=461 981 All participants were men	on exposure and duration The occupational section of the questionnaire included 3 questions on occupation: the current occupation; the last occupation, if retired; the job held for the longest period of time, if different from the other 2	Hypertensive heart disease ICD: 401–405 Other heart disease ICD: 390–398, 415–429 Cerebro-vascular disease ICD: 430–438 Arterio-sclerosis ICD:440 Other vascular disease ICD: 441–459	>15 years: 1.68 (1.06; 2.66) Relative risk of death from cerebrovascular disease among auto mechanics likely to be exposed to gasoline as well as diesel engines exhausts, RR (95% CI) 0.84 (0.48; 1.49) *Diesel Exhaust Exposure and Mortality, RR* Ischemic heart disease: 0.98 Hypertensive heart disease: 1.34 Other heart disease: 0.94 Cerebrovascular disease: 1.61, P <0.05 Arteriosclerosis: 3.12, p<0.05 Other vascular disease: 0.72	
Boice et al 1999 [13] USA	Retrospective cohort mortality study The mean follow-up time was 24 years Aircraft manufacturing industry 1960–1996	Participants were workers employed for at least 1 year at a large aircraft manufacturing facility in California on or after 1 January 1960 n=77 965 (total cohort) 15 488 women 62 477 men Chromate, n=3 634	**Chromate, TCE, PCE and mixed solvents** Factory job titles were classified as to likely use of chemicals, and internal Poisson regression analyses were used to compute mortality risk ratios for categories of years of exposure to chromate,	**Heart disease and cerebrovascular disease, mortality** The mortality experience of the workers was determined by examination of national, state, and company records to the end of 1996	Standardised mortality ratios for workers employed in aircraft manufacturing for at least 1 year since 1960 and followed up to the end of 1996 with potential routine exposure to selected agents (sex and race combined). SMR (95% CI) **All heart disease** Chromate: 0.96 (0.87; 1.06) Trichloroethylene: 0.85 (0.78; 0.94) Perchloroethylene: 0.84 (0.71; 0.99) Mixed solvents: 0.90 (0.85; 0.96) **Cerebrovascular disease** Chromate: 0.73 (0.55; 0.96) Trichloroethylene: 0.66 (0.50; 0.85) Perchloroethylene: 0.86 (0.56; 1.27) Mixed solvents: 0.77 (0.65; 0.90)	–

		Trichloro-ethylene, n=2 267 Perchloro-ethylene, n=2 631 Mixed solvents, n=9 201	Trichloroethylene, Perchloroethylene, and mixed solvents, with unexposed factory workers serving as referents Walkthrough surveys, interviews, industrial hygiene files, job descriptions and other historical documents were reviewed. Also, job code and title combinations were obtained from personnel records, and job changes. From this information job families was identified as were job titles with potential for the exposure of interest, and duration of exposure Individual workers were then classified	Cause of death, coded according to the international classification of diseases (ICD) code in use at the time of death All heart disease was defined as ICD-9 codes 390–398, 402, 404, 410–429 Cerebrovascular disease was defined as ICD-9 codes 430–438)		

			into categories of routine, intermittent, or no likely exposure to chromate, TCE, PCE and mixed solvents, and the duration of exposure to each substance was determined			
Bond et al 1992 [105] USA	Cohort Chemical industry Average follow-up of 30.9 years per Worker 1937–1977	Participants were male chemical workers who were potentially exposed to styrene and related materials for a year or more between 1937–1971 n=2 904 All participants were men	**Styrene** Manufacturing jobs held between 1937–1977 were grouped according to similar exposure profiles to form 57 categories of common exposure experiences. Each of the categories was individually evaluated by an industrial hygienist and was assigned an exposure intensity code with respect to the 5 chemical agent groupings (combinations of styrene and other chemicals)	**Diseases of the circulatory system and arterio-sclerotic heart disease** Cause-specific mortality among the cohort was compared against that of the white male population of the United States Code of the International Classification of Diseases, 8th revision: Diseases of the circulatory system (390–458)	Summary of mortality among the styrene-based products cohort relative to that among unexposed workers from the Michigan manufacturing location. RR (95% CI) Diseases of the circulatory system 0.90 (0.81; 1.01) Arteriosclerotic heart disease 0.86 (0.76; 0.98)	–

				Arteriosclerotic heart disease (410–413)		
Braeckman et al 2001 [14] Belgium	Prospective cohort Viscose rayon factory Year performed not stated	Participants were workers in a viscose rayon factory (viscose preparation, spinning, and bleaching departments) that were exposed to carbon disufide Control subjects were males working in processing factories without occupational exposure to chemicals known to be noxious for the cardiovascular system n=122 (85 exposed and 37 non-exposed) All participants were men	**Carbon disulfide (CS_2)** Exposure to CS_2 was assessed using personal monitoring pumps and charcoal tubes. These tubes were fixated on the collar of the worker in order to measure the CS_2 concentration outside the respirators or were fixated on the face of the worker in order to measure CS_2 inside the respirators	**Blood pressure** Systolic and diastolic blood pressure (Korotkoff phase I and V) readings were registered in supine position after 5 min rest with a mercury manometer. All readings were done by a physician	Difference between workers exposed to carbon disulfide and the reference group according to Mann-Whitney U test Systolic blood pressure: n.s Diastolic blood pressure: n.s	Difference between workers exposed to carbon disulfide and reference group according to Mann-Whitney U test, adjusted for age, pack-years, alcohol, and ethnic descent Systolic blood pressure: n.s Diastolic blood pressure: n.s
Brown et al 1987 [16] USA	Prospective cohort study	Participants were dry clean workers exposed to PCE	**Perchloro-ethylene (PCE)** Workers had been employed	**Mortality from circulatory**	Mortality of circulatory system diseases SMR (95% CI) PCE: 70 (60; 82)	–

	Average follow-up time not stated Dry cleaning 1959–1982	n=1 690 (exposed cohort) Participant included both males and females (numbers not stated)	at least a year prior to 1960* at a shop were PCE was the primary solvent. Solvent history was available for at least half of the shops *A gradual shift from petroleum to PCE which increased in the early 1960s	**system diseases** Deceased subjects were identified by death certificate and cause of death was coded by a trained nosologist according to ICD. Those lost to follow-up were considered alive Expected death rates for 1975–1982 was based on US deaths occurrence through 1978		
Brown et al 2015 [15] USA	Prospective cohort study Median follow-up was 8 years Aluminium smelter 1996–2012	Participants were hourly workers employed at 1 of 11 US aluminum smelters and fabrication facilities for more than 2 years between January 1, 1996, and December 31, 2012	**Total particulate matter (TPM)** Each job was associated with a time-invariant exposure level to TPM based upon 8 385 personal samples collected by the company at 11 facilities	**Ischemic heart disease** Incident ischemic heart disease was defined by any of the following events: (1) insurance billing claim for a indicative procedure,	Risk ratio of occupational exposure to PM2.5 by facility type and cut-off Level. RR (95% CI) **Ischemic Heart Disease** *Smelter* Median 1.77 mg/m^3: 1.39 (0.81; 2.39) 10th 0.16 mg/m^3: 1.77 (1.03; 3.06) *Fabricator* Median 0.20 mg/m^3: 1.14 (0.80; 1.63) 10th 0.06 mg/m^3: 1.45 (1.13; 1.86)	–

		Eligible workers, regardless of hire date, were followed for incidence of IHD after a 2-year washout period, implemented to remove prevalent cases of heart disease from the cohort. Follow-up for each worker began at the later of January 1, 1998, or 2 years after hire and ended at termination of employment n=12 547 (5 426 smelter workers and 7 121 fabrication workers	between 1980–2011. Additional modeling and expert judgment were used to generate estimates of TPM and % $PM_{2.5}$ from jobs without measured values. Each job was assigned a confidence level by industrial hygienists and researchers reflecting the method used to determine the exposure level. The analysis was restricted to subjects who ever held a job with a high confidence level	such as revascul-arization, angioplasty, or a bypass, (2) face-to-face visit with a provider with a relevant International Classification of Diseases (ICD) diagnosis code, (3) hospital-ization for more than 2 days with the relevant ICD admitting code, or (4) matching record of death from the National Death Index with a relevant cause of death		
Burstyn et al 2005 [17] Denmark, Finland, France, Germany, Israel, The	Prospective cohort Average follow-up 17 years Asphalt workers 1953–2000	Persons included in the historical cohort were first employed between 1913–1999 in companies	**Benzo(a)pyrene and coal tar** Exposures to benzo(a)pyrene were assessed quantitatively using measurement-driven exposure	**Mortality from cardiovascular r diseases and ischemic heart disease** A follow-up for mortality was	Exposure to polycyclic aromatic hydrocarbons and mortality from cardiovascular diseases using relative risks in Poisson regression models. RR (95% CI) adjusted for country, calendar period, age, and duration of employment **Diseases of the circulatory system** *Coal tar, cumulative exposure (unit-years*)	–

Netherlands and Norway		applying and mixing asphalt The duration of employment for inclusion in the cohort was 1 work season n=12 367 All participants were men Cohort members accumulated 193 889 person-years of observation	models. Exposure to coal tar was assessed in a semi quantitative manner on the basis of information supplied by company representatives Exposures were reconstructed by using information about changes in asphalt paving technology in each company over time, the relation between production characteristics and exposure levels, and job histories Occupational histories were coded on the basis of information from personnel records according to classifications of jobs	conducted, including data on primary causes of death Causes of death were coded according to the International Classification of Diseases, 9th Revision (ICD-9). Mortality from diseases of circulatory system was identified by ICD-9 codes 390–459, and mortality from ischemic heart disease was restricted to ICD-9 codes 410–414	0–0.29: 1.04 (0.75; 1.43) 0.30–0.73: 1.41 (0.99; 2.00) 0.74–1.41: 1.41 (0.96; 2.08) 1.42–2.09: 1.36 (0.89; 2.08) ≥2.10: 1.31 (0.86; 1.99) p for trend 0.11 *Coal tar, average exposure (dimensionless units)* 0–0.12: 1.02 (0.72; 1.44) 0.13–0.25: 1.11 (0.71; 1.72) 0.67–0.33: 2.01 (1.29; 3.14) 0.34–0.99: 1.35 (0.84; 2.18) ≥1:00: 1.85 (1.17; 2.91) p for trend<0.001 *Benzo(a)pyrene, cumulative exposure (ng/m^3 years)* 189–501: 1.08 (0.85; 1.38) 502–931: 1.06 (0.80; 1.42) 932–2012: 1.24 (0.89; 1.71) ≥2013: 1.42 (0.96; 2.09) p for trend 0.09 *Benzo(a)pyrene, average exposure (ng/m^3)* 68–105: 1.30 (1.01; 1.67) 106–146: 1.55 (l.18; 2.05) 147–272: 1.45 (1.09; 1.93) ≥273: 1.58 (1.16; 2.15) p for trend <0.001 **Ischemic heart disease** *Coal tar, cumulative exposure (unit-years)* 0–0.29: 1.00 (0.66; 1.52) 0.30–0.73: 1.29 (0.82; 2.01) 0.74–1.41: 1.45 (0.90; 2.32) 1.42–2.09: 1.41 (0.84; 2.36) ≥2.10: 1.48 (0.90; 2.44) p for trend 0.07	

			constructed for the study. In the current analysis, only men who appeared to have been exclusively employed in asphalt paving were included		*Coal tar, average exposure (dimensionless units)* 0–0.12: 1.01 (0.65; 1.57) 0.13–0.25: 1.09 (0.64; 1.85) 0.67–0.33: 1.80 (1.04; 3.10) 0.34–0.99: 1.47 (0.84; 2.58) ≥1:00: 1.64 (0.94; 2.84) p for trend 0.04 *Benzo(a)pyrene, cumulative exposure (ng/m^3 years)* 189–501: 0.99 (0.72; 1.36) 502–931: 1.22 (0.86; 1.74) 932–2012: 1.24 (0.82; 1.85) ≥2013: 1.58 (0.98; 2.55) p for trend 0.06 *Benzo(a)pyrene, average exposure (ng/m^3)* 68–105: 1.13 (0.82; 1.55) 106–146: 1.33 (0.94; 1.90) 147–272: 1.20 (0.84; 1.71) ≥273: 1.64 (1.13; 2.38) p for trend 0.02	
Calvert et al 2011 [18] USA *Note:* update of Brown and Kaplan 1987	Prospective cohort study Average follow-up time not stated Dry cleaning 1959–2004	Participants were dry clean workers exposed to PCE. These workers were not known to ever have been exposed to carbon tetrachloride or trichloroethylene and all had worked for at least 1 year prior to 1960 in a shop using PCE as the	**Perchloro-ethylene (PCE)** Workers had been employed at least a year prior to 1960* at a shop were PCE was the primary solvent. Solvent history was available for at least half of the shops *A gradual shift from petroleum to PCE which	**Hypertension and mortality from several conditions** Causes of death were coded according to ICD-9 codes Prior to 1979, vital status was determined using national records from the Social Security	Standardised mortality ratios for dry clean workers employed for minimum 1 year prior to 1960 and followed up to the end of 2004 with potential routine exposure to PCE. SMR (95% CI) adjusted for age, race, sex and calendar-time *Diseases of the heart* 1.01 (0.92; 1.11) *Ischaemic heart disease* 1.10 (0.99; 1.22) *Diseases of the circulatory system* 0.92 (0.77; 1.08) *Cerebrovascular disease* 0.91 (0.75; 1.11)	–

		primary cleaning solvent Participant were identified from dry cleaning union records in 4 US cities n=1 704 (exposed cohort) 1 112 women 592 men	increased in the early 1960s	Administration, etc. Status from 1979–2004 was determined from the National Death Index. To evaluate incidence of hypertension, the cohort was linked to the REMIS Expected death rates were calculated on the basis of age, race, sex and calendar-time specific national rates	*Diseases of arteries, veins and pulmonary circulation* 0.84 (0.56; 1.22) Standardised incidence ratios for dry clean workers employed for minimum 1 year prior to 1960 and followed up to the end of 2004 with potential routine exposure to PCE. SIR (95% CI) *Hypertension* 1.98 (1.11; 3.27)	
Calvert et al 1998 [19] USA Data on additional diagnoses are presented in the article	Case-control Chemical plants Participants worked at the plants between 1951–1972. Analysis was made in 1987	Participants were living individuals (workers) employed more than 15 years earlier in the production of TCP or 1 of its derivatives, which were contaminated with TCDD, and an unexposed comparison group	**Dioxin** 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) Blood was obtained from the participants after fasting and analysed for TCDD Lower TCDD was defined as serum TCDD <238 pg/g lipid	**Several diagnoses** Information on worker and referent health status was collected through a comprehensive set of standardized interviews and medical examinations A participant was defined	Distribution of the cardiovascular outcomes among workers and referents. OR (95% CI) **Workers with lower serum TCDD** Myocardial infarction: 1.28 (0.67; 2.45) Arrhythmia: 0.89 (0.51; 1.53) Hypertension: 1.14 (0.79; 1.66) **Workers with higher serum TCDD** Myocardial infarction: 1.91 (0.84; 4.38) Arrhythmia: 0.91 (0.40; 2.07) Hypertension: 1.46 (0.84; 2.52) **All workers** Myocardial infarction: 1.49 (0.83; 2.68) Arrhythmia: 0.89 (0.54; 1.48) Hypertension: 1.22 (0.86; 1.72)	Parameter estimates from adjusted logistic regression models. OR (95% CI) **Workers with lower serum TCDD** Myocardial infarction: 1.14 (0.29; 4.49) Arrhythmia: 0.98 (0.56; 1.70) Hypertension: 1.34 (0.89; 2.02) **Workers with higher serum TCDD** Myocardial infarction: 1.09 (0.23; 5.06) Arrhythmia: 0.77 (0.34; 1.78) Hypertension: 1.05 (0.58; 1.89)

		To constitute the referent (comparison) group, 1 individual with no self-reported occupational exposure to TCDD-contaminated substances was sought from within the residential neighbourhood of each worker; this individual matched the worker in age (within 5 years), race, and gender n=543, 208 workers with lower serum TCDD 66 workers with higher serum TCDD 260 referents 33 women 510 men	and higher TCDD was defined as serum TCDD <238 pg/g lipid	as having a history of myocardial infarction if the individual had either reported that a physician had diagnosed this condition or had ECG evidence of a previous myocardial infarction A participant was defined as having a history of cardiac arrhythmia if he/she reported that a physician had ever diagnosed this condition, or if he/she had ECG evidence of an arrhythmia A participant was defined as having hypertension if 1 of the following 3 criteria was	Myocardial infarction includes those with self-reported and/ar ECG diagnosis Arrhythmia included those with self-reported and/or ECG diagnose Hypertension included those with self-reported, current (systolic, and/or current diastolic hypertension **Subcohort of workers only exposed to PCE** Diseases of the heart: 1.08 (0.91; 1.27) Ischaemic heart disease: 1.24 (1.03; 1.48) Diseases of the circulatory system: 0.77 (0.54; 1.06) Cerebrovascular disease: 0.74 (0.48; 1.10) Diseases of arteries, veins and pulmonary circulation: 0.60 (0.22; 1.30)	

				satisified: a) a self reported history of physician-diagnosed hypertension; b) the lowest of 2 brachial artery systolic pressure readings taken while the participant was in sitting position >140 mm Hg or c) the lowest of 2 brachial artery diastolic pressure readings taken while the participant was in sitting position >90 mm Hg		
Carreon et al 2014 [20] USA Note: See also study by Prince et al (partly the same population, this study has	Cohort study Follow-up time not specified Chemical manufacturing plant 1946–2006	Participants were workers employed at a New York State chemical manufacturing plant between 1946–2006 58% of the participants were also	**Carbon disulphide** Any worker assigned to the Rubber Chemicals department from 1954–1994 were considered as exposed to carbon	**Coronary artery disease mortality** Vital status was ascertained through 2007 by linking with records of a national statistics' death index	Coronary artery disease mortality in groups defined by exposure to carbon disulfide and shift work. Cutpoint based on median exposure duration among long-term coronary artery disease decedents. SMR (95% CI). Selected results for coronary artery disease mortality were indirectly adjusted for cigarette smoking All workers SW<4y, CS2:<4y: 1 (reference) SW<4y, CS2≥4y: 0.78 (0.43; 1.40) SW≥4y, CS2<4y: 1.05 (0.61; 1.82)	–

longer follow-up time)		exposed to vinyle chloride in1974 or earlier 69% of the participants were also exposed to o-toluidine to at least low extent n=1 874 135 women 1 739 men	disulfide. Multiple jobs assigned to other departments and departments whose work was conducted throughout the plant were also considered exposed to carbon disulfide, as specified in the article The years, departments and jobs were selected based on reported use of carbon disulfide at the plant and partial industrial hygiene data	Causes of death were coded to the revision of the International Classification of Diseases in effect at the time of death	SW≥4y, CS2≥4y: 1.97 (0.84; 4.65) Workers employed≥90 days SW<4y, CS2:<4y: 1 (reference) SW<4y, CS2≥4y: 1.10 (0.57; 2.10) SW≥4y, CS2<4y:1.41 (0.77; 2.60) SW≥4y, CS2≥4y: 2.70 (1.05; 6.93)	
Charles et al 2010 [21] USA	Prospective cohort. Data from the Honolulu Heart Program Average follow-up time not specified 1965–1998	Participants were identified through elected services records from World War II 66% of the participants had jobs involving manual labor, 7.8% were in professional	**Pesticide, metal, and solvent** Information on occupational exposure was collected during baseline examination (1965–1968). Industrial hygienists assessed the	**Mortality due to circulatory diseases, coronary heart disease (CHD), and stroke** Mortality data were obtained through a comprehensive surveillance	Hazard ratio at each level of the 3 exposures relative to the zero exposure intensity level, 1965–1998. HR (95% CI) For all data below, none exposure was set to 1.00 (reference) **Pesticide exposure** *Circulatory diseases (0-year lag*)* Low: 1.01 (0.68; 1.53) Medium: 1.25 (0.95; 1.65) High: 1.48 (1.04; 2.12)	Hazard ratios of the 3 exposures HR (95% CI) adjusted for education, smoking status, triglycerides, physical activity, alcohol intake, and systolic blood pressure For all data below, none exposure was set to 1.00 (reference) **Pesticide exposure** *Circulatory diseases (0-year lag*)* Low: 0.89 (0.57; 1.32) Medium: 1.19 (0.90; 1.59)

		occupations, 9% were clerks, 7.6% were managers, 7.3% were salesmen, and 2% were technicans n=7 540 All participants were men	potential for pesticide, metal, and solvent exposure in each reported occupation. They created 4 levels of exposure to each agent: none (0), low (1–39), medium (40–79) or high (≥80)	system (1998). Underlying cause of death was determined by a panel of study physicians and classified according to ICD-8 (diseases of the circulatory system codes 390–459)	*(15-year lag*)* Low: 1.07 (0.81; 1.41) Medium: 1.34 (0.89; 2.01) High: 2.89 (0.93; 8.97) *CHD (0-year lag*)* Low: 1.27 (0.75; 2.16) Medium: 1.21 (0.80; 1.82) High: 1.11 (0.61; 2.02) *(15-year lag*)* Low: 1.11 (0.75; 1.65) Medium: 1.06 (0.55; 2.05) High: 2.02 (0.29; 14.37) *Stroke (0-year lag*)* Low: 0.79 (0.37; 1.66) Medium: 1.33 (0.68; 2.06) High: 1.93 (1.15; 3.23) *(15-year lag*)* Low: 1.08 (0.69; 1.69) Medium: 1.65 (0.91; 3.01) High: 5.26 (1.31; 21.10) **Metal exposure** *Circulatory diseases (0-year lag*)* Low: 0.81 (0.71; 0.92) Medium: 1.08 (0.90; 1.29) High: 1.13 (0.82; 1.56) *(15-year lag*)* Low: 0.86 (0.76; 0.98) Medium: 1.35 (1.02; 1.83) High: 1.29 (0.49; 3.46) *CHD (0-year lag*)* Low: 0.73 (0.60; 0.89) Medium: 0.99 (0.75; 1.30) High: 1.07 (0.67; 1.72)	High: 1.39 (0.95; 1.95) *(15-year lag*)* Low: 0.97 (0.73; 1.30) Medium: 1.23 (0.82; 1.89) High: 1.97 (0.63; 6.12) *CHD (0-year lag*)* Low: 1.23 (0.72; 2.09) Medium: 1.18 (0.78; 1.80) High: 1.06 (0.58; 1.93) *(15-year lag*)* Low: 1.06 (0.71; 1.58) Medium: 1.01 (0.52; 1.96) High: 1.38 (0.93; 9.83) *Stroke (0-year lag*)* Low: 0.61 (0.27; 1.37) Medium: 1.21 (0.76; 1.91) High: 1.73 (1.03; 2.91) *(15-year lag*)* Low: 0.92 (0.58; 1.49) Medium: 1.48 (0.81; 2.70) High: 3.70 (0.92; 14.96) **Metal exposure** *Circulatory diseases (0-year lag*)* Low: 0.83 (0.72; 0.95) Medium: 0.95 (0.78; 1.16) High: 1.08 (0.78; 1.51) *(15-year lag*)* Low: 0.86 (0.76; 0.99) Medium: 1.18 (0.88; 1.60) High: 1.03 (0.33; 3.20) *CHD (0-year lag*)* Low: 0.75 (0.61; 0.93) Medium: 0.89 (0.66; 1.19)

					(15-year lag)* Low: 0.74 (0.62; 0.90) Medium: 1.24 (0.80; 1.93) High: 1.95 (0.63; 6.08) *Stroke (0-year lag*)* Low: 0.92 (0.74; 1.13) Medium: 1.15 (0.85; 1.54) High: 1.12 (0.66; 1.92) *(15-year lag*)* Low: 1.00 (0.82; 1.21) Medium: 1.46 (0.91; 2.35) High: 0.92 (0.13; 6.57) **Solvent exposure** *Circulatory diseases (0-year lag*)* Low: 0.78 (0.69; 0.89) Medium: 0.98 (0.84; 1.16) High: 1.14 (0.92; 1.42) *(15-year lag*)* Low: 0.85 (0.75; 0.96) Medium: 1.22 (0.97; 1.54) High: 1.98 (1.14; 3.43) *CHD (0-year lag*)* Low: 0.78 (0.65; 0.94) Medium: 0.82 (0.63; 1.05) High: 1.11 (0.80; 1.52) *(15-year lag*)* Low: 0.76 (0.63; 0.90) Medium: 1.21 (0.87; 1.69) High: 2.46 (1.22; 4.97) *Stroke (0-year lag*)* Low: 0.86 (0.69; 1.06) Medium: 1.13 (0.87; 1.47) High: 1.29 (0.91; 1.83)	High: 1.03 (0.63; 1.69) *(15-year lag*)* Low: 0.74 (0.61; 0.91) Medium: 1.12 (0.72; 1.75) High: 1.45 (0.36; 5.83) *Stroke (0-year lag*)* Low: 0.92 (0.74; 1.15) Medium: 1.01 (0.73; 1.39) High: 1.08 (0.63; 1.86) *(15-year lag*)* Low: 0.98 (0.80; 1.21) Medium: 1.28 (0.79; 2.08) High: 0.95 (0.13; 6.75) **Solvent exposure** *Circulatory diseases (0-year lag*)* Low: 0.83 (0.72; 0.95) Medium: 0.97 (0.81; 1.15) High: 1.05 (0.83; 1.32) *(15-year lag*)* Low: 0.88 (0.77; 1.00) Medium: 1.09 (0.86; 1.39) High: 1.76 (0.99; 3.12) *CHD (0-year lag*)* Low: 0.83 (0.68; 1.02) Medium: 0.81 (0.61; 1.06) High: 1.06 (0.75; 1.48) *(15-year lag*)* Low: 0.79 (0.65; 0.96) Medium: 1.15 (0.81; 1.62) High: 2.27 (1.07; 4.84) *Stroke (0-year lag*)* Low: 0.89 (0.71; 1.13) Medium: 1.11 (0.84; 1.46)

					(15-year lag)* Low: 0.98 (0.81; 1.19) Medium: 1.31 (0.91; 1.91) High: 1.32 (0.42; 4.12) *Latency intervals were used in analysis. With 0-year lagging the entire period of exposure is investigated and with 15-year, the exposure period excluding the 15 years immediately prior to death is investigated	High: 1.15 (0.80; 1.67) *(15-year lag*)* Low: 1.01 (0.82; 1.23) Medium: 1.12 (0.76; 1.67) High: 1.18 (0.38; 3.71)
Chen et al 1988 [22] USA	Cohort study Average follow-up time not specified Chemical manufacturing plant 1950–1982	Participants were workers from a chemical manufacturing plant (E. I. Du Pont de Nemours & Co) Expected number of deaths was based on Du Pont company rates, adjusted for age and time period n=3 859 (DMF only+DMF/ACN) Gender not stated	**Dimethyl-formamide (DMF) and acrylonitrile (ACN)** The sample was divided into tree groups based on exposure: -*DMF only* (workers only exposed to DMF) - *DMF/ACN* (workers exposed to DMF and ACN), and - *DMF only+DMF/CAN* *Exposure information is presented in a previous paper	**Ischemic heart disaease mortality and cerebrovascular disease mortality** Terminated employees were identified by national records. Death certificates were obtained from state health departments Underlying cause of death was coded by trained nosologists according to ICD	Difference in mortality ratio between exposed workers observed from 1950 through 1982 and controls *Ischemic heart disease* DMF-only: p<0.05 (higher death rate for observed cases, 77 observed versus 57.3 expected) DMF/CAN: p<0.01 (higher death rate for observed cases, 72 observed versus 54.9 expected) *Cerebrovascular disease* DMF-only: n.s. (9 observed versus 7.7 expected) DMF/ACN: n.s. (4 observed versus 6.6 expected)	–
Cocco et al 1994 [23]	Cohort study 28 years	Participants were male workers in 2	**Dust** The ores extracted in	**Cardio-vascular diseases**	Standardized mortality ratios and 95% confidence intervals by cause of death: total cohort. SMR (95% CI)	–

Italy	Lead and zinc mines 1960–1988	metal mines located in Sardinia, Italy, with at least 1 year of employment between 1932–1971, and still employed on 1 January 1960, or who had worked a minimum of 12 months consecutively between 1 January 1960 and 19 September 1971 n=4 740 All participants were men	both mines are mainly blende and galena (lead and zinc sulphides), and the matrix consists of dolomite and limestone (mainly carbonates) in mine A, and metamorphic schist (mainly quartz and syderite) in mine B Mean respirable dust concentrations in underground workplaces were declining from 2.5–2.6 mg/m^3 in 1962–1970, to 1.6–1.8 mg/m^3 from 1971 onwards. Concentrations around 3.5 mg/m^3 were retrospectively estimated to have occurred in 1945–1960 Dust concentrations at surface workplaces	The underlying causes of death were coded by an expert nosologist according to ICD-9) Cardiovascular diseases were codes 390–459	Cardiovascular diseases Underground 0.66 (0.56; 0.77) Surface 0.57 (0.46; 0.71)	

			averaged <1 mg/m^3 in both mines from the 1970s No data were available on polycyclic aromatic hydrocarbons released by diesel engines in the mining environment. Diesel powered transport equipment came into use in the early 1970s, when 48% of cohort members were still employed. The main gases from diesel exhausts (carbon monoxide, nitrogen, and sulphur oxides) in the underground air environment were below the threshold limit value time weighted average limits in both mines			

Cooper at al 1985 [24] USA *Note:* SMR data on cumulative years of exposure for both cohorts is available (CI is not stated)	Prospective cohort study Follow-up time 34 years Lead production facilities 1946–1980	Participants derived from 6 lead production facilities and 10 battery plants Comparison with US white male rates n=6 819 (4 519 battery plant workers and 2 300 smelter workers) All participants were men	**Lead exposure** There was no way of estimating actual lead exposure except from some data on urinary lead concentrations (available for 2 275 men) and on blood lead concentration (available for 1 860) No separate analysis of death were made for those with monitoring information but the information served as evidence that much of the population had been exposed to lead amounts above current standards.	**Mortality from several diseases** Causes of death were coded by an experiences nosologist, in conformity with ICD-7 (with ICD-8 translated into ICD-7 codes) Vascular lesions, central nervous system (330–334) All diseases, circulatory system (400–468) Hypertensive heart disease (440–443) Other hypertensive disease (444–447)	Standardized mortality ratio for 2 cohorts of lead workers employed for at least 1 year between 1946–1970. SMR (95% CI) *Vascular lesions, central nervous system* Lead battery plants: 93 (77; 111) Lead production facilities: 132 (98; 175) *Circulatory system (all diseases)* Lead battery plants: 100 (93; 108) Lead production facilities: 91 (79; 103) *Arteriosclerotic heart disease* Lead battery plants: 94 (86; 102) Lead production facilities: 76 (64; 89) *Hypertensive heart disease* Lead battery plants: 128 (89; 178) Lead production facilities: 203 (113; 335) *Other hypertensive disease* Lead battery plants: 320 (197; 489) Lead production facilities: 475 (218; 902) ***Years of employment*** ***1–9, 10–19 and ≥20 years*** *Vascular lesions, central nervous system* Lead battery plants: 82 (100; 89) Lead production facilities: 119 (133; 146) *Circulatory system (all diseases)* Lead battery plants: 80 (109; 105) Lead production facilities: 87 (115; 102) *Arteriosclerotic heart disease* Lead battery plants: 62 (111; 99) Lead production facilities: 69 (115; 80)	–
Costello et al 2015 [27] USA	Prospective cohort	Participants were hourly workers employed at a	**Metal work fluids** Quantitative levels of	**Mortality from ischaemic heart disease**	Association of cumulative exposure to metalworking fluid (mg/m^3-years) with ischaemic heart disease mortality. HR (95% CI)	–

Note: same population as in article by Costello 2015	Average follow-up time not specified Automobile manufacturing plants 1941–1995	automobile manufacturing plant for at least 3 years and hired between 1938–1981. Follow-up began 3 years after hire and ended at the time of death or 1994 n=39 412 4 797 women (3 517 white women and 1 280 black women) 34 614 men (28 478 white men and 6 136 black men)	exposure to each metalworking fluid class for plant, department and job were estimated over time, based on 541 personal and area samples for PM3.5 (mg/m^3) collected by industrial hygienists. On the basis of plant records, a type of fluid (straight, soluble or synthetic) was assigned to each plant-year, department-year, job-year and calendar-year specific exposure category. For jobs with mixed exposures the percentage of time spent using each type of fluid was estimated Annual average exposure to each type of	Data on vital status and cause of death were obtained through the Social Security Administration, the National Death Index, plant records, death certificates and state mortality files. The outcome of interest for this analysis is mortality from ischemic heart disease (ICD-9 410–414)	adjusted for type of metalworking fluid, manufacturing plant, year and age *White women* Soluble (mg/m^3-years) 0: 1.00 0–0.76: 1.84 (0.76; 4.50) 0.77–1.80: 2.40 (0.97; 5.91) 1.81–3.44: 2.44 (0.96; 6.22) >3.44: 1.89 (0.74; 4.86) Synthetic (mg/m^3-years) 0: 1.00 0–0.65: 1.29 (0.80; 2.07) >0.65: 1.37 (0.82; 2.29) *Black women* Data not analysed due to small sample size *White men* Soluble (mg/m^3-years) 0: 1.00 0–0.76: 1.01 (0.85; 1.21) 0.77–1.80: 1.00 (0.84; 1.19) 1.81–3.44: 0.94 (0.78; 1.12) >3.44: 1.01 (0.87; 1.18) Synthetic (mg/m^3-years) 0: 1.00 0–0.65: 1.01 (0.90; 1.13) >0.65: 0.92 (0.79; 1.06) *Black men* Soluble (mg/m^3-years) 0: 1.00 0–0.76: 1.16 (0.67; 2.01) 0.77–1.80: 0.96 (0.54; 1.73) 1.81–3.44: 1.33 (0.79; 2.24) >3.44: 1.01 (0.64; 1.61) Synthetic (mg/m^3-years)	

			fluid and size fraction was calculated for each participant by combining work histories with the estimated exposure concentrations. Cumulative exposure is the sum of annual exposures		0: 1.00 0–0.65: 1.38 (0.64; 2.97) >0.65: 3.29 (1.49; 7.31)	
Costello et al 2013 [26] USA *Note: same population as in article by Costello 2013* *Note*: Data on exposure over time periods also available in the article	Prospective cohort study 54 years Automobile workers 1941–1995	Participants were employees at 1 of 3 Michigan automobile manufacturing plants for at least 3 years and hired between January 1, 1938 and January 1, 1985 n=39 412 4 808 women 34 604 men	**Particulate matter from air pollution** Quantitative levels of exposure to metalworking fluid class were estimated over time, based on personal and area samples for particulate matter (mg/m^3) collected by industrial hygienists. Exposures were characterized by particle-size fraction Among years with any active working time, current exposure	**Ischemic heart disease** Outcome was obtained through the Social Security Administration, the National Death Index, plant records, and state mortality files Cause of mortality was obtained from state vital records, death certificates, and the National Death Index The outcome of interest for	Hazard ratios for ischemic heart disease mortality and cumulative exposure to 3 types of metal working fluid particulate matter (<3.5 µm diameter) in the cohort. HR (95% CI) **Air pollution mg/m^3- years** ***Straight*** 0: 1.00 >0–0.065: 1.09 (0.95; 1.25) 0.066–0.20: 1.16 (1.01; 1.34) 0.21–0.48: 0.97 (0.84; 1.12) 0.49–1.64: 1.05 (0.92; 1.20) >1.64: 1.07 (0.93; 1.22) ***Soluble*** 0: 1.00 >0–0.96: 1.03 (0.87; 1.21) 0.97–2.24: 0.99 (0.84; 1.17) 2.25–4.42: 0.99 (0.84; 1.17) 4.43–7.98: 1.02 (0.86; 1.20) >7.98: 1.00 (0.85; 1.18) ***Synthetic*** 0: 1.00 >0–0.053: 1.03 (0.87; 1.23) 0.054–0.20: 1.10 (0.92; 1.30) 0.21–0.49: 0.95 (0.80; 1.13)	Hazard ratios for ischemic heart disease mortality and cumulative exposure to straight metal working fluid particulate matter (<3.5 µm diameter) in the cohort. The model was weighted by the stabilized inverse probability of staying at work. HR (95% CI) **Air pollution mg/m^3- years** ***Straight*** 0: 1.00 <0.06: 1.42 (1.04; 1.94) 0.07–0.22: 1.13 (0.83; 1.55) 0.23–0.68: 0.88 (0.64; 1.21) 0.68–2.77: 1.29 (0.97; 1.72) >2.77: 1.53 (1.15; 2.05)

			represents the exposure the subject would have had if he/she had worked the whole year whereas annual exposures (summed for cumulative exposure) were weighted by the percentage of the year the subject had actively worked	this analysis was mortality from ischemic heart disease (ICD-9 410–414)	0.50–0.95: 0.95 (0.80; 1.14) >0.95: 0.95 (0.80; 1.12)	
Costello et al 2014 [25] USA *Note*: data also available for categories by quartiles of exposure among cases above reference level 0.05 mg/m^3	Prospective cohort 10 years Aluminium industry 1998–2008	Participants were US aluminium workers in 8 study plants primarily engaged in smelting aluminium or fabricating processes involving aluminium-related products Hourly workers enrolled in a primary insurance plan and employed for at least 2 years during follow-up were	**Fine particulate matter** Fine particulate matter ($PM_{2.5}$) in air pollution, primarily from combustion sources, was assessed A job exposure matrix was constructed for arithmetic mean total particulate matter by distinct exposure group. The company had developed an industrial hygiene database of	**Ischemic heart disease** Incident ischemic heart disease was identified from medical claims data from 1998–2008 Actively employed workers were followed for incidence of ischemic heart disease identified from health insurance claims through 2009 or until they	Hazard ratios for incident ischemic heart disease. HR (95% CI) adjusted for age, race, gender, calendar year, smoking, facility type, body mass index, job grade and past exposure **All recent exposures** ***Recent $PM_{2.5}$ (mg/m^3), categories by quintiles of exposure among cases*** ≤0.11: 1.0 >0.11–0.22: 1.05 (0.81; 1.35) >0.22–0.45: 1.23 (0.92; 1.63) >0.45–1.47: 1.06 (0.78; 1.45) >1.47: 1.09 (0.73; 1.62) **Only recent exposures assessed with high confidence** ***Recent $PM_{2.5}$ (mg/m^3), categories by quintiles of exposure among cases*** ≤0.12: 1.0 >0.12–0.23: 1.05 (0.80; 1.39) >0.23–0.50: 1.04 (0.77; 1.41) >0.50–1.63: 1.29 (0.88; 1.88) >1.63: 1.21 (0.78; 1.88)	The paper presents figures with penalized spline of the adjusted hazard ratio for ischemic heart disease and recent $PM_{2.5}$ in a Cox model for males restricted to exposures measured with the highest confidence. The HR for $PM_{2.5}$ and incident IHD rose in fabrication to 1.5 at 1.25 mg/m^3 and was statisticalle significant throughout most of the exposure range. The exposure response in the smelters was approximately linear and rose to an HR of 1.5 at 9 mg/m^3, but was only statistically significant around the mean

		considered for the cohort Participants were at work for 2 disease-free years after 1st January 1996, before entering follow-up The median year of birth was 1955 n=11 966 2 194 smelters 8 290 fabrication workers 960 refinery workers 522 other workers 1 917 women 10 049 men	over 300 000 samples collected over the past 25 years To estimate $PM_{2.5}$, side-by-side personal size-selective sampling was conducted in 2010 and 2011 in 8 facilities	left work (whichever occurred first) Health insurance claims for a relevant procedure (revascular-ization, angioplasty, or bypass), hospitalizatio n for 2 or more days or a face-to-face visit with an ICD-9 code for ischemic heart disease (codes 410–414) comprised an ischemic heart disease diagnosis	Hazard ratios for ischemic heart disease according to cumulative $PM_{2.5}$ (mg/m^3-years). HR (95% CI) adjusted for age, race, gender, calendar year, smoking, body mass index, job grade and facility type **Cumulative exposure** ≤1.89: 1.0 >1.89–4.52: 0.89 (0.70; 1.12) >4.52–10.51: 0.81 (0.64; 1.03) >10.51–35.58: 0.82 (0.63; 1.07) >35.58: 0.80 (0.59; 1.07)	
Cragle et al 1984 [28] USA	Cohort study Lithium isotope separation plant using mercury 1953–1979	Participants were white male employees working at plant for at least 4 months between 01/01 1953, and 30/04 1958 n=5 663	**Mercury** *Mercury exposure group*: Workers who had been monitored with mercury urinalyses since 1953 (n=1 918) or since 1955 (n=215); total	Causes of mortality from death certificates and vital status were obtained from Social Security Administratio n records and were coded	Standardized mortality ratios computed for each group using U.S. white male mortality rates for comparisons. SMR **Vascular lesions of the central nervous system** Mercury exposure group: 1.00 Subgroup with high exposure: 0.87 Subgroup employed ≥1 year: 0.91 Potential risk group: 0.58 Never at risk group: 0.78	–

