**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| ***Ore mining*** | | | | | | | | |
| McLaughlin *et al*. (1992) China | Lung | 254 metal miner cases in a case control study of metal mines and potteries nested in the cohort study by Chen *et al*. (1992). Cohort defined by employment 1972–74 and followed up to 1989. Information on tobacco use, medical history, and demographics by questionnaire. In original cohort study vital status and cause of death obtained for 95 and 87% respectively | 1118 controls matched on decade of birth and mine or factory in same cohort who survived the case | Quantitative estimates of cumulative respirable silica dust exposures (μg-yr/$m^3$) and confounders based on company industrial hygiene records and special sampling | **Cumulative silica dust exposures:**<br>***Tungsten***<br>0 (μg-yr/$m^3$)<br>0.1–8.69<br>8.70–26.2<br>≥ 26.3<br>***Iron-copper***<br>0 (μg-yr/$m^3$)<br>0.1–8.69<br>8.70–26.2<br>≥ 26.3<br>***Tin***<br>0 (μg-yr/$m^3$)<br>0.1–8.69<br>8.70–26.2<br>≥ 26.3 | <br><br>1.0<br>1.4 (0.88 – 2.14)<br>1.1 (0.69 – 1.64)<br>0.5 (0.32 – 0.74)<br>(trend $P$ = 0.01)<br><br>1.0<br>1.3 (0.88 – 1.83)<br>1.3 (0.81 – 2.0)<br>1.3 (0.81 – 1.56)<br>(trend NS)<br><br>1.0<br>1.5 (0.89 – 2.47)<br>1.9 (1.19 – 2.90)<br>3.1 (2.12 – 4.23)<br>(trend $P$ = 0.004) | Smoking but not occupational confounders | Trend with increasing exposure found for tin only. Trend also found with arsenic exposure. Silica, arsenic, and PAH exposures highly correlated. Exposures to asbestos, nickel, talc, and cadmium reported to be minimal. |
| Reid & Sluis-Cremer (1996) South Africa | Lung | 159 gold miner cases nested within a cohort of 4 925 gold miners born 1916–1930 with mortality follow-up 1970–1989 | 318 controls matched 2:1 on cases by birth year | Quantitative assessment of cumulative respirable dust exposures (mg/$m^3$) from converted particle count measurements | **Cumulative dust exposure up to 5 years before death of case:**<br>Continuous | <br>1.19 (0.97–1.70) | Smoking | No arsenic in the dust. Potential exposure to radon. |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| Hnizdo *et al*. (1997) South Africa | Lung | 78 cases among gold miners examined medically 1968 – 72, aged 45 – 54, with ≥ 10 years working in gold mines and minimal exposure in other mines | 386 controls, matched 5:1 by year of birth and survival of the case | Quantitative estimates of respirable mixed dust exposure derived from personal measurements (mg-yr/$m^3$). Uranium exposures derived similarly | **Cumulative mixed dust exposures:** *Unadjusted for silicosis*<br>< 2.7 (mg-yr/$m^3$)<br>2.7 – 4.3<br>4.4 – 6.3<br>≥ 6.3 | <br>1.0<br>1.83 (0.8 – 0.41)<br>1.85 (0.8 – 4.3)<br>3.19 (1.3 – 7.6) | Adjusted for smoking and uranium production but not for radon | No association with uranium production. Postulated that silica might be a surrogate for radon exposure |
| Carta *et al*. (2001) Italy | Lung | 34 cases in a nested case control study of a cohort of Sardinian miners and quarrymen who had radiologically-defined silicosis between 1964 and 1970. Vital status to 1997, 98% traced, cause of death for 100% | Controls matched 4:1 from the cohort on year of birth (2 years) and survival of the case | Quantitative estimates of respirable mixed dust exposure derived from personal measurements (g-hr/$m^3$). Radon exposures derived similarly | **Cumulative total dust exposures:**<br>≤ 10 (g-hr/$m^3$)<br>> 10<br>Linear continuous | <br>1.0<br>1.30 (0.71 – 2.68)<br>1.003 (NS) | Smoking, airflow obstruction, radon, silicosis severity | Small study and limited range of exposure caused by restriction to silicotics may have reduced study power, although effects observed for radon and airflow obstruction |