		All participants were men	numberwas 2 133 A subgroup had exposure above plant action value (0.3 mg HG/L), n=858 A subgroup was employed in mercury process for 1 year or more, n=1 741 *Potential risk group:* not-monitored workers who had had some period of work in potentially exposed positions, n=270 *Never at risk group*: Nonmonitored group, n=3 260 Average length of mercury exposure was 3.73 years, with a median of 2.03 years	following ICD-10 Expected deaths were computed for each group using U.S. white male mortality rates for comparisons	Exposure to mercury vapors at the plant was not related to any excess of deaths from diseases determined to be target organs for mercury (e.g. central nervous system). No excesses were found when level of exposure and length of exposure were considered	
Craig et al 1985 [29]	Cohort 16 years	Participants were men younger than	Nitroglycerine and ethylene glycole dinitrate	**Ischemic heart disease, acute**	Observed and expected numbers of deaths. Expected values, based on county population	–

Great Britain	Exposives factory 1965–1980	65 years at an exposives factory The population used for comparison was the mal population in the county were the factory was situated n=4 061 workers at the factory All participants were men	Exposure was assessed by job category. Job descriptions and locations were examined by the personel department at the factory 3 categories were applied: 1) exposed to both nitroglycerine and ethylene glycole dinitrate (EGDN) in a proportion 4:1, 2) exposed only to nitroglycerine and 3) not exposed the neither substance The management subdivide the emplyees in high or low exposure	**myocardial infarction and cerebro-vascular disease** Mortality data was based on ICD-8: ischemic heart disease 410–414, acute myocardial infarction 410 and cerebro-vascular disease 430–438	and internal controls respectively, are given in parentesis **Ischemic heart disease** *Men aged 50–54 years. Expected values based on internal controls. None of the excesses are statistically significant* Not exposed: 145 Both nitroglycerine and EGDN, low: 12 (8) Both nitroglycerine and EGDN, high: 22 (21) Only nitroglycerine, low: 1 (2) Only nitroglycerine, high: 9 (8) *Men aged 15–49 years. Expected values based on internal controls* Not exposed: 35 Both nitroglycerine and EGDN, low: 6 (5) Both nitroglycerine and EGDN, high: 12 (7) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 2 (3) *Men aged 15–49 years. Expected values based on population in the county of the factory* Not exposed: 35 Both nitroglycerine and EGDN, low: 6 (6) Both nitroglycerine and EGDN, high: 12 (8) Only nitroglycerine, low: 0 (1) Only nitroglycerine, high: 2 (3) **Acute myocardial infarction** *Men aged 50–54 years. Expected values based on internal controls. None of the excesses are statistically significant* Not exposed: 123 Both nitroglycerine and EGDN, low: 11 (7) Both nitroglycerine and EGDN, high: 19 (18) Only nitroglycerine, low: 1 (1) Only nitroglycerine, high: 6 (7) *Men aged 15–49 years. Expected values based on internal controls. The excess in mortality for*	

					younger workers exposed to both nitroglycerine and EGDN is statistically significant (p<0.05) Not exposed: 34 Both nitroglycerine and EGDN, low: 5 (5) Both nitroglycerine and EGDN, high: 12 (6) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 1 (2) *Men aged 15–49 years. Expected values based on population in the county of the factory. The excess in mortality for younger workers exposed to both nitroglycerine and EGDN is statistically significant (p<0.01)* Not exposed: 34 (49) Both nitroglycerine and EGDN, low: 5 (5) Both nitroglycerine and EGDN, high: 12 (7) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 1 (3) **Cerebrovascular disease** *Men aged 50–54 years. Expected values based on internal controls. None of the excesses are statistically significant* Not exposed: 44 Both nitroglycerine and EGDN, low: 3 (2) Both nitroglycerine and EGDN, high: 7 (7) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 6 (3) *Men aged 15–49 years. Expected values based on internal controls* Not exposed: 3 Both nitroglycerine and EGDN, low: 0 (0) Both nitroglycerine and EGDN, high: 0 (0) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 1 (0) *Men aged 15–49 years. Expected values based on population in the county of the factory* Not exposed: 3 (12)	

					Both nitroglycerine and EGDN, low: 0 (2) Both nitroglycerine and EGDN, high: 0 (2) Only nitroglycerine, low: 0 (0) Only nitroglycerine, high: 0 (1)	
Cypel et al 2016 [30] Vietnam	Cohort study Approximately 40 years Army veterans 1965 to 2013	Participants were Army Chemical Corps veterans who sprayed defoliant in Vietnam Veterans were identified based on a review of morning reports of units stationed in Vietnam and data on army personnel with military occupational specialty codes showing chemical operations involvement Eligible veterans was restricted to men who had a minimum of 18 months active US Army service between 1965 to 28, 1973 n= 3 086	**2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD)** Data were assessed by a survey (mail or computerized assisted telephone interview) Self-reported herbicide-spray-status was obtained from ‘‘yes/no’’ responses to the survey questions (described in the article) Spray-history was verified against serum 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)	**Blood pressure** Data were assessed by a survey (mail or computerized assisted telephone interview), medical records review/ abstraction, and in-home examination study Physician-diagnosed hypertension was obtained from the survey based on whether a veteran reported that a doctor, nurse, or healthcare professional ever told the veteran that he had high	Multiple Logistic Regression, OR (95% CI) Herbicide sprayer vs nonsprayer: 1.74 (1.44; 2.11) Vietnam service vs no Vietnam service: 1.26 (1.05; 1.53) Vietnam sprayers vs. Vietnam nonsprayers: 1.77 (1.35, 2.30) Non-Vietnam sprayers vs.non-Vietnam nonsprayers: 1.72 (1.31, 2.26) Vietnam sprayers vs. non-Vietnam sprayers: 1.29 (0.95, 1.74) Vietnam nonsprayers vs.non-Vietnam nonsprayers: 1.25 (0.99, 1.59) Vietnam nonsprayers vs.non-Vietnam sprayers: 0.73 (0.53, 0.99) Vietnam sprayers vs.non-Vietnam nonsprayers: 2.21 (1.76, 2.77)	-

		All participants were men		blood pressure or hypertension Self-reported physician-diagnosed-hypertension was confirmed by blood pressure measurement by trained medical technicians and medical record reviews		
Davies et al 1984 [31] Great Britain	Cohort study 15 years Lead chromate pigments factories 1930 and 1981	Participants were men employed at tree factories during the specified time span n=1 077 All participants were men	**Lead** Factories were making lead chromate pigments, which in recent decades were generally based on lead nitrate produced on site from metallic lead and nitric acid. Previously lead acetate had been more often used, and there had been some use of litharge. The most severe lead exposures	**Hypertensive disease and cerebrovascular disease, mortality** The 3 factories provided a total of 57 cases of non-fatal clinical lead poisoning. All 57 men were successfully traced as dead or as alive on 31 December 1981, and certified causes of	Observed and expected deaths from lead related causes. Ration between observed deaths and expected deaths Hypertensive disease: 0 Cerebrovascular disease: 4.10, p<0.001 Cerebrovascular disease - Early cohorts Men with lead intoxication; O/E 7/1.74**, other men O/E 23/24 79 Cerebrovascular disease - Late cohort (lower exposure): O/E 1/6.52 This is suggestive of dose-response	–

			usually occurred during the grinding or handling of the dried colours No data are available on lead in air levels during the period when lead poisoning cases were occurring. 2 of the factories also made zinc chromate pigments	death were ascertained Hypertensive disease were ICD-9 codes 401–405 and cerebrovascular disease were 430–438		
Delzell et al, 2005 [32] USA and Canada	Prospective cohort Follow-up time varied between subjects Synthetic rubber industry 1944–1998	Participants were men employed for at least 1 year at any of 8 North American synthetic rubber industry plants in the United States and in Canada n=16 579 All participants were men	**Styrene** Work histories covered the time period 1943–1991 and consisted of information on each job Quantitative estimates of exposure to styrene and 1.3-butadiene was developed by identifying for each plant-specific job its component tasks that entailed exposure and documenting	**Ischemic heart disease mortality** Information on subjects' vital status came mainly from employment and pension records and from record linkages with national registers The codes from NDI-Plus was used to determine the International Classification of Diseases	Ischemic heart disease by cumulative exposure (ppm-year) and lifetime average intensity of exposure (ppm) to styrene. Active group; includes person-year and deaths occurring up to 31 d after subjects' employment separation dates. RR (95% CI) *Styrene ppm-year* >0–<3.78: 0.95 (0.81; 1.13) 3.78–<10.23: 1.03 (0.88; 1.22) 10.23–<25.95: 0.99 (0.84; 1.17) 25.95–<60.67: 0.95 (0.80; 1.12) >60.67: 1.07 (0.90; 1.27) *Styrene ppm (lifetime)* >0–<0.86: 1.06 (0.89; 1.25) 0.86–<1.47: 0.93 (0.79; 1.10) 1.47–<2.53: 0.91 (0.77; 1.07) 2.53–<5.50: 0.99 (0.83; 1.17) >5.50: 1.14 (0.96; 1.35) Data is listed for all ischemic heart disease. In the article, data is also presented for acute and	–

			historic changes in those tasks; calculating 8-hour time weighted average exposure in parts per million (ppm) for each combination of job and calendar year at each plant and compiling these into job-exposure matrices (JEMs); and linking the year- and job-specific agent exposure estimates in the JEMs with each subject's work history and follow-up experience to obtain time-dependent cumulative exposure and average intensity of exposure estimates	(ICD) code for each decedent's underlying cause of death. For U.S. deaths before 1979, a nosologist assigned an ICD code to the underlying cause of death. Cause of death codes for Canadian decedents came from a national register. All codes for U.S. and Canadian decedents were based the revision of the ICD in effect at the time of death	chronic ischemic heart disease. A relationship was found between lifetime average intensity of styrene (ppm)and chronic ischemic heart disease among men younger than 55 years (p=0.003)	
Dixit et al 2016 [33] Several countries	Cohort 2013-2014	Participants were enrolled in the Health eHeart Study, an internet-based,	Second hand smoke Second hand smoke was assessed	Atrial fibrillation Prevalent atrial fibrillation	Odds ratio described graphically People smoked at work, closed enough to see/smell the smoke: p= 0.069	-

		longitudinal cardiovascular cohort study Participants were recruited from cardiology and general medicine clinics, academic institutions, lay press, and social media, and through partnerships with advocacy groups and medical organizations n= 4 976 2 748 women and 2 228 men	through a validated 22-question survey	was assessed by self-report, with validation of a subset by review of electronic medical records	People smoke in the same room at work: p= 0.148	
Du et al 2012 [34] China	Prospective cohort study Average follow-up time was 26 years Asbestos miners 1981–2010	Participants were asbestos workers in a chrysotile asbestos mine in China who had been working in the mine for at least a year between 1981–1988 The workers were divided into groups based on exposure status	**Chrysotile asbestos** The dust concentrations in the different work areas measured in 2009 ranged between 4.33–196.67 mg/m^3, while the fiber data was not available, which were much higher than the National Standard	**Mortality from cardio-vascular disease or cerebro-vascular disease** Causes of death were obtained fromdeath registration and municipal hospitals. The response rate was 82.8%. The diagnosis of asbestos	Standardized mortality ratios of major causes by exposure to asbestos. Age, gender, and death-period were standardized. Exposed group vs National standard. SMR (95% CI) *Cardiovascular disease* 1.27 (0.96; 1.63) *Pulmonary heart disease* 2.70 Described as statistically significant excess mortality but no further information was presented *Cerebrovascular disease* 1.38 (1.03; 1.79) Relative risk for cause-specific mortality in relation to direct exposure to asbestos,	–

		Exposed group were frontline workers who worked directly on mining or processing asbestos products *Control group* were workers in management or service departments not directly exposed to asbestos n=1 257 194 women 1 063 men		for the miners was made by the Pneumoconiosis Diagnosis Panel according to the Chinese Diagnosis Criteria of Pneumoconiosis by Radiograph (GB5908-86) Expected number of deaths were was calculated by person-years at risk stratified into 5-year intervals by age and calender time and the multiplied by age-, gender-, and cause - specific national death rates of 1981–2010	adjusted for gender, smoking status and employment years. Exposed group vs Control group. RR (95% CI) *Cardiovascular disease* 1.30 (0.79; 2.14) *Cerebrovascular disease* 1.75 (1.00; 3.08)	
Ellingsen et al 1993 [36] Norway	Cohort study Chloralkali plant using mercury cell process	Participants were male workers at 2 Norwegian chloralkali plants first	**Chloralkali and mercury** A cumulative urinary mercury dose was calculated for	**Several conditions** Causes of mortality were recorded via	Standardized mortality ratios (SMR) and incidence rates (SIR) by death cause for mercury exposed males, working for more than 1 year at 2 Norwegian chloralkali plants between 1953–1988. SMR (95% CI)	–

	1953–1988	employed before 1980 and exposed to mercury vapour for more than 1 year The mean time of employment among the 674 workers employed for the first time before 1980 was 9.6 (range 1.0–38.9) years n=674 All participants were men	each subject based on the quarterly mean individual urinary mercury concentration (~20 000 measurements) Individual missing data was replaced by individual average calculated from the mean values from 2 quarters before and after the missing period The mean cumulative urinary mercury dose was 3 700 nmol/l. The individual mean urinary mercury concentration each year was 465 nmol/l Nearly 30% of the subjects in the restricted cohort have at least once exceeded the urinary mercury	death certificates coded by the Central Bureau of Statistics of Norway according to current ICD-7 system Mortality rate was then compared with expected rates in Norwegian male population, based on age specific national rates for 5 year age groups for each calendar year from 1953 Diseases of the circulatory system (codes 330–334, 400–468) Arterio-sclerotic and degenerative heart	**Diseases of the circulatory system** All: 0.87 (0.70; 1.06) *by years of employment* *<5 years, SMR:*0.94 (0.65; 1.31) *5–14 years, SMR:* 0.99 (0.67; 1.39) *≥15 years SIR:* 0.69 (0.44; 1.01) *by time since first employment at the plant* *<10 years, SMR:* 0.68 (0.30; 1.35) 10–14 years, SMR: 0.84 (0.53; 1.26) *≥20 years, SMR:* 0.91 (0.69; 1.17) **Arteriosclerotic and degenerative heart disease** All: 0.94 (0.73; 1.19) *by years of employment* *<5 years, SMR:* 0.87 (0.54; 1.31) *5–14 years, SMR:* 1.05 (0.66; 1.57) *≥15 years SIR:* 0.91 (0.57; 1.38) There was a highly significant association between the number of years employed and cumulative urinary mercury dose (Pearson's r=0.69, p<0.01, n=657)	

			concentration of 1 500 nmol/l during their biological surveillance	disease (codes 420–422)		
Enterline et al 1990 [38] USA *Note:* same sample as in the article by Tsai et al. 1996, but with shorter time to follow-up	Prospective cohort study Follow-up time varied between subjects Oil company plants 1948–1983	Participants were workers from 2 different Shell chemical plants, who had at least 3 months of employment where exposure to epichlorohydrin could have occured n=863 (exposed group) All participants were men	**Epichlorohydrin** Potential exposure to epichlorohydrin (EHC) Based on each worker's job with the highest potential level of exposure, a panel of industrial hygiene personnel and current and former employees assigned every employee into 1 of the 5 potential exposure categories: heavy, moderate, light, none, or unknown During early production periods exposures were sufficiently high to be a source	**Mortality to different causes of death** Cause of death was coded according to ICD-8: cerebro-vascular disease (430–438) heart disease (390–398, 400.1, 400.9, 402, 404, 410–414, 420–429) rheumatic heart disease (390–398), ischaemic heart disease (410–414), and hypertension w/o heart disease (400.0, 400.2, 400.3, 401, 403) Expected deaths were calculated from the	Mortality in workers with probable exposure to epichlorohydrin. SMR (CI not stated), p **SMRs for 4 follow-up periods:** *Stroke* 1948–*19*75: 63.7, n.s 1948–*19*77: 50.5, n.s 1948–*19*79: 60.6, n.s 1948–*19*83: 130.4, n.s *All heart disease* 1948–*19*75: 58.0, p<0.05 1948–*19*77: 69.2, n.s 1948–*19*79: 68.7, p<0.05 1948–*19*83: 72.2, p<0.05 *Coronary heart disease* 1948–*19*75: 70.0, n.s 1948–*19*77: 83.9, n.s 1948–*19*79: 84.3, n.s 1948–*19*83: 75.7, n.s *Other heart disease* 1948–*19*75: – 1948–*19*77: 14.3, p<0.05 1948–*19*79: 11.4, p<0.01 1948–*19*83: 52.4, n.s **SMRs for time since first exposure to ECH** *Cerebrovascular disease* Total: 128.9, n.s <20 years: 168.8, n.s ≥20 years: 98.4, n.s *Heart disease* Total: 67.9, p<0.05	–

			of irritation (10–20 ppm)	mortality and population data system maintained by the department of biostatistics, Graduate School of Public Health, University of Pittsburgh. Expected deaths are based on white male death rates	<20 years: 63.6, n.s ≥20 years: 70.9, n.s *Ischaemic heart disease* Total: 72.3, n.s <20 years: 71.1, n.s ≥20 years: 73.3, n.s *All other heart disease* Total: 50.6, n.s <20 years: – ≥20 years: 64.6, n.s **SMRs for 20 years or more since first exposure:** *Cerebrovascular disease* None to light: 82.0, n.s Moderate to heavy: 121.9, n.s *Heart disease* None to light: 39.2, p<0.05 Moderate to heavy: 105.4, n.s	
Ehrlich et al 1998 [35] South Africa	Prospective cohort study Follow-up time varied between subjects Acid battery production plant 1974–1994	Participants were employees at a lead acid battery plant. All permanent production workers were invited to participate, as well as temporary workers (mainly on short contracts) over 37 years of age n=382 (359 were	**Lead (Pb)** Exposures include inorganic Pb fume and Pb oxide, and 25–35% sulphuric acid by weight in water Lead absorption measures included current blood Pb and zinc protoporphyrin concentrations. The variables were: (a)	**Blood pressure** A calibrated mercury sphygmo-manometer was used to measure blood pressure. A trained nurse, with the aid of a double headed stethoscope, recorded systolic pressure at the beginning	There was no significant association between any of the Pb exposure measures and either systolic or diastolic blood pressure, or hypertension defined categorically	–

		permanent and 23 temporary employees) All participants were men	cumulative blood Pb (µg.y/dl), the sum of average blood Pb in each year over all such years of employment, and, (b) historical blood Pb, calculated by dividing the cumulative blood Pb by duration of exposure	of the consecutive run of Korotkow sounds (phase I) and diastolic pressure as the dis-appearance of the sounds (phase V). The average of 3 readings was used for systolic and diastolic blood pressure in the analysis		
Englander et al 1988 [37] Sweden	Prospective cohort study Follow-up time varied between subjects Sulphuric acid factory 1960–1985	Participants were male workers, employed for at least 6 months during the period 1961–1981, in a sulphuric acid factory n=400 All participants were men	**Sulphur dioxide (dust)** Since 1969, fairly extensive measurements have been performed in the respiratory zone of the workers The median level of *total dust*, over the years, was 2.2 mg/m^3 (time-weighted average), of *respirable dust* 0.6 mg/m^3, of *sulphur dioxide*	**Cardio-vascular diseases mortality** Death certificates was coded according to ICD-8, cardio-vascular diseases (390–458) Expected mortality for the period 1961–1985 was calculated using calendar-year,	Standardized mortality ratio in a cohort of workers in a sulphuric acid plant. SMR, p *Cardiovascular diseases* All: 1.33, p=0.17 ≥5 years latency period: 1.51, p=0.05 Relationship between time of employment and risk of death. A latency time of ≥5 years have been applied. SSMR (=SMR standardised to the age distribution in the <2 years group). SMR (SSMR); p for trend in SSMR *Cardiovascular diseases (years of employment)* *<2 years: 1.29 (1.29)* 2–5 *years* 1.50 (1.72) >5 *years*: 1.57 (1.82) p for trend >0.05	–

			3.6 mg/m^3, and of *arsenic* 11 mg/m^3	cause and 5 year age-group specific mortaliry rates for males in the country		
Eskenazi et al 1988 [39] USA	Case control Follow-up time not stated General population, pregnant women 1980–1984	Participants were women with a first prenatal visit to private or midwifery practices, or health organisation between the years 1980–1982 Participants were drawn from a prospective cohort study of pregnant women that was conducted at an American university Each of the solvent-exposed women were matched to 2 unexposed women with respect to race, gravidity, marital status, and being	**Solvents** Exposure to solvents was based on the review of job descriptions by 2 industrial hygenists. Based on the women's job description, they judged if the participants had been exposed to substational levels of solvents (defined as apprioximately 1/3 of the respective threshold limit value)	**Cardio-vascular pregancy complications** Information was obtained from daily deliveries at the university hospital were the study was conducted Data regarding pregnancy outcomes were abstracted from the mothers' medical charts and coded according to ICD-9	Maternal complications during pregnancy. Exposed group vs. unexposed group. p *Cardiovascular pregnancy complications* p=0.04 *Hypertension* n.s *Preeclampsia* p=0.03 Association of solvent exposure and maternal complications. RR (95% CI) *Hypertension:* 2.3 (0.8; 6.9) *Preeclampsia:* 5.3 (1.1; 26.7)	Association of solvent exposure, and maternal complications. Adjusted for induced abortion, alcohol use, weeks gestation at interview, employed at interview, pill used in previous years, smoked, and parity. RR (95% CI) *Hypertension:* 3.0 (0.9; 9.9) *Preeclampsia:* 3.9 (2.4; 5.4)

		within 2 years of each maternal stage at the estimated date of conception n=270 (90 solvent-exposed and 180 unexposed) All participants were women				
Fanning 1988 [40] United Kingdom	Cohort study Follow-up time not stated Battery factories 1926–1985	Participants derived from battery factories that were part of an international group of companies in with a number manufacturing facilities. Employees from all the UK companies were eligible to join a common pension scheme. This scheme was the main source of data on deaths occuring between 1926–1985 Analysis was restricted to	**Lead exposure** Job titles in the battery factories and advice from experienced physicans and managers enabled a division to be made between those employees considered to have had a high or moderate level of lead exposure and those with no exposure. As a general indication of the level of lead exposure of the 2 groups, the range of blood levels over the	**Mortality to selected diseases** The underlying cause of death was coded in accordance with ICD-9 Diseases of the circulatory system: codes 390–459 Hypertensive diease: codes 401–405 Ischaemic heart disease: codes 410–414	Association with cause of death between 1926–1985 and lead exposure in male lead workers. OR (CI not stated) *Diseases of the circulatory system* 1.07, n.s *Hypertensive disease* 1.26, n.s *Ischaemic heart disease* 0.96, n.s *Cerebrovascular disease, depending on year of death* 1926–1945: 4.57, n.s. 1946–1965: 1.94 p<0.05 1966–1985: 0.96, n.s. Total period: 1.24, n.s.	–

		deaths that occured among men n=2 073 (867 exposed to lead and 1 206 unexposed) All participants were men	past 20 years (when such routine monitoring was made) would have been 40–80 µg/100 ml in exposed workers and less than 40 µg/100 ml in unexposed	Cerebrovascular disease: codes 430–438		
Finkelstein et al 2004 [41] Canada	Retrospective cohort study 25 years Construction work 1975–2000	Participants were members of construction trade unions in Ontario The ‘‘exposure’’ variable was membership in the Union of Operating Engineers Analysis of the occupational distribution of deaths attributed to ischemic heart disease (IHD) was undertaken using MOR methodology, a form of case-control analysis [Walter, 1986]. All subjects were from the cohort, and no	**Diesel exhaust** Operating engineers are workers who operate and maintain heavy earthmoving equipment such as cranes, bulldozers, graders, and backhoes. Most of this equipment is powered by diesel engines and workers may thus be exposed to diesel exhaust The potential for exposure depends upon the nature of the job and the equipment	**Ischemic heart disease** The deaths included in this analysis are those identified at the Ontario Mortality Registry using probabilistic matching methodology The underlying cause of death, as coded by the Provincial nosologists, was utilized in the analyzes For ischemic heart disease ICD-9 codes 410–414 was applied	Mortality odds ratios for ischemic heart disease and myocardial infarction mortality, comparing the heavy equipment operators to other workers. MOR (95% CI) **Ischemic heart disease** Ages 25–64: 1.47 (1.17; 1.84), Ages 65 or more: 1.20 (0.96; 1.50), All ages combined: 1.32 (1.13; 1.55) **Myocardial infarction** Ages 25–64: 1.43 (1.07; 1.90), Ages 65 or more: 1.06 (0.79; 1.43), All ages combined: 1.23 (1.00; 1.51)	–

		external reference population was utilized. Cases were defined to be those subjects in the cohort who died from ischemic heart disease Controls were subjects in the cohort who died from any cause other than heart disease, with a few notable exceptions n=1 009 deaths Mortality among heavy equipment operators (n=1 009 deaths) was compared with mortality among other construction union members (n=6 291)				
Flesch-Janys et al 1995 [42] Germany	Prospective cohort study Chemical plant	Participants were male workers exposed for at least 3 months	**Dioxins (PCDD/F, TCDD)** First, definitions of 14 production	**Cardio-vascular diseases, ischemic**	Relative risk of cardiovascular diseases, ischemic heart diseases and other cardiovascular diseases mortality in relation to quintiles of extimated exposure levels (ng/kg of blood fat) at the end of exposure above	–

	1952–1992	between 1952–1984 in a chemical plant An unexposed cohort of gas workers with similar socioeconomic background served as an external reference group n=1 189 (exposed cohort) All participants were men	departments of the plant were developed from an analysis of the production process by an industrial hygienist. These definitions included measurements of PCDD/F in the various products Second, each worker was assigned the time he had spent in each departement. These duration estimates were derived from personnel records supplied by the company and in worker's interviews Third, concentrations of PCDD/F in adipose tissue or whole blood were determined for 190 workers	**heart diseases** Vital status was assessed by direct contact or through community registries. Causes of death were derived from records obtained from a hospital or family doctor by a pathologist. Causes of death was coded according to ICD-9 (all cardiovascular diseases: 390–459, ischemic heart diseases: 410–414) Available medical records or death certicifcates were reviewed for 138 of 162 members of the chemical	german median background levels using the cohort of gas workers as a reference, 1952–1992. RR (95% CI) ***TCDD*** *Cardiovascular diseases* 1st: 1.22 (0.81; 1.83) 2nd: 0.88 (0.54; 1.44) 3rd: 1.35 (0.91; 2.01) 4th: 1.64 (1.12; 2.39) 5th: 1.53 (0.95; 2.44) 6th: 1.96 (1.15; 3.34) p for trend=0.01 *Ischemic heart diseases* 1st: 1.43 (0.83; 2.44) 2nd: 0.81 (0.41; 1.61) 3rd: 1.18 (0.65; 2.16) 4th: 0.90 (0.47; 1.75) 5th: 1.61 (0.85; 3.04) 6th: 2.48 (1.32; 4.66) p for trend <0.01 *Other cardiovascular diseases* 1st: 1.02 (0.54; 1.92) 2nd: 0.98 (0.49; 1.97) 3rd: 1.54 (0.90; 2.64) 4th: 2.52 (1.57; 4.06) 5th:1.46 (0.72; 2.94) 6th: 1.24 (0.45; 3.40) p for trend=0.27 **All PCDD/F combined (total toxic eduivalencies)** *Cardiovascular diseases* 1st: 0.93 (0.57; 1.50) 2nd: 0.92 (0.59; 1.46) 3rd: 1.48 (1.01; 2.17) 4th: 1.55 (1.07; 2.24) 5th: 1.63 (1.01; 2.64) 6th: 2.06 (1.23; 3.45)	

			Forth, PCDD/F levels for these workers were estimated at the end of exposure based on an algoritm Finally, estimated levels for all members of the cohort were obtained ***Exposure quintiles*** *TCDD* 1st: 0–2.8 2nd: 2.81–14.4 3rd: 14.5–49.2 4th: 49.3–156.7 5th: 156.8–334.6 6th: 344.7–3890.2 *PCDD/F combined (total toxic eduivalencies)* 1st: 1.0–12.2 2nd: 12.3–39.5 3rd: 39.6–98.9 4th: 99.0–278.5 5th: 278.6–545.0 6th: 545.1–4361.9	cohort and a random sample (n=32) of the control cohort. The review was conducted by an independent pathologist blinded to cohort membership	p for trend <0.01 *Ischemic heart diseases* 1st: 1.02 (0.54; 1.95) 2nd: 0.96 (0.51; 1.82) 3rd: 0.97 (0.52; 1.81) 4th: 1.13 (0.64; 2.00) 5th: 1.73 (0.92; 3.27) 6th: 2.72 (1.49; 4.98) p for trend <0.01 *Other cardiovascular diseases* 1st: 0.84 (0.40; 1.74) 2nd: 0.91 (0.48; 1.75) 3rd: 2.05 (1.26; 3.36) 4th: 2.07 (1.27; 3.38) 5th: 1.53 (0.73; 3.20) 6th: 1.19 (0.44; 3.26) p for trend=0.30	
Franco et al 1982 [43] Italy	Case control Follow-up time not stated	Participants were male workers in a viscose plant. Workers were	**Carbon disulphide (CS_s)** From 1963–1979 environmental	**Blood pressure** Diastolic and systolic blood pressure were	Group difference between CS_s – exposed workers and referents *Systolic blood pressure:* n.s	–

	Viscose factory 1963–1979	matched for age, height, and weight to referents working in the textile section of the same plant and not exposed to CS_s Mean age of workers was 40.2 years n=140 (70 CS_s – exposed workers and 70 referents) All participants were men	measures of CS_s were made occasionally Mean CS_s measures at the worksites were: Year: mg/m^3 1970–*19*71: 83 1972: 24.9 1979: 23.6 Mean CS_s measures for the center of the aisle was: 1972: 11.1 1979: 2.8 Since the plant had not been changed substantially during 1972–*19*79, 1 may assume that the CS_2 concentrations have been similar through out that period	measured by the same physican according to the American Heart Association	Diastolic blood preassure: n.s	
Friesen et al 2009 [46] Australia	Prospective cohort study Mean follow-up time ws 16.2 years. Bauxite mining and aluminium factory	Participants were male workers from 4 bauxite mines and 3 alumina refineries employed on or after 1983. Work history and smoking	**Alumina and bauxite dust** Before 1998 the measurements were primarily collected using a close-faced 37 mm cassette for total dust, which have	**Cerebro-vascular, circulatory, and cardio-vascular diseases mortality** The cohort was linked to national	Relative risk for selected mortality sites by cumulative alumina and bauxite exposure (mg/m^3-years). RR (95% CI) adjusted for age and calendar year **Cerebrovascular diseases** *Alumina* Note exposed: 1 Low: 2.2 (0.4; 12) Medium: 10.8 (1.9; 60)	Relative risk for selected mortality sites by cumulative alumina and bauxite exposure (mg/m^3-years) and adjusted for age, calendar year, and smoking status. RR (95% CI) **Cerebrovascular diseases** *Alumina* Note exposed: 1 Low: 2.7 (0.5; 15)

	1983–2002	status was self-reported in interviews Mean age at study entry was 32 years n=5 770 All participants were men	been found to underestimate the inhalable fraction. After 1998 dust was collected using an inhalable sampling head The annual mean was calculated for each site/job task/year. For tasks with measurements, the first 3 years of measurement were averaged and assigned to to all prior years. Tasks with no monitoring data were determined by a hygienist A job exposure matrix was created and the cumulative exposure to bauxite and alumina was calculated for each individual	mortality registries from 1983–2002	High: 4.2 (0.8; 24) Trend, p-value: 0.02 *Bauxite* Note exposed: 1 Low: 2.3 (0.4; 14) Medium: 2.1 (0.4; 11) High: 1.9 (0.3; 11) Trend, p-value: 0.40 **All circulatory diseases** *Alumina* Note exposed: 1 Low: 0.8 (0.4; 1.6) Medium: 1.8 (0.9; 3.6) High: 1.7 (0.8; 3.6) Trend, p-value: 0.07 **All cardiovascular diseases** *Alumina* Note exposed: 1 Low: 0.7 (0.3; 1.5) Medium: 1.5 (0.7; 3.2) High: 1.5 (0.7; 3.4) Trend, p-value: 0.26	Medium: 8.7 (1.5; 49) High: 4.2 (0.7; 21) Trend, p-value: 0.04 Bauxite Note exposed: 1 Low: 2.5 (0.4; 15) Medium: 2.2 (0.4; 11) High: 2.4 (0.4; 15) Trend, p-value: 0.27 **All circulatory diseases** *Alumina* Note exposed: 1 Low: 0.8 (0.4; 1.6) Medium: 1.7 (0.9; 3.4) High: 1.6 (0.8; 3.3) Trend, p-value: 0.11 **All cardiovascular diseases** *Alumina* Note exposed: 1 Low: 0.7 (0.3; 1.5) Medium: 1.4 (0.7; 3.0) High: 1.4 (0.7; 3.3) Trend, p-value: 0.33
Friesen et al 2010 [44]	Prospective cohort study	The participates had worked for 3 or more years	**Benzo[a]pyrene exposure**	**Ischemic heart disease, acute**	Mortality according to past cumulative (5-year lag) and recent cumulative benzo[a]pyrene exposure, µg/m^3-year, in male aluminium	–

Canada	The mean age to follow-up was 23.5 years (maximum 47 years) Aluminium industry 1954–1999	at the aluminium smelter or its power-generating station between 1954–1997 Work histories through 1999 were abstracted from company records. Using probabilistic linkage, the cohort was linked to the Canadian national mortality database (1957–1999) Mean age 32.4 years n=6 423 All participants were men	A benzo [a] pyrene job exposure matrix was developed using approaches to maximize the personal exposure measurements that had been collected by the company and Work Safe British Columbia from the mid-1970s onwards. Pre-1977 exposure levels were backwards-extrapolated from 1977 exposure levels. The job- and time-period-specific exposure levels were linked to each worker's work history for calculation of cumulative and current exposure levels	**myocardial infarction and cerebro-vascular disease** All causes of death were recoded to ICD-9. Follow-up began from the point at which the worker attained 3 years of employment and extended to the earliest of the following dates: the date of death, the date last known to be alive, or December 31, 1999 For ischemic heart disease mortality, internal comparisons were conducted: 1) all ischemic heart disease occurring from 1957 onwards; 2) acute	smelter workers. HR (95% CI) adjusted for smoking **All Ischemic heart disease (1957 onward)** 0: 1.00 >0–<7.79: 1.11 (0.76; 1.62) 7.79–<24.3: 1.48 (1.01; 2.17) 24.3–<66.7: 1.28 (0.86; 1.91) ≥66.7: 1.62 (1.06; 2.46) Continuous: 1.002 (1.000; 1.005) **Acute myocardial infarction (1969 onward)** 0: 1.00 >0–<7.51: 1.14 (0.71; 1.82) 7.51–<27.7: 1.21 (0.75; 1.96) 27.7–<67.4: 1.36 (0.84; 2.22) ≥67.4: 1.46 (0.87; 2.45) Continuous: 1.001 (0.997; 1.005) **Cerebrovascular disease** 0: 1.00 >0–<11.4: 0.88 (0.42; 1.83) 11.4–<76.8: 0.65 (0.31; 1.34) ≥76.8: 1.42 (0.67; 2.99) Trend: 0.20	

				myocardial infarction occurring from 1969 onwards		
Friesen et al 2007 [45] Canada *Note:* Same population as Friesen et al. 2010	Prospective cohort study The mean time to follow-up was 23.5 years (maximum 47 years) Aluminium industry 1954–1999	Participants had worked for 3 or more years at the aluminium smelter or its power-generating station between 1954–1997 n=6 423 All participants were men	**Benzene-soluble material (BSM) and benzo(a)pyrene (BaP)** Job exposure matrices with dimensions for job, department and time period for BSM and BaP were developed independently of each other. The job exposure matrix was applied to each worker's work history record and aggregated over the worker's employment to obtain each worker's cumulative exposure	**Acute myocardial infarction mortality** Cause of death were recoded to ICD-9: Acute myocardial infarction (410) Cohort members were linked using probabilistic linkage techniques to the National Mortality Database (1954–1999) to ascertain their cause of death	Mortality according to acute myocardial infarction and expose to cumulative BSM (μg/m^3 year) and cumulative BaP (μg/m^3 year). Log-linear model. Change in –2 log likelihood*; B (slope parameter from model); SE **Benzene-soluble material (BSM)** 1.52; 0.0124; 0.0098 **Benzo(a)pyrene (BaP)** 1.32; 0.0020; 0.0017 *Change in –2 log likelihood compared with the model with age, calendar year and smoking status but no exposure (BaP and BSM were modestly associated with AMI)	Mortality according to acute myocardial infarction and expose to cumulative BSM (μg/m^3 year) and cumulative BaP (mg/m^3 year). Log–log model. change in –2 log likelihood*; B (slope parameter from model); SE **Benzene-soluble material (BSM)** 1.84; 0.0943; 0.0691 **Benzo(a)pyrene (BaP)** 1.88; 0.0611; 0.0447 *Change in –2 log likelihood compared with the model with age, calendar year and smoking status but no exposure
Frost et al 2008 [47] Great Britain	Prospective cohort study Follow-up not stated	All workers in The Great Britain Asbestos Survey employed in asbestos removal work	**Asbestos** At each medical examination workers completed the survey questionnaire,	**Circulatory disease, ischaemic heart disease, and cerebrovascular disease**	Standardised mortality all asbestos removal workers and a subcohort of workers with detailed questionnair. SMR (95% CI) *Circulatory disease* All workers: 114.0 (107.0; 121.4)	Relative risks of mortality for stripping/removal workers, using Poisson regression analyses, adjustment for age, calendar period, and sex. RR (95% CI) *Circulatory disease*

	Asbestos manufacturing industry 1971–2005	Participants were workers only ever employed as asbestos strippers. This was a subcohort from the The Great Britain Asbestos Survey where asbestos manufacturing workers were initially invited to participate in the survey with voluntary medical examinations at 2 yearly intervals n=31 302 n=52 387 in the entire cohort Gender not stated	which included information about asbestos exposures. For removal workers this included information on the type of dust suppression technique used, the kind of respirator used and the weekly hours spent in a stripping enclosure while removal was going on Only workers who filled in detailed questionnaire on exposure and work practices were included in analysis by duration of exposure and weekly hours spent stripping	m**ortality** Survey participants were flagged for death registrations at the National Health Service Central Register. Deaths occurring until December 2005 were included in the analysis The expected number of deaths was calculated using the 5-year age-, period- and sex-specific mortality rates for England and Wales, and for Scotland	Subcohort workers with detailed questionnaire: 102.1 (89.9; 115.2) *Ischaemic heart disease* All workers: 113.3 (104.9; 122.3) Workers with detailed questionnaire: 102.4 (87.7; 119.0) *Cerebrovascular disease* All workers: 125.0 (105.6; 146.9) Subcohort workers with detailed questionnaire: 118.3 (84.9; 160.5)	Length of exposure in years <10: 1.0 10–: 1.0 (0.7; 1.4) 20–: 0.8 (0.5; 1.2) 30–: 0.6 (0.4; 0.9) 40+: 0.9 (0.6; 1.5) *Ischaemic heart disease* Length of exposure in years <10: 1.0 10–: 1.2 (0.8; 1.7) 20–: 0.6 (0.3; 1.0) 30–: 0.6* (0.3; 1.0) 40+: 1.1 (0.6; 1.9) Weekly hours spent stripping/removal work <10: 1.0 10–: 1.0 (0.5; 1.8) 20–: 0.9 (0.6; 1.6) 30–: 1.6 (1.0; 2.6) 40+: 1.9 (1.2; 2.8) *Cerebrovascular disease* Length of exposure in years <10: 1.0 10–: 0.8 (0.3; 1.9) 20–: 0.7 (0.3; 1.5) 30–: - 40+: -
Gallagher et al 2012 [48] China	Prospective cohort study 11 years Textile industry 1989–2000	Participants were female textile workers initially enrolled in a randomized trial of breast self-examination	**Air pollution** Air pollution was assessed in time period-specific cotton dust exposure levels based on 2 400 historic	**Cardio-vascular disease mortality** Cause of death information was collected	Hazard ratios for stroke mortality and estimated cumulative exposure to endotoxin and cotton dust. HR (95% CI) adjusted for age at baseline and smoking **Endotoxin (EU/m³ x year)** Unexposed: 1.00 >0–2 275.08: 0.71 (0.33; 1.53)	–

		from textile factories in Shanghai The cohort included 267 400 women who were active or retired workers as of 1989–1991 Quantitative endotoxin and cotton dust measures were available for a subcohort The median age at enrolment was 43 (30 to over 60 years old) n=267 400 female textile workers out of which spinning, weaving or knitting: Cotton (n=86 265; also exposure to endotoxins), mixed fibers n=26 262, wool n=29 181, synthetic fibers n=11 335; all exposed to particulate	cotton dust measurements made by Chinese factory inspectors in 56 cotton factories between 1975–1999 Dust estimates were converted to endotoxin estimates using measurements made in 3 Shanghai factories (Astrakianakis et al, 2006) and job-specific endotoxin surveys (Olenchock et al, 1983; Kennedy et al, 1987; Christiani et al, 1993, 1999) Exposure estimates for all jobs that each woman held throughout the textile industry work history were summed to obtain cumulative exposure estimates for	from the Shanghai Textile Industry Bureau's Tumour and Death Registry Cardio-vascular disease outcomes were 1 or more causes of death by ICD-9 code The outcomes included ischemic heart disease mortality (ICD-9 codes 410–414), ischemic stroke mortality (434), and mortality from intracerebral haemorrhage (431)	>2 275.08–2 943.66: 1.07 (0.49; 2.32) >2 943.66–5 167.85: 0.91 (0.43; 1.95) >5 167.85: 1.52 (0.72; 3.24) **Cotton dust (mg/m³ x year)** Unexposed:1.00 >0–85.64: 0.75 (0.35; 1.61) >85.64–118.06: 1.12 (0.52; 2.41) >118.06–175.94: 1.14 (0.53; 2.48) >175.94: 1.15 (0.54; 2.42)	

		matter (organic dust All participants were women	both types of exposures			
Gardner et al 1986 [49] Great Britain	Cohort study asbestos cement factory 1941–1983	Participants were employed between 1941–1983 at an asbestos cement factory in England n=2 167 650 women 1 517 men	**Asbestos** The production process incorporated the use of chrysotile asbestos fibre only, except for a small amount of amosite during 4 months in 1976. Measured airborne fibre concentrations available since 1970 from personal samplers showed mean levels below 1 fibre/ml, although higher levels had probably occurred previously in certain areas of the factory	**Circulatory diseases** Each person identified in the factory records was traced where possible at a national register For circulatory diseases ICD-9 codes 390–459 were applied	Mortality among the workforce Circulatory diseases Women SMR: 119 (comparison based on local rates from Tamworth Municipal Borough) Observed/expected number of deaths based on rates from England and Wales (95% CI) 1.16 (0.87; 1.51) Men SMR: 118 Observed/expected number of deaths (95% CI) 0.87 (0.74; 1.01)	–
Gerhardsson et al 1995 [50] Sweden	Prospective cohort study Median duration of follow-up was 13.8 years	Participants were lead battery workers, employed for at least 3 months	**Lead** Since 1969 blood lead sampling has usually been performed every 2–*3*	**Cardio-vascular diseases, ischaemic heart diseases, and cerebro-**	Mortality in workers exposed to lead in comparison with the counrty population, during the follow-up period 1969–1989. SMR (95% CI) **Cardiovascular diseases** Total cohort: 1.46 (1.05;2.02)	–

	Lead smeltery 1942–1989	during 1942–1987 n=664 (201 workers first employed up to 1969, 463 workers employed after 1969) All participants were men	months over the years A blood lead index was calculated for each lead worker in the cohort and the cumulative lead exposure intensity over time and peak blood lead concentrations that exceeded 3.4 µmol/l were used as exposure variables The lead exposure among the smelter workers had gradually declined during the follow-up period from a mean concentration of 3.0 µmol Pb/l in 1969 to 1.6 µmol/l in 1985	**vascular diseases mortality** Information on causes of death 1969–*19*89 was obtained from Statistics Sweden. The death certificates were coded according to ICD-8 Cardio-vascular diseases: codes 390–458 Ischaemic heart diseases: codes 410–414 Cerebrovascular diseases: codes 430–438 Expected mortality for the period 1969–*19*89 was calculated by calendar year, cause, and 5-	First employed ≤1969: 1.54 (1.07; 2.20) First employed ≥1970: 1.19 (0.48; 2.46) **Ischaemic heart diseases** Total cohort:1.72 (1.20; 2.42) First employed ≤1969: 1.81 (1.22; 2.65) First employed ≥1970: 1.38 (0.51; 3.00) **Cerebrovascular diseases** Total cohort: 0 (0.00; 1.23) First employed ≤1969: 0 (0.00; 1.55) First employed ≥1970: 0 (0.00; 5.95)	

				year age group specific mortalities for the country population		
Gibbs et al 2014 [51] Canada	Cohort Aluminum smelting facilities 1950–2004	Participants were workers at 7 aluminum reduction plants n=5 977 All participants were men	Aluminum The exposure assessment methodology was based on measurement data averaging, a deterministic mathematical model using process-related correction factors, and expert-based extrapolation, previously described by Lavou´e et al.1 Estimates of exposure to coal tar pitch volatiles (CTPVs) were derived from measurements of benzene-soluble materials (BSM) and of B(a)P in the workplace. Predictions of past BSM and B(a)P concentrations were made on the basis of	Ischemic heart disease and cerebrovascular disease Mortality data for the period 1950–2004 were obtained from the Canadian National Mortality Database managed by Statistics Canada. Diagnoses after 2000 were obtained, coded to the 10th revision of the International Classification of Diseases (ICD-10). Diagnoses before 2000 were retrieved as originally coded in the registries (in	Standardized mortality ratios in men working in combined fixed and dynamic cohorts of aluminum reduction plants. SMR (95% CI) **Ischemic heart disease** *Fixed cohorts (years)* 1950–1999: 92.3 (87.2; 97.6) 2000–2004: 83.4 (66.2; 103.6) 1950–2004: 91.7 (86.7; 96.8) *Dynamic cohorts (years)* 1950–1999: 86.5 (75.4; 98.7) 2000–2004: 83.1 (62.4; 108.4) 1950–2004: 85.8 (76.0; 96.6) **Cerebrovascular disease** *Fixed cohorts (years)* 1950–1999: 113.6 (101.4; 126.8) 2000–2004: 68.9 (42.1; 106.4) 1950–2004: 109.4 (98.0; 121.7) *Dynamic cohorts (years)* 1950–1999: 112.9 (82.9; 150.1) 2000–2004: 61.1 (26.4; 120.5) 1950–2004: 100.5 (75.7; 130.8)	–

			work environment parameters considered most likely to influence past worker exposures. Concentrations were estimated for each job and period that job existed, and cumulative exposures to BSM and B(a)P were calculated using each individual's work history and the estimated concentrations associated with each job and period. This report uses only B(a)P as the index of CTPV exposure as the previous follow-up did not suggest major differences between B(a)P and BSM	ICD-8 or ICD-9 codes) and recoded as ICD-10 codes by Statistics Canada schemic heart disease (I20–I25, I51.6) Cerebrovascular disease (I60–I69)		
Glenn et al 2006 [52] South Korea	Prospective cohort 5 years	Participants were employed at 26 lead-using facilities	**Lead** Subjects completed a standardized questionnaire	**Systolic blood pressure** Blood pressure (systolic and	Association of tibia lead with systolic blood pressure. Longer-term changes associated with cumulative dose. β (95% CI)	–

	A lead-exposed occupational cohort at lead-using facilities 1997–2001	On average, participants were 41 years old at baseline and had worked 8.5 years in lead-exposed jobs 575 workers with complete data for 3 visits. Of these, 504 workers had current inorganic lead exposure and 71 workers no longer had positions with lead exposure. During the study, 78 additional workers left their lead-exposed jobs. All "retired" workers were retained in the cohort and were included in the analyses n=575 140 women 435 men	concerning personal characteristics and behaviours, medical history, and an occupational history Tibia lead was measured using x-ray fluorescence in units of micrograms of lead per gram of bone mineral (μ,g/g)	fifth Korotkoff diastolic) was measured at each visit using a sphygmo-manometer Hypertension was defined as a report of a physician's diagnosis of hypertension or taking medications to control high blood pressure	Final models included covariates for visit, baseline age, baseline age squared, categories of lifetime alcohol consumption, body mass index, gender, and use of blood pressure-lowering medications Controlling for the baseline association of recent dose with baseline blood pressure: –0.02 (–0.03–0.004) Controlling for the baseline association of cumulative dose with baseline blood pressure: –0.02 (–0.03–0.01) Controlling for the baseline association of cumulative dose with baseline blood pressure, *excluding hypertension*: –0.02 (–0.03–0.002)	
Graham et al 2004 [53]	Prospective cohort study	Participants were all men who had	**Dust (granite, quartz)**	**Mortality due to all diseases of the**	Mortality in workers exposed to granite dust comparison with all white males in the United States. SMR (95% CI)	–

USA	Granite industry Cases were identified 1950–1996	worked in the Vermont granite industry. The cohort included men who had been exposed to high levels of granite dust prior to 1940 (high-exposure group) and those employed at dust levels after 1940 (low-exposure group) n=5 408 workers (2 539 deaths) All participants were men	Exposure before and after 1940, when dust controls were introduced and exposures were reduced by 80–90%. Before 1940, general stone shed air contained 20 million particles/cubic foot (mppcf) (approximately equivalent to 0.2 mg/m of quartz), and pneumatic chisel workers were exposed on average to 60 mppcf (approximately equivalent to 0.6 mg/m of quartz). Other workers had variable exposures After 1940, a period of decline occurred in dust levels and then stabilized in approximately 1955, when average dust levels were 5–6	**circulatory system and to ischemic heart disease** Vital status of workers was identified by death certificated ascertained from Vermont department of health (has reciprocal notification agreements regarding deaths occurring out of state), obituary notices, and the National Death Index Death certificates were analysed by a qualified nosologist and underlying cause of death was coded according to the ICD-8	**All diseases of the circulatory system** Total cohort: 0.79 (0.74; 0.85) High-exposure group: (first employed ≤1940): 0.94 (0.86; 1.03) Low-exposure group: (first employed ≥1940): 0.63 (0.56; 0.70) **Ischaemic heart diseases** Total cohort: 0.74 (0.69; 0.80) High-exposure group: (first employed ≤1940): 0.89 (0.81; 0.97) Low-exposure group: (first employed ≥1940): 0.58 (0.51; 0.65) **Silicosis** Total cohort: 20.55 (15.39; 26.87) High-exposure group: (first employed ≤1940): 27.38 (20.32; 36.10) Low-exposure group: (first employed ≥1940): 3.98 (0.82; 11.64)	

			mppcf (equivalent to 0.05-0.06 mg/m^3 of quartz)			
Gustavsson et al 1990 [56] Sweden	Cohort study Gas industry 1966 to 1986	Participants were workers at a Swedish gas production company, employed for at least one year between 1965 and 1972 n=295 All participants were men	**Benzo(a)pyrene** The exposure to benzo(a)pyrene (BaP) was measured in the current plant by area sampling on top of the ovens. In 1964 a mean level of 4.3 µg/m^3 of BaP was detected, (range 0.007-33 µg/m^3). In 1965 lower levels were found, mean 0.52 µg/m^3, range 0.021-1.29 µg/m^3 Generator gas for heating the coke ovens was produced in the steam and generator department. Generator gas contains - around 30% carbon monoxide	**Several** Expected numbers of deaths were based on local death rates among occupationally active men Outcome was traced by a register of the living population at Stockholm County Administration Board, death and burial books at the clerical parishes, and Stockholm City Archives. Underlying causes of deaths were obtained from Statistics Sweden Mortality was investigated from 1966 to 1986	Mortality among the gas workers between 1966 and 1986. Reference rates based on local mortality adjusted for occupational activity **Circulatory disease** All employed: 127 (90; 174) Employed 1-29 years: 84 (34; 174) Employed ≥30 years: 175 (100; 284) **Ischemic heart disease** All employed: 125 (83; 181) Employed 1-29 years: 81 (26; 190) Employed ≥30 years: 167 (83; 299) **Cerebrovascular disease**: 152 (56; 331)	

			Measurements of the carbon monoxide content in exhaled air was measured among the workers at the coke ovens and at the steam and generator central in 1967, which indicated that only a few of these workers were exposed to carbon monoxide in significant amounts			
Gustavsson et al 2001 [55] Sweden	Case-control. Data extracted from the SHEEP study General population 1992–1994 Male cases were identified 1992–1993 and female cases 1992–1994	Participants were Swedish citizens living in Stockholm County who were 45–70 years of age and free of clinically diagnosed myocardial infarction n=2 993 **Cases** Women: 398 Men: 937 **Controls** Women: 538	**Exposure to different chemical substances** Participants answered questionnaires on lifetime occupational history, description and duration of work tasks and specific occupational exposures. A senior industrial hygienist examined the questionnaires	**Myocardial infarction** Cases were persons surviving at least 28 days after the infarction, identified from coronary or intensive care units at the emergency hospitals in Stockholm County or from a computerized hospital	Estimates for myocardial infarction according to the highest intensity of exposure during at least 1 year of work. RR (95% CI) adjusted for age group, sex, year of enrollment and hospital catchment area **Highest intensity of exposure** *Motor exhaust (mg of CO/m^3)* Unexposed: 1 >0–2.2: 1.04 (0.78; 1.40) 2.3–3.3: 1.54 (1.15; 2.09) 3.4–6.8: 1.73 (1.29; 2.31) 6.9–11.3: 1.51 (1.01; 2.25) ≥11.4: 1.15 (0.77; 1.71) *Combustion products other than motor exhaust (mg of respirable particles/m3)* Unexposed: 1.0 >0–0.9: 1.15 (0.93; 1.42) 1.0–2.4: 1.76 (1.32; 2.34)	Estimates for myocardial infarction according to the highest intensity of exposure during at least 1 year of work. RR (95% CI) adjusted for age group, 6, year of enrolment, hospital catchment area, smoking, alcohol drinking, hypertension, overweight, diabetes mellitus and physical activity at leisure time **Highest intensity of exposure** *Motor exhaust (mg of CO/m3)* Unexposed: 1.0 >0–2.2: 0.95 (0.69; 1.29) 2.3–3.3: 1.34 (0.98; 1.83) 3.4–6.8: 1.36 (0.99; 1.85) 6.9–11.3: 1.24 (0.81; 1.9) ≥11.4: 0.98 (0.64; 1.50)

		Men: 1 120	and assessed the probability and the intensity of occupational exposure to substances by the expert rating method The intensity of exposure to motor exhaust was assessed by a job-exposure matrix (Siemiatycki 1996) The other factors were estimated in a semi-quantitative way based on exposure levels reported for a limited number of occupations in which the respective exposure was common	discharge register Standardized diagnostic criteria to define myocardial infarction were used Controls were selected through computerized population register at the time of case identification	≥2.5: 2.18 (1.30; 3.64) *Organic solvents (hygienic effect)* Unexposed: 1.0 >0.5[1]–0.19: 1.32 (1.08; 1.61) 0.2–0.5: 1.13 (0.82; 1.55) ≥0.5: 1.6 (1.04; 2.48) *Lead (mg/m3)* Unexposed: 1.0 >0–0.03: 0.94 (0.75; 1.18) ≥0.04: 1.17 (0.75; 1.82) *Dynamite* Unexposed: 1.0 Exposed: 1.55 (0.92; 2.61) [1] Probably meant to be 0.05 **Cumulative exposures** *Motor exhaust (mg of CO/m3-year)* Unexposed: 1 >0–15.4: 1.07 (0.92; 1.40) 15.4–60: 1.54 (1.20; 1.99) ≥61: 1.55 (1.20; 2.00) *Combustion products other than motor exhaust (mg of respirable particles/m3-year)* Unexposed: 1.0 >0–1.64: 1.17 (0.90; 1.53) 1.65–6.74: 1.40 (1.06; 1.85) ≥6.75: 1.60 (1.22; 2.08) *Organic solvents (hygienic effect-years)* Unexposed: 1.0 >0–0.55: 1.50 (1.16; 1.95) 0.56–1.9: 1.09 (0.82; 1.44) ≥2.0: 1.31 (1.01; 1.69) *Lead (mg/m3-years)* Unexposed: 1.0	*Combustion products other than motor exhaust (mg of respirable particles/m3)* Unexposed: 1.0 >0–0.9: 1.0 (0.80; 1.25) 1.0–2.4: 1.42 (1.05; 1.92) ≥2.5: 2.11 (1.23; 3.60) *Organic solvents (hygienic effect***)** Unexposed: 1.0 >0.5[1]–0.19: 1.26 (1.02; 1.55) 0.2–0.5: 1.05 (0.76; 1.47) ≥0.5: 1.49 (0.94; 2.35) *Lead (mg/m3)* Unexposed: 1.0 >0–0.03: 0.88 (0.69; 1.12) ≥0.04: 1.03 (0.64; 1.65) *Dynamite* Unexposed: 1.0 Exposed: 1.49 (0.86; 2.56) [1] Probably ment to be 0.05 **Cumulative exposures** *Motor exhaust (mg of CO/m3-year)* Unexposed: 1 >0–15.4: 0.98 (0.74; 1.31) 15.4–60: 1.32 (1.01; 1.73) ≥61: 1.21 (0.92; 1.59) *Combustion products other than motor exhaust (mg of respirable particles/m3-year)* Unexposed: 1.0 >0–1.64: 1.00 (0.75; 1.33) 1.65–6.74: 1.22 (0.91; 1.64) ≥6.75: 1.35 (1.02; 1.79) *Organic solvents (hygienic effect-years)* Unexposed: 1.0

					>0–0.04: 0.86 (0.64; 1.15) ≥0.05: 1.10 (0.84; 1.45) *Dynamite* Unexposed: 1.0 Duration <7.0 years: 1.31 (0.63; 2.74) Duration ≥7.0 years: 1.82 (0.87; 3.82)	>0–0.55: 1.50 (1.14; 1.96) 0.56–1.9: 1.00 (0.74; 1.34) ≥2.0: 1.20 (0.92; 1.58) *Lead (mg/m3-years)* Unexposed: 1.0 >0–0.04: 0.81 (0.60; 1.11) ≥0.05: 1.00 (0.74; 1.34) *Dynamite* Unexposed: 1.0 Duration <7.0 years: 1.20 (0.56; 2.57) Duration ≥7.0 years: 1.83 (0.85; 3.92)
Gustavsson et al 2013 [54] Sweden	Cohort study Chimney sweeps 1991–2005	Participants were male Swedish chimney sweeps and union members between 1918–2006. They were followed from the later of the following dates: first membership/employment period or 01/01 1991, and up to 31/12 2005	**Several exposures assumed** (not measured): Dust, particulate matter <2.5 µm, combustion-generated particles, polycyclic aromatic hydrocarbons, arsenic, chromium, cadmium, nickel, lead and asbestos	**Acute first-time myocardial infarction** incidence (lethal and non-lethal) as obtained by the Swedish National Board of Health and Welfare's nationwide register of first-time, since 1987 Causes of mortality obtained from the nationwide Swedish registers of Total Population and Causes of	Standardised incidence ratios for total cohort of Swedish chimney sweeps and by employment duration. SIR (95% CI) **Myocardial infarction** 0–9 years employment: 1.53 (1.28; 1.81) 10–19 years employment: 1.28 (0.99; 1.63) 20–29 years employment: 1.18 (0.86; 1.59) >30 years employments: 1.39 (1.08; 1.76) Total cohort: 1.39 (95% CI 1.24; 1.55)	–

				Deaths and classified according to the International Classification of Diseases		
Harding et al 2012 [57] Great Britain	Prospective cohort study Asbestos workers Median follow-up of 19 years 1971–2005	Workers taking part in voluntary medical surveillance for the early detection of asbestos-related disease were included in the survey, as well as workers undergoing regular statutory medical examinations as required by asbestos licensing regulations n=98 912 Both men and women participated	**Asbestos** At the time of a medical examination, workers were invited to participate in the survey and to complete a survey questionnaire The questionnaire requested information on duration of occupational exposure to asbestos, current job type and smoking history	Ischemic heart disease and cerebro-vascular disease mortality Cerebrovascular diseases codes 430–438 for ICD-9 and codes I60–I69 for ICD-10) Ischemic heart disease codes 410–414 (ICD-9) and codes I20–I25 (ICD-10) Mortality was identified through the underlying cause of death. Deaths occurring to the end of 2005 were included	Standardized mortality ratios, unadjusted. SMR (95% CI) **Ischemic heart disease** *Women*: 1.89 (1.62; 2.19) *Men*: 1.39 (1.35; 1.43) **Cerebrovascular disease** *Women*: 2.04 (1.64; 2.51) *Men*: 1.63 (1.52; 1.73) Poisson regression analysis. For each disease, the data represent a single model including job, year of birth, duration of exposure, sex, age attained and smoking status. RR (95% CI) **Ischemic heart disease** *Women and men, duration of exposure (years)* <10: 1.0 10–19: 1.16 (1.03; 1.30) 20–29: 1.10 (0.98; 1.24) 30–39: 1.15 (1.02; 1.30) 40+: 1.25 (1.10; 1.42) **Cerebrovascular disease** *Women and men, duration of exposure (years)* <10: 1.0 10–19: 1.20 (0.93; 1.56) 20–29: 1.05 (0.80; 1.37) 30–39: 1.04 (0.79; 1.38) 40+: 1.22 (0.93; 1.62)	Standardized mortality ratios, smoking adjusted. SMR (95% CI) SMR (95% CI) **Ischemic heart disease** *Women*: 1.61 (1.38; 1.87) *Men*: 1.28 (1.24; 1.32) **Cerebrovascular disease** *Women*: 1.86 (1.49; 2.28) *Men*: 1.51 (1.42; 1.61)

				Workers were flagged for death registrations with a national health service register		
Hart et al 2013 [58] USA	Cohort Trucking industry 1985–2000	Partcipants were men with at least 1 year of work in a trucking industry job who also had ambient pollution information available at their home address Participants were unionised employees working in 1985 from 4 large national companies n=52 345 All participants were men	**Vehicle exhaust** Exposure was categorised into 8 job categories based on a review of job titles and duties from a 2001–2005 industrial hygiene exposure assessment and information on the historical use of diesel and other vehicles by workers in the cobort Each worker could potentially accumulate exposure in multiple job categories throughout his career	**Ischemic heart desease** The National Death Index record axis data, 1985–2000, that lists both underlying and contributing causes of death and identified all deaths with IHD (ICD-9 410–414.9, ICD-10 120–125.9) was used	Ischemic heart desease mortality associated with any work in each major job title, follow-up 1985–2000. HR (95% CI) using regression coefficients from Cox proportional hazards regression models stratified on age in 1985, decade of hire and caiendar time, with risk sets by attained age, and adjusted for race Long haul: 1.39 (1.18; 1.64) Pick-up and delivery: 1.11 (0.96; 1.28) Dockworker: 1.31 (1.13; 1.52) Combination: 1.07 (0.90; 1.28) Mechanic: 1.05 (0.80; 1.3 8) Hostler: 1.01 (0. 77; 1.33) Clerk: 0.60 (0.41; 0.87)	Ischemic heart desease mortality associated with any work in each major job title, follow-up 1985–2000. HR (95% CI) additionally adjusted for the healthy worker survivor effect (total years on work, years off of work), census region and the 1985–2000 average ambient PM_{10}, $N0_2$ and $S0_2$ values at the last known residential address Long haul: 1.44 (1.22; 1.70) Pick-up and delivery: 1.11 (0.96; 1.28 Dockworker: 1.30 (1.12; 1.51) Combination: 1.11 (0.93; 1.32) Mechanic: 1.10 (0.84; 1.44) Hostler: 1.04 (0.79; 1.37) Clerk: 0.63 (0.43; 0.91)
He et al 1994 [60] China	Case-control study Women with full time jobs	Participants were women with full time	**Passive smoking at work**	**Coronary heart disease** The final diag-nosis was	Passive smoking. Crude OR (95% CI) *Passive smoking at work* All: 2.45 (1.23; 4.88)	Passive smoking at work. OR (95% CI) adjusted for age, history of hypertension, type A personality, total

	December 1989–November 1992	jobs, who had never smoked Cases were patients with coronary heart disease (non-fatal, incident cases) from the 3 large teaching hospitals Controls were from 3 sources: patients admitted because of suspected or diagnosed coronary heart disease but confirmed to be normal after coronary arteriography (no coronary stenosis at all); other medical outpatients attending cardiology departments and a random sample of healthy subjects from a community screening programme for coronary heart disease. The	A standardised questionnaire was designed to collect information on passive smoking at work The interviews were carried out by 3 trained interviewers	myocardial infarction according to WHO criteria or coronary stenosis confirmed by coronary arteriography	Husband *not* smoking: 2.53 (0.82; 7.83) Husband smoking: 4.18 (1.63; 10.92)	cholesterol, high density lipoprotein and passive smoking from husband 2.36 (1.01; 5.55)

		latter 2 groups were confirmed to be free of coronary heart disease by WHO criteria 9 and normal exercise electro-cardiography n=185 59 patients and 126 controls All participants were women				
He et al 2012 [59] China	Cohort study 7-year follow-up Machinery factory 1976–1994	Participants worked at a machinery factory in Xi'an who were never smokers both in 1976 and in 1994. Never smoking was defifi ned as not smoking currently or having smoked <100 cigarettes in their lifetime n=910 471 women 439 men	**secondhand smoke** Secondhand smoke exposure was defifined as exposure to another person's tobacco smoke in the workplace For at least 15 min daily for >1 day every week and the duration of SHS exposure was for at least 2 years since 1976	**Ischemic stroke** Vital status was traced by 2 senior physicians of the factory hospital. Causes of death were obtained from hospital death certificates or death certifi cates in the local police department Stroke was defined using World Health Orga-nization Multinational Monitoring of Trends and	Major causes of death by second hand smoke exposure at work. RR (no confidence interval) Ischemic stroke: 3.05	–

				Determinants in Cardio-vascular Disease (MONICA) criteria		
Hein et al 2007 [61] USA	Cohort study Asbestos textile plant 1916–1977	Participants were workers exposed to chrysotile in a South Carolina asbestos textile plant The original cohort was defined as all white male workers employed in textile production operations for at least 1 month between 1940–1965, with vital status follow-up through 1975. The cohort was expanded to include white and non-white males and white females and vital status follow-up was extended through 1990 n=3 072	**Asbestos** The study plant produced asbestos products and asbestos textile products Detailed work histories listing beginning and ending dates in departments and operations were available for each member of the cohort. A job exposure matrix was available to link with the detailed work histories to calculate cumulative exposure to chrysotile. Chrysotile exposure concentrations (expressed as fibres longer than 5 micrometers per millilitre of	**Several diseases, mortality** Workers were followed up for mortality through 2001 For the update, names of cohort members were submitted to a national death index for determination of vital status from 1991–2001 All deaths were coded according to the revision of the International Classification of Diseases (ICD) in effect at the time of death	Mortality through 2001 based on US mortality rates for selected causes of death among workers in the South Carolina asbestos textile workers cohort. SMR (95% CI) **All workers combined** Diseases of the heart: 1.20 (1.10; 1.30) Ischaemic heart disease: 1.20 (1.10; 1.32) Hypertension*: 1.63 (0.87; 2.78) Cerebrovascular disease: 1.29 (1.08; 1.53) **Women** Diseases of the heart:1.12 (0.97; 1.29) Ischaemic heart disease: 1.11 (0.94; 1.30) Hypertension*: 1.40 (0.38; 3.59) Cerebrovascular disease: 1.19 (0.90; 1.56) **White men** Diseases of the heart: 1.38 (1.23; 1.55) Ischaemic heart disease: 1.39 (1.22; 1.58) Hypertension*: 1.10 (0.13; 3.99) Cerebrovascular disease: 1.70 (1.25; 2.24) **Non-white men** Diseases of the heart: 0.95 (0.77; 1.15) Ischaemic heart disease: 0.92 (0.72; 1.17) Hypertension*: 2.10 (0.84; 4.33) Cerebrovascular disease: 1.03 (0.69; 1.49) *without heart disease	–

		1 265 women 1 807 men	air) were estimated using statistical modelling of nearly 6 000 industrial hygiene sampling measurements taken over the period 1930–1975 and analysed using phase contrast microscopy. Exposure concentrations were considerably higher before 1940, before engineering dust control measures were put into place Cumulative exposure, fibre-years/ml median was 4.4 (white men), 4.2 (white women), 14.5 (non-white men) and 5.9 (non-white women)			
Hernberg et al 1976 [62] Finland	Cohort study 8 years follow-up	Participants were male workers in a	**Carbon disulphide** The plant chemists had	**Coronary heart disease** The causes of deaths during	Rate ratio for mortality to coronary heart disease 1942–1975, exposed cohort vs controls. Rate ratio (95% CI)	–

Note: same study population as Hernberg 1970, this is an 8-year follow-up *Also same population as in the articles by Tolonen et al 1975 and Nurminen et al*	Viscose plant 1942–1975	viscose rayon plant Inclusion criteria: (1) age 25–64 years at the time of examination (1967/1968); (2) 5 years or more of exposure to CS_2 in the most heavily polluted departments during any period between 1942–1967; (3) those who had died before reaching the age of 65 years The exposed subjects were matched with controls with histories of no exposure. These were selected from the employee rolls of a paper mill located in the same city n=686 (343 exposed workers and 343 controls)	collected air samples routinely since 1945 At 1975, 48% of the intial sample were still empolyed at the plant. Also, the exposure status of the viscose workers change radically during the last 3 years and only 19% of the men were still exposed to CS_2, which levels had decreased drastically	the follow-up periond from June 1967–May 1975 were verified from death certificates and classified according to ICD-8 No 1 was lost during the follow-up	Total period: 2.2 (1.0; 4.8) Last 3 years: 1.0 (confidence intervals not stated)	