| Chen & Chen (2002) China | Lung | 130 (M) cases employed in 4 tin mines at least one year between 1972 and 1974. Based on extended follow-up of tin miners in Chen *et al*. (1992) to 1994. See entry for McLaughlin *et al*. (1992) in this table for details on the cohort. | 627 controls, matched ~5:1 on decade of birth and mine, and living at time of case diagnosis | Quantitative estimates of total mixed dust exposures and confounders derived from company industrial hygiene records and special sampling; mean(sd) = 112.4 (92.9) mg-yr/$m^3$ (cases), 98.6 (75.4) (controls) | **Cumulative total dust exposures:** ***Unadjusted for silicosis***<br>< 0.1 (mg-yr/$m^3$)<br>0.1 – 14.9<br>50 – 119.9<br>≥ 120 | <br>1.0<br>2.1 (1.1 to 3.8)<br>1.7 (0.9 to 3.1)<br>2.8 (1.6 to 5.0) | Smoking | Exposures to radon were low and no carcinogenic PAHs were detected. Correlation between arsenic and silica exposures prevented adjustment for arsenic |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| Chen *et al*. (2007) China | Lung | 391 (M) metal miner cases in a case control study of metal mines and potteries nested in the cohort study by Chen *et al*. (1992) with an extended follow-up to 1994. See entry for McLaughlin *et al*. (1992) in this table for details on the cohort. | 1420 (M) matched controls selected by density sampling ~4:1 before 1989 and 3:1 thereafter by decade of birth and mine or factory | Quantitative estimates of cumulative respirable silica dust exposures and confounders based on company industrial hygiene records and special sampling; mean = 4.8 mg-yr/$m^3$ (tungsten), 2.6 (tin), 0.2 (iron/copper) | **Cumulative silica dust exposures:**<br>***Tungsten***<br>0 (mg-yr/$m^3$)<br>0.1 – 1.1<br>1.1 – 2.6<br>2.6 – 5.4<br>5.4 – 10.1<br>10.1 – 72.4<br>***Iron-copper***<br>0 (mg-yr/$m^3$)<br>0.1 – 1.1<br>1.1 – 2.6<br>2.6 – 5.4<br>5.4 – 10.1<br>10.1 – 72.4<br>***All***<br>0 (mg-yr/$m^3$)<br>0.1 – 1.1<br>1.1 – 2.6<br>2.6 – 5.4<br>5.4 – 10.1<br>10.1 – 72.4 | <br><br>1.0<br>2.0 (0.97 – 4.19)<br>1.4 (0.64 – 2.81)<br>0.6 (0.32 – 1.30)<br>0.8 (0.42 – 1.51)<br>1.0 (0.55 – 1.66)<br><br>1.0<br>0.7 (0.24 – 2.08)<br>1.0 (0.31 – 3.28)<br>1.4 (0.33 – 5.50)<br>-<br>-<br><br>1.0<br>1.40 (0.81 – 2.43)<br>1.54 (0.90 – 2.63)<br>1.30 (0.75 – 2.24)<br>1.18 (0.68 – 2.06)<br>1.50 (0.83 – 2.72) | Smoking, inorganic arsenic, radon, and 'carcinogenic PAHs' | Silica, arsenic, and PAH exposures highly correlated. (0.57–0.80, 0.83, and 0.55 for PAHs, arsenic, and raon). Analysis adjusting only for smoking showed exposure-response, but not after adjustment for other confounders. Tin worker results could not be adjusted for arsenic because of collinearity. No examination of silicosis. The 'All' group includes data from pottery workers. Continuous variable for silica exposure was non-significant in all models |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| ***Ceramics*** | | | | | | | | |