		All participants were men				
Hernberg et al 1970 [117] Finland *Note: same study population as Hernberg 1976* *Also same population as in the articles by Tolonen et al and by Nurminen et al*	Cohort study Viscose plant 1942–1967	Participants were male workers in a viscose rayon plant. Inclusion criteria: (1) age 25–64 years at the time of examination (1967/1968); (2) 5 years or more of exposure to CS_2 in the most heavily polluted departments during any period between 1942–1967; (3) those who had died before reaching the age of 65 years The exposed subjects who were alive were matched with controls with histories of no exposure. These were selected from the employee rolls of a paper mill located in the same city,	**Carbon disulphide** The plant chemists had collected air samples routinely since 1945. CS_2 and H_2S concentrations were determined separately using a titrimetric xanthate method. An index of exposure dosage was calculated for every exposed subject from his work history using the CS_2+H_2S measures Index calculations were based on number of months worked and annual arithmetic mean of the CS_s+H_2S concentration	**Myocardical infarction and blood preassure** Death certificates were obtained for the exposed men who had died before reaching the age of 65 years Coronary history was obtained by using a WHO questionnaire (World Health Organization, 1963) Blood pressure was measured with wrap-around cuffs and a stethoscope. 2 successive recordings were made. The diastolic readings were made when Korotkoff's	Mortality to myocardical infarction adjusted for age and year of death (observed vs expected) **Mortality to myocardical infarction** p=0.0018 (the proportion of coronary deaths was higher than expected on the basis of general mortality statistics) **Exposed group vs controls** *History of myocardical infarction* Total: n.s Spinners: n.s Exposed ≥15 years: n.s Dosage index >245: n.s *Systolic blood pressure* Total: p<0.001 Spinners: p<0.05 Exposed ≥15 years: n.s Dosage index >245: n.s *Diastolic blood pressure, 4th phase* Total: p<0.001 Spinners: p<0.001 Exposed ≥15 years: p<0.001 Dosage index >245: p<0.001 *Diastolic blood pressure, 5th phase* Total: p<0.001 Spinners: p<0.05 Exposed ≥15 years: p<0.05 Dosage index >245: p<0.001	–

		without any knowledge of the health of the control n=734 (48 dead exposed subjects, 343 exposed workers and 343 controls) All participants were men		sounds disappeared The expected number of coronary deaths, adjusted for age and year of death was based on official statistics for the general male population		
Hertzman et al 1997 [63] Canada	Prospective cohort study Average years of follow-up was 24.5 Sawmill 1950–1990	Participants were sawmill workers from 11 chloro-phenateusing mills, all who had worked for at least 1 year in a study mill between January 1, 1950, and December 31, 1985 The male population of British Columbia served as the external comparison group n=23 829	**Chlorophenate** Each worker's cumulative exposure history was calculated based on old records and key informants to reconstruct industrial histories for each mill. Time lines were for the introduction of chloro-phenate and changes in formulation, application technology, and locations and exposure-constant time periods was	**Circulatory system disease, mortality** Vital status was identified by the British Columbia Death File and the Canadian Mortality Data Base. Those sawmill workers who were lost to follow-up but who had social insurance numbers were linked by Statistics Canada to confirm vital status	Standardized mortality ratios for cause-specific mortality among the British Columbia sawmill workers cohort, by employment in 11 chlorophenate-using. SMR (95% CI) *Circulatory system disease* Person-years to last known year: 1.14 (1.10; 1.18) Person-years to 1990: 0.74 (0.71; 0.76)	–

		All participants were men	identified for a given job title. Summary exposure score was then calculated for each job title in each time period. This score, which combined then averaged the worker raters' estimates of duration and frequency of exposure for each job title, was interpreted as a number of "exposure hours per year"	Causes of death were coded according to ICD-8		
Hertz-Picciotto et al 2000 [64] USA	Retrospective cohort study 36 years Industry 1940–1976	Participants were white males employed 1 year or more during 1940–1964 at a single copper smelter in the US Number of persons included is not stated 72 946 person-years	**Arsenic exposure** Arsenic exposures were assigned on the basis of departmental air monitoring data, urinary arsenic data and work history of each employee	**Several outcomes** Deaths were identified from company files and social security files Circulatory disease mortality included ICD-8 codes 390–458 Further division was made in to	Disease rate ratio in relation to cumulative arsenic exposure. Baseline. RR (95% CI) adjusted for age and year of hire **Circulatory disease** ***Cumulative arsenic exposure (µg As/m^3-years)*** <750: 1.0 750–1 999: 0.90 (0.63; 1.3) 2 000–3 999: 0.90 (0.64; 1.3) 4 000–7 999: 0.99 (0.70; 1.4) 8 000–19 999: 0.95 (0.67; 1.4) ≥20 000: 1.0 (0.70; 1.5) **Cardiovascular disease** ***Cumulative arsenic exposure (µg As/m^3-years)*** <750: 1.0 750–1 999: 0.86 (0.56; 1.3) 2 000–3 999: 0.95 (0.64; 1.4) 4 000–7 999: 1.1 (0.77; 1.7)	Disease rate ratio in relation to cumulative arsenic exposure. 20 year lag. RR (95% CI) adjusted for age, year of hire, work status and healthy worker survivor effect **Circulatory disease** ***Cumulative arsenic exposure (µg As/m^3-years)*** <750: 1.0 750–1 999: 0.94 (0.70; 1.3) 2 000–3 999: 1.1 (0.82; 1.5) 4 000–7 999: 1.2 (0.90; 1.7) 8 000–19 999: 1.4 (1.0; 1.9) ≥20 000: 1.3 (0.86; 2.0) **Cardiovascular disease** ***Cumulative arsenic exposure (µg As/m^3-years)***

				cardiovascular disease (410–414 and 420–429) and cerebrovascular disease (430–438)	8 000–19 999: 1.2 (0.83; 1.9) ≥20 000: 1.3 (0.86; 2.1) **Cerebrovascular disease** ***Cumulative arsenic exposure (µg As/m³-years)*** <750: 1.0 750–1 999: 0.80 (0.37; 1.7) 2 000–3 999: 0.70 (0.33; 1.5) 4 000–7 999: 0.53 (0.24; 1.2) 8 000–19 999: 0.32 (0.13; 0.77) ≥20 000: 0.45 (0.18; 1.1)	<750: 1.0 750–1 999: 0.90 (0.64; 1.3) 2 000–3 999: 1.1 (0.78; 1.6) 4 000–7 999: 1.4 (0.98; 2.0) 8 000–19 999: 1.7 (1.2; 2.5) ≥20 000: 1.5 (0.95; 2.5) **Cerebrovascular disease** ***Cumulative arsenic exposure (µg As/m³-years)*** <750: 1.0 750–1 999: 1.0 (0.54; 2.0) 2 000–3 999: 1.1 (0.55; 2.1) 4 000–7 999: 0.60 (0.27; 1.3) 8 000–19 999: 0.71 (0.31; 1.6) ≥20 000: 0.67 (0.23; 1.9)
Hilt et al 1999 [65] Norway	Prospective cohort study Welding factory 1960–1993	Participants were men who had worked at a welding plant for more than 1 year since 1960, and who were under the age of 70 years at the time of the study in 1993 The control group consisted of randomly chosen men from the census register of the town where the plant was located, and categorically matched for age. Inclusion criteria were	**Dust** Data on occupational exposure factors were collected from a questionnaire. In addition, information on exposure was gathered by an experienced occupational hygienist The main welding methods were tungsten inert gas welding (TIG), metal inactive/active gas welding (MIG/MAG), and manual	**Cardio-vascular diseases** Data on the occurrence of cardio-vascular diseases and related symptoms were collected from both groups by means of a mailed self-administered questionnaire In order to assure the quality of the questionnaire data regarding	Prevalence ratio of cardiovascular diseases and related symptoms in stainless steel metal workers and a control group from the general population. PR (95% CI) adjusted for age and smoking habits Myocardial infarction: 1.37 (0.7; 2.8) High blood pressure: 1.08 (0.7; 1.6) Angina pectoralis: 0.64 (0.2; 2.8) Multiple logistic regression for outcome variables consistent with ischemic heart disese for the study group versus the control group, for 10 years of employment at the plant, for directly and indirectly exposed members of the study group as compared with the unexposed, and for specific exposure factors or actvities. OR (95% CI) adjusted for age, amount of current smoking, length of education, and first degree relatives with cardiovascular diseases under the age of 50 **Angina pectoris** Study versus control: 2.5 (1.1; 5.8) 10 y employment: 1.1 (0.7; 1.7)	–

		that these participants should not be metal workers, and they should have been occupationally active for at least 5 years since 1960 n=1 225 (236 from study group and 989 from control group) All participants were men	metal arc welding (MMA). While the amount of MMA welding had decreased, the proportions of the welding methods used at the time of the study were 60% TIG, 10% MIG/MAG, and 30% MMA. Dust measurements in 1976 showed a mean concentration of total dust of 4.1 mg/m3, and of total chromium and nickel of 0.1 and 0.04 mg/m3 respectively The study group was divided into: probably no exposure to dust or gases, probably indirect exposures only, and workers who had been working directly with welding or grinding	cardiovascular diseases morbidity, the researchers obtained permission from each participant to check their health information with their appointed general practitioners	Indirectly exposed: 3.0 (0.6; 13.9) Directly exposed: 2.1 (0.7; 6.1) Any welding: 1.4 (0.5; 4.2) Stainless steel welding: 1.6 (0.5; 5.0) Grinding: 3.3 (1.3; 8.5) Insulation with foam: 2.3 (1.03; 5.0) **Myocardial infarction** Study versus control group: 2.4 (1.1; 4.9) 10 y employment: 1.3 (1.0; 1.7) Indirectly exposed: 3.6 (1.1; 11.5) Directly exposed: 2.4 (1.0; 5.9) Any welding: 1.8 (0.7; 4.4) Stainless steel welding: 1.8 (0.7; 4.7) Grinding: 2.5 (1.1; 5.9) Insulation with foam: 4.0 (2.2; 7.2)	
Hogstedt et al 1977	Case – referent study	Participants were male	**Nitroglycerine-nitroglycol**	**Ischemic heart disease**	Risk ratio for exposure of men who died 1955–1977. RR (95% CI)	–

[67] Sweden *Note: Same population as Hogstedt et al 1984*	Dynamite industry 1921–1975	factory workers with more than 1 year employment, aged 36–70 who died between the years 1955–1975 in Noraberg (8 000 habitants) Some cases, not being classifiable were excluded from the study. In additions, all explosion accident were excluded and people with diabetes mellitus n=353 deaths (169 cases, who died in cardio-cerebrovascular diseases, and 184 referents, subjects who died from alla other diseases) All participants were male	Type of occupation and duration of employment were assessed by a company representative through available registers Exposure were classified as fulltime or part-time based on type of work in the factory Full-time exposed persons received exposure partly by inhalation, partly through the skin whereas part-time exposure is referring to temporary exposure	**and cerebro-vascular diseases** Death due to ischemic heart disease and cerebro-vascular diseases was classified according to WHO (ICD 410–412, 427–428, 430–438) The diagnoses from the death records were classified as to the under-lying cause of death without knowledge of work exposure	*Ischemic heart disease+Cerebrovascular diseases* Exposed vs non-exposed: 3.2 (1.4; 7.3) *Ischemic heart disease* Exposed vs non-exposed: 3.4 (1.5; 7.8) Crude rate ratio for exposure of men who died in cardio-cerebrovascular diseases 1955–1977 *Ischemic heart disease+Cerebrovascular diseases* Exposed vs non-exposed: 2.5 (p <0.01) *Ischemic heart disease* Exposed vs non-exposed: 2.7 (p <0.01) *Cerebrovascular diseases* Exposed vs non-exposed: 1.6 (n.s)	
Hogstedt et al 1979 [66] Sweden	Prospective cohort study Dynamite industry	Participants were factory workers employed for at	**Nitroglycerine-nitroglycol** First measure was made in	**Circulatory system** Deseasesed men were	SMRs for number of deaths before the age of 80 in people exposed or unexposed to nitroglycerine-nitroglycol compared with the national average.	–

	1927–1977	least on year, in 1927 or later Expected number of deaths were calculated by multiplying person-years of observation within 5-year age categories during the respective calender years of the study period by the cause-, sex-, and age-specific death rates n=143 (88 exposed and 55 unexposed) OBS. No direct comparisons were made All participants were male	1954 and the 8-hour time-weighted-average in the breathing zone might be calculated in the range og 1–2.5 mg nitrates/m^3. Until the 1950's all work was done without protective equipment and over the yers skin absorbtion has probably been extensive Job titles from the company records were classified according into exposure categories by a reference group of workers with very long experience in ther factory	traced through the death and burial books of the parishes Death certificates have been checked with the SNCBS and the officially determinded underlying cause of death has been used and classified according to the 1965 revison of ICD (410–438 circulatory system)	***Circulatory system*** *Exposed dynamite workers* Observed 1951–*19*64: Observed 2 versus expected 2.8, n.s Observed 1965–*19*77: Observed 9 versus expected 4.5, p<0.05 *Dynamite workers with at least 10 years of exposure* Observed 1951–*19*64: Observed 2 versus expected 2.6, n.s Observed 1965–*19*77: Observed 8 versus expected 3.6, p<0.05 *Unexposed dynamite workers* Observed 1951–*19*64: Observed 1 versus expected 1.3, n.s Observed 1965–*19*77: Observed 3 versus expected 2.7, n.s	
Hogstedt et al 1984 [68] Sweden *Note: Extended study, same population as*	Case – referent study Dynamite industry 1921–1980	Participants were male factory workers with more than 1 year employment, aged 36–70 who died between the	**Nitroglycerine-nitroglycol** The mean 8-hour tme-weighted average concentrations of nitrate esters for different job	**Ischemic heart disease and cerebrovascular diseases** The diagnoses from the death records were	Crude rate ratio for exposure of men who died in cardio-cerebrovascular diseases. CRR (95% CI) *Cerebrovascular diseases (ICD 430–38)* 1976–1980: 4 (n.s) 1955–1980: 2.9 (0.9; 6.4) *Cardio-vascular diseases (ICD 410–12, 427–28)*	Risk ratio after stratification for age at death based on Mantel-Haenszel procedure. RR_{MH} (95% CI) *Cardio-vascular diseases (ICD 410–12, 427–28)* 1955–1980: 3.2 (1.5; 7.3)

Hogstedt et al 1977		years 1955–1980 in Noraberg (8 000 habitants) n=440 deaths (217 cases, who died in cardio-cerebrovascular diseases, and 223 referents, subjects who died from alla other diseases)	types during 7-year periods 1958–1978 were calculated from a large number of semiannually measured short-time samples in the factory The air concentrations were estimated to have been in the range of 0.2–1.1 mg/m^3	classified as to the under-lying cause of death accoring to WHO classification (ICD 410–412, 427–428, 430–438)	1976–1980: 2.6 1955–1980: 2.7 (1.4; 5.4)	
Hooiveld et al 1998 [69] The Netherlands	Retrospective cohort study 1955–1991 Mean follow-up was 22.3 years Industry	Participants were male workers in a chemical industry involved in the synthesis and formulation of phenoxy herbicides In 1963, an uncontrolled reaction occurred in factory. An explosion followed and chemicals were released Contract workers, hired	**Phenoxy herbicides** An extensive company questionnaire was used for assessment of exposure. For each individual, the definition of exposure status was based on a detailed occupational history, including periods of employment in different departments and position held, as well as	**Death due to circulatory system, ischemic heart disease, cerebro-vascular disease and other heart disease** The diseases were defined according to ICD-9; circulatory system (390–459), ischemic heart disease (410–414), cerebrovascular disease (430–438) and other	Relative risks for causes of death for workers with medium and high TCDD* levels compared with workers with low TCDD levels, based on model-predicted TCDD levels for 1 031 male workers, 1955–1991. RR (95% CI) *TCDD: 2,3,7,8-tetrachlorodibenzo-p-dioxin **Circulatory system** Medium: 1.8 (1.0; 3.6) High: 2.3 (1.2; 4.3) **Ischemic heart diseases** Medium: 1.8 (0.8; 4.1) High: 3.1 (1.4; 6.5) **Cerebrovascular disease** Medium: 2.9 (0.8; 11.6) High: 1.5 (0.3; 7.1) **Other heart disease** Medium: 1.5 (0.2; 10.4) High: 0.7 (0.1; 7.9)	Relative risks for causes of death for workers with medium and high TCDD levels compared with workers with low TCDD levels, based on model-predicted TCDD levels for 1 031 male workers, 1955–1991. RR (95% CI) adjusted for age, calendar period at end of follow-up and time since first exposure/employment **Circulatory system** Medium: 1.5 (0.8; 2.8) High: 1.5 (0.8; 2.9) **Ischemic heart diseases** Medium: 1.5 (0.7; 3.6) High: 2.3 (1.0; 5.0) **Cerebrovascular disease** Medium: 2.0 (0.5; 8.2) High: 0.8 (0.2; 4.1) **Other heart disease**

		to assist in cleaning up after the accident, were included. Other contract workers were excluded n=1 031 All participants were men	exposed to an accident	heart disease (415–429)		Medium: 1.1 (0.2; 7.6) High: 0.4 (0.0; 4.9)
Ibfelt et al 2010 [70] Denmark	Prospective cohort study 19 years Industry 1987–2006	Participants were male metal workers, born before 1964. They were working with welding in 75 welding companies in Denmark (excluding shipyards). Participants were employed for a minimum of 1 year Participants were identified from the computerized files of the nationwide Danish Pension Fund The mean age was 42 years	**Particles** A questionnaire elicited information on the welding material used, the welding process, the first year of welding, number of years welding in various decades, use of exhaust ventilation and welding in confident spaces The total exposure of particles was calculated by extrapolation, on the assumption of a declining trend	**Cardio-vascular disease** Information on the occurrence of cardiovascular disease was retrieved by linkage to the Danish National Patient Registry by personal identification numbers Outcomes were coded according to ICD-8 and ICD-10: acute myocardial infarction (419 and I21, angina pectoris (413,	Adjusted hazard rate ratios for cardiovascular disease among welders according to accumulated exposure to particles. HRR (95% CI) adjusted for calendar year, tobacco smoking, alcohol consumption and use of hypertension or “heart” medicine **Acute myocardial infarction** ***Exposure to particles (mg/m³ x years)*** 0–10: 1.00 10–50: 1.11 (0.65; 1.89) 50–100: 1.43 (0.85; 2.41) >100: 1.03 (0.61; 1.74) **Angina pectoris** ***Exposure to particles (mg/m³ x years)*** 0–10: 1.00 10–50: 1.23 (0.73; 2.08) 50–100: 1.41 (0.84; 2.36) >100: 1.21 (0.72; 2.03) **Chronic ischemic heart disease** ***Exposure to particles (mg/m³ x years)*** 0–10: 1.00 10–50: 2.51 (1.15; 5.49) 50–100: 2.79 (1.29; 6.04) >100: 1.70 (0.78; 3.72)	–

		n=3 499 All participants were men	in exposure in all welding processes by use of an exposure matrix	I20), chronic ischemic heart disease (412, I25), cerebral infarct (433 and 434, I63)	**Cerebral infarct** ***Exposure to particles (mg/m³ x years)*** 0–10: 1.00 10–50: 1.32 (0.58; 3.01) 50–100: 1.17 (0.52; 2.67) >100: 1.54 (0.70; 3.39)	
Ilar et al 2014 [71] Sweden *Note*: Results from different years since exposure cessation is also available in the article *Note*: Partly the same population as in Gustavsson 2001	Case-control study (SHEEP) General working population 1992–1994	Participants were Swedish citizens aged 45–70 years who resided in Stockholm county during 1992–1994, and population controls 1 control per case, matched on gender, age and hospital attachment area was randomly selected from the study base within 2 days of the inclusion of a case. All controls were initially checked for myocardial infarction n=3 878 (1 643 cases and 2 235 controls) 1 309 women (538 cases and	**Motor exhaust** The exposure to motor exhaust was assessed via a job exposure matrix using elemental carbon to quantify the occupational exposure to motor exhaust The core of the particles in motor exhaust is elemental carbon to which organic compounds like PAHs attach, whereas the gas phase is a mixture of a very large number of chemical compounds including oxides of nitrogen, carbon dioxide, and carbon monoxide	**Myocardial infarction** Myocardial infarction was assessed by a method described in previous studies (Gustavsson et al, 2001 and Reuterwall et al, 1999)	Odds ratios of non–lethal myocardial infarction according to the highest average intensity to motor exhaust during at least 1 year of work. OR (95% CI) crude model **Motor exhaust** *Elemental carbon exposure (µg/m³)* Unexposed: 1.00 Ever exposed: 1.28 (1.08; 1.53) >0–21.9: 1.01 (0.77; 1.33) 22.0–42.0: 1.42 (1.10; 1.83) >42.0: 1.43 (1.10; 1.87) Test for trend p=0.002 Odds ratios of non–lethal myocardial infarction subdivided by cumulative exposure to motor exhaust at work *Cumulative elemental carbon exposure (µg/m³-years)* Unexposed: 1.00 >0–202: 1.08 (0.82; 1.41) >202–710: 1.38 (1.06; 1.79) >710: 1.40 (1.08; 1.83) Test for trend p=0.075	Odds ratios of non–lethal myocardial infarction according to the highest average intensity to motor exhaust during at least 1 year of work. OR (95% CI) adjusted for sex, age group, hospital catchment area and year of enrollment, smoking, alcohol drinking, diabetes mellitus, physical inactivity at leisure time, hypertension and overweight **Motor exhaust** *Elemental carbon exposure (µg/m³)* Unexposed: 1.00 Ever exposed: 1.09 (0.91; 1.32) >0–21.9: 0.93 (0.69; 1.25) 22.0–42.0: 1.14 (0.87; 1.50) >42.0: 1.21 (0.91; 1.59) Test for trend p=0.139 *Cumulative elemental carbon exposure (µg/m³-years)* Unexposed: 1.00 >0–202: 0.97 (0.73; 1.30) >202–710: 1.17 (0.89; 1.54) >710: 1.14 (0.86; 1.50)

		771 controls) and 2 569 men (1 105 cases and 1 464 controls)				
Jansson et al 2012 [72] Sweden	Cohort study (follow up of an earlier study by Gustavsson et al) Followed for mortality from 1952 through 2006	Participants were male Swedish chimney sweeps who were members of the national trade union n= 1 087 All participants were men	Chimney sweeps (proxy for exposure to particles, dust levels of 3–19 mg/m^3)	The cohort was linked to the nationwide Causes of Death Register to identify underlying causes of deaths Standardised mortality ratios (SMRs) were estimated using the Swedish male population as reference.	Standardised mortality ratios (SMRs) and 95% CIs for the total cohort of Swedish chimney sweeps and for those first employed after 31 December 1950. SMR (95% CI) Circulatory system disease Total: 1.18 (1.10; 1.27) First employed after 1950: 1.19 (1.00; 1.40) Ischemic heart disease Total: 1.20 (1.10; 1.32) First employed after 1950: 1.29 (1.04; 1.58) Cerebrovascular disease Total: 0.96 (0.78; 1.16) First employed after 1950: 1.21 (0.76; 1.83)	-
Jäppinen et al 1990 [73] Finland	Cohort study Based on the follow-up separate analyses were accomplished of those with a follow-up time of 5 years or less, 6–15 years, and more than 15 years	Participants were Finnish sulphite and sulphate mill workers who had been employed continuously for at least 1 year between 1945–1961	**Sulphur dioxide, hydrogen sulphide and organic sulphides** The basic source of the data was the company employment files	**Diseases of the circulatory system and ischaemic heart disease** National death rates were used for comparison and mortality was followed up until 1981	SMRs by duration of employment, referred to the population of Finland, in men exposed to hydrogen sulphide, organic sulphides and sulphur dioxide. SMR (95% CI) **Diseases of the circulatory system** *Hydrogen sulphide, organic sulphides* 1–4 years of employment: 216 (99; 410) >5 years of employment: 136 (91; 197) All: 150 (105; 206) *Sulphur dioxide* 1–4 years of employment: 164 (66; 339)	–

	Sulphite mills 1945–1981	Exposed to sulphur dioxide: 2 268 person-years Exposed to hydrogen sulphide and organic sulphides: 4 179 person-years All participants were men	1 cohort was formed of male workers who had had a higher than average exposure to sulphur dioxide within the sulphite mill. These were workers in the digesting, screening, washing, evaporation, and acid preparation departments Another cohort was formed of male workers who had had a higher than average exposure to hydrogen sulphide and organic sulphides within the sulphate mills. They worked in the digesting, washing, evaporation, and cooking liquor preparation departments	The personal data were confirmed from a national register and, when necessary, from local population registers. The cause of death was retrieved through the Central Statistical Office Deaths were classified by 8th revision of ICD: diseases of the circulatory system (codes 390–458), ischaemic heart disease (codes 410–414) The general Finnish population was used for reference	>5 years of employment: 112 (65; 179) All: 123 (79; 184) **Ischaemic heart disease** *Hydrogen sulphide, organic sulphides* 1–4 years of employment: 254 (102; 523) >5 years of employment: 129 (77; 205) All: 150 (97; 222) *Sulphur dioxide* 1–4 years of employment: 172 (56; 402) >5 years of employment: 136 (73; 233) All: 145 (86; 229) SMRs referred to the population of Finland in men exposed to hydrogen sulphide and organic sulphides >5 years and followed up for more than 15 years. SMR (95% CI) **Diseases of the circulatory system** *Hydrogen sulphide, organic sulphide* 173 (109; 262) **Ischaemic heart disease** *Hydrogen sulphide, organic sulphide* 162 (88; 272)	

Järup et al 1998 [74] Sweden	Cohort study Battery factory employed between 1940 and 1980, mortality followed up until 1992	Participants were workers employed for at least one year in the nickel-cadmium battery factory between 1931 and 1982 n=869 Original cohort n=900 183 women and 717 men	**Nickel hydroxide and cadmium oxide** A detailed description of the production history was compiled and provided the foundation for a consensus approach in which exposure concentrations were assigned to 23 generic job titles in three periods for cadmium and nickel exposure on two separate categorical scales. Quantitative estimates of breathing zone concentrations of cadmium and nickel for each category of the scales were made from personal and selected fixed point workroom monitoring data covering the period 1946–92. These estimates	**Ischemic heart disease and cerebro-vasular disease** Vital status and causes of death were obtained from the Swedish cause of death registry. Regional reference rates were used to compute the expected numbers of deaths All causes of death were recoded to the eighth revision of the international classification of diseases (ICD-8).	Standardized mortality rates in male battery workers (1951–92), regional reference rates, Kalmar county. SMR (95% CI) **Ischemic heart disease** Women: 75 (24.5; 176) Men: 116 (96.2; 140) **Cerebrovasular disease** Women: 134 (36.5; 176) Men: 78 (46.7; 121)	

			were linked to the combinations of generic job titles and periods to form a job-exposure matrix, which was applied to the individual work histories. The resulting individual exposure profiles for cadmium and nickel were used for the calculation of estimated cumulative exposures			
Järup et al 1989 [75] Sweden	Cohort study Smelter industry 1928 through 1967 (mortality was followed through 1981)	Participants were male Swedish smelter workers employed for at least 3 months from 1928 through 1967 n=3 916 All participants were men	**Arsenic** Arsenic levels in the air of all workplaces within the smelter were estimated for three different time periods. Using this exposure matrix and detailed information of the work history, cumulative arsenic	**Ischemic heart disease and cerebro-vascular disease** Standardized mortality ratios were calculated for several dose categories using age-specific mortality rates from the county where the	Cumulative arsenic exposure and risks of dying from ischemic heart disease or cerebrovascular disease among Ronnskar smelter workers. SMR (95% CI) Ischemic heart disease Cumulative arsenic exposure, mg x years/m^3 <25 : 100 (78; 127) 0.25-<1: 115 (86; 150) 1-<5: 98 (79; 119) 5-<15: 102 (80; 129) 15-<50: 113 (93; 136) 50-<100: 157 (94; 245) ≥100: 112 (68; 172) Total: 107 (97; 117) Cerebrovascular disease Cumulative arsenic exposure, mg x years/m^3 <25 : 108 (65; 168)	-

			exposure could be computed for each worker	smelter was situated The causes of death were obtained from the computerized files maintained by the Swedish National Central Bureau of Statistics. All causes of death were coded according to ICD 8	0.25-<1: 161 (97; 252) 1-<5: 93 (60; 138) 5-<15: 71 (40; 117) 15-<50: 122 (86; 168) 50-<100: 143 (46; 334) ≥100: 69 (19; 178) Total: 106 (88; 126)	
Kawachi et al 1997 [76] USA	Prospective cohort study. Data from the Nurses' Health Study 10 years Health care 1982–1992	Participants were nurses from the health study cohort established in 1976 when 121 700 female registered nurses 30–55 years of age completed mailed questionnaires requesting information	**Passive smoking** The passive smoking was assessed by a questionnaire. The questions are stated in the article. The questionnaires were mailed every 2 years to the cohort	**Coronary heart disease** End points comprised incidence cases of nonfatal myocardial infarction and fatal coronary heart disease occurring after the return to the 1982	Among women exposed to second- hand smoke only at work the multivariate relative risks of total coronary heart disease were assessed. OR (95% CI) **Second-hand smoke** Occasionally exposed: 1.49 (0.71; 3.14) Regularly exposed: 1.92 (0.88; 4.18)	–

		about risk factors for coronary heart disease and other diseases Age: 36–61 years n=32 046 All participants were women		questionnaire but before June 1, 1992 All women who reported having a nonfatal myocardial infarction were asked for permission to review medical records. Cases were confirmed if they met the diagnostic criteria of the World Health Organization. Medical records were reviewed by physicians who were blinded to exposer status		
Kazantis et al 1988 [77] Great Britain	Prospective cohort study Different occupations 1942–1984	A cohort of almost 7 000 male workers bom before 1940 and exposed to cadmium for more than 1 year between 1942–1970 was initially	**Cadmium** Jobs were classified according to the level of past cadmium exposure into the 3 groups high, medium and low, and the years at risk	**Hypertensive and cerebro-vascular disease** Deaths were coded by underlying and other causes according to the 8th	Cause-specific mortality in relation to cadmium exposure (1943–1984)., SMR (95% Cl) **Hypertensive disease** Ever high: 124 (3; 692) Ever medium: 168 (72; 331) Always low: 112 (77; 147) Total: 119 (85; 152) **Cerebrocavsular disease** Ever high: 42 (5; 151)	–

		followed with regard to mortality experience to the end of 1979 This cohort study was updated for a further 5 years to include all deaths to the end of 1984	were divided on the basis of these categories into 3 groups	revision of ICD. Expected numbers were calculated from mortality rates for the population of England and Wales corrected to the 8th revision ICD codes, and regional variation in mortality was taken into account	Ever medium: 79 (50; 119) Always low: 78 (66; 90) Total: 77 (66; 89)	
Keil et al 2016 [78] USA	Cohort Copper smelter 1938–1990	Study population is a cohort of workers from a Participantes were white, male individuals who worked at least 1 year at acopper smelting facility in Anaconda, Montana between 1938 and 1956 The median age at entry was 32, and the median time of work	**Arsenic** Exposure to arsenic was quantified using work area measurements from using a series of 702 measurements of airborne arsenic trioxide (As_2O_3) made between 1943 and 1958. The measurements were used to estimate a timeweighted airborne concentration for each work	**Heart disease** Causes of death were classified according to the International Classification of Disease (ICD) codes as assigned to the underlying cause of death noted on death certificates	The authors estimate that eliminating arsenic exposure at work would have prevented 7.2 (95% CI: –1.2, 15) deaths due to heart disease by age 70 per 1 000 workers	–

		prior to study entry was 1 year n=8 014 All participants were men	area corresponding to 0.29 mg/m^3 (light) 0.58 mg/m^3 (medium) and 11.4 mg/m^3 (heavy). Authors created a quantitative exposure metric in mg/m^3 -years as the product of the duration of work and the airborne concentration in each area The observed median cumulative arsenic exposure across all person time in the study was 1.7 mg/m^3 -years (interquartile range: 0.87, 4.1). The predicted exposure under the "natural course" intervention corresponded well with the observed data			
Ketchum et al 2005	Prospective cohort study	Participants were veterans	**Herbicides**	**Circulatory disease**	Relative risk for circulatory disease mortality, followed-up until December 1999, for Vietman	–

[79] Vietnam	Herbicides spraying in military service 1962–1999	of Operation Ranch Hand, the unit responsible for aerially spraying herbicides in Vietnam in 1962–1971 The comparison population consisted of of Air Force veterans in the same calendar period that the Ranch Hand unit was active in Vietnam. Comparison veterans were stationed throughout Southeast Asia, were not involved with spraying herbicides, and were demo-graphically similar to Ranch Hand veterans n=20 340 (exposed: 1 262 and unexposed: 19 078) Gender not stated in the article	Dioxin levels were measured in serum collected from veterans who completed a physical examination in 1987. Additional measurements were made in 1992 and 1997 Each veteran was assigned to 1 of 4 dioxin exposure categories (comparison, background, low, and high) based on his cohort (Ranch Hand or comparison), dioxin concentration, and half-life-extrapolated initial dioxin concentration	Underlying causes of death were coded in accordance with the rules and conventions of ICD-9	veterans exposed to herbicides compared with an unexposed cohort between 1962–1971. RR (95% CI) adjusted for for military occupation, year of birth, smoking history, and family history of heart disease ***Circulatory diseases (by exposure category)*** Total: 1.3 (1.0; 1.6) Background: 0.8 (0.4; 1.8) Low: 1.8 (0.9; 3.5) High: 1.5 (0.7; 3.3) ***Circulatory diseases (by disease category)**** Atherosclerotic heart disease: 1.7 (1.1; 2.5) Cardiomyopathy: 0.5 (0.1; 3.8) Cerebrovascular disease: 2.3 (0.9; 6.0) Hypertensive disease: 2.5 (0.6; 10.8) Other circulatory diseases: 2.4 (0.8; 6.7) Total: 1.7 (1.2; 2.4) *Only participants enlisted as ground crew (n=11 311)	

Kobal et al 2004 [80] Slovenia	Cohort study Mercury mining 1959–2000	Participants were miners (both active and retired) not exposed to mercury for at least 8 months The controls were workers with no occupational exposure to mercury n=112 (54 miners and 58 controls) All participants were men	**Mercury (Hg^0)** Environmental data was collected from 1959–2000 from workload records and daily reports on Hg^0 measurements in the workplace. For exposure records, duration and level of exposure were calculated for each miner following environmental indicators of past exposure	**Blood pressure** A medical examination included a medical history and an evaluation of some behavioural and biological risk factors for cardiovascular disease	Difference between miners and controls. p-values are reported Systolic blood pressure: $p<0.01$ Diastolic blood pressure: $p<0.01$ The mean values of systolic and diastolic blood pressure were significantly higher among miners compared to the controls, but no significant difference between the prevalence of hypertension (blood pressure over 140/90) was found	–
Koskela et al 1994 [81] Finland	Prospective cohort study 15 years Industry 1950–1987	Participants were men hired in 1950–1972 by 20 iron, steel and nonferrous foundries (included in the mortality study of Finnish foundry worker in 1973) Age of the participants were not stated in the article n=2 857	**Carbon monoxide (CO) exposure** Carbon monoxide exposure was measured in 1972 in 52 iron, 10 steel, and 5 copper alloy foundries. The hygienic standard for CO was exceeded in the air and in blood COHb content of the workers. On the	**Medication for hypertension** The vital status of the cohort members was traced through the Population Data Register Data on specially compensated medication was obtained from the	The age–standardized incidence rate for compensated medication for hypertension was 4.7 for the workers whose exposure time was less than 1 year and who were not exposed to CO and 9.4 for those who were regularly exposed to CO for at least 5 years. RR (95% CI): 2.0 (1.28; 2.92) Among the iron foundry workers the corresponding rates were 4.7 and 9.9. RR (95% CI): 2.1 (1.24; 3.38)	–

		All participants were men	basis of CO measurements the foundry occupations were classified into categories	Social Insurance Institution		
Koskela et al 2000 [83] Finland *Note: This cohort is a subcohort from Koskela 1994*	Prospective cohort study 20 years Foundry workers 1973–1993	Participants were male foundry workers in Finland who participated in a health examination in 1973. They were hired in 1950–1972 and were still actively working in the foundries n=931 All participants were men	**Carbon monoxide** In 1972, CO levels were measured in 52 iron, 10 steel, and 5 nonferrous foundries On the basis of these CO measurements, the foundry occupations were divided into 3 categories: 1) regular CO exposure: casters, furnacemen, and knockout men. 2) occasional or slight CO exposure: fettlers, truck drivers, crane operators, and loader drivers; and. 3) no CO exposure: floor molders,	**Ischemic heart disease mortality** Participants completed a questionnaire on symptoms and diagnosed diseases, including a history of chest pain and cardiovascular diseases diagnosed by a physician. Blood pressure measurement was included in the health examination, as was a 12-lead resting ECG Vital status was traced through a population information system. Causes of	Risk of mortality from ischemic heart disease. RR (95% CI) *Carbon monoxide exposure* Regular: 2.15 (1.00; 4.63) Occasional: 1.80 (0.91; 3.57) Indicative myocardial infarction, RR *Carbon monoxide exposure* Regular: 1.87 (not significant) Occasional: 1.84 (not significant) Suggestive coronary heart disease, RR *Carbon monoxide exposure* Regular: 2.00 (not significant) Occasional: 1.89 (not significant)	–

			machine molders, coremakers, ingot casters, and other workers To analyze the data, the lifetime time-weighted average of the 3 CO exposure scores obtained until the end of 1992 from the questionnaire was used to define the main exposure category for each worker	death categorized according to the ICD-8th, Revisions were acquired from Statistics Finland, and codes 410.00–414.99 were classified ischemic heart disease Morbidity from cardiovascular diseases during follow-up was measured with the use of medication, for which special compensation is granted by the national sickness insurance law (coronary heart disease, cardiac insufficiency, cardiac arrhythmia, and hypertension)		

Koskela et al 1990 [82] Finland *Note: The study cohort is also, included in the articles Koskela et al 2000 and Koskela et al 2005*	Prospective cohort Cotton mill 1950–1985	The population comprised all women exposed to raw cotton who had been hired between 1950–1971 by the 5 Finnish cotton mills. The minimum exposure period was 5 years Comparison group consisited of a similarly selected group of 398 female paper box assembly workers not exposed to dust The cohort was also compared with national registers on death, disability, and free medication for chronic diseases n=1 463 (1 065 exposed, 398 nonexposed) All participants were women	**Dust** A survey of dust concentrations in the 5 cotton mills had been performed in 1972. The mean dust concentrations were 2 mg/m^3 for bale opening, 33 mg/m^3 for carding, 1–8 mg/m^3 for spinning, and 2 mg/m^3 for the winding phases of work About 80% of the subjects worked under conditions where the dust concentrations were higher than the Finnish hygienic standard (1 mg/m^3). The median exposure time was 28–0 years (range 5–38) for living workers and 22.4 years (range 5–29) for workers who had died	**Cardio-vascular diseases** The causes of death were ascertained from death certificates according to ICD-8 The death certificates were obtained from the Central Statistical Office of Finland. The causes of disability and free medication under the national sickness insurance law were obtained from the Finnish Social Insurance Institution	SMR for the cohort of cotton mill workers at the end of 1985. The period of entry was defined as 1950–1971. SMR Cardiovascular diseases: 77, n.s Ischaemic heart disease: 74, n.s Cerebrovascular disease: 91, n.s The numbers of certain diseases with the entitlement to free medication from national sickness insurance on 31 December 1981 compare with the national age specific rates for the same date *Hypertension*: n.s Odds ratios and significances of the differences in the occurrence of diagnosed diseases reported on the questionnaire by the cotton mill workers and by the nonexposed comparison group. OR Heart diseases: 1.0, n.s Cerebrovascular diseases: 1.3, n.s Hypertension: 0.8, n.s	–

Koskela et al 2005 [84] Finland *Note: This study comprises several study cohorts, including same cohort as in Koskela et al 1990 and 1 in Koskela et al. 2000*	Prospective cohort Different industries 1940–1992	The population comprised a sample of 6 022 current and former workers from a total of 22 000 members of 6 cohorts variously exposed to dust (granite workers, foundry workers, cotton mill workers, iron foundry workers, metal product workers, and electrical workers) All cohorts were followed until the end of 1992 n=6 022 Participants were men and women	**Dust** Dust exposure was defined as exposure years multiplied by dust concentration 10 mg-y/m^3 or dust category, or as exposure years only Workers were divided into high and low exposure and groups Granite workers: high ≥20 mg/m^3; low <20 mg/m^3 Cotton mill workers: high >10 mg/m^3; low ≤10 mg/m^3 Foundry workers: high comprised floor and machine moulders and fettlers and the labourers assisting them; low comprised core makers, furnace men,	**Cardio-vascular diseases and ischaemic heart disease** The vital statuses and addresses of the workers were traced through the Population Information System Causes of death were obtained from Statistics Finland. Causes of disability were available from the Social Insurance Institution. Cardio-vascular diseases was available from the death and disability registers. Data on medicines for which special compensation is granted under the National	Age adjusted incidence rates per 100 000 person-years for cardiovascular diseases, high vs low dust exposure categories. Rate ratio (95% CI) adjusted for age *All cardiovascular diseases* Granite workers: 1.1 (0.89; 1.29) Cotton mill workers: 1.2 (0.68; 2.25) Foundry workers: 1.0 (0.79; 1.14) Metal workers: 1.2 (1.04; 1.35) *Ischaemic heart disease* Granite workers: 1.2 (0.94; 1.58) Cotton mill workers: 1.9 (0.47; 7.45) Foundry workers: 0.9 (0.71; 1.16) Metal workers: 1.4 (1.19; 1.74)	–

			casters, truck drivers, others Metal workers: high comprised iron foundry workers; low comprised metal product and electrical workers	Sickness Insurance Act from the Social Insurance Institution. The diseases were coded according to the ICD-8		
Kotseva 2001 [85] Bulgaria	Case–control study Viscose rayon plant	The participants consisted of workers exposed to carbon disulfide, with a minimum of 1 year's work in a viscose rayon plant The reference group was age and gender-matched plastic industry workers without occupational contact with noxious chemicals n=282 (141 exposed, 141 nonexposed) Participants were men och women	**Carbon disulfide (CS_2)** Concentrations of CS_2 were assessed using stationary measurements and personal sampling methods. Personal breathing zone samples from some workers within each job category were collected with NIOSH type 100/50 mg charocoal absorption tubes at a flow rate of up to 50ml/min using calibrated Gilian low-flow sampling pumps. Charcoal samples were	**Hypertension and coronary heart disease** Blood preassure measuremnts and routine ECG at rest were performed. Hypertension was deter-mined according to the classification of the American Heart Association. Arterial. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood	Prevalence odds ratios of qualitative cardiovascular outcomes versus the degree of exposure to carbon disulfide. OR (95% CI) *Hypertension* CS_2 index <100 vs controls: 0.96 (0.38; 2.41) CS_2 index ≥100 vs controls: 1.47 (0.64; 3.36) All exposed vs controls: 1.21 (0.60; 2.47) *Coronary heart disease* CS_2 index <100 vs controls: 0.98 (0.38; 2.52) CS_2 index ≥100 vs controls: 1.73 (0.75; 4.01) All exposed vs controls: 1.34 (0.64; 2.77)	–

			desorbed with a toluene and analysed by a gas chromate-graphy according to NIOSH method	pressure ≥90 mmHg The probability of coronary heart disease was determined by means of the WHO standardized cardio-vascular questionnaire and the ECGs were coded separately by 2 trained physicans		
Kreuzer et al 2015 [86] Germany	Retrospective cohort study 62 years Industry 1946–2008	Participants were former employees of a uranium mining company in East Germany, who had worked for at least 6 months during 1946–1990. The cohort include workers from different types of work places (under-ground, open pit, surface and milling) n=4 054	**External radiation exposure** To determine the silica dust, a comprehensive job exposure matrix was used. The matrix assigned an average annual exposure value to each facility, work place and job type	**Cardio-vascular diseases** Information on the underlying cause of death, coded according the International Classification of Disease (ICD-10), was based on death certificates from the Public Health offices and their archives and the	Excess relative risk estimates per cumulative silica dust exposure (mg/m^3-years). ERR (95% CI) **Cardiovascular disease (I00–I99)** Silica dust:–0.0017 (–0.014; 0.011), p<0.05 **Ischemic heart disease (I20–I25)** Silica dust: 0.0012 (–0.018; 0.021), p<0.05 **Cerebrovascular disease (I60–I69)** Silica dust: 0.0035 (–0.025; 0.032), p<0.05 *Note*: point estimate data has been updated compared to the data stated in the article, after personal correspondence with M Kreuzer	–

		All participants were men		autopsy files from the local pathology archive		
Laden et al 2007 [87] USA	Prospective cohort Trucking industry 1985–2000	Participants were unionized trucking industry employees from 4 national trucking companies who had worked for at least 1 day in 1985 n=36 299 All participants were men	**Fine particulate air pollution (particulate matter from vehicle exhausts)** Detailed work history information for all employees were obtained. Information was available on each individual included date of hire, last date of work, and daily job title and terminal Participants were grouped into different groups based on job titles, duties, and job location in the unionized trucking industry	**Circulatory system disease, ischemic heart disease and cerebro-vascular disease** Vital status, date of death, and cause-specific mortality from 1985–2000 was obtained through searching the National Death Index Expected numbers of all and cause-specific deaths were calculated stratifying by race, 10-year age group, and calendar period using U.S. national reference rates	Cause-specific mortality in the Trucking industry particle study cohort, 1985–2000. SMR (95% CI) *Circulatory system diseases (ICD-9: 390–459)* Total: 0.73 (0.69; 0.76) *Ischemic heart disease (ICD-9: 410–414)* Total: 1.41 (1.33; 1.49) All drivers: 1.49 (1.40; 1.59) Dockworker: 1.32 (1.15; 1.52) Shop workers: 1.34 (1.05; 1.72) *Cerebrovascular disease (ICD-9: 430–438)* Total: 0.69 (0.59; 0.80)	–

Landen et al 2011 [88] USA *Note*: additional data on coal region is available in the article	Prospective cohort study 14 years Coal mines 1969–1993 Baseline 1969–1971	Participants were under-ground coal miners from 31 coal mines in the USA Participants who had missing or invalid data on vital status, smoking or coal dust exposure were excluded n=9 971 No information was given on participants´ gender	**Coal dust exposure** Using a job exposure matrix, the time worked in each job title was multiplied by job title specific exposure estimate Job title specific estimate based on dust samples collected in US mines between year 1969–1971	**Ischemic heart disease mortality** Vital status was obtained through the social security administratio n files of the united mine workers of America welfare and retirement fund, state vital statistics offices and the national death index database Deaths categorized as ischemic heart disease were ICD-8th and 9th revision, code 410–414	Cox proportional hazard ratio for ischemic heart disease mortality by quartiles of dust exposure. HR (95% CI) adjusted for age, body mass index and smoking **Dust exposure (mg-year/m^3)** ≤20: 1.0 20.1–63.5: 1.58 (1.09; 2.30) 63.6–97.4: 1.81 (1.23; 2.66) >97.4: 1.92 (1.29; 2.86) Coal particulate is mechanically generated and particle size distribution within PM_{10} would tend toward larger particles	–
Lanes et al 1990 [89] USA	Prospective cohort study Cellulose fiber production plant 1954–1986	Participants were employees at a cellulose fiber production plant employed in the preparation or extrusion areas for at least 3 months	**Methylene chloride** An industrial hygiene survey conducted in 1977 reported 8h time-weighted average exposures ranging from below	**Hypertension, cerebro-vascular disease, and ischemic heart disease mortality** Vital status of the cohort for the period 1954–1986 was	Cause-specific mortality in cellulose fiber production workers in 1986. The period of exposure was defined as 1954–1977. SMR (95% CI) *Hypertension without heart disease* 3.20 (0.54; 10.66) *Cerebrovascular disease* 0.56 (0.21; 1.25) *Ischemic heart disease*	–

		between 1954–1977 The mortality of the cohort was compared with mortality rates the population of York County, South Carolina n=1 271 720 women 551 men	detectable limits up to 1 700 ppm for methylene chloride, 1 600 ppm for aceton, and 140 ppm for methanol among the workers in the extrusion and preparation areas	ascertained by comparing the cohort roster with decedent records of the plant, the national death index, and the Social Security Admini-stration A nosologist reviewed the death certificates and assigned codes to the underlying cause of death and contributing causes of death in accordance with the ICD-9 revision	0.90 (0.62; 1.27)	
Langseth et al 2006 [90] Norway	Cohort study Pulp and paper mill 1951–2000	Participants were women first employed between 1920–93 n=3 143 women	**Paper dust** Information about each cohort member was obtained from personnel record files in the mills in order to identify employment	**Several** Data on cause and date of death were added by linkage to the Cause of Death Register using unique personal identification	Standardised mortality ratios for selected causes of death in female pulp and paper workers in Norway by duration of employment. SMR (95% CI) **All cardiovascular diseases** *Pulp and paper workers* < 3 years employment: 1.33 (1.00; 1.74) ≥3 years employment: 1.14 (1.01; 1.29) Total: 1.17 (1.05; 1.30)	

			periods and job categories. Exposure to microorganisms and endotoxins may be related to respiratory symptoms38 and both have been considered as a potential exposure in paper mills.	numbers. The follow-up period was 1951–2000. Standardised mortality ratios (SMRs) with 95% confidence intervals (95% CIs) were calculated using the national female mortality rates as reference.	*Paper department workers* < 3 years employment: 1.36 (0.96; 1.88) ≥3 years employment: 1.15(0.97; 1.37) **Ischaemic heart disease** *Pulp and paper workers* < 3 years employment: 1.73 (1.18; 2.44) ≥3 years employment: 1.12 (0.94; 1.35) Total: 1.22 (1.03; 1.43) *Paper department workers* < 3 years employment: 1.94 (1.24; 2.89) ≥3 years employment: 1.21 (0.93; 1.56) **Cerebrovascular diseases** *Pulp and paper workers* < 3 years employment: 0.83 (0.40; 1.52) ≥3 years employment: 1.22 (0.97; 1.51) Total: 1.16 (0.94; 1.42)	
Laplanche et al 1992 [91] France	Cohort study with age-matched control group 7 years follow-up Industry 1981–1988	Participants were workers exposed to vinyl chloride and non-exposed controls matched for age (to 2 years), plant, and physician The exposed group consisted of 40–55 year old employees exposed to vinyl chloride at the time of inclusion in 1980–1981 or previously	**Vinyl chloride** Interviews for initial data collection were conducted by the physician of each plant over a 1 year period	**Cardio-vascular disease** (except Raynaud's disease) ICD-9 codes 390–459 were applied The subjects were followed up yearly for 7 years from the time of inclusion until December 1988 for vital status and health and occupational	Relation between exposure to vinyl chloride and occurrence of cardiovascular disease (except Raynaud's disease) during the 7 year follow-up. RR (95% CI) adjusted for age, duration of activity as a blue collar worker, foreign origin, marital state, place of residence, paternal or maternal history of cancer, cardiovascular, respiratory, or other disease, smoking habits, subject considered ethylic by the plant physician, and alcohol consumption Exposed in relation to 1976 Non-exposed: 1.0 After: 0.8 (0.4; 1.9) <10 y before: 1.3 (0.9; 1.9) ≥10 y before: 1.5 (1.1; 2.1)	–

		Controls were employees who had never been exposed to vinyl chloride n=2 200 Gender not stated		state. Causes of death as well as pathological findings were coded according to the 9th revision of the International Classification of Diseases (ICD-9).		
Larson et al 2010 [92] USA	Prospective cohort study Vermiculite mining and processing 1920–2006	Participants were vermiculite workers exposed to *Libby amphibole* some time between 1920–1990. Data derived from the Agency for toxic subsetances and disease registry Multiple cause death rates for the U.S. 1960–2002 population were used as reference n=1 862 Gender not stated	**Cumulative fiber exposure** Historical air sampling data were used to estimate the 8-hour time-weighted average fiber exposure for all areas of the vermiculite operation for various time periods in the company's history. The proportion of each day spent at each location was calculated for each job title, and an 8-hour time-weighted average exposure was estimated for	**Mortality to heart diseases, diseases of the circulatory system, and hypertension without heart disease** The National Death Index was used to determine vital status of each worker and death certificates were acquired for 80% of the desceased workers Immediate and under-lying causes of deaths were coded	Cause-specific mortality in the Libby Vermiculite worker cohort in December 2006. SMR (95% CI) *Heart diseases* 0.9 (0.9; 1.0) *Ischemic heart disease* 0.7 (0.6; 0.8) *Other heart diseases* 1.5 (1.2; 1.8) *Diseases of the circulatory system* 1.4 (1.2; 1.6) *Hypertension without heart disease* 1.7 (1.2; 2.4) Estimated RR (95% CI) for the effect of cumulative fiber exposure with 20 years lag (fibers/ml-years) *Cardiovascular disease* <1.4: 1.0 1.4–<8.6: 1.3 (1.0; 1.6) 8.6–<44.0: 1.3 (1.0; 1.6) ≥44.0: 1.5 (1.1; 2.0)	

			each job held by the worker by length of time (in years) spent at that job Finally, lifetime cumulative fiber exposure for each worker was obtained by summing the cumulative fiber exposure for each job that worker held	by a certified nosologist using ICD-9. For workers whome we were unable to obtain a death certificate, the researchers relied on National Death Index codoed causes of death	Model p-value 0.0067	
Levine et al 1986 [93] USA	Cohort study Ammunition plants 1951–1980	Participants derived from 2 different plants, 1 located in Joliet and 1 in Radford. All men with high to moderate levels of exposure to dinitrotoluene during 1950s were enrolled in the study cohort Expected deaths were determined by applying age- and calendar year-specific mortality rates for US white	**Dinitrotoluene (DNT) exposure** Batch processes had been used in boths plants, and there were considerable potential for exposure to the skin and respiratory tract. Exposure magnitude and duration over a typical work shift (exposure intensity) were estimated using data derived from process records and the assessments of persons familiar with the	**All cause-mortality** Vital status of cohort members were ascertained using a variety of sources: plant personnel records, the Social Security Administratio n, the Veterans Administratio n, The National Death index, state registrars, and telephone surveys.	Cause-specific mortality by years since entry into cohorts. SMR, p-value **Joliet** *Circulatory system* 0–15 year since entry: 0, n.s >15 year since entry: 175, p≤0.05 *Chronic rheumatic heart disease* 0–15 year since entry: - >15 yeaer since entry: - *Ischemic heart disease* 0–15 year since entry: - >15 year since entry: 178, n.s *Cerebrovascular disease* 0–15 year since entry: - >15 year since entry: *Residual circulatory system* 0–15 year since entry: - >15 year since entry: -	–

		males to the person-years of observation accumulated by the cohorts through Dec 31, 1980 n=457 (156 at Joliet and 301 Radford) All participants were men	operations. DNT-related jobs at Joliet- those on the DNT production lines- were judged by the authors to afford high levels of of DNT exposure. Jobs at Ratford were categorized by plnt technical personnel according to opportunity for exposure: high, moderate, low, or none	Underlying and contributed causes of death were coded by a trained nosologist, according to the ICD-8	**Radford** *Circulatory system* 0–15 year since entry: 99, n.s >15 year since entry: 150, p≤0.001 *Chronic rheumatic heart disease* 0–15 year since entry: - >15 year since entry: - *Ischemic heart disease* 0–15 year since entry: 93, n.s >15 year since entry: 149, p≤0.01 *Cerebrovascular disease* 0–15 year since entry: - >15 year since entry: 78, n.s *Residual circulatory system* 0–15 year since entry: - >15 year since entry: 206, p≤0.05 Mortality from ischemic heart disease more than 15 years following cohort entry by exposure duration and intensity (both cohorts). SMR, p-value *Ischemic heart disease* Exposure duration ≤5 months High intensity: 131, n.s Only high intensity: 135, n.s Mixed intensity: - Total intensity: 103 Exposure duration >5 months High intensity: 224, p≤0.05 Only high intensity: 205, p≤0.05 Mixed intensity: 153, p≤0.05 Total intensity: 168, p≤0.01 Total exposure duration High intensity: 178, n.s	

					Only high intensity: 175, p≤0.05 Mixed intensity: 140, n.s Total intensity: 154, p≤0.01	
Liddell et al 1980 [94] Canada *Note:* same population as in article by McDonald et al	Cohort study Chrysotile mines 1966–1975	Participants were men born 1891–1920 and employed for at least 1 month in the chrysotile mines and mills of Quebec 2 study cohorts were set up. Cohort 1 was a subset of the mortality cohort of all 11 379 subjects, born 1891–1920, who had worked for at least a month in the Quebec asbestos production industry. Only men qualified for the present subset, and they had to have had at least 1 chest radiograph taken while still employed in the industry	**Asbestos** Data on the dust exposure accumulated during the gross service was available (see also article by McDonald for more information about measurements)	**Heart disease and cerebro-vascular disease, mortality** ICD-8 codes 400–443 were applied for heart disase and codes 330–334 for cerebrovascular diseases The numbers expected on the basis of male mortality in Quebec	Deaths from specific causes related to dust exposure. Risks relative to normals, or to unexposed, and χ2 statistics **Diseases of heart** All workers SMR: 1.36 Normal radiograph SMR: 1.20 Less than normal SMR: 1.83 RR:1.53 χ2: 25.25 Cause-specific mortality in cohort I in relation to radiological abnormality **Cerebrovascular diseases** All workers SMR: 1.44 Normal radiograph SMR: 1.39 Less than normal 1.62 RR:1.17 χ2:0.59	–

		Cohort 2 was selected from the 1 015 men still employed in November 1966 who had been the subjects in cross-sectional studies of respiratory symptoms and function and had had their 1966 routine chest radiograph assessed by 6 readers n=4 559 All participants were men				
Liu et al 2014 [95] China	Prospective cohort Average follow-up was 35 years Mines and factories 1960–2003	Participants were workers at 29 Chinese metal mines and pottery factories who had worked for 1 year or more between 1960–1974 The cohort was retrospectively followed to 1960 and prospectively followed to 2003	**Crystalline silica** Occupational dust monitoring data were used to create a job-exposure matrix that included facility-, job-, and year-specific crystalline silica concentrations By linking the job-exposure matrix and work history, the researchers	**Heart disease mortality** Trained local occupational physicians traced the vital status during the follow-up. Underlying causes of death (99% complete) were obtained from local hospital records, employment	Association between crystalline silica exposure and mortality from heart disease among the entire cohort quartile of cumulative silica exposure (defined as mg/m^3-years according to exposure distribution among silica-exposed subjects) with unexposed subjects as a reference. HR (95% CI) *Pulmonary heart disease* 0.01–0.75: 0.92 (0.67; 1.26) 0.76–1.84: 1.39 (1.08; 1.79) 1.85–5.37: 2.47 (2.01; 3.03) >5.37: 5.46 (4.52; 6.61) *p-trend:* <0.001 *Ischemic heart disease* 0.01–0.75: 1.02 (0.76; 1.38) 0.76–1.84: 1.41 (1.09; 1.83)	–

		n=42 572 total, 85% men n=15 092 not exposed, 71% men n=8 633 with lifetime highest silica exposure of ≤0.1 mg/m^3, 95% men	defined the lifetime highest silica exposure (mg/m^3) for each worker as the highest silica concentration among all job titles Cumulative silica exposure for each worker (mg/m^3-years) was calculated	information, or oral reports from colleagues or next-of-kin The ICD-10 was used to classify the causes of death. Causes of deaths was divided into those resulting from heart disease (ICD-10 codes: I00–I09, I11, I13, and I20–I51), pulmonary heart disease (I26, I27), ischemic heart disease (I20–I25), hypertensive heart disease (I11), and other heart disease (I00–I09, I13, I28–I51)	1.85–5.37: 1.03 (0.78; 1.35) >5.37: 0.70 (0.51; 0.96) p-trend: 0.002 *Hypertensive heart disease* 0.01–0.75: 0.93 (0.60; 1.43) 0.76–1.84: 0.69 (0.47; 1.04) 1.85–5.37: 0.81 (0.59; 1.11) >5.37: 0.77 (0.57; 1.05) p-trend: 0.01 Association between a subcohort of cumulative crystalline silica exposed subjects with a lifetime highest silica exposure of >0.1 mg/m^3 and mortality, with unexposed subjects as a reference. HR (95% CI) *Pulmonary heart disease* 0.04–1.73: 1.24 (0.95; 1.63) 1.74–3.80: 2.03 (1.61; 2.55) 3.81–7.06: 3.77 (3.06; 4.65) >7.07: 5.80 (4.75; 7.07) *p-trend:* <0.001 *Ischemic heart disease* 0.04–1.73: 1.18 (0.87; 1.59) 1.74–3.80: 1.05 (0.78; 1.43) 3.81–7.06: 0.90 (0.65; 1.24) >7.07: 0.61 (0.42; 0.89) *p-trend:* <0.006 negative trend *Silicosis* 0.04–1.73: 1.00 1.74–3.80: 6.31 (4.86; 8.21) 3.81–7.06: 15.17 (11.80;19.52) >7.07: 19.30 (14.90; 24.99) *p-trend:* <0.001 Association between a subcohort of cumulative crystalline silica exposed subjects with a lifetime highest silica exposure of ≤0.1	

					mg/m^3 and mortality, with unexposed subjects as a reference. HR (95% CI) *Pulmonary heart disease* 0.01–0.33: 1.46 (0.83; 2.58) 0.34–0.55: 1.51 (0.80; 2.86) 0.56–0.87: 1.29 (0.72; 2.29) >0.87: 2.40 (1.51; 3.83) *p-trend:* <0.001 *Ischemic heart disease* 0.01–0.33: 1.04 (0.63; 1.72) 0.34–0.55: 1.13 (0.68; 1.90) 0.56–0.87: 1.52 (1.02; 2.27) >0.87: 1.60 (1.07; 2.40) *p-trend:* <0.001 *Silicosis* 0.01–0.33: 1.00 0.34–0.55: 0.43 (0.11; 1.65) 0.56–0.87: 1.12 (0.44; 2.86) >0.87: 1.60 (0.64; 4.01) *p-trend:* 0.04	
Lundstrom et al 1997 [96] Sweden	Prospective cohort study Copper–lead smelter 1928–1987	Participants were male blue-collar workers, first employed for at least 3 months during the period 1928–1979 at a smelter in northern Sweden. This cohort had been monitored for their blood lead concentration (B-Pb) since 1950 and had	**Lead exposure** The blood-lead level (B-Pb) was analyzed by emission spectrometry from 1950–1969, and since 1967 atomic absorption has been used. The annual mean (arithmetic) B-Pb value of the lead smelter workers was 3.0 µmol/l in 1950 and	**Cause-specific mortality** Information about mortality in 1955–1987 was gathered from the Cause-of-Death Register at Statistics Sweden. The death certificates were coded according to the 8th	Mortality in the total cohort of lead-exposed workers and in the highest exposed subgroup in comparison with the county population, during the follow-up period of 1955–1987. SMR (95% CI) *Cardiovascular diseases (ICD-8: 390–458)* Total cohort: 0.9 (0.8; 1.0) Highest exposed: 0.8 (0.7; 1.0) *Ischemic heart diseases (ICD-8: 410–414)* Total cohort: 0.8 (0.7; 1.0) Highest exposed: 0.7 (0.6; 0.9) *Cerebrovascular diseases (ICD-8: 430–438)* Total cohort: 0.8 (0.6; 1.2) Highest exposed: 0.9 (0.5; 1.4)	–

		mainly been employed in the lead-exposed departments n=3 979 All participants were male	approximately 1.6 µmol/l in 1987	revision of the International Classification of Diseases (ICD-8) The expected mortality for the period 1955–1987 was calculated with the use of moltality rates specific for calendar year, cause, gender, and 5-year age groups in the county population		
MacMahon et al 1988 [97] USA	Prospective cohort study Rayon industry 1957–1983	Participants were white men exposed to carbon disulfide in the US rayon industry, employed in shift-work for a 12-month period between 1957–1979 The cohort was compared with the US population, matched for gender, age and study period	**Carbon disulfide** A list of job titles was prepared by a knowledgeable person and each job title was assigned to 1 of 5 categories of probalble exposure. Each job title held by each individual was so coded and an estimated duration of	**Mortality to circulatory diseases, artero-sclerotic heart disease, and cerebro-vascular disease** Deaths to mid-1983 were ascertained by the Social Security Administratio n and the National Death Index.	Mortality in the total cohort of carbon disulfide-exposed workers through July 1, 1983. SMR (95% CI) *All circulatory diseases* 104 (98; 110) *Arteiosclerotic heart disease* 104 (97; 112) *Cerebrovascular disease* 108 (90; 128) Mortality in the total cohort of workers by level of exposure to carbon disulfide. SMR, p-value (differs from 100) *All circulatory diseases* Most exposed: 114, p<0.05	–

		n=10 418 All participants were men	exposure level was computed for each individual. This classification by exposure level cannot be interpreted as indicative of the actual exposure of any person, only as a ranking of the general level of exposure likely to be experienced by the class of individuals with similar job title relative to other classes	The underlying causes of death were coded according to the ICD-8	Least exposed: 88, p<0.05 No exposure: 119, p<0.05 *Arteiosclerotic heart disease* Most exposed: 124, p<0.01 Least exposed: 85, p<0.05 No exposure: 108, n.s *Cerebrovascular disease* Most exposed: 103, n.s Least exposed: 89 No exposure: 149	
Malcolm et al 1982 [98] Great Britain	Cohort study Lead acid battery companies 1925–1976	Participants were workers from 4 lead acid battery companies Participants represented 13 865 men years experience as pensioners The 2 largest companies also had secondary lead smelting operations	**Lead** Over the years, various methods of monitoring the health and absorption of lead was used Before 1927 the principal method was monthly clinical screening that included the early detection of significant symptoms of lead poisoning	**Several conditions** The death certificates of those who died during the study period were mostly obtained from the company pension scheme	A significant excess of deaths from cerebrovascular accidents was found among pensioners dying between 1925–1976 in the most exposed group, but not among men in the same exposure group dying in employment There was no significant excess of deaths from hypertensive disease, nor any other circulatory disease There was no excess of observed to expected deaths among any of the 3 groups of women in any of the cause groups examined *Highest exposure group* 1925–1976 43 observed, 33.69 expected, p=0.055 1925–1961 24 observed, 9.3 expected, p<0.001	–

		n=1 898 254 women 1 644 men	In 1927 punctuate basophil counting was started, supported by haemoglobin estimations In 1964 facilities for lead in blood analysis became available for all the lead workers		There is an excess in the most exposed group, but this is not statistically significant (p=0.055) Most of this excess occured in the 65–69 years old group where 18 deaths were observed compared with 7.5 expected, (p<0.001). In the 70 and over age group 25 deaths were observed compared with 26.19 expected	
Manuwald et al 2012 [99] Germany	Prospective cohort 23 years Chemical plant 1952–2007	Participants were all persons employed in a chemical plant on a full-time basis for a minimum of 3 months between 1952–1984 when the plant was closed down Subjects entered the cohort at the date of their first employment in the plant n=1 589 398 women 1 191 men	**2,4,5-trichlorophenoxyacetic acid (TCDD)** The factory produced various herbicides and insecticides, including 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), the production of which started in 1952 The intensity of e'xpasure ta TCDD has been estimated retrospectively for the different workplaces in the plant in a	**Diseases of circulatory mortality** Mortality follow-up was performed for the period from 1952 up to the reference date of 31 December 2007 Standardised mortality ratios (SMRs) was calculated using the population of Hamburg as reference	Diseases of circulatory system mortality. Comparison by TCDD levels. Quartile of estimated cumulative job exposure (TCDD based on blood fat). SMR (95% CI) **Women** No exposure: 0.6S (0.35; 1.20) >0–<19.5 ppt: 0.79 (0.39; 1.41) <19.5–<78.3 ppt: 0.78 (0.46; 1.23) >78.3 ppt: 0.73 (0.44; 1.15) No trend test, only 7 cases **Men** No exposure: >0–<13.1 ppt: 1.14 (0.86; 1.49) <13.1–<77.4 ppt: 1.20 (0.92; 1.53) <77.4–<334.5 ppt: 1.55 (0.97; 2.35) >334.5 ppt: 1.44 (0.89; 2.20) Trend test: p=0.3	–

			previous analysis, based an dioxin analyses in blood or fat tissue samples (Becher et al, 1998)	The causes of death in the study group were determined by an experienced physician (pathologist) from the death certificates following international coding rules in accordance with the ICD-9		
Marsh et al 2009 [100] USA	Retrospective cohort 54 years Mining industry 1946–2000	Participants were male workers employed 3 or more years in a copper mine or smelter mill between 1946–1996 n=2 422 All participants were men	**Arsenic** Historical exposures were estimated for arsenic Company recorded job titles were used to generate a job dictionary and the job dictionary was used to generate a job exposure matrix, based on the relative exposure intensities as these intensities changed over time	**Cerebro-vascular disease** A vital status tracing protocol was used to identify deaths among cohort members with unconfirmed vital status. The protocol relied on national-scale sources, eg the National Death Index Deaths were coded by a trained	Risk of cerebrovascular disease by arsenic exposure metric, total cohort. Internal comparisons (1946–2000). RR (95%CI) **Duration of exposure (years)** >0–6.535: 1.36 (0.72; 2.55) >6.536: 1.84 (0.96; 3.52) **Cumulative exposure (µg/m³-years)** <0.046: 1.08 (0.46; 2.52) 0.046–0.413: 1.46 (0.62; 3.46) 0.414–0.721: 3.70 (1.65; 8.30) ≥0.722: 1.33 (0.56; 3.13) **Average intensity (µg/m³)** Exposed: 1.46 (0.89; 2.41) Key results of exploratory analysis of mortality in relation to occupational exposure to arsenic, total cohort, External comparisons 1960–2000. SMR (95% CI) **Cerebrovascular disease** Unexposed:93 (67; 125)	–

			Standardized mortality ratios (SMRs) were computed based on US and local county rates and modelled internal relative risks	nosologist to the underlying cause of death using ICD rules in effect at time of death The total and cause-specific mortality was examined using a modified life table procedure from the Occupational Cohort Mortality Program [Marsh et al., 1988b] Person-years at risk contributed by each study member were jointly classified by race, age group, calendar time, duration of employment, and the time since first employment	Exposed: 152 (99; 225)	

Matanoski et al 2003 [101] USA	Case–cohort study Industry 1943–1982	Participants were male workers employed during 1943–1984 in 2 styrene-butadiene rubber-manufacturing plants in the United States The cases included men who died from ischemic heart disease and a 15% random sample of all 6 587 male workers (997 men) who were ever employed in the same period, representing 997 in the subcohort Among 498 cases, 71 were also part of the subcohort. These cases were included as references in the comparison subcohort until the time of death, at which	**Styrene exposure** A job dictionary was developed for this industry. Each job was assigned a unique job code. All jobs were reviewed and ranked from 0–10 for both styrene and butadiene. A detailed job history for each subject was abstracted and coded based on the job dictionary Measurement data for styrene and butadiene were collected for many of the jobs from different sources. For any job where there were no measurements, the z-score method (Tao et al., 1996) was used to estimate the exposures. This method	**Ischemic heart disease** The vital status of each worker was determined through the death notification system and vital status records of the national registers and through follow-up by local plant beneficiary records and motor vehicle administratio n records. Direct follow-up was conducted for individuals with unknown vital status Ischemic heart disease cases were defined as deaths with ICD-8 codes 410–414, including acute myocardial infarction (ICD-8 code	Risk of ischemic heart disease from cumulative styrene exposure (ppm-year) among active workers in 2 rubber plants, United States, 1943–1982. Relative hazard (95% CI). Butadiene excluded from the model **Acute ischemic heart disease** Employed for ≥2 years: 1.04 (1.00; 1.09) Employed for ≥5 years: 1.04 (1.00; 1.08) *Time-weighted intensity for recent 2 years* *Employed for ≥2 years* <0.10 ppm: 1.00 0.10–<0.20 ppm: 1.24 (0.36; 4.33) 0.20–<0.30 ppm: 2.95 (1.02; 8.57) ≥30 ppm: 4.30 (1.56; 11.84) *Employed for ≥5 years* <0.10 ppm: 1.00 0.10–<0.20 ppm: 1.25 (0.36; 1.35) 0.20–<0.30 ppm: 3.00 (1.03; 8.73) ≥30 ppm: 4.24 (1.54; 11.68) **Chronic ischemic heart disease** Employed for ≥2 years: 0.99 (0.93; 1.05) Employed for ≥5 years: 0.99 (0.93; 1.05)	Risk of ischemic heart disease from cumulative styrene exposure among active workers in 2 rubber plants, United States, 1943–1982. Relative hazard (95% CI) adjusted for butadiene **Acute ischemic heart disease** Employed for ≥2 years: 1.08 (1.02; 1.14) Employed for ≥5 years: 1.07 (1.02; 1.13) **Chronic ischemic heart disease** Employed for ≥2 years: 0.98 (0.90; 1.07) Employed for ≥5 years: 0.98 (0.90; 1.07)

		point they were included as cases The follow-up for each individual began at the time of first hire and ended at the subject's death or at the termination of the study 1982 n=1 495 498 cases and 997 controls All participants were men	assumed that the relative exposure of a job was similar across the industry because tasks associated with the job were similar, but individual plants might have had overall differences in the actual levels of the chemicals	410), other acute and sub-acute forms of ischemic heart disease (ICD-8 code 411), chronic ischemic heart disease (ICD-8 code 412), angina pectoris (ICD-8 code 413), and asymptomatic ischemic heart disease		
McDonald et al 1993 [102] Canada *Note:* same population as in article by Liddell et al, 1980	Cohort study Chrysotile mines 1966–1988	Participants were men born 1891–1920 and employed for at least 1 month in the chrysotile mines and mills of Quebec n=11 000 All participants were men	**Asbestos** Estimates of dust concentrations, year by year, for each of the more than 5 000 jobs in the industry was based on about 4 500 midget impinger dust counts from annual surveys in all companies, 1949–1966	**Ischaemic heart disease, mortality** ICD-8 codes 410–414 were applied The numbers expected on the basis of male mortality in Quebec	Deaths by selected causes and place of employment in relation to exposure (million particles per cubic foot x years) accumulated to age 55, 20 years or more after first employment. SMR **Ischaemic heart disease** *Asbestos mine and mill* <30: 0.82 30–<100: 0.86 100–<300: 0.97 ≥300: 1.17 *Thetford Mines* <30: 0.98 30–<100: 1.15 100–<300: 1.18 ≥300: 1.24 *Asbestos factory*	–