| McLaughlin *et al*. (1992) China | Lung | 62 pottery worker cases in a case control study of metal mines and potteries nested in the cohort study by Chen *et al*. (1992). Cohort defined by employment 1972 – 74 and followed up to 1989. Information on tobacco use, medical history, and demographics by questionnaire. In original cohort study vital status and cause of death obtained for 95 and 87% respectively | 238 controls matched on decade of birth and mine or factory in same cohort who survived the case | Quantitative estimates of cumulative respirable silica dust exposures (μg-yr/$m^3$) and confounders based on company industrial hygiene records and special sampling | **Cumulative silica dust exposures:** ***Potteries*** 0 (μg-yr/$m^3$) 0.1 – 8.69 8.70 – 26.2 ≥ 26.3 | 1.0 1.8 (1.04 – 2.87) 1.5 (0.99 – 2.18) 2.1 (0.80 – 4.12) (trend NS) | Smoking, age | Silica and PAH exposures correlated, r = 0.56 in potteries. Adjustment for PAHs reported to raise the silca RRs. Exposures to asbestos, nickel, talc, and cadmium reported to be minimal |
| Cherry *et al*. (1998) Stoke on Trent, United Kingdom | Lung | 52 (M) cases born 1916–45, employed in the pottery, refractory, and sandstone industries, and included in a medical surveillance programme of dusty trades. Nested within a cohort study having 99% traced and cause of death for 97% | 197 (M) controls from same population as cases matched 3:1 or 4:1 on date of birth and date of first exposure (both achieved within 4 years). Never smokers, those whose entire silica exposure outside pottery industry, and those who died before case excluded | Quantitative estimates of cumulative respirable silica dust exposure (μg-yr/$m^3$), average intensity, and duration of exposure using work histories and measured counts converted to mass and later mass concentrations | **Cumulative silica dust exposures:** Linear continuous (μg-yr/$m^3$) **Average intensity (concentration):** Linear continuous (μg-yr/$m^3$) | 1.01 (0.85 – 1.19) 1.67 (1.13 – 2.47) | Smoking amount conditioned logistic regression | Silica exposures ranged from 800 down to 50 μg-yr/$m^3$ from the 1930s to the 1990s. All smokers. Lagged exposure results very similar to those with no lag. Some workers probably exposed at some time to cristobalite and/or tridymite. No mention of confounders |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| Ulm *et al*. (1999) Germany | Lung | 114 (M, F) non-silicotic ceramic worker cases from a medical monitoring programme for silicosis as part of a case control study of stone, quarry, and ceramic workers. Silicosis defined as ≥ 1/1 on ILO scale | 564 (M, F) controls frequency matched on sex, year or birth, and smoking, from same underlying population as cases. | Dust measurements and subjective assessments of silica dust levels combined with work histories to give cumulative exposures; median = 2.97 mg-yr/m$^3$ (cases), 2.88 (controls) | **Cumulative silica dust exposures:**<br>***Ceramics***<br>≤ 2.88 (mg-yr/m$^3$)<br>> 2.88<br>***All***<br>< 1.56 (mg-yr/m$^3$)<br>1.56 – 2.88<br>2.89 – 4.68<br>> 4.68 | <br><br>1.00<br>1.05 (0.59 – 1.86)<br><br>1.00<br>0.95 (0.48 – 1.53)<br>0.92 (0.44 – 1.61)<br>1.04 (0.53 – 1.89) | Asbestos, PAHs, radon, diesel exhaust, welding fumes, and heavy metals reported as recorded but unclear if all used in the modeling. Models also included age of exposure onset, year of first exposure, duration of exposure, and latency | Cases smoked more cigarettes than controls and ex-smokers among controls had stopped smoking earlier. No effect seen among ceramic workers for average exposure or peak exposure. The 'All' group includes stone and quarry workers |