			Each man's dust exposure was calculated, in terms of million particles per cubic foot x years accumulated to age 55. The calculation took into account the fraction of the year worked, the average dust concentration for the particular job and year, and the weekly hours worked during the period in question		<30: 1.11 30–<100: 0.67 100–<300: 0.92 ≥300: 2.92 *Complete cohort* <30: 0.92 30–<100: 0.97 100–<300: 1.09 ≥300: 1.24 **Cerebrovascular disease** *Complete cohort* <30: 0.89 30–<100: 0.79 100–<300: 1.16 ≥300: 1.62	
McElvenny et al 2015 [103] Great Britain	Cohort study Mean follow-up length was 29.2 years Workers 1975–2011	Participants were workers who had been monitored for lead via blood lead level measurements. Data was derived from the British Health and Safety Executive cohort data file Great Britain population was used as external	**Lead exposue** 2 distinct exposure estimates were used in the study. First, blood lead levels (BLL) were used (each participants mean and maximum BLL in nmol/dL). In addition, codes for the process or activity the workers	**Mortality to circulatory diseases, ischaemic heart disease and cerebro-vascular disease** Data derived from the British Health and Safety Executive cohort data file	Mortality in lead exposed workers 1975–2011. SMR (95% CI) *Circulatory system diseases* Males: 105 (99; 111) Females: 102 (88; 118) Total: 105 (99; 110) *Ischemic heart disease* Males: 106 (99; 114) Females: 102 (82; 127) Total: 106 (99; 113) *Cerebrovascular disease* Males: 119 (101; 139) Females: 105 (75; 146) Total: 116 (100; 134)	Cox regression analysis for the disease groups of a priori interest, with ischeamic heart disease and cerebrovascular disease. HR (95% CI) adjusted for age and sex *Circulatory system diseases* Mean BLL: 1.30 (1.17; 1.44) Maximum BLL: 1.25 (1.14; 1.37) Assessed lead level Medium vs low: 1.09 (0.95; 1.24) High vs low: 1.11 (0.94; 1.30) *Ischemic heart disease* Mean BLL: 1.30 (1.17; 1.43) Maximum BLL: 1.23 (1.11; 1.34)

		compatator (with stratification by sex, 5-year age band and calendar year) n=9 122 Participants were both women and men	undertook as well as the industry sector they were working in was available Second, risk assessment data and Health and Safety Executive National Exposure database were used to develop an exposure classification based on categorization of the process and industry in which workers were classified as high, medium, or low exposure			Assessed lead level Medium vs low: 1.02 (85; 1.18) High vs low: 1.02 (0.82; 1.22) *Cerebrovascular disease* Mean BLL: 1.15 (0.83; 1.28) Maximum BLL: 1.23 (0.98; 1.48) Assessed lead level Medium vs low: 1.25 (0.87; 1.62) High vs low: 1.50 (1.07; 1.93)
Mills et al 2009 [104] USA *Note*: Data on specified chemicals are stated in the article .	Prospective cohort study. Data from the Agricultural Health study cohort The median mortality follow-up was 11.8 years The median incidence follow-up was 5.0 years	Participants were licensed male pesticide applicators, primarily farmers with pesticide licenses, in North Carolina and Iowa n=54 069 (mortality analysis)	**Pesticides** All agricultural exposure information was collected at enrollment, including self-reported ever use of 50 pesticides In addition, detailed application	**Myocardial infarction** Deaths from myocardial infarction were recorded from state and national death records Myocardial infarction mortality was	Association of myocardial infarction mortality and incidence with personal lifetime days of pesticide use and high exposure events among male pesticide applicators. HR (95% CI) **Myocardial infarction mortality** ***Lifetime exposure, day*** 0–50: 1.00 51–100: 1.06 (0.76; 1.47) 101–250: 0.93 (0.71; 1.23) >250: 0.97 (0.75; 1.26) ***High - pesticide - exposures event*** No: 1.00	–

	Farming 1993–2006	 n=32 024 (incidence analysis) All participants were men	information was collected for 22 pesticides at enrollment, including years used, days per year used, and use in the last year Detailed information on the remaining 28 chemicals was collected on a take-home questionnaire completed by approximately 40% of the cohort	defined as any death attributed primarily to myocardial infarction or with myocardial infarction listed as a contributing cause on the death certificate by ICD-10, codes I21–I22 Nonfatal myocardial infarction incidence was determined on the basis of a positive response on the 5-year follow-up questionnaire to the question, ‘‘Has a doctor or other health professional ever told you that you had a heart attack (or myocardial infarction)?’’	Yes: 0.88 (0.57; 1.38) ***Pesticide - poisoning event*** No: 1.00 Yes: 1.40 (0.66; 2.97) **Myocardial infarction incidence** ***Lifetime exposure, day*** 0–50: 1.00 51–100: 1.10 (0.83; 1.46) 101–250: 1.14 (0.91; 1.42) >250: 1.19 (0.96; 1.47) ***High - pesticide - exposures event*** No: 1.00 Yes: 1.12 (0.84; 1.48) ***Pesticide - poisoning event*** No: 1.00 Yes: 0.94 (0.48; 1.82)	

Mørck et al 1988 [108] Denmark	Cohort study Printing plant	Participants were male workers from 2 photograhpic printing plants n=262	**Toluene (methyl-benzene)** Each participant was given an exposure score based on interview information which included a detailed analysis of the subjects' actual and previous locations during working hours. The assessment of exposure as a function of the location of the workers during working hours and the type of work processes they were involved in was based on previous measurements of toluene air concentrations carried out by the Danish National Institute of Occupational Health	**Blood pressure** Blood pressure was measured using Hawksley random spymomanometer by a trained observer	Correlation between exposition score and blood pressure Systolic blood pressure: r=0.191, p=0.0019 Diastolic blood pressure: r=0.096, p=0.123	Partial correlation coefficients with exposition score taking into account the effect of alcohol consumption, smoking, age, log height, and log weight Log systolic BP: r=0.191, p<0.01 Log diastolic BP: r=0.096, n.s
Moulin et al 1993 [106] France	Historical prospective method	Participants were welders and manual workers	**Welding fumes** 3 factories, where shipyards most commonly	**Ischaemic heart diseases, mortality**	SMR (95% CI) among welders Ischaemic heart diseases: 1.51 (1.00; 2.18) Cerebrovascular diseases: 0.93 (0.42; 1.76)	–

	13 years Welding in factories and shipyards 1975–1988	employed in 13 factories in France The welding exposed cohort consisted of all male workers who were employed as welders at the date of beginning of the follow-up period. For each welder, 3 controls were selected at random among the nonwelders employed at this date The cohorts were restricted to workers with at least 1 year of employment The data were collected from the personnel registers 13 factories including 3 shipyards n=9 404 2 721 welders and 6 683	used mild steel and low alloyed (chromium or nickel) steels, sometimes coated with antirust paints Main materials of 2 other factories were mild steel, stainless steel, armoured steels, and aluminium The other factories produced different types of products for metallurgy, using mild steel, stainless steel, or aluminium as base materials The main welding techniques initially were manual metal arc welding and oxy-acetylene welding to a lesser extent. Due to technical developments, other welding processes have	The vital status of the subjects was determined from information provided by the administrative records of the factories, by the registry office of the subjects' birthplaces, and by the national file The following ICD-8 codes were used för ischaemic heart diseases: 410–414 Expected numbers were calculated using national death rates	SMRs for selected causes among welders and controls by duration of employment and by time since first employment. SMR **Ischaemic heart diseases** *Welders* <10 years:0.00, n.s. 10–19 years: 1.00, n.s. ≥20 years: 1.79, p<0.05 *Controls* <10 years: 1.27, n.s. 10–19 years: 0.63, n.s. ≥20 years: 0.96, n.s. SMRs for selected causes among controls and sub-groups of welders including shipyard welders (5 year lag) SMR (95% CI) with adjustment for sex, age, and calendar time Controls 0.91 (0.68; 1.20) Part time welders only or boilermakers only: 1.50 (0.49; 3.49) Mild steel welders only 1.50 (0.82; 2.52) Ever stainless steel welders 1.71 (0.78; 3.25) Predominantly chromium VI 1.78 (0.49; 4.56)	

		manual workers (controls) All participants were men	been introduced: tungsten inert gas, metal inert gas, and metal active gas Welders may also be exposed to asbestos Some were also exposed to paints, which may have resulted in additional exposure to organic contaminants or to metals such as chromium			
Murray et al 1993 [107] South Africa	Case-control Gold mining 1974–1988	Participants were white South African gold miners aged 45 and over who underwent a full necropsy between 1974–1988 The original criteria of Fulton et a for right ventricular hypertrophy were applied to define cases. The control	**Silicosis** Silicosis was assessed macroscopically and confirmed microscopically. It was graded as slight, moderate, or extensive, based on the profusion' of nodules. Eleven subjects with massive fibrosis were included in the extensive disease category	**Ischaemic heart disease and cor pulmonale** Ischaemic heart disease was classified as minimal disease-slight coronary artery atheroma; moderate disease-moderate coronary artery atheroma; and severe	Frequency of ischaemic heart disease Cases/Controls Severe 101/80 Moderate 19/31 Minimal 197/160 None 74/70 Total 391/341 χ_2=5.87, df=3, p=0.1179 Predictors of cor pulmonale. OR (95% CI) Silicosis, slight: 1.46 (1.03; 2.08) Silicosis, moderate: 1.63 (0.93; 2.84) Silicosis, extensive: 4.95 (2.92; 8.38)	–

		subjects had normal hearts n=732 (391 cases and 341 controls) Gender not stated		disease-pronounced atheroma, extensive myocardial fibrosis, or myocardial infarction		
Neophytou et al 2014 [109] USA	Prospective cohort study 14 years Industry 1998–2012	Participants were hourly workers at 11 US plants of the same aluminum company. Workers had to be enrolled in the primary insurance plan and employed for at least 2 years during follow-up to be eligible. To exclude prevalent cases, a 2–year washout period without any ischemic heart disease claims was required A requisition was that the person was actively employed and without any previously	**Exposure to particles** A job exposure matrix was created to determine the average annual particulate matters with aerodynamic diameter of 2.5 µm or less ($PM_{2.5}$) In short, concentration (in mg/m^3) was assigned to district exposure groups within each plant. The procedure to create the matrix is described elsewhere (Coles et al., 2008)	**Ischemic heart disease** Incidence of ischemic heart disease cases was identified from health insurance claims through 2012 or until the date of active employment termination Ischemic heart disease cases were defined as subjects with insurance claims for relevant procedures for 2 or more days, or face-to-face visits The following codes were applied: ICD-	Association between binary exposure to $PM_{2.5}$ defined by 10th-percentile cutoff and incidence ischemic heart disease in a cohort of actively employed US aluminum workers stratified by facility type. Data adjusted for sex, race, smoking status, body mass index, job grade, plant, and risk score. No censoring weights **Risk of ischemic heart disease** ***HR (95% CI)*** Smelting: 1.98 (1.18; 3.32) Fabrication: 1.38 (0.98; 1.94) **$PM_{2.5}$ exposure mg/m^3, Cox regresssion** <0.260: 1.00 0.260–1.469: 1.51 (0.98; 2.37) 1.470–1.959: 1.73 (1.06; 2.86) 1.960–2.589: 1.53 (0.97; 2.45) ≥2.590: 1.53 (0.97; 2.46)	Association between binary exposure to $PM_{2.5}$ and incidence ischemic heart disease in a cohort of actively employed US aluminum workers stratified by facility type. Data adjusted for sex, race, smoking status, body mass index, job grade, plant, and risk score. All terminations **Risk of ischemic heart disease** ***HR (95% CI*)** Smelting: 1.87 (1.10; 3.18) Fabrication: 1.35 (0.95; 1.91) **Probability of remaining uncensored associated with exposure to $PM_{2.5}$** ***OR (95% CI)*** Smelting: 1.07 (0.95; 1.20) Fabrication: 1.05 (0.94; 1.16)

		ischemic heart disease event The mean age was 44 years n=6 348 (12 949 at baseline) 1 737 women and 11 212 men at baseline		9, admission codes 410–414 or ICD-10, codes I20–I25		
Nishiwaki et al 2004 [110] Japan	Prospective cohort study Mean follow-up time was 4 years Viscose rayon factories 1992–1999	Participants were male workers exposed to CS_2 and male referent workers in 11 Japanese viscose rayon factories. None of the subjects had any medical history of cerebrovascular and cardiovascular diseases, including medically treated hypertension at baseline, determined by checking companies' medical records and through a self	**Carbon disulphide, CS_2** Among the CS_2 exposed workers, 251 remained to be exposed to CS_2 until the end of the observation period (exposed workers), and 140 workers had their exposure truncated because 4 factories discontinued production of rayon fibres around 1994–1995 for economic reasons (ex-exposed workers) CS_2 concentrations	**Hyperintense spots** so-called ''silent cerebral infarctions" Brain MRI was performed twice at the baseline and the follow-up surveys at 8 hospitals near the factories MRI films were sent to a neuro-radiologist for evaluation. He evaluated (a) hyperintense spots in T2 weighted images (HIS) in the cerebrum,	Intra-individual changes in hyperintense spots on T2 weighted images over 6 years. OR (95% CI), adjusted for age Ex-exposed: 1.24 (0.68; 2.27) Exposed: 2.56 (1.59; 4.10)	Intra-individual changes in hyperintense spots on T2 weighted images over 6 years. OR (95% CI), adjusted for age, smoking, alcohol intake, body mass index, education level, shift work, systolic blood pressure, and HDL cholesterol Ex-exposed: 1.33 (0.70; 2.54) Exposed: 2.27 (1.37; 3.76)

		administered questionnaire Mean age was approximately 35 years n=666 (217 exposed, 125 ex-exposed, and 324 referent subjects) All participants were men	in the workers' breathing zone were measured twice a year with a Parkin-Elmer diffusive sampler tube. The level of 2-thiothiazolidine-4-carboxylic acid (TTCA), a metabolite of CS_2, in urine was also determined twice a year as a biological monitoring parameter. Individual exposure level was represented by the arithmetic mean of TTCA and CS_2 concentration for 6 years	brain stem, and cerebellum, and (b) atrophy of the cerebrum, for the baseline and follow-up films The diagnostic criteria for HIS were those recommended by the study subgroup on diagnostic criteria, pathogenesis, and management for asymptomatic cerebrovascular diseases		
Notkola et al 1987 [111] Finland	Cohort study Farming 1979–1983	Participants were men included in the Finnish farm register on 31 December 1978 All persons or corporations owning farms were included in the register, in which a farm	**Organic and microbial du st s and endotoxins** According to the article "Farmers, especially on small livestock-producing farms, are exposed to various organic	**Cardio-vascular diseases mortality** Data on all deaths during 1979–1983, causes of death, etc, were obtained from death certificate	Mortality by cause of death in 1979–1983 among male farmers aged 35–64 years on the basis of farm register data with the data on all economically active males in Finland in 1976–1980 as reference. SMR (95% CI) Cardiovascular diseases: 96 (94; 98)	–

		refers to the totality of cultivated land that has a field and garden area which surpasses 1 ha. Information is collected each year with a postal questionnaire Only the active men aged 35–64 years whose occupation was listed in the farm register as farmer were included n=100 435 All participants were men	and microbial dusts and endotoxins" No measurements were conducted	data recorded in the Finnish register on causes of death. For the classification of deaths the International Classification of Diseases (ICD), 8th revision, was used The following causes of death were considered: cardiovascular diseases (ICD-8, 390–459)		
Nugteren et al 2012 [112] The Netherlands	Population-based prospective cohort study 4 years General working population 2002–2006	Participants were all pregnant women who had an expected delivery date between April 2002 and January 2006 and lived in Rotterdam The study included	**Several factors** Information was collected by questionnaire completed during mid-pregnancy Items on physically demanding work were based on the Dutch Musculoskeletal	**Pregnancy induced hypertension and pre-eclampsia** Information on pregnancy complications was obtained from medical records Women who delivered in hospital and	Associations in a birth cohort study among pregnant women on chemical exposure and hypertensive disorders during pregnancy. OR (95% CI) **Pregnancy induced hypertension** ***Exposure to chemicals (JEM)*** PAH: 2.99 (0.91; 9.77) Pesticides: – Phthalates: – Organic solvents: 0.72 (0.22; 2.29) Alkylphenolic: 1.04 (0.32; 3.34) Metals: – Any chemicals: 1.05 (0.45; 2.44)	Associations in a birth cohort study among pregnant women on physically demanding work, chemical exposure and hypertensive disorders during pregnancy. OR (95% CI) adjusted for maternal age, educational level, parity, ethnicity and body mass index **Pregnancy induced hypertension** ***Exposure to chemicals (JEM)*** PAH: 2.64 (0.74; 9.35) Pesticides: – Phthalates: – Organic solvents: 0.94 (0.29; 3.09) Alkylphenolic: 1.56 (0.46; 5.29)

		women who were prenatally enrolled, with paid employment before or during pregnancy, with no history of pre-existing hypertension and with a spontaneously conceived singleton live born pregnancy Women were excluded if they had twin pregnancies, a pregnancy of non–spontaneous origin, fetal death, if a mother already was included in the study with an earlier pregnancy or if the women had pre–existing hypertension n=4 465 All participants were women	Questionnaire (Hildebrandt et al., 2001) and concerned manual handling, standing, walking, driving, night shifts, and working hours Further questions on job title, type of business, name of employer, and activities in the job were used to classify jobs into the Dutch Classification of Occupations and subsequently to link these codes to a Job-Exposure-Matrix for chemical exposure	who had chronic hypertension or were reported to have experienced pregnancy induced hypertension (>140/90 mm Hg) or hypertension related complications (pre-eclampsia, proteinuria, eclampsia, and/or HELLP syndrome), were selected from hospital registries. Their individual medical records were studied by qualified medical doctors Pregnancy induced hypertension, preeclampsia and eclampsia were defined according to the criteria of	**Preeclampsia** ***Exposure to chemicals (Job-Exposure-Matrix)*** PAH: 1.28 (0.17; 9.43) Pesticides: 3.14 (0.42; 23.73) Phthalates: 1.05 (0.14; 7.72) Organic solvents: 0.96 (0.30; 3.08) Alkylphenolic: 0.91 (0.22; 3.75) Metals: 2.72 (0.65; 11.43) Any chemicals: 1.17 (0.46; 2.93)	Metals: – Any chemicals: 1.22 (0.51; 2.94) **Preeclampsia** ***Exposure to chemicals (JEM)*** PAH: 0.89 (0.12; 6.75) Pesticides: 3.15 (0.38; 25.94) Phthalates: 0.82 (0.11; 6.16) Organic solvents: 0.92 (0.28; 3.04) Alkylphenolic: 0.81 (0.19; 3.45) Metals: 2.21 (0.50; 9.67) Any chemicals: 1.04 (0.40; 2.68)

				the International Society for the Study of Hypertension in Pregnancy and according to criteria of the College of Obstetricians and Gynecologists		
Nurminen et al 1976 [113] Finland *Note: same study population as the 2 articles by Hernberg et al and the article by Tolonen et al*	For information on the population and measurements, see articles by Hernberg and Tolonen				Coronary mortality among the CS_2 exposed and non-exposed cohorts and various derived estimates of death rates. Data from an 8-year follow-up OR: 2.3	-
Olsen et al 1994 [114] USA	Retrospective cohort Industry 1957–1986	Participants were male employees who had a minimum of 1 month work experience between 1957–1986 in the production or use of epichlorohydrin and allyl chloride and 1	**Epichlorohydrin and allyl chloride** All plants and departments where employees had the potential for exposure to epichloro-hydrin and allyl chloride from 1956–1986 were identified.	**Several conditions** Sources of information for vital status follow-up included company records, the Social Security Administratio n, and the National Death index	Deaths by cause, for 2 latency periods in mortality study of workers with potential exposure to epichlorohydrin and allyl chloride. RR (95% CI) *3-year latency* Diseases of the circulatory system 1.22 (0.88; 1.69) Arteriosclerotic heart disease 1.07 (0.68; 1.67) Cerebrovascular disease 0.36(0.05; 2.45)	–

		year total employmenl duration at Dow Chemical's Texas Operations n=1 064 All participants were men	This involved a review of computerized and bard copy work history records, plant rosters, personal interviews and industrial hygiene data. This resulted in the development of a jobexposure matrix which consisted of 3 basic sections: epoxy resins, glycerine, and the allyl chloride/epichlo ro-hydrin production areas	Death certificates were obtained and coded according to the International Classification of Diseases rules in effect at the time of death by nosologists experienced in multiple revision coding Expected deaths were calculated by indirect standardizatio n methods using U.S. white and black male mortality rates Diseases of the circulatory system (ICD-8 codes 390–458). Arterio-sclerotic heart disease (ICD-8 codes 410–	*15-year latency* Diseases of the circulatory system 1.41 (0.93; 2.13) Arteriosclerotic heart disease 1.36(0.79; 2.36) Cerebrovascular disease No data	

				413). Cerebro-vascular disease (ICD-8 codes 430–438)		
Ott et al 1996 [115] Germany	Retrospective cohort Industry 1953–1992	Participants were workers at a TCD factory n=243 All participants were men	2,3,7,8 tetrachloro-dibenzopdioxin (TCDD) In 1953, an accident occurred in a trichlorophenol (TCP) production unit. Byproducts that escaped in the accident contaminated surfaces of the enclosed production The agent most likely to have caused these responses was TCDD, a byproduct p of TCP production Workers were biomonitored 30 years after the accident	Diseases of circulatory system and Ischaemic heart disease Expected deaths based on age, sex, and calendar period specific death rates for the former West Germany, 1952–1992	Standardised mortality ratios by cause of death and TCDD dose group, 1953–1992. SMR (95% CI) **TCDD µg/kg body weight** *Diseases of circulatory system* All: 0.8 (0.6; 1.2) <0.1: 0.8 (0.4; 1.4) 0.1–0.99: 1.0 (0.5; 1.7) ≥1: 0.8 (0.4; 1.3) *Ischaemic heart disease* All: 0.7 (0.4; 1.1) <0.1: 0.9 (0.3; 1.8) 0.1–0.99: 0.7 (0.2; 1.7) ≥1: 0.6 (0.2; 1.3)	–
Parkinson et al 1987 [116]	Cross-sectional study with a retrospective approach	Participants were workers at 3 battery plants (exposed to	**Occupational lead exposure** Time weighted average blood	**Diastolic and systolic blood pressure**	Inter-correlation among blood pressure, exposure, and risk factors for all lead and control workers combined (n=428)	Multiple regression of diastolic blood pressure on lead exposure. Standardised regression coefficients, β. Controlled for age, education, income,

USA	Industry 1982	lead) and non-exposed workers from a truck side frame manufacturing plant with no known neurotoxic exposures, such as lead, arsenic, or solvents The workers participating in the study were white men aged 18–60, and employed at the plant for at least 1 year n=428 (270 exposed and 158 non-exposed workers) All participants were men	lead values (a cumulative index derived for the lead exposed workers from all blood lead values since date of hire taken by the lead battery companies) The median number of blood lead values was 14, and in 40 workers blood lead values had been determined on more than 60 occasions	Subjects were examined before work or during the weekend in an office maintained for the study Blood pressure was measured 3 times, at 5 minute intervals, with the worker sitting. The 1st and 4th Korotkoff sounds were used as indicators of systolic and diastolic blood pressure, respectively	Systolic blood pressure - lead: 0.10 ($p<0.05$) Diastolic blood pressure - lead: 0.11 ($p<0.05$)	cigarette use, alcohol use, exercise and body mass *Diastolic blood pressure* Lead vs control: β=–0.029 Time weighted average for blood lead calculated for lead workers only: β=0.070 not significant *Systolic blood pressure* Lead vs control: β=0.15 Time weighted average for blood lead calculated for lead workers only: β=0.09 not significant
Pepllonska et al 2001 [118] Poland	Retrospective cohort 45 years Industry 1950–1995	Participants were male workers at a Polish factory manufacturing rayon and viscose products Participants were identified through the	**Carbon disulfide** The chemical agent was assessed through air monitoring	**Death caused by diseases of the circulatory system** The vital status of the participants were ascertained on the basis of data	Observed deaths in the cohort of male workers by level of exposure. SMR (95% CI) **Diseases of the circulatory system** Non exposed: 97 (80; 117) Intermittent exposure: 111 (89; 136) High exposure: 126 (111; 142) **Hypertensive disease** Non exposed: 0 Intermittent exposure: 0 High exposure: 64 (21; 149)	–

		company's personnel files Eligibility required continuous work for at least 12 months during 1950–1985, employment in production or maintenance departments and residence in the town during employment n=2 878 All participants were men		obtained from the local register of town residents. The causes of death were coded according to ICD-9 Mortality data were analyzed according to a standard person-years method, including only persons aged up to 80 years	**Ischemic heart disease** Non exposed: 106 (73; 148) Intermittent exposure: 113 (76; 162) High exposure: 119 (94; 149) **Cerebrovascular disease** Non exposed: 233 (160; 327) Intermittent exposure: 174 (103; 275) High exposure: 209 (157; 273) **Atherosclerosis** Non exposed: 46 (24; 80) Intermittent exposure: 60 (30; 107) High exposure: 81 (57; 112)	
Peplonska 1996 [119] Poland	Mortality cohort study Viscose rayon plants 1970–1990	Total mortality and selected cause specific mortality risk were assessed in a historical cohort study. All persons enrolled in the cohort were employees of 6 viscose rayon plants in Poland The study cohort consisted mainly of process workers	**Carbon disulfide, CS_2** Chronic occupational CS_2 poisoning diagnosed during the years 1970–1990 An essential element in the diagnosis of chronic CS2 poisoning was the nervous system pathology in the form of organic changes or	**Several diseases of the circulatory system** Mortality assessment was based on the standardized mortality ratio using the person-years method. The general population of Poland was the reference population	Mortality from selected causes in the cohort of men with reported chronic carbon disulfide poisoning. SMR (95% CI) *Women* Diseases of the circulatory system: 166 (95; 270) Ischaemic heart disease: 149 (31; 435) Cerebrovascular disease: 286 (115; 589) *Men* Diseases of the circulatory system: 139 (125; 154) Ischaemic heart disease: 137 (114; 164) Cerebrovascular disease: 188 (143; 242)	–

		performing the jobs involving the highest exposure to carbon disulfide n=2 291 169 women 2 122 men	severe functional disorders	Diseases of the circulatory system were based on the ICD codes 390–459, ischaemic heart disease (ICD codes 410–414), and cerebrovascular disease (ICD codes 430–438)		
Persson et al 2007 [120] Sweden	Retrospective cohort Up to 37 years Industry 1955–1992	Participants were workers at 3 major old mills in the middle of Sweden The first calendar year with complete files based on year of leaving work was chosen for each mill as the beginning of cohort inclusion Furthermore, at least 1 year of continuous employment and a Nordic ethnicity according to	**Sulfate and sulphite** An industrial hygienist gathered detailed information about present and historical exposures and the processing conditions from the 3 mills	**Mortality in ischemic heart disease and cerebro-vascular disease** Information on workers who fulfilled the inclusion criteria were collected from the mills The cohort was followed-up for mortality in the national Causes-of-Death Register. The underlying causes of death given in	Standardized mortality ratios for men in 3 Swedish pulp and paper mills, by exposure. SMR (95% CI) **Ischemic heart disease** Sulfate digestion: 165 (112; 236) Sulfite digestion: 98 (32; 229) **Cerebrovascular disease** Sulfate digestion: 49 (6; 176) Sulfite digestion: 91 (2; 509)	–

		name were applied as inclusion criteria n=7 107 757 women 6 350 men		the register were recoded when necessary by a trained physician into the 8th revision of ICD Deaths in diseases of the circulatory system (ICD-8, 390–458) were considered along with ischemic heart diseases (ICD-8, 410–414) and cerebro-vascular diseases (ICD-8, 430–438)		
Peters et al 2013 [121] Australia	Prospective cohort Miners 1961–2009	Participants were a cohort of male gold miners was established from surveys of respiratory symptoms, smoking and lung function performed in the 60-ties and 70-ties	**Aluminium dust** Duration of aluminium exposure was calculated by summing the years a subject worked in a job where he indicated the inhalation of aluminium dust. The number of months worked	**Several cardio-vascular conditions, mortality** The cohort was linked to a national mortality database Expected numbers of deaths were	Mortality among underground gold miners **not** treated with aluminium dust inhalation (SMR 95% CI) *Cardiovascular disease* 1.26 (1.12; 1.41) *Cerebrovascular disease* 1.43 (1.16; 1.78) *Pneumoconiosis* 13.5 (9.76; 18.8)	–

		Analyses were limited to ever underground miners 1 cohort comprised 1247 underground gold miners not treated with aluminium dust inhalation. Another cohort comprised 647 underground gold miners treated with aluminium dust inhalation n=1 894 All participants were men	underground for each miner was assigned based on the job descriptions	estimated using age-specific and period-specific mortality rates calculated for the Western Australian male population in 5-year periods from 1970–2009 Causes of death were coded according to the ICD revision in force at the time of death Mortality from cardiovascular diseases (ICD-9 410–429; ICD-10 120–52), cerebrovascular diseases (ICD-9 430–438; ICD-lO 160–69)	Mortality among underground gold miners treated with aluminium dust inhalation. Hazard ratios. HR (95% CI) adjusted for year of birth *Cardiovascular disease* Ever aluminium: 1.19 (0.99; 1.44) 1–9 years aluminium: 1.23 (0.99; 1.54) ≥10 years aluminium: 1.15 (0.91; 1.45) Continuous (years of aluminium dust exposure): 1.02 (1.00; 1.04) *Cerebrovascular disease* Ever aluminium: 0.89 (0.63; 1.27) 1–9 years aluminium: 0.79 (0.51; 1.25) ≥10 years aluminium: 1.01 (0.65; 1.55) Continuous (years of aluminium dust exposure): 0.99 (0.96; 1.03)	
Piccotto et al 2016 [122] USA	Prospective cohort study Automobile plant	Participants were autoworkers at 3 plants in	**Straight metalworking fluids exposure**	**Ischemic heart disease mortality**	Ratio of median survival time if always exposed to straight metalworking fluids at a $PM_{3.5}$ concentration of 1 mg/m^3 versus never	–

	1938–1994	Michigan, who were hired between 1938–1982 and worked for at least 3 years Follow-up for mortality continued through 1994 n=38 666 Gender not stated	Job histories were available from company records, as were air sampling measures of total particular matter collected over decades. These historical particulate matter data were combined with size fractions of particulate matter collected in the mid-1980s by research industrial hygienists. The resulting time-dependent job-exposure matrix was combined with job records to estimate annual averages of daily exposure to each fluid type in each calendar year for each worker	Ischemic heart disease was coded according to ICD-9, codes 410–414	exposed (the ration is less than 1 if the exposure is harmful). Ratio (95% CI) Ischemic heart disease 0.41 (0.17; 0.99)	
Prince et al 2000 [123] USA	Retrospective cohort mortality study	Participants were workers employed in the rubber chemicals	``**Rubber chemicals''** The chemical plant under study opened in	**Ischemic heart disease** Ischemic heart disease (IHD)	Selected causes of death among New York rubber manufacturing workers. SMR (95% CI) **Ischemic heart disease** Definitely exposed 1.51 (0.94; 2.3)	–

	``Rubber chemicals'' manufacturing 1946–1991	department from 1946–1988. They were followed through December 31, 1994 n=708 25 women 683 men	1946 for the production of polyvinyl chloride. Beginning in 1957, the plant made an antioxidant for use in tire manufacturing from ortho-toluidine (o-toluidine), aniline, hydroquinone, and toluene. A rubber accelerator was produced since 1970 from carbon disulfide, sulfur, aniline, benzothiazole, and a proprietary chemical According to the article: ”A potential cardiovascular risk factor that affects the rubber chemicals department is exposure to CS_2”	mortality (ICD 410–414, 9th revision) The standardized mortality ratio (SMR) analyses were based on reference rates in the United States population	*Duration employed in department (for definietely exposed)* <5 years: 1.6 5–15 years: 0.6 >15 years: 2.3	

			Based on 8-hour TWAs, the investigators reported maximum exposures of 0.67 ppm CS_2			
Radican et al 2008 [124] USA	Cohort Industry Exposure 1952-56 Follow-up 1990 to 2000	Participants were civilians employed at an aircraft maintenance facility for at least one year between 1 January 1952 and 31 December 1956 - Follow-up 1990 (948 exposed workers) - Follow-up 2000 (1 282 exposed) n=14 455 3 725 women 10 730 men	**Trichloro-ethylene** Information on exposures, chemicals, jobs, processes and other relevant information was collected from sources such as worker compensation files, histories and telephone books of the facility, organization charts, technical orders and position descriptions. Walk through surveys and interviews of long term employees were also conducted For TCE a more detailed approach was also taken that	**Ischemic heart disease** Mortality data was assessed from a central computerized index of death record information for the entire US,based on the Ninth Revision of the International Classification of Diseases (ICD-9) for the years 1979–1998 and the Tenth Revision of the International Classification of Diseases (ICD-10) for 1999 and later	Cox model hazard ratios (95% confidence intervals) for selected causes of death among workers exposed to trichloroethylene, 1990 follow up, and Cox model hazard ratios (95% confidence intervals) and numbers of cases, 2000 follow up. time variable = age; covariate = gender HR (95% CI) Ischemic Heart Disease (410–414) (410–414) (I20–I25) Follow-up 1990 HR: 1.1 (0.9; 1.2) Follow-up 2000 1.09 (0.99; 1.21)	Cox model hazard ratios (95% confidence intervals) for selected causes of death among workers exposed to trichloroethylene, 1990 follow up, and Cox model hazard ratios (95% confidence intervals) and numbers of cases, 2000 follow up. time variable = age, covariates = gender and race, included all races HR (95% CI) Follow-up 1990 HR: 1.1 (1.0; 1.3) Follow-up 2000 No data

			identified the frequency and pattern of exposure based on the job tasks			
Randem et al 2003 [125] USA	Cohort study Asphalt work, private and public 1970–1996	Participants were male asphalt workers from 11 private asphalt companies, the Public Roads Administration and the Oslo Road Maintenance Service Company records were used to identify the workers. In the state-owned companies, expert panels consisting of earlier leaders and workers were used to identify earlier asphalt workers, since job code on personnel records did not specify asphalt work n=8 610	**Asphalt, PAH and bitumen** In most of the analyses time since first employment in asphalt work, decade of employment in asphalt, duration of employment (under or over 6 months), and job class were used as surrogates for exposure A study-specific job exposure matrix was used for individual–individual exposure estimates	**Several conditions** The cause-specific deaths observed in the study cohort were compared with the expected figures calculated from 5-year period- and age-specific national death rates The grouping of causes of death was based on ICD-9. The observation period spanned the 8th, 9th, and 10th revision of the ICD codes Circulatory system (codes 390–459), hypertension	Standardized mortality ratio among asphalt workers. SMR (95% CI) **All asphalt workers** Circulatory system: 0.93 (0.83; 1.03) Hypertension: 0.84 (0.34; 1.72) Ischemic heart disease: 0.86 (0.75; 0.98) Cerebrovascular disease: 1.14 (0.89; 1.43) Other disease of heart: 1.02 (0.71; 1.43) Standardized mortality ratio among asphalt workers. SMR (95% CI) **Workers with more than 6 months of employment in asphalt jobs** **Circulatory diseases** *Cumulative exposure to bitumen fume (bitumen units x years)* Unknown: 1.28 (0.64; 2.29) 0–9.9: 0.94 (0.79; 1.13) 10–25.89: 0.94 (0.76; 1.14) >25.9: 0.86 (0.69; 1.06) *Cumulative exposure to PAH (PAH units x years)* Unknown: 1.28 (0.64; 2.29) 0–23.49: 0.96 (0.80; 1.14) 23.5–59.9: 0.94 (0.76; 1.15) >60: 0.83 (0.67; 1.04)	–

		All participants were men		(codes 401–405), ischemic heart disease (codes 410–414), cerebrovascular disease (codes 430–438), other disease of heart (codes 415–429)		
Reid et al 1996 [126] South Africa	Cohort and cas-control study 20 years Gold mining 1970–1990	Participants were white gold miners aged 39–54 years working in the vicinity of Johannesburg Participants were working in the mines in 1970 with at least 85% of service on gold mines and at least 15% of shifts underground These restrictions resulted in a population in which over 99% of the shifts worked were in gold mines and 88% were underground	**Dust** Dust exposures estimated by thermal precipitator count of respirable mass after acid treatment are used to calculate dust exposure in h-mg/m^3 per shift The typical gold miner of this study serves 27 years underground and was exposed to 3–7 y-mg/m'=7 104 h-mg/M3 (after acid treatment) According to the author" In South Africa gold miners	**Several diseases** Ischaemic heart disease was based on ICD-9 codes 410–414, hypertensive disease on codes 400–405 and cerebrovascular disease on codes 430–438 In the cohort study, miners were compared with the reference population of all white men in the Republic of South Africa	SMR:s in the cohort of gold miners from selected causes. SMR (95% CI) Ischaemic heart disease: 124.1 (115.0; 133.7) p<0.0001 Hypertensive disease 95.1 (55.4; 152.3), n.s. Cerebrovascular disease 108.8 (89.3; 131.2), n.s. Relative risks for gold miners in case-contoll comparisson. RR (95% CI) Underground decades (2 400 shifts) 5 years before case death Ischaemic heart disease: 0.97 (0.83; 1.1)	Relative risks for gold miners in case-contoll comparisson. RR (95% CI) adjusted for smoking Underground decades (2 400 shifts) 5 years before case death 0.98 (0.83; 1.2)

		n=4 925 All participants were men	work in stressful conditions of heat and humidity and are exposed to low levels of dust containing a moderate amount of silica and to low concentrations of radon daughters. They are also exposed to unknown concentrations of other atmospheric pollutants, such as nitrous fumes from the blasting operations and exhaust fumes where diesel engines are used"	The following procedure was applied for the in the case-control part of the study: For each case, with cause of death assessed according to the best available information, a set of controls was selected strictly at random from among all those miners born in the same year as the case and who survived the case		
Rinsky et al 2013 [127] USA	Prospective cohort study. Data from the agricultural health study (AHS) cohort Mainly farming The median follow-up was 13 years 1993–2008	Participants were mainly farmers and commercial pesticide applicators living in North Carolina and Iowa Participants with missing data on	**Pesticide use** Information on pesticide use was obtained through a self-administered questionnaire. Participants reported lifetime days of pesticide use, including total	**Stroke mortality** Mortality from stroke was obtained through the national death registries, with the ICD-9 codes 430–438 or ICD-10 code I60–I69	Association between pesticides use activities and stroke mortality. HR (95% CI) adjusted for state, smoking status and alcohol consumption **Lifetime exposure** <50 days: 1 51–100 days: 1.56 (1.04; 2.33) 101–250 days: 1.13 (0.79; 1.62) >250 days: 1.09 (0.78; 1.55) **Years mixed applied pesticides** ≤5 years: 1 6–10 years: 1.41 (0.87; 2.3)	–

	(participants enrolled 1993–1997)	smoking or alcohol consumption were excluded n=51 603 All participants were men	years and days per year Participants reported ever use of the 50 most commonly used pesticides and duration of use for 22 of those pesticides	as the underlying or contributing causes of death on the death certificates	11–20 years: 0.93 (0.58; 1.48) 21–30 years: 1.14 (0.72; 1.78) >30 years: 1.31 (0.84; 2.02) **High pesticide exposure event** No: 1 Yes: 0.91 (0.52; 1.58) **Pesticide use** No: 1 Yes: 1.73 (0.77; 3.92)	
Ronneberg et al 1995 [128] Norway	Retrospective cohort study Aluminum industry using prebaked carbon anodes 1962–1991	Participants were all men hired in 1922 or later and employed for at least 6 months continuously at a Norwegian aluminium smelter between 1922–1975 n=1 085 All participants were men	**Coal tar pitch volatiles and pot emissions** The intensity of exposure to coal tar pitch volatiles and pot emissions (fluorides, sulphur dioxide, carbon monoxide) had been assessed on a semiquantitative scale from 0–100 for all jobs in the smelter. Dose-response relations between specific exposures and causes of death were investigated with cumulative exposure as a surrogate for dose. This was	**Ischaemic heart disease and cerebro-vascular diseases, mortality** The underlying cause of death was obtained from the Central Bureau of Statistics with the personal identification number for all deaths the Central Bureau of Statistics had revised and coded the underlying cause according to the current revision of the International Classification	SMRs for selected clauses in 1962–1991 among 661 aluminium smelter workers with at least 3 years' employment, by cumulative exposure (intensity-year) to coal tar pitch volatiles and pot emissions **Ischaemic heart disease** *Coal tar pitch volatiles* Zero, all: 0.84 Low: 0.94 High: 0.98 p-value for trend: 0.52, n.s. *Pot emissions* Zero, all: 1.12 Low: 0.82 High: 0.75 p-value for trend: 0.10, n.s. **Cerebrovascular diseases** *Coal tar pitch volatiles* 0–19 years time window for exposure to observation Zero, all: 1.18 Low: 0.36 High: 0.00 p-value for trend: 0.08 40+years time window for exposure to observation	–

			calculated in intensity-years as the prod product of exposure intensity and duration summed for all jobs held at the smelter	of Diseases (ICD) from the medical death certificates and necropsy reports. The observation period spanned the 7th, 8th, and 9th revision of the ICD	Zero, all: 0.94 Low: 1.79 High: 1.47 p-value for trend: 0.79 *Pot emissions* 0–19 years time window for exposure to observation Zero, all: 0.99 Low: 0.93 High: 1.68 p-value for trend: 0.03 40+years time window for exposure to observation Zero, all: 0.52 Low: 1.67 High: 2.04 p-value for trend: 0.03	
Rosenlund et al 2001 [129] Sweden	Case-control study. Data from the SHEEP study General working population 1970–1993	Participants were never-smoking Swedish citizens age 45–70 years who resided in Stockholm county during 1992–1993 Cases were identified from the coronary and intensive care units at the department of internal medicine at the emergency hospital in Stockholm County, the	**Environmental tobacco smoke** A postal questionnaire with a telephone follow-up provided information on environmental tobacco smoke exposure and other potential risk factors for myocardial infarction	**Myocardial infarction** Outcome was nonfatal and fatal first events of myocardial infarction The diagnosis criteria for myocardial infarction used to determine case inclusion were those applied by the Swedish Association of Cardiologists**.** They required	Odds ratio for myocardial infarction associated with exposure to environmental tobacco smoke. OR (95% CI) adjusted for age, hospital catchment area, body mass index, socioeconomic status, job strain, hypertension, diet and diabetes mellitus **Work place exposure to tobacco smoke** ***Women*** Never exposed: 1.00 Ever exposed: 0.94 (0.59; 1.50) Currently exposed: 1.31 (0.62; 2.79) Previous exposure: 0.90 (0.54; 1.51) ***Men*** Never exposed: 1.00 Ever exposed: 1.14 (0.78; 1.67) Currently exposed: 1.39 (0.86; 2.25) Previous exposure: 1.05 (0.69; 1.61) ***Both genders (also adjusted for gender)*** Never exposed: 1.00	–

		Hospital Discharge Register for the county or death certificates from the national Cause of Death Register 1 control per case, matched on gender, age and hospital attachment area, was randomly selected from the study base within 2 days of the inclusion of a case. All controls were initially checked for myocardial infarctions n=1 011 (334 cases and 677 controls) 411 women 690 men		at least 2 of 3 conditions to be met regarding certain symptoms, specific blood enzyme changes, or specific electrocardio-gram changes. In addition myocardial necrosis detected at autopsy that could be related to the time of disease onset was also included	Ever exposed: 1.07 (0.80; 1.44) Currently exposed: 1.31 (0.89; 1.95) Previous exposure: 1.02 (0.74; 1.40)	
Sakr et al 2009 [130] USA *Note*: Results also available for more	Retrospective cohort study 5, 10, 15 and 20 years Industry	Participants were all employees who had ever worked at the Washington work plant	**Ammonium perfluoro-octanoate (APFO)** APFO is a bio persistent surfactant used in the	**Ischemic heart disease** Causes of death were determined primarily through a company	Relative risk estimates for mortality from ischemic heart disease by estimated cumulative exposure category, including increasing 5–years lags of exposure. RR (95% CI) adjusted for race and calendar year **4th quartile**	–

categories (quartiles) and lagged exposure periods	1948–2002	between 1948–2002 The median age was 27 years n=4 747 105 women 4 642 men	manufacture of several types of fluorpolymers Time-dependent APFO exposure was estimated from detailed work histories for all employees using an exposure reconstruction model developed from occupational information and serum PFOA data	mortality database of death certificates submitted with life insurance claims filed by beneficiaries of deceased employees and pensioners Additionally, the US National Death Index was used to ascertain cause of death The diagnosis was according to ICD-9, codes 410.0–414.9 and ICD-10 codes I20–I25.9 listed as the underlying cause of death	***Quartiles determined by <u>case</u> distribution of estimated cumulative exposure*** 5–years lag, ≥6.51 ppm-years: 1.1 (0.7; 1.7) 20–years lag, ≥5.08 ppm-years: 0.8 (0.5; 1.5) ***Quartiles determined by <u>cohort</u> distribution of estimated cumulative exposure*** 5–years lag, ≥7.06 ppm-years: 1.1 (0.7; 2.0) 20–years lag, ≥5.45 ppm-years: 1.3 (0.8; 2.3)	
Sali et al 1999 [131] Seven European countries	Cohort study Man made vitreous fibre factories	Participants were male workers employed for at least 1 year during the period 1933–	**Man made vitreous fibres** No direct measures of exposure to man made vitreous fibres	**Several diseases** Trained nosologists abstracted the underlying	Mortality from selected non-neoplastic causes of death. SMR (95% CI) **Diseases of the circulatory system (I00–I99)** Rock (slag) wool:0.99 (0.91; 1.08) Glass wool:1.05 (0.97; 1.14) Continuous filament:1.22 (0.94; 1.55)	–

		1977 in 13 factories in Denmark, Finland, Norway, Sweden, the United Kingdom, Germany, and Italy. 7 factories produced rock or slag wool, 5 factories glass wool, and 2 factories continuous filament n=11 373 All participants were men	or other workplace pollutants were conducted. Exposure was assessed by indirect indicators, such as time since first employment, duration of employment, and technological phase at first employment	cause of death from the death certificates, with the exception of Denmark where routine data were used. Causes of death were coded according to the version of the international classification of diseases (ICD) in use at the time of death. In this report, the causes are presented according to the 10th revision of the ICD (ICD-10).8 Age, calendar period, country and sex specific national mortality reference data were derived from the Mortality Data Bank of the World Health	**Ischaemic heart disease (I20–I25)** Rock (slag) wool:0.97 (0.87; 1.08) Glass wool: 1.05 (0.95; 1.15) Continuous filament: 1.43 (1.06; 1.88) **Cerebrovascular disease (I60–I69)** Rock (slag) wool: 0.95 (0.77; 1.17) Glass wool: 1.05 (0.86; 1.26) Continuous filament: 1.21 (0.60; 2.16) Relative risks of mortality from ischaemic heart disease according to time since first employment, duration of employment, and technological phase at first employment RR (95% CI) **Rock (slag) wool** Duration of employment (1–4 years is reference=1.0) 5–9 years: 1.0 (0.7; 1.3) 10–19 years: 0.9 (0.6; 1.2) ≥20 years: 0.7 (0.5; 1.9) Trend: p=0.07 **Glass wool** Duration of employment (1–4 years is reference=1.0) 5–9 years: 0.8 (0.7; 1.1) 10–19 years: 1.0 (0.8; 1.4) ≥20 years: 1.2 (0.8; 1.7) Trend: p=0.5 **Continuous filament** Duration of employment (1–4 years is reference=1.0) 59 years: 1.3 (0.6; 2.9) 10–19 years: 2.5 (1.0; 6.2) ≥20 years: 1.6 (0.3; 8.7)	

				Organisation and used to compute expected deaths Standardised mortality ratios (SMRs) were calculated with national mortalities for reference; an internal exposure-response analyses based on multivariate Poisson regression models was also conducted		
Sanden et al 1993 [132] Sweden	Prospective cohort follow-up time varied between the participants shipyard workers 1977–1988	Participants were shipyard workers, earlier exposed to asbestos, who participated in a health monitoring programme between 1977–1979 n=1 725 All participants were men	**Asbestos** Exposure to asbestos stopped at the shipyards in 1972. After that time only a few men who pulled down asbestos in ships at a repair yard were exposed The determinants were diagnosed	**Ischemic heart disease mortality** ischemic heart disease was based on ICD, codes 410–414 The cases of ischemic heart disease were found by linking the national identity	Mortality from ischemic heart disease. RR (95% CI), stratified for age and smoking habits Asbestosis or suspected asbestosis Total: 3.1 (95% CI 1.5; 6.4) Ex-smokers and current smokers born between 1910 and 1919: 4.3 (2.0; 9.3)	–

			with asbestoses at a health checkup between 1977–1979	number of each man with the Swedish Death Register. The underlying cause of death was used The observation period for each man was the time between the health checkup and 31 December 1988 or the point of death or emigration if this occurred before the end point		
Schwartz et al 2000 [133] USA	Cohort study Follow-up time not specified Organolead manufacturing industry 1960–1983	Participants were workers who had ever been employed in the facility on or after January 1, 1950, were male, and were between the ages of 40–70 years in 1995 A total ol 3 223 workers were	**Lead** During the third year of the study, tibial lead was measured by 109Cd K x-ray fluorescence (XRF) at the mid-tibial shaft we estimated DMSA-chelatable lead burden as 4-hr	**Blood pressure** Blood pressure was measured by a trained technician at each yearly study visit, as well as during the bone-lead-measurement visit with a	Logistic regression modeling results identifying predictors of hypertension in 543 former organolead manufacturing workers. OR (95% CI) controlled for age, body mass index, lifetlme alcohol consumption, and diabetes Blood lead–systolic: 0.95 (0.87; 1.03) Blood lead (3 outliers excluded)–systolic: 2.90 (1.48; 5.71) Tibial lead–systolic: 1.03 (0.87; 1.24) Tibial lead–cheletable lead–systolic: 0.99 (0.96; 1.01)	Logistic regression modeling results identifying predictors of hypertension in 543 former organolead manufacturing workers. OR (95% CI) controlled for age, body mass index, lifetlme alcohol consumption, and diabetes Blood lead (3 outliers excluded) x age interaction– systolic: 0.98 (0.97; 0.99) Tibial lead x age interaction–cheletable lead–systolic: 1.00 (0.99; 1.00)

		selected randomly for recruitment: 703 were enrolled in the study n=543 (total cohort 703 men) All participants were men The 543 workers who received tibial lead measurements were primarily Caucasian and had a mean age of 57.6 years	lead excretion following oral administration of 10 mg/kg DMSA DMSA=meso 2.3-dimercapto-sucdnic acid	sphygmomanometer and an appropriately sized cuff Hypertension was defined as having systolic blond pressure >160 mm Hg, having diastolic blood pressure >96mm Hg, or currently taking medications for high blood pressure	Blood lead–DSMA–cheletable lead–diastolic: 1.01 (0.99; 1.02) Tibial lead–DSMA–cheletable lead–diastolic: 0.96 (0.87; 1.07)	Tibial lead x age interaction–DSMA–cheletable lead–diastolic: 1.00 (0.99; 1.01)
Sjogren et al 2003 [136] Sweden	Cohort study 5 and 25 years Agricultural industry 1970–1995	Participants were male and female livestock and agricultural workers. The age of livestock and agricultural workers as well as referents was 20–64 years at the time of entry Livestock and agricultural workers were identified as 2 separate	**Organic dust** No measurements of organic dust were conducted According to the article "agricultural workers and farmers are exposed to organic dust containing endotoxins and other microbial agents, such as mites, and	**Ischaemic heart disease** Ischaemic heart disease (IHD) was defined as code 410–414 of the International Classification of Diseases, 7th and 8th revisions (ICD-7 and 8) The livestock and agricultural	Standardised mortality ratios of ischaemic heart disease in different cohorts of workers followed until the end of 1995. SMR (95% CI) *Livestock workers in 1970 census* Women: 1.10 (0.98; 1.23) Men: 1.06 (0.95; 1.18) *Agricultural workers in 1970 census* Women: 0.98 (0.94; 1.03) Men: 1.00 (0.96; 1.05) *Agricultural and livestock workers in 1990 census* Women: 2.64 (0.72; 6.77) Men: 1.20 (0.79; 1.76)	–

		occupational groups in the National Census of 1970. These workers were followed from 1970–1995. In the later census of 1990, these 2 groups of workers were merged into 1, which was followed until the end of 1995 The referent groups comprised all gainfully employed men and women identified in the respective census n=7 520 272 3 308 444 women (1970: 1 260 583 and 1990: 2 020 548) 4 211 828 men (1970: 2 047 861 and 1990: 2 163 967)	moulds, which may cause allergic and inflammatory respiratory diseases such as asthma, chronic bronchitis, and allergic alveolitis"	workers were identified with their 10 digit identification number, and the established cohorts were linked to the Cause of Death Register during the period of follow-up		

Sjogren et al 2013 [135] Sweden *Note*: the article also presents data for those exposed for 5 years or longer	Prospective cohort 18 years General working population 1987–2005	Participants were all manual workers in the Swedish National Census 1980, who were alive as of 1 January 1987 The cohort was restricted to manual workers, including skilled and unskilled workers in the production and service sector White-collar workers, professionals, self-employed and farmers were excluded The mean age at entrance was 46 years n=983 409 498 745 women 494 674 men	**Small (<1µm) and large (>1µm) particles** A job-exposure matrix for exposure to small and large particles was developed by experienced occupational hygienists by combing relevant occupational exposure information from a Swedish job-exposure matrix developed for an occupational cancer study and an airway irritant job-exposure matrix	**Ischemic and hemorrhagic stroke** First time events of Ischemic or hemorrhagic stroke were identified through linkage to the Hospital Discharge Register and the National Cause of Death Register Disease was coded according to ICD-9 and ICD-10 Ischemic stroke or cerebral infarction: ICD-9 code 434 and ICD-10 code I63 Hemorrhagic stroke: ICD-9 code 431 and ICD-10 code I61	Hazard ratios for stroke among workers exposed to small (<1µm) and large (>1µm) particles. HR (95% CI) adjusted for age, socioeconomic groups and residential population density **Ischemic stroke** ***Women-ever exposed-small particles*** All: 1.00 (0.95; 1.05) Low exposure: 0.98 (0.93; 1.04) Medium to high exposed: 1.20 (1.01; 1.44) ***Women - ever exposed - large particles*** All: 1.11 (1.07; 1.15) Low exposure: 1.11 (1.07; 1.14) Medium to high exposed: 1.10 (1.03; 1.18) ***Men - ever exposed - small particles*** All: 1.06 (1.02; 1.10) Low exposure: 1.09 (1.04; 1.15) Medium to high exposed: 1.02 (0.98; 1.07) ***Men - ever exposed - large particles*** All: 1.05 (1.00; 1.11) Low exposure: 1.10 (1.05; 1.16) Medium to high exposed: 1.05 (0.99; 1.10) **Hemorragic stroke** ***Women - ever exposed - small particles*** All: 1.11 (0.99; 1.25) Low exposure: 1.10 (0.98; 1.24) Medium to high exposure: 1.19 (0.82; 1.75) ***Women - ever exposed - large particles*** All: 1.15 (1.07; 1.24) Low exposure: 1.14 (1.05; 1.23) Medium to high exposed: 1.13 (0.97; 1.31) ***Men - ever exposed - small particles*** All: 1.04 (0.97; 1.13) Low exposure: 1.11 (1.00; 1.23)	–

					Medium to high exposure: 0.97 (0.88; 1.07) ***Men - ever exposed - large particles*** All: 1.02 (0.92; 1.13) Low exposure: 1.04 (0.93; 1.16) Medium to high exposure: 1.00 (0.89; 1.12)	
Sjogren et al 2002 [134] Sweden	Prospective cohort study Welding 1970–1995	Participants were male welders and gas cutters identified in the National Censuses, 1 cohort from 1970 and 1 from 1990 The cohorts were compared with all gainfully employed men in Sweden for each age stratum n=59 790 (1970 cohort: 31 722, 1990 cohort: 28 068) All participants were men	**Welding fumes** For the 1970 chohort census employment was identified based on responses to a questionnaire In the 1990 Census information about employment was based on data received by Statistics Sweden from employers	**Ischemic heart disease** Participants were linked to the Cause of Death Register during the period of follow-up Ischemic heart disease was defined as code 410–414 of the ICD-7 and ICD-8	Mortality in 2 cohorts of welders and gas cutters followed until the end of 1995. SMR (95% CI) *Ischemic heart disease* 1970 Census: 1.06 (1.02; 1.11) 1990 Census: 1.35 (1.10; 1.64)	–
Spirtas et al 1991 [137] USA	Retrospective cohort study Aircraft maintenance facility 1952–1982	Participants were civilian employees who worked for at least 1 year at Hill Air Force Base, Utah, between 1952–1956	**Trichloro-ethylene (TCE)** 2 industrial hygienists conducted walkthrough surveys of the base, interviewed	**Several diseases, mortality** Vital state of cohort members was determined through a number of	Cause specific SMRs for workers exposed to TCE. SMR (95%CI) adjusted for age and calendar period *White women* Cerebrovascular disease 83 (52; 125) All heart diseases 89 (69; 112) Rheumatic heart disease 93 (40; 184) Ischaemic heart disease 90 (67; 117)	–

		n=12 538 3 138 women 9 400 men Persons of unknown race have been combined with white persons for purposes of statistical analysis. The combined group of white workers and persons of unknown race is called whites in this report	long term employees, and reviewed industrial hygiene files, position descriptions, and other historical documents from the base to obtain information on departments (called organisations by the air force), job titles and tasks, numbers of employees, operations, chemicals used, monitoring results, and engineering controls Quantitative assessments of actual exposure levels of trichloro-ethylene (TCE) could not be made. For each combination of job and organisation, an assessment was made as to whether it had	sources, mainly national registers The underlying and contributory cause(s) of death were determined by a nosologist according to the rubrics of the International Classification of Diseases (ICD) in effect at the time of death	All other heart disease 82 (30; 178) *White men* Cerebrovascular disease: 83 (66; 103) All heart diseases: 97 (89; 104) Ischaemic heart disease: 98 (90; 107) Chronic disease of endocardium; other myocardial disease: 37 (8; 109) Hypertension with heart disease: 73 (24; 171) All other heart disease: 58 (38; 86), p<001. Hypertension without heart disease: 27 (1; 149) SMRs among workers by cumulative exposure to TCE. SMR **Ischaemic heart disease** *Women, cumulative exposure* <5: 107 5–25: 13 >25: 99 Total exposure: 90 Exposure trend: n.s *Men, umulative exposure* <5: 94 5–25: 94 >25: 105 Total exposure: 98 Exposure trend: n.s.	

			frequent or infrequent peak exposures to TCE, or continuous, or intermittent low level exposures to TCE, or both. From these patterns of use, exposure indices were developed that reflected comparative differences in exposure			
Stayner et al 1992 [138] USA	Retrospective cohort mortality study Army munitions facility 1949–1982 n=15 654 5 529 nitroglycerin, 4 989 dinitrotoluene, and 5 136 unexposed workers All participants were men	Participants were current and former white male workers from a United States Army munitions facility involved in the production of propellants used in munitions and rocket motors	**Nitroglycerin, dinitrotoluene** An environmental survey was conducted by industrial hygienists. Personal air-monitoring samples for nitroglycerin were collected and analysed for 92 individuals from 10 different job locations. The time-weighted averages of the nitroglycerin exposure were found to range	**Several conditions** The vital status of the cohorts was ascertained as of 31 December 1982 through national registers. Death certificates were obtained from state vital statistics bureaus and coded by a trained nosologist using the International Classification	Standardized rate ratios for ischemic heart disease and cerebrovascular disease by high and low nitroglycerin exposure. SRR (95% CI) **Ischemic heart disease** An increased ischemic heart disease mortality was found among workers younger than 45 years and actively exposed to nitroglycerin 3.30 (1.29; 8.48) *High exposure to nitroglycerine* <1 year: 1.09 1–5 years: 0.92 >5 years: 0.52 Total:0.96 *Low exposure to nitroglycerine* <1 year: 1.43 1–5 years: 1.08 >5 years: 1.43 Total:1.14 **Cerebrovascular disease** *High exposure to nitroglycerine*	–

			from 0.001–0.028 ppm. 3 groups were formed: (i) workers probably exposed to nitroglycerin, (ii) workers probably exposed to dinitrotoluene, (iii) workers not exposed to either dinitrotoluene or nitroglycerin	of Diseases revision in effect at the time of death A modified life-table program was used to compute the expected numbers of deaths by multiplying mortality rates specific for cause, 5-year age groups, and 5-year calendar groups from data on white males in the US population by the corresponding person years distribution of the study population	<1 year: 0.77 1–5 years: 0.73 >5 years: 0.63 Total: 0.77 *Low exposure to nitroglycerine* <1 year: 0.60 1–5 years: 1.06 >5 years: 1.97 Total: 0.93 *Exposure to dinitrotoluene* An increased cerebrovascular mortality was found among workers 55–59 years old and exposed to dinitrotoluene: 4.46 (1.11; 17.84) Standardlzed mortality ratios (SMR) and standardized rate ratios (SRR) for cardiovascular and cerebrovascular causes of death. Standardized for age and calendar year *Nitroglycerin·exposed* Cerebrovascular disease, SMR: 0.90, SRR:0.87 lschemic heart disease, SMR: 1.07, SRR:1.07 Chronic disease of the endocardium, SMR: 0.74 Other myocardial degeneration, SMR: 0.58 Hypertension with heart disease, SMR: 0.52 Hypertension without heart disease, SMR: 0.47 Disease of the arteries and veins, SMR: 1.02 Other diseases of the heart: SMR: 0.87 *Dinitrotoluene exposed* Cerebrovascular disease, SMR: 0.95, SRR:0.89 Ischemic heart disease, SMR: 0.98, SRR:0.99 Chronic disease of the endocardium, SMR: 0.46 Other myocardial degeneration, SMR: 1.41 Hypertension with heart disease, SMR: 0.21 Hypertension without heart disease, SMR: 1.17 Disease of the arteries and veins, SMR: 1.08 Other diseases of the heart: SMR: 0.95	

Steenland et al 1992 [140] USA *Note: same population as in study by Bertke, 2016*	Cohort study Smelter work 1940–1982	Participants were male hourly smelter workers who worked in a lead-exposed department for at Jeast 1 year, with at least 1 day of employment at the smelter between 1940–1965 n=1 990 All participants were men	**Lead** The cohort was heavily exposed to lead. A 1975 industrial hygiene survey showed average airborne lead concentrations of 3.1 mg/m^3. Blood leads in 1976 averaged 56.3 µg/100 ml High-lead departments were defined as those in which the average airborne lead concentrations during the 1975 survey exceeded 0.2 mg/m^3, or in which 50% or more of the jobs had average levels more than twice the existing standard Arsenic exposures in this cohort were relatively low, averaging 14 µg/m^3 in 1975	**Several conditions** Follow-up of the cohort was conducted via national registers The following ICD-9 codes were applied: ischemic heart disease (410–414), hyper-tension with heart disease (402, 404), hypertension with no heart disease (401, 403, 405) and cerebro-vascular disease (430–438)	Mortality results. SMR (95% CI) **Ischemic heart disease** Entire cohort: 0.94 (0.84; 1.05) High Lead Exposure Suboohort: 0.99 (0.87; 1.12) **Hypertension with heart disease** Entire cohort: 0.97 (0.53; 1.63) High lead exposure subcohort: 1.18 (0.60; 2.05) **Hypertension with no heart disease** Entire cohort: 1.73 (0.63;3.77) High lead exposure subcohort: 1.18 (0.60; 2.05) **Cerebrovascular disease** Entire cohort: 1.05 (0.82; 1.32) High lead exposure subcohort: 2.49 (0.24; 3.52) Mortality results for selected causes by duration of exposure, for the entire cohort. SMR (no CI presented) **Ischemic heart disease** 1–5 years: 1.02 5–20 years: 0.92 >20 years: 0.86 **Hypertension with heart disease** 1–5 years: 0.60 5–20 years: 0.90 >20 years: 1.57 **Hypertension with no heart disease** 1–5 years: 1.54 5–20 years: 1.51 >20 years: 2.38	–

			Cadmium exposures were also relatively low, averaging 113 µg/m^3 in 1975		**Cerebrovascular disease** 1–5 years: 0.83 5–20 years: 1.01 >20 years: 1.41	
Steenland et al 1996 [141] USA	Prospective cohort 7 years General working population 1982–1989	Participants were 30 years of age and older and enrolled nationwide in the US. The mean age at enrolment was 56 years Persons who reported having smoked were excluded. Also, those not currently employed at baseline were excluded Those with a heart disease at baseline were kept in the analysis n=275 060 196 350 women 78 710 men	**Passive smoking at work** Passive smoking was assessed by questionnaire. Items are described in the article	**Death in coronary heart disease** Death certificates were obtained and coded by a nosologist to the 9th revision of ICD. The codes applied were 410–414	Results for self-reported exposure to environmental smoke from cigarettes. RR (95% CI) controlled for age, self-reported history of illness (heart disease, hypertension, diabetes and arthritis), body mass index, educational level, aspirin use, diuretic use, liquor and wine consumption, employment status, exercise and oestrogen use **Women** ***All*** Currently exposed at work: 1.06 (0.84; 1.34) ***Age <65 years*** Currently exposed at work: 1.09 (0.70; 1.52) **Men** ***All*** Currently exposed at work: 1.03 (0.89; 1.19) ***Age <65 years*** Currently exposed at work: 1.10 (0.92; 1.31)	–
Steenland et al 1999 [139] USA	Cohort mortality analysis Industry	Participants were male workers from 8 US chemical plants that	**2,3,7,8-tetrachloro-dibenzo-pdioxin (TCDD)**	**Death from ischemic heart disease and cerebro-**	Cohort mortality results when exposed to 2,3,7,8-tetrachlorodibenzo-pdioxin. SMR (95% CI) **TCDD exposure**	–

	1942–1984	produced TCDD-contaminated products (including Agent Orange) from 1942–1984 Documentation of ever having worked in a TCDD-exposed job was required for inclusion Workers with exposure to both penta-chlorophenol and TCDD were eliminated to avoid possible confounding of any TCDD effects by penta-chlorophenol n=5 132 (total cohort) All participants were men	Exposure to TCDD was assessed by a job-exposure matrix assigning each worker a quantitative exposure score for each day worked The score was based on the concentration of TCDD present in process materials, the fraction of the day the worker worked on the specific process, and a qualitative contact level based on estimates of the TCDD contamination reaching skin areas or inhaled	**vascular disease** Follow-up through 1993 was conducted via Social Security death files, the National Death Index, and the Internal Revenue Service Death (underlying cause) from ischemic heart disease was based on ICD-9, codes 410–414 Life table analyses, stratified for race, age, and calendar time and, using the US population as a comparison, were conducted for the entire cohort for 92 underlying causes of death by using a	Ischemic heart disease: 1.09 (1.00; 1.20) Cerebrovascular disease: 0.96 (0.74; 1.21) Cox regression results for the exposure–level subcohort for ischemic heart disease. RR (95% CI) by cumulative exposure score category Septile 1 (0–<19): 1.00 Septile 2 (19–139): 1.23 (0.75; 2.00) Septile 3 (139–<581: 1.34 (0.83; 2.18) Septile 4 (581–<1 650): 1.30 (0.79; 2.13) Septile 5: (1 650–<5 740): 1.39 (0.86; 2.24) Septile 6 (5 740–<20 200): 1.57 (0.96; 2.56) Septile 7 (≥20 200): 1.75 (1.07; 2.87)	

				National Institute for Occupational Safety and Health life table program		
Suadicani et al 1995 [142] Denmark	Cohort. Data from the Copenhagen Male Cohort Study 6 years General working population Baseline: 1985–1986 Follow-up: 1991	Participants were Caucasian men aged 53–75 years without overt cardio-vascular disease employed at 14 large work-places in Copenhagen, Denmark The mean age was 63 years Workers with angina pectoris, acute myocardial infarction, stroke and/or intermittent claudication were excluded n=2 974 All participants were men	**Soldering fumes, organic solvents and lifting** All men were interviewed by a physician at baseline Occupational exposure to noise was assessed by questionnaire (items stated in the article) Long-term exposure was defined as exposure for 5 years or longer Short-term exposure was excluded from the analyses	**Ischemic heart disease** A register follow-up in the Danish Central Person Register was carried out on morbidity and mortality between 1985–1986 and 1991 Information on hospital admission for non-fatal acute myocardial infarction and death certificate diagnoses within the follow-up period was obtained from national registers Included diagnoses were codes	Association between occupational exposure and risk of Ischemic heart disease in 6 years. RR (95% CI) adjusted for age, social class, use of tobacco, alcohol, physical activity, blood pressure, body mass index, several blood markers and retirement status **Soldering fumes** 0–4 years: 1.0 5–15 years: 2.0 (0.9; 4.7) ≥16 years: 2.2 (1.2; 4.0) **Organic solvents** 0–4 years: 1.0 5–15 years: 1.0 (0.4; 2.5) ≥16 years: 2.2 (1.2; 3.9)	–

				410–412 from the ICD-8		
Suadicani et al 2002 [143] Denmark	Prospective cohort study 8-year follow-up 1985–1986	Participants were men without overt cardiovascular disease living in the Copenhagen area The Copenhagen Male Study was set up in 1970 as a prospective cardiovascular cohort study of men with a mean age of 48 years. In 1985–1986 a new baseline was established. All survivors from the 1970 study were traced. All survivors (except 34 emigrants) from the original cohort were invited to take part in this study Men who at baseline had a history of overt cardio-vascular disease: acute	**Several exposures** Information on occupational exposure to soldering fumes, welding fumes, organic solvents, asbestos, glass fibre and plastic fumes was obtained from the questionnaire. Long-term exposure was defined as frequent (several times a week) occupational exposure for at least 5 years	**Myocardial infarction and ischaemic heart disease** For all who reported admission to hospital because of acute myocardial infarction before the start of the study, the hospital records were checked. The diagnosis was accepted if at least 2 of the following symptoms/signs were recorded: retrosternal pain lasting more than 20 min, typical, serial electrocardiographic changes in more than 2 electro-cardiograms, acute increase of relevant	History of disease according to long-term occupational exposure to airborne pollutants (>5 years of exposure several times a week or more vs. no exposure) and ABO phenotype. OR (95% CI) **Myocardial infarction-O phenotype** Soldering fumes: 3.0 (1.6; 5.8) Welding fumes: 2.1 (1.05; 4.3) Organic solvents: 1.8 (0.9; 3.6) Asbestos: 2.2 (0.7; 6.3) Work with glass fibre: 4.9 (1.0; 23.0) Plastic fumes: 8.3 (2.6; 27.0) **Myocardial infarction-Other phenotypes** Soldering fumes: 0.7 (0.3; 1.7) Welding fumes: 0.8 (0.4; 1.9) Organic solvents: 0.4 (0.1; 1.1) Asbestos: 0.6 (0.1; 2.3) **Ischaemic heart disease-O phenotype** Soldering fumes: 1.8 (1.0; 3.2) Welding fumes: 1.1 (0.6; 2.2) Organic solvents: 1.1 (0.6; 2.1) Asbestos: 2.0 (0.8; 4.8) Work with glass fibre: 1.4 (0.2; 11.7) **Ischaemic heart disease-Other phenotypes** Soldering fumes: 1.05 (0.5; 2.2) Welding fumes: 1.0 (0.5; 2.1) Organic solvents: 1.3 (0.7; 2.5) Asbestos: 0.5 (0.1; 2.3)	–

		myocardial infarction, angina pectoris, stroke or intermittent claudication were excluded n=3 321 men		serum enzymes Information on angina pectoris, stroke and intermittent claudication was established from a questionnaire		
Sugimoto et al 1978 [144] Japan	Cohort study Rayon factory	Participants were male workers exposed to carbon disulfide in a rayon filament factory and male nonexposed workers from a nearby cuprammonium rayon factory The subjects, whose ages ranged between 35–54 years, were randomly selected from their respective population pools. Their medical histories were not taken into consideration in the selection	**Carbon disulfide** The workers were interviewed for an accurate occupational history concerning exposure to carbon disulfide, and the data were rechecked against their employment records From the available data on carbon disulfide concentration in the polluted departments or at sites at which "exposure" work is done,	**Blood pressure and myocardial ischemia** Blood pressure measurements were made according to a standard procedure with a sphygmomanometer and a stethoscope. Blood pressures were measured by the same observer for both groups after the subjects had rested for at least 15 minutes	Differences between exposed and nonexposed workers, and among exposed workers (Index of exposure dosages: ≤99, 100–199, ≥200). P-value *Systolic blood pressure* Exposed vs. nonexposed: n.s There were no differences between the exposure indexes among the exposed group *Diastolic blood pressure* Exposed vs. nonexposed: n.s There were no differences between the exposure indexes among the exposed group *Myocardial ischemia* Exposed vs. nonexposed: n.s There were no differences between the exposure indexes among the exposed group	–

		n=810 (420 exposed and 390 nonexposed) All participants were men	the annual changes in exposure levels have been calculated since the commencement of production. The index of exposure dosages was calculated for every worker from his work history, the time-weighted carbon disulfide concentrations being the criterion	Minnesota codes of the resting and postexercise ECGs accepted as indicative of myocardial ischemia The ECG response of exposed and nonexposed subjects to single-load submaximal exercise was determined during bicycle ergometry		
Swaen et al 1994 [145] The Netherlands	Retrospective cohort Up to 40 years Industry 1947–1988	Participants were male production and maintenance workers from a Dutch viscose textile plant, employed for at least half a year between 1947–1980 n=3 322 All participants were men	**Carbon disulfide** The study population was subdivide into exposure groups based on occupation Results of area monitoring were available, based on different methods depending on when the measurements had been conducted	**Diseases of the circulatory system** The cause of death was ascertained from a national statistical register	Mortality for 3 exposure groups. SMR **Diseases of the circulatory system** Not exposed: 94.4 Intermittent exposure: 84.6 Continuous exposure: 114.6	–