| Chen *et al*. (2007) China | Lung | 120 (M) pottery worker cases in a case control study of metal mines and potteries nested in the cohort study by Chen *et al*. (1992) with an extended follow-up to 1994. See entry for McLaughlin *et al*. (1992) in this table for details on the cohort. | 459 (M) matched controls selected by density sampling ~4:1 before 1989 and 3:1 thereafter by decade of birth and mine or factory | Quantitative estimates of cumulative respirable silica dust exposures and confounders based on company industrial hygiene records and special sampling; mean = 2.1 mg-yr/m$^3$ | **Cumulative silica dust exposures:**<br>***Potteries***<br>0 (mg-yr/m$^3$)<br>0.1 – 1.1<br>1.1 – 2.6<br>2.6 – 5.4<br>5,4 – 10.1<br>10.1 – 72.4<br>***All***<br>0 (mg-yr/m$^3$)<br>0.1 – 1.1<br>1.1 – 2.6<br>2.6 – 5.4<br>5.4 – 10.1<br>10.1 – 72.4 | <br>1.0<br>0.7 (0.25 – 1.98)<br>0.7 (0.29 – 1.81)<br>0.7 (0.25 – 2.19)<br>0.5 (0.15 – 1.84)<br>0.9 (0.19 – 4.32)<br><br>1.0<br>1.4 (0.81 – 2.43)<br>1.54 (0.90 – 2.63)<br>1.30 (0.75 – 2.24)<br>1.18 (0.68 – 2.06)<br>1.50 (0.83 – 2.72) | Smoking, inorganic arsenic, radon, and 'carcinogenic PAHs' | Silica and PAH exposures (r = 0.57–0.80 in the overall study). Analysis adjusting only for smoking showed exposure-response, but not after adjustment for other confounders. Tin worker results could not be adjusted for arsenic because of collinearity. No examination of silicosis. The 'All' group includes data from metal mines. Continuous variable for silica exposure was non-significant in all models |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| ***Stone quarries*** | | | | | | | | |
| Ulm *et al*. (1999) Germany | Lung | 133 (M) non-silicotic quarry and stone worker cases as part of a case control study of stone, quarry, and ceramic workers. Silicosis defined as ≥ 1/1 on ILO scale | 231 (M) controls matched on smoking from a population of those involved in accidents at work or to and from work | Dust measurements and subjective assessments of silica dust levels combined with work histories to give cumulative exposures; median = 2.97 mg-yr/m$^3$ (cases), 2.88 (controls) | **Cumulative silica dust exposures:**<br>***Quarry & stone***<br>≤ 2.88 (mg-yr/m$^3$)<br>> 2.88<br>***All***<br>< 1.56 (mg-yr/m$^3$)<br>1.56 – 2.88<br>2.89 – 4.68<br>> 4.68 | <br><br>1.00<br>0.86 (0.38 – 1.95)<br><br>1.00<br>0.95 (0.48 – 1.53)<br>0.92 (0.44 – 1.61)<br>1.04 (0.53 – 1.89) | Asbestos, PAHs, radon, diesel exhaust, welding fumes, and heavy metals reported as recorded but unclear if all used in the modeling. Models also included age of exposure onset, year of first exposure, duration of exposure, and latency | Cases smoked more cigarettes than controls and ex-smokers among controls had stopped smoking earlier. No effect seen among ceramic workers for average exposure or peak exposure. The 'All' group includes ceramic workers |
| ***Sand & Gravel*** | | | | | | | | |
| McDonald *et al*. (2005) United States | Lung | 105 (M) cases among a cohort of sand and gravel workers who were employed between 1940 and 1979 for 3 years or more in 1 of 8 sand-producing plants and a large associated office complex. 