Sweetnam et al 1987 [146] Great Britain	Prospective cohort 32 years Viscose rayon factory Period off follow-up 1950–1982	Participants were men who were employed at 1 factory for at least 1 year between 1 January 1945 and 31 December 1949 n=2 848 All participants were men	**Carbon disulphide (CS_2)** For each man, the data collected included date of birth, date and cause of death, and the description and dates of every job that he had had in the viscose rayon factory Staff were simply divided into 2 groups as process workers (with some exposure to CS_2) and nonprocess workers (with no exposure to CS_2) An exposure score was also calculated for all operators. Measurement of doses by means of personal monitors has been carried out only during the past 6 years. Before this,	**Ischemic heart disease mortality** All causes of death were coded to the ICD classification by 1 experienced coder. The revision used was that in force at the time that the death occurred. Thus a death may have been coded to the 6th, 7th, 8th, or 9th revisions of the ICD	SMRs by exposure score among operatives; all ages. SMR **Ischemic heart disease** *The entire period* Exposure score 0–99: 100 100–199: 116 200–299: 138 ≥300: 144 Test for trend: p<0.05 **Ischemic heart disease** *The last 2 years* Exposure score 0: 115 1–23: 106 24–47: 104 48–71: 169 72–96: 236 Test for trend: p<0.01	–

			measurements of environmental levels of CS_2 were done on an area basis. Taking into consideration personal monitoring results, previous area measurements, and the nature of the work, each job was allocated a CS_2 rating			
Svendsen et al 1987 [147] USA	Cohort study Working population 1973-1982	Participants were men aged 35-57 years, recruited in 18 US cities. They were screened to select those in the upper 10-15 per cent of a risk score distribution derived from Framingham data, based on serum cholesterol concentration, cigarette smoking, and diastolic blood pressure	**Passive smoking at work** The participants were asked the smoking status of most of their coworkers	**Coronary heart disease** Classification of cause of death was performed by a committee of three cardiologists. They used hospital records, physicians' reports, next-of kin interviews, death certificates, and autopsy reports, when available	The relative risk for the endpoint death, for men whose coworkers smoked compared with men whose coworkers did not smoke, adjusted for age and wife's smoking status is described. RR (95% CI) Coronary heart disease death: 2.6 (0.5; 12.7) Fatal or nonfatal coronary heart disease: 1.4 (0.8; 2.5)	

		Participants were free of overt coronary heart disease by history and resting electrocardiogram Participants reported that they did not smoke cigarettes, pipes, cigars, or cigarillos. n=12 866 men (1 236 never smokers of which 906 had a majority of smoking co-workers)				
Svensson et al 1989 [148] Sweden	Prospective cohort study Stainless steel factory 1927–1983	Exposed cohort: Participants were male production workers who handled stainless steel, mainly from sinks and saucepans Reference population: Expected mortality for the period 1951–1983 was	**Dust** 3 coded categories of workers (grinders, brushers/polishers, and welders) were considered to have high exposure to dust, chromium and nickel. Grinders and brushers/polishers were also exposed to the	**Cardio-vascular diseases** Death certificates coded according to ICD-8. In 32% of the cases, the death certificate information was based upon autopsy	Mortality in workers exposed to dust. SMR (95% CI) **Cardio-vascular diseases** All: 0.81 (0.65; 1.00) ≥5 years exposure, >20 years latency period: 0.84 (0.60; 1.17)	–

		calculated using calendar year, cause of death-, and 5-year age-group specific mortality rates for males in Blekinge county, Sweden n=1 164 (exposed subjects) All participants were men	dust of grinding material and agents. Welders were also exposed to welding fumes. Another group was exposed indirectly to intermediate levels of same agents. There was also a group whose exposure were low			
Takebayashi et al 2004 [149] Japan	Prospective cohort study Mean follow-up time was 4 years Viscose rayon factories 1992–1999	Participants were male workers exposed to CS_2 and male referent workers in 11 Japanese viscose rayon factories. None of the subjects had any medical history of cerebrovascular and cardiovascular diseases, including medically treated hypertension at baseline, determined by checking companies'	**Carbon disulphide, CS_2** Among the CS_2 exposed workers, 251 remained to be exposed to CS_2 until the end of the observation period (exposed workers), and 140 workers had their exposure truncated because 4 factories discontinued production of rayon fibres around 1994–1995 for economic reasons (ex-	**Ischaemia and blood pressure** Incidence of ischaemic findings, defined as Minnesota codes I, IV1–3, V1–3 (at rest and after the load), or receiving treatment for ischaemia Blood pressure was measured by a doctor with a sphygmo-manometer. Aortic stiffness was	Effects of exposure to carbon disulphide on the risk factors of cardiovascular disease in the follow-up survey. Difference between non-exposed, exposed and ex-exposed workers *Blood pressure, p value (ANOVA) for difference between 3 groups of workers (non-exposed, exposed and ex-exposed)* Systolic blood pressure: 0.05, p<0.05 Diastolic blood pressure: 0.23, n.s. Difference between non-exposed, exposed and ex-exposed workers: *Coronary artery, p value (χ2 test or Fisher's exact method) for difference between the 3 groups* Ischaemic signs (defined as Minnesota codes I, IV1–3, V1–3 or receiving treatment for ischaemia) Incidence over 6 years: 0.02, p<0.05 Ischaemia (defined as rigorous ECG findings such as ST depression >2 mm or receiving treatment)	–

		medical records and through a self administered questionnaire Mean age was approximately 35 years n=666 (217 exposed, 125 ex-exposed, and 324 referent subjects) All participants were men	exposed workers) CS_2 concentrations in the workers' breathing zone were measured twice a year with a Parkin-Elmer diffusive sampler tube. The level of 2-thiothiazolidine-4-carboxylic acid (TTCA), a metabolite of CS_2, in urine was also determined twice a year as a biological monitoring parameter. Individual exposure level was represented by the arithmetic mean of TTCA and CS_2 concentration for 6 years	evaluated by measuring carotid-femoral pulse wave velocity. Ultrasound measurement of the stiffness of the carotid artery was also done to obtain blood flow rate, maximal velocity of the blood, and stiffness parameter	Incidence over 6 years: 0.97, n.s. The authors conclude that incidence of ischaemic findings was significantly higher in the exposed workers	
Telisman et al 2004 [150] Croatia	Prospective cohort study Industry	Participants were male industrial workers Cases were workers, employed at	**Lead** Exposure to lead was measured by blood Pb (BPb), activity of d-aminolevulinic acid	**Blood pressure** Systolic and diastolic blood pressure was measured using a	Significance of the difference between lead workers and reference subjects (z, P) Systolic blood pressure: 1.177, P>0.20 Diastolic blood pressure: 0.037, P>0.90	Multiple regression adjusted for BMI, smoking, alcohol, haematocrit and B-Cadmium, s-Zink and S-Cupper *Lead in blood* Not significant (estimate not presented) *Erythrocyte protoporphyrin*

		the present work place for ≥2 years, with slight to moderate occupational exposure to lead Referents were workers not occupationally exposed to lead n=151 (100 cases and 51 controls) All participants were men	dehydratase (ALAD), erythrocyte protoporphyrin (EP) Lead workers were had occupational exposure to lead during 5.2–21 years Lead workers had been regularly controlled for lead exposure in our laboratory for more than 15 years. Their long-term average BPbvalues were <400 mg/l	standardized method according to the WHO recommendations. 2 consecutive blood pressure readings were performed and the mean values were used for the calculations	There was a difference between lead workers and reference subjects for all 2 biomarkers of lead exposure (p<0.0001) Correlation coefficient (Pearson) *Lead in blood* Systolic 0.069 (ns) Diastolic 0.027 (n.s.) *Erythrocyte protoporphyrin* Systolic 0.191, p=0.06 Diastolic 0.068, n.s.	Systolic: β:0.330, p=0.001 Diastolic: β: 0.195, p=0.05
Theriault et al 1988 [151] USA	Case-control Aluminum industry 1975–1983	Participants were men employed at an aluminum plant Cases were selected from the company absenteeism list with reason given as angina pectoris or myocardial infarction	**Several chemicals** Detailed occupational histories were supplied by the company Industrial hygienists classified each job according to the estimated level of exposure to	**Angina pectoris and myocardial infarction** Confirmation of diagnoses was made by the researchers reviewing the medical records of the company	Risk of heart disease associated with chemical and physical contaminants. OR (95% CI) **Chemicals** Total dust: 0.93 (0.70; 1.24) Fluorides: 0.87 (0.70; 1.08) Sulfur dioxide: 1.16 (0.92; 1.46) Carbon monoxide: 1.18 (0.86; 1.62) Benzene-soluble materials: 0.98 (0.81; 1.18) **Reduction plant workers** All: 1.72 (1.09; 2.97) Söderberg potroom 1.71 (1.07; 2.72) Prebake 2.26 (1.27; 4.02) Pot lining 1.73 (0.94; 3.17) Electrode plant 1.68 (0.98; 2.88)	–

		Referents were employees at the same plan who were not known to suffer from either ischemic heart disease or peripheral vascular disease. 2 referents were matched for each case according to birth date, hiring date and length of service n=881 (306 cases and 575 controls) All participants were men	each of several chemicals		**Duration of work in reduction plant** <1 year 1.00 1–4 years 2.22 (1.17; 4.20) 5–9 years 1.98 (0.92; 4.28) 10–14 years 1.39 (0.62; 3.13) 15–19 years 0.80 (0.36; 1.80) 20+years 1.86 (1.05; 3.28)	
Tollestrup et al 1995 [152] USA		The study cohort included 1 225 individuals who had livecl in the Wenatchee area of Washington State cluring the 1938 apple growing season	**Lead arsenate spray and residue** 3 levels of exposure (ie., orchardist, intermediate, consumer) were defined, based upon the use of lead arsenate pesticide spray before and during the 1938	**Several** The cause of death was coded for all clecedents, using the fnternational Classification of Diseases (9th revision).	Crude mortality rates per 100 000 person-months of follow-up and cox proportional hazards survival analysis for orchardists and intermediatcs compared with consumers **Coronary heart diseases** *Women* Orchardist: 0.80 (0.31; 2.11) Intermediate: 0.98 (0.49; 1.94) *Men* Orchardist: 1.23 (0.73; 2.06) Intermediate: 1.94 (1.08; 3.48) **Other heart diseases**	-

		n= 1 225 (893 with occupational exposure) 225 women and 667 men	apple growing season Mean blood lead (µg/l) for the 3 exposure groups were (women/men): Consumers: 25.8/26.3 Orchardists: 34.4/43.9 Intermediate: 21.9/29.5 Urinary arsenic (µg/l) for the 3 exposure groups were (women/men): Consumers: 69.4/88.0 Orchardists: 118.1/176.4 Intermediate: 69.3/103.5 Consumers included individuals whose occupations did not bring them into contact with lead arsenate spray (e.g., school teachers, store clerks, housewives).		*Women* Orchardist: 0.93 (0.29; 3.00) Intermediate 0.63 (0.26; 1.54) *Men* Orchardist: 1.40 (0.54; 3.62) Intermediate: 1.87 (0.66; 5.34) **Stroke** *Women* Orchardist: 0.95 (0.33; 2.36) Intermediate: 1.02 (0.52; 1.98) *Men* Orchardist: 1.65 (0.65; 4.20) Intermediate: 2.18 (0.72; 6.57)	

			Orchardists were individuals who prepared and applied lead arsenate spray during the 1938 growing season. lntermediates included individuals who had not used lead arsenate spray during 1938 or who had infrequent exposure to lead arsenate sp ray. This group included retired orchardists who had long histories of using the· spray prior to 1938, as well as warehouse workers who had experienced infrequent exposures			
Tolonen et al 1979 [153] Finland *Note: same population as in the 2*	Cohort study 5-year follow-up Viscose plant 1942–1975	Participants were male workers in a viscose rayon plant The exposed cohort	**Carbon disulphide** The concentrations of carbon disulphide and hydrogen sulphide in the	**Coronary heart disease** The causes of deaths were verified from death certificates and classified	A 5-year follow-up showed that more men had died from coronary heart disease in the exposed group compared to the control group ($p<0.007$). Other causes of death were evenly distributed	–

articles by Hernberg et al and the article by Nurminen		comprised 343 men with at least 5 years' exposure to carbon disulphide during any period between 1942–1967. All of the men were or had been employed by the same viscose rayon plant. They were all individually matched with workers from a nearby paper-mill by age, district of birth, and type of work n=686 (343 exposed workers and 343 controls) All participants were men	workroom air had been measured regularly since 1950, and about 4 000 measurements were available. In all probability the concentrations had been very high in the 1940s, between 20–40 ppm in the 1950s, and about 10–30 ppm from 1960 onwards.	according to ICD-8	In addition, more nonfatal first infarctions had occurred in the exposed group as compared to the control group On re-examination in 1972, more of the exposed men, as compared to the controls, had a history of angina ($p<0.0002$). The mean systolic and diastolic blood pressures were slightly higher in the exposed group ($p<0.001$ and $p<0.01$, respectively) The relative risk was 4.8 for fatal attacks, 3.7 for all infarctions, 2.8 for nonfatal infarctions, 2.2 for angina, and 1.4 for ECG findings indicative of coronary heart disease	
Toren et al 2007 [154] Sweden	Retrospective cohort 32 years Construction 1971–2002	Participants were male construction workers Persons who emigrated were excluded. Those examined at	**Several chemicals** The definition of exposure to inorganic dust (asbestos, man-made mineral fibres, dust from cement,	**Mortality to ischaemic heart disease and cerebro-vascular disease** Mortality data was gathered from the	Mortality from ischaemic heart disease and cerebrovascular disease. RR (95% CI) adjusted for smoking, age, hypertension and body mass index **Ischaemic heart disease** Occupational exposure to particulate air pollution: 1.12 (1.10; 1.14) Inorganic dust: 1.13 (1.10; 1.16)	Poisson regression models. In addition to the listed predictors, the models were adjusted for age using 13 5-year categories (20–84 years). RR (95% CI) **Ischaemic heart disease** Inorganic dust: 1.07 (1.03; 1.12) Gases and irritants: 1.03 (0.97; 1.09) Fumes: 1.05 (1.00; 1.10)

		baseline before the age of 15 or after the age of 67 years were likewise excluded n=248 087 All participants were men	concrete and quartz), wood dust, fumes (metal fumes, asphalt fumes and diesel exhaust) and gases and irritants (organic solvents and reactive chemicals) was based on a job-exposure matrix with focus on exposure in the mid-1970s The occupational title at the time of the first health examination was used. A job exposure matrix was developed. The matrix was based on exposure estimations by industrial hygienists, where each occupation was studied at visits to occupational sites	National Cause of Death Register Diagnoses was based on the ICD, categories 410–412 (ICD-9) and I21–I25 (ICD-10) for ischaemic heart disease and categories 430–438 (ICD-9) and I60–I69 (ICD-10) for cerebro-vascular disease	Gases and irritants: 1.12 (1.07; 1.17) Fumes: 1.12 (1.08; 1.16) Diesel exhaust: 1.18 (1.13; 1.24) Asphalt fumes: 1.12 (0.96; 1.30) Metal fumes: 1.01 (0.95; 1.08) Wood dust: 1.12 (1.04; 1.20) **Cerebrovascular diseases** Occupational exposure to particulate air pollution: 0.97 (0.93; 1.01) Inorganic dust: 0.97 (0.92; 1.02) Gases and irritants: 0.98 (0.89; 1.07) Fumes: 1.03 (0.95; 1.11) Diesel exhaust: 1.09 (0.99; 1.20) Asphalt fumes: 1.18 (0.86; 1.58) Metal fumes: 0.92 (0.80; 1.05) Wood dust: 0.91 (0.79; 1.04)	Wood dust: 1.01 (0.93; 1.10) **Cerebrovascular disease** Inorganic dust: 0.95 (0.87; 1.04) Gases and irritants: 1.05 (0.94; 1.17) Fumes: 1.09 (0.95; 1.26) Wood dust: 0.86 (0.72; 1.02)

Tsai et al 1996 [155] USA *Note:* same sample as in the article by Enterline et al 1990, but with longer time to follow-up	Prospective cohort study Oil Company plants 1948–1993	Participants were workers who had at least 3 months of employment where exposure to epichloro-hydrin could have occured. Some of the exposed workers also had a potential exposure to allyl chloride n=863 (exposed group) All participants were men	**Epichlorohydrin** Potential exposure to epichlorohydrin Based on each worker's job with the highest potential level of exposure, a panel of industrial hygiene personnel and current and former employees assigned every employee into 1 of the 5 potential exposure categories: heavy, moderate, light, none, or unknown	**Cerebro-vascular disease and heart diseases, mortality** Data was obtained from the national death index and the Social Security Administratio n Cause of death was coded according to ICD-8: cerebrovascul ar disease (codes 430–438) and all heart disease (codes 390–398, 400, 401, 404 and 410–414)	Mortality in workers exposed to epichlorohydrin. SMR (95% CI) **Cerebrovascular disease** All: 66.7 (28.8; 131.4) *≥20 years since first potential exposure* All: 41.3 (11.2; 105.7) None to light exposure: 24.3 (6.2; 135.5) Moderate/heavy exposure: 62.1 (12.8; 181.4) **All heart disease** All: 64.5(50.7; 81.8) *≥20 years since first potential exposure* All: 63.3 (48.3; 82.9) None to light exposure: 59.5(37.7; 89.3) Moderate/heavy exposure: 75.7 (51.8; 106.7)	–
Wang et al 2013 [156] China	Prospective cohort Follow-up was 37 years Chrysotile textile factory 1972–2008	Participants were workers at an asbestos textile factory in China, where only chrysotile was used since 1958 to manufacture asbestos textiles, friction and rubber	**Chrysotile asbestos** The concentrations of dust and fibers measured at different workshops periodically were generally far higher than the Chinese	**Several conditions** The expected number of deaths was calculated based on person years multiplied by the Chinese nationwide gender-and	Standardized mortality ratios for non-malignant diseases in asbestos textile worker cohort, China, 1972–2008. SMR (95% CI) Pulmonary heart disease Women: 8.33 (2.29, 30.39) based on 2 cases Men:13.09 (9.46, 18.12) based on 36 cases Other diseases of the heart Women: 1.02 (0.35, 2.99) based on 3 cases Men: 0.87 (0.52, 1.43) based on 15 cases	–

		materials, and construction materials, such as asbestos cement and tiles In addition to detailed information on workers' occupational history and personal data that were collected from either personnel records or individual contacts, workers' vital status was verified by using a combination of active follow-up and record linkages to death certificates kept in the factory n=854 277 women 577 men	national standards. A measurement conducted in 2002 indicated that the asbestos fiber concentrations in air samples were 18 f⁄cm^3 in the raw material section, and 6 f⁄cm^3 in the textile section; the fiber concentrations in personal samples were 6 and 8 f⁄cm^3 in the 2 sections, respectively. Analysis of available chrysotile samples by X-ray diffraction and transmission electron microscopy indicated a very low level of tremolite contamination	cause-specific mortality rates adjusted for 5-year age groups For those deceased, the date and underlying cause of death were retrieved and verified from hospitals and a local death registry. All causes of deaths were coded according to ICD-10 Pulmonary heart disease: I26 Other diseases of the heart: I11, I09, I25, I50 Cerebro-vascular diseases: I62–63	Cerebrovascular diseases Women: 0.28 (0.05, 1.61) based on 1 case Men: 0.98 (0.65, 1.48) based on 22 cases	

Weiss et al 1988 [157] USA	Prospecitive cohort 5 years Police 1969–1975	Participants were policemen at the Boston Police Department n=89 70 individuals provided 162 pairs of data (consecutive examinations) for the regression All participants were men	**Lead** Blood lead values (in µg/100 mL) were collected only in year 2. Based on the distribution of blood lead values in our sample and in that of the United States population, blood lead values were divided into high (≥30 µg/100 mL) and low (≥20 and ≤30 µg/100 mL) groups for purposes of the regression analysis. These 2 groups were compared with our reference group in which values were <20 µg/100 mL	**Blood pressure** Blood pressure (in mm Hg) was recorded in years 2 through 5. The mean of triplicate measures of systolic pressure and diastolic pressure at each visit was used for analysis	Relationship of systolic blood pressure in Boston policemen at time t to prior systolic blood pressure, body mass index, age, smoking, and blood lead. Coefficient, SE, p **Blood lead** Low (20–29 µg/100 mL) 0.224, 2.251 p=0.921 High (≥30 µg/100 mL) 5.804, 2.748 p=0.036	Relationship of systolic blood pressure in Boston policemen at time t to independent variables excluding influential points **Blood lead** Low (20–29 µg/100 mL) –1.415, 2.233 p=0.527 High (≥30 µg/100 mL) 4.467, 2.672 p=0.097
Welch et al 1982 [158] USA	Cohort study Copper smelter 1943–1977	Participants were employees at a copper smelter The sample selected from 2 composite categories	**Arsenic** Departments with similar concentrations were combined into 4 categories of exposure: 1) low (<100	**Ischemic Heart disease** Death certificates were obtained from county. and state vital statistics	Mortality for smelter workers by TWA and Ceiling arsenic categories defined as of entry into cohort. SMR **Ischemic Heart disease** *TWA (time weighted average)* <100: 126, significant at 0.05 level 100–500: 135, significant at 0.01 level 500–4 999: 169, significant at 0.01 level	–

		(heavy and other). Men in the heavy exposure category had spent at least 24 months in the arsenic kitchens, Cottrells, or arsenic roaster. Men in the other group might have worked in these departments for less than 24 months, as well as in departments where arsenic concentrations were categorized as medium or light. All men in the heavy category and a 20% random sample of the rest of the group were selected n=1 800 All participants were men	µ.g/m^3, 2) medium (100–499 µg/m^3), 3) high (500–4 999 µg/m^3) and 4) very high (≥5 000 µg/m^3). 3 indices of individual arsenic exposure were developed: time-weighted average, 30-day ceiling, and cumulative	departments to ascertain the exact date and cause of death, and were. coded·by nosologists Mortality was compared to that of men in the State of Montana using the modified lifetable-method	≥5 000: 148, significant at 0.05 level *Ceciling level* Low (<100): 108, n.s Medium (100–499):126, n.s. High (500–4 999):159, significant at 0.01 level Very hig (≥5 000): 171, significant at 0.01 level	

Vena et al 1998 [159] 12 Countries	Prospective cohort The follow-up was conducted according to a similar methodology in the different countries and extended from 1939–1992 but varied by cohort Workers exposed to certain chemicals 1939–1992	Participants were workers exposed to phenoxyacid herbicides and chlorophenols The cohort included any worker ever employed in production or spraying of phenoxyacid herbicides except those in selected cohorts for which minimum employment periods were specified n=21 863 Both women and men participated, but the number of participants of each sex is not specified	**Dioxin** Exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin or higher chlorinated dioxins (TCDD/HCD) was discerned from job records and company questionnaires	**Several outcomes** Underlying cause of death was retrieved from the death certificate records in each country and coded according to the revision of ICD in effect at the time of death Diseases of the circulatory system was defined as ICD-9 code 390–459, ischemic heart disease was code 410–414, cerebrovascular disease was coded 430–438 and other diseases of the heart was code 415–429	Poisson regression analyses of mortality from selected causes among workers in the IARC international cohort study and indices of exposure to TCDD or HCD, 1939–1992. RR (95% CI) adjusted for age, gender, country, calendar period, employment status, and years since first exposure and duration of exposure to phenoxy herbicides or chlorophenols **All circulatory disease** ***Exposure to TCDD/HCD*** Yes: 1.51 (1.17; 1.96) ***Duration of TCDD/HCD exposure, years*** <1: 1.00 1–4: 1.16 (0.98; 1.38) 5–9: 1.32 (1.08; 1.60) 10–19: 1.28 (1.05; 1.55) ≥20: 0.96 (0.73; 1.27) **Ischemic heart disease** ***Exposure to TCDD/HCD*** Yes: 1.67 (1.23; 2.26) ***Duration of TCDD/HCD exposure, years*** <1: 1.00 1–4: 1.05 (0.86; 1.29) 5–9: 1.17 (0.92; 1.48) 10–19: 1.21 (0.96; 1.53) ≥20: 0.98 (0.70; 1.36) **Cerebrovascular disease** ***Exposure to TCDD/HCD*** Yes: 1.54 (0.83; 2.88) ***Duration of TCDD/HCD exposure, years*** <1: 1.00 1–4: 1.13 (0.70; 1.82) 5–9: 1.39 (0.83; 2.32) 10–19: 1.22 (0.72; 2.08) ≥20: 0.30 (0.10; 0.91)	–

					Standardized mortality ratios for major causes of death by gender among workers in the IARC international cohort study exposed to phenoxy herbicides or chlorophenols, 1939–1992. SMR (95% CI) **Women** Circulatory system disease: 1.00 (0.73; 1.32) Ischemic heart disease: 1.07 (0.68; 1.59) Cerebrovascular disease: 0.73 (0.33; 1.38) Other diseases of the heart: 0.92 (0.34; 2.00) **Men** Circulatory system disease: 0.91 (0.87; 0.95) Ischemic heart disease: 0.92 (0.87; 0.98) Cerebrovascular disease: 0.86 (0.76; 0.97) Other diseases of the heart: 1.11 (0.95; 1.29) Standardized mortality ratios (1939–1992) for specific causes among workers in the IARC international cohort study exposed to phenoxy herbicides or chlorophenols by exposure to TCDD or HCD. SMR (95% CI) **Workers exposed to TCDD/HCD** Circulatory system disease: 0.94 (0.88; 0.99) Ischemic heart disease: 0.97 (0.90; 1.04) Cerebrovascular disease: 0.84 (0.71; 0.98) Other diseases of the heart: 1.20 (1.01; 1.42)	
Veremulen et al 1978 [160] Belgium	Cohort study Silicosis patients	Participants were all ambulatory patients who were examined at the Fund od Occupational Diseases since the mechano-graphical records were	**Silicosis** Patients had radiological signs of silicosis of at least stage m (ie. rounded opacities exceeding about 1.5 mm diameter extended over	**Electrocardio-graphic signs of chronic cor pulmonale** The electro-cardiogram was read by several cardiologists and the results were	A correlation between the duration of exposure and chronic cor pulmonale could not be found The authors concluded that chronic cor pulmonale is an infrequent and mostly late complication of silicosis	–

		used in 1972, in whom the diagnoses of silicosis was made and a complete record was obtained n=40 376 Gender not stated	at least 2 intercoastal spaces)	recordedin a standardized way on the optical reading sheet. On this sheet several objective data including the signs of chronic cor pulmonale were recorded. In the last section the cardiologist must express his opinion as to the presence and severity of chronic cor pulmonale		
Wiebert et al 2012 [161] Sweden	Prospective cohort 18 years Manual work 1987–2005	Participants were all manual workers in the Swedish National Census 1980, who were alive on 1 January 1987. White-collar workers, professionals, self-employed and farmers were not included n=1 725 671	**Particles** Information on occupation was obtained from the population censuses in 1980, 1985 and 1990. A job exposure matrix was developed by the authors by combining relevant occupational particle exposure information	**Heart disease** First-time events of acute myocardial infarction (ICD-9 code 410 and ICD-10 code I21) or other ischemic heart diseases (ICD-9 codes 411–414 and ICD-10 codes I20, I22–I25) during 1987–	Hazard ratios for heart disease in occupations were at least 80% of the workers are exposed to small (<1 µm) or large (>1 µm) particles. HR (95% CI). Data for women and men are adjusted for age and particle size. Data for all workers are also adjusted for sex **All - Acute myocardial infarction** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.12 (1.09; 1.16) Large particles: 1.16 (1.13; 1.19) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.22 (1.14; 1.32) Large particles: 1.17 (1.11; 1.24) **Women - Acute myocardial infarction**	Hazard ratios for heart disease in occupations were at least 80% of the workers are exposed to small (<1 µm) or large (>1 µm) particles. HR (95% CI). Data for women and men are adjusted for age, sex, particle size, socioeconomic group and residential area. Data for all workers are also adjusted for sex **All - Acute myocardial infarction** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.12 (1.09; 1.15) Large particles: 1.14 (1.10; 1.18) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.21 (1.11; 1.31)

		741 631 women 984 040 men	from a Swedish job exposure matrix developed for the Nordic occupational cancer study and airway-irritant job exposure matrix	2005 were identified through linkage to the Hospital Discharge Register and the National Cause of Death Register	***Ever exposed (unexposed: 1.00)*** Small particles: 1.30 (1.12; 1.51) Large particles: 1.22 (1.18; 1.27) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.37 (0.89; 2.10) Large particles: 1.30 (1.18; 1.42) **Men - Acute myocardial infarction** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.10 (1.06; 1.13) Large particles: 1 19 (1.07; 1.14) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.16 (1.07; 1.26) Large particles: 1.11 (1.04: 1.19) **All - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.13 (1.10; 1.16) Large particles: 1.11 (1.09; 1.13) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.25 (1.17; 1.33) Large particles: 1.14 (1.08; 1.21) **Women - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.24 (1.11; 1.39) Large particles: 1.13 (1.10; 1.16) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.20 (0.88; 1.64) Large particles: 1.22 (1.14; 1.30) **Men - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.11 (1.09; 1.14) Large particles: 1.10 (1.05; 1.11) ***Exposed ≥5 years (unexposed: 1.00)***	Large particles: 1.14 (1.06; 1.23) **Women - Acute myocardial infarction** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.30 (1.12; 1.51) Large particles:1.17 (1.09: 1.26) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.50 (0.95; 2.37) Large particles: 1.39 (1.17; 1.66) **Men - Acute myocardial infarction** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.10 (1.07; 1.14) Large particles: 1.13 (1.08; 1.18) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.18 (1.07; 1.29) Large particles: 1.11 (1.02; 1.21) **All - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.13 (1.11; 1.16) Large particles: 1.12 (1.09; 1.51) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.23 (1.17; 1.31) Large particles: 1.14 (1.10; 1.19) **Women - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.24 (1.10; 1.39) Large particles: 1.12 (1.06; 1.18) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.34 (0.96; 1.86) Large particles: 1.33 (1.18; 1.51) **Men - Ischemic heart disease** ***Ever exposed (unexposed: 1.00)*** Small particles: 1.12 (1.08; 1.15)

					Small particles: 1.23 (1.14; 1.32) Large particles: 1.12 (1.05; 1 20)	Large particles: 1.12 (1.09; 1.16) ***Exposed ≥5 years (unexposed: 1.00)*** Small particles: 1.19 (1.12; 1.27) Large particles: 1.10 (1.04; 1.16)
Wilcosky et al 1983 [162] USA	Prospective cohort study 15 year follow-up Rubber industry 1921–1978	Participants were white male production workers in a large rubber- and tire-manufacturing plant. The workers had to be between 40 and 80 years of age and working on the plant or had retired following at least 10 years of employment n=1 282 All participants were men	**Solvents** 25 different solvents had been identified to exist in the plant during the period 1921–1976. Exposure to different solvents was determined by combining information on starting and edning date of the job, department code, job title and the solvent charts	**Ischemic heart disease mortality** The Occupational Health Studies Group followed the cohort and a trained nosologist coded all deaths under the ICD-8	Mortality during 1964–1978 for workers exposed in the rubber industry. Rate ratio, χ^2, p-value, adjusted for age *Ischemic Heart disease* Carbon disulphide: 0.9, 0.32, n.s Gasoline: 1.3, 4.28, p<0.05 Ethanol: 1.4, 5.69, p<0.05 Phenol: 1.6, 9.20, p<0.01 Perchloroethylene: 0.4, 5.05, p<0.05 Methylene Cholride: 1.0, 0.01, n.s Mineral Spirits: 1.5, 1.44, n.s	–

Virtanen et al 2002 [163] Finland	Prospective cohort. Data from the Finnish Longitudinal Census file 13 years Working men 1981–1994	Participants were between 25–64 years in 1980. They had the same occupation in both 1975 and 1980 Mining work, military work and agricultural work were excluded n=507 000 All participants were men	**Several occupational factors** Data on working condition came from a job exposure matrix developed by the Finnish Institute for occupational health Data on occupation was assessed by a questionnaire developed within the Finnish Longitudinal Census study	**Cardio-vascular death** Causes of death were retrieved from the national register and the Finnish translation of the ICD-9 was used for disease classification Cardio-vascular death included acute myocardial death (codes 390–459), acute myocardial infarction (410) and cerebrovascular deaths (430–438)	Rate ratio of work exposure on mortality. Disease group/exposure variable level. RR (95% CI) **All cardiovascular diseases** ***Chlorinated hydrocarbon solvents*** Low, unexposed:1.00 High: 1.09 (0.98; 1.21) ***Cadmium (unexposed: 1.00)*** High: 1.01 (0.93; 1.10) ***Diesel exhaust (unexposed:1.00)*** Exposed: 1.06 (1.00; 1.14) ***Lead (unexposed:1.00)*** Low: 1.00 (0.93; 1.08) High: 1.12 (1.03; 1.23) **Myocardial infarctions** ***Chlorinated hydrocarbon solvents*** Low, unexposed:1.00 High: 1.09 (0.95; 1.25) ***Diesel exhaust (unexposed:1.00)*** Low: 1.07 (0.95; 1.20) High: 1.09 (0.95; 1.24) ***Lead (Unexposed:1.00)*** Low: 1.01 (0.93; 1.10) High: 1.13 (1.00; 1.28) **Cerebrovascular disease** ***Arsenic*** Low, unexposed:1.00 High: 1.04 (0.75; 1.45) ***Cadmium (unexposed: 1.00)*** High: 1.07 (0.91; 1.24) ***Diesel exhaust (unexposed:1.00)***	–

					Exposed: 1.12 (0.97; 1.29) ***Lead (low, unexposed:1.00)*** High: 1.24 (1.00; 1.55) ***Organic solvents (low, unexposed:1.00)*** High: 1.11 (0.92; 1.35)	
Wong et al 1994 [164] USA	Cohort Reinforced plastics and composites industry 1948–1989	The cohort consisted of male and female employees who were exposed to styrene for at least 6 months between 1948–1977 at 30 participating manufacturing plants in the United States n=15 826	**Styrene** Monomer and other chemicals Effect at the time of death. Person-years of observation started after 6 months of exposure to styrene, and ended on the date of death or 31 December 1989 (whichever was earlier) The first year of styrene use ranged from 1948–1968 A cumulative exposure in ppm-years, calculated as	**Several** Causes of death were coded according to the revision of the International Classification of Diseases (ICD) in effect at the time of death Expected deaths were based on United State national age-sex-cause-race-year-specific death rates	Observed and expected deaths by cause, SMR (95% CI) for all cohort members Cerebrovascular disease: 111.4 (87.0; 140.6) All heart disease: 101.0 (92.3; 110.4) Ischaemic heart disease: 103.7 (93.7; 114.4) Chronic endocardial disease; other myocardial insufficiency: 126.1 (72.1; 204.8) Hypertension with heart disease: 185.9 (110.2; 293.8) All other heart disease: 76.8 (57.3; 100.7) Observed deaths and SMRs by cause and duration of exposure to styrene for all cohort members Cerebrovascular disease <1 year: 131.5 1.1–1.9 years: 92.8 2–4.9 years: 111.0 5–9.9 years 82.1 >10 years: 135.5 All heart disease <1 year: 121.1 1.1–1.9 years: 105.4 2–4.9 years: 110.3 5–9.9 years 86.0	–

			the sum of products of time-weighted average and duration of exposure of each job, was developed for each cohort member		>10 years: 84.9 Ischaemic heart disease <1 year: 129.4, p<0.05 1.1–1.9 years: 113.8 2–4.9 years: 108.7 5–9.9 years 85.8 >10 years: 85.6 Chronic endocardial disease; other myocardial insufficiency <1 year: 89.2 1.1–1.9 years: 95.8 2–4.9 years: 276.0, p<0.05 5–9.9 years 82.7 >10 years: 65.8 Hypertension with heart disease <1 year: 283.9 1.1–1.9 years: 59.1 2–4.9 years: 216.3 5–9.9 years 165.1 >10 years: 190.6 All other heart disease <1 year: 70.7 1.1–1.9 years: 66.7 2–4.9 years: 90.1 5–9.9 years: 80 9 >10 years: 72.7 Observed deaths and SMRs by cause and cumulative styrene exposure (ppm-years) for all cohort members Cerebrovascular disease <10.0: 101.9 10.0–29.9: 95.8 30.0–99.9: 103.0 >100.0: 141.9	

					All heart disease <10.0: 117.5 10.0–29.9: 96.7 30.0–99.9: 101.4 >100.0: 91.0 Ischaemic heart disease <10.0: 121.6 10.0–29.9: 104.9 30.0–99.9: 104.7 >100.0: 86.9 Chronic endocardial disease; other myocardial insufficiency <10.0: 75.5 10.0–29.9: 140.1 30.0–99.9: 109.2 >100.0: 170.3 Hypertension with heart disease <10.0: 278.1, p<0.05 10.0–29.9: 90.3 30.0–99.9: 109.8 >100.0: 271.7, p<0.05 All other heart disease <10.0: 82.5 10.0–29.9: 52.0 30.0–99.9: 89.6 >100.0: 79.7 In general, results for both men and women were similar and, therefore, results are not presented separately by sex Authors conclude that the increased mortality was not likely to be related to exposure to styrene	

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