99% traced; cause of death established for 100%. Extended follow-up of McDonald *et al*. (2001) | Up to two controls on plant, matched on age (5 years) and date of first employment (5 years), with survival past case | Quantitative estimates of cumulative respirable silica dust exposure (μg-yr/m$^3$) based on company work histories and industrial hygiene records | **Cumulative silica dust exposures:**<br>≤ 700 (μg-yr/m$^3$)<br>> 700 – ≥ 1 800<br>> 1 800 – ≥ 4 500<br>> 4 500 | <br>1.00<br>1.10<br>1.77<br>2.64 (trend *P* = 0.06) | Smoking | Slightly more significant but less monotonic trend for 15 year lag cumulative exposure. Significant trend for average exposure but not for years employed |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| Steenland & Sanderson (2001a) United States | Lung | 75 cases among a cohort of sand and gravel workers from 18 plants with > 6 months of employment from 1960 – 1988 with follow-up to 1996 | 100 controls per case matched on race, sex, and date of birth (5 years) | Quantitative estimates of cumulative respirable silica exposure (mg-yr/m$^3$) based on company work histories and industrial hygiene information | **Cumulative silica dust exposure:**<br>*Unlagged*<br>≤ 0.18 (mg-yr/m$^3$)<br>> 0.18- ≤ 0.59<br>> 0.59- ≤ 1.23<br>> 1.23<br><br>*Lagged 15 years*<br>≤ 0.18 (mg-yr/m$^3$)<br>> 0.18- ≤ 0.59<br>> 0.59- ≤ 1.23<br>> 1.23<br><br>**Average silica exposure:**<br>≤ 0.023<br>> 0.023- ≤ 0.046<br>> 0.046- ≤ 0.065<br>> 0.065 | <br>1.0<br>1.28 (0.65–2.51)<br>0.73 (0.36–1.49)<br>1.70 (0.88–3.25)<br>*P* = 0.04 for trend<br><br>1.0<br>1.35 (0.72–2.54)<br>1.63 (0.83–3.18)<br>2.00 (1.00–4.01)<br>*P* = 0.06 for trend<br><br>1.0<br>0.92 (0.42–2.00)<br>1.44 (0.72–2.86)<br>2.26 (1.17–4.38)<br>*P* = 0.003 for trend | None | |
| ***Other*** | | | | | | | | |
| Xu *et al*. (1996) China | Lung | 552 M and 58 F cases diagnosed 1987 – 93 at ages 30–70 with at least 10 years employment among current and former employees of a large iron and steel complex. Response rate was 95.8% | 1100 controls from same population group matched by gender and birth year. Response rate was 94.4% | Interviews used to obtain work histories, which were linked with extensive monitoring data to give cumulative total silica (mg-yr/m$^3$) and BaP exposures | **Cumulative silica dust exposures:**<br>Non-exposed<br>< 3.7 (mg-yr/m$^3$)<br>3.7 – 10.39<br>10.4 – 27.71<br>≥ 27.71 | <br>1.0<br>1.7 (1.2 – 2.4)<br>1.5 (1.0 – 2.1)<br>1.5 (1.0 – 2.1)<br>1.8 (1.2 – 2.5)<br>(trend *P* = 0.007) | Year and smoking but not BaP nor asbestos | Non-exposed reported to be those in administrative and low exposure jobs. Similar trend seen for total mixed dust. Clearer and slightly more significant trend for BaP. Correlation between BaP and silica exposures not reported |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| Watkins *et al.* (2002) United States | Lung | 31 WM and 8 BM cases among workers at 28 roofing and asphalt manufacturing facilities recorded in a mortality surveillance system for a large company | 110 WM and 23 BM controls from same programme who were not cases matched up to 4:1 by sex, race, and year of birth (2 years) | Quantitative estimates of cumulative respirable silica dust exposure (mg-days/$m^3$) based on company work histories and industrial hygiene records. Similar process for asphalt fumes | **Cumulative silica dust exposures:** ***Method I*** 0 (mg-days/$m^3$) 10 – 99.9 > 100 ***Method II*** 0 (mg-days/$m^3$) 1 – 9.9 10 – 99.9 > 100 | 1.00 1.53 (0.19 – 10.76) 1.31 (0.52 – 3.29) 1.00 0.70 (0 – 11.21) 0.46 (0.01 – 4.80) 1.49 (0.60 – 3.74) | Smoking and asphalt fumes | Small study with low exposures. Nearly all (> 90%) cases and controls fell into the 0 and > 100 groups. Main interest was in asphalt fumes, which showed no significant elevations in lung cancer risk |
| Westberg and Bellander (2003) Sweden | Lung | 31 cases among workers from 7 aluminum foundry hired before 1994 and with 1 year of employment | 233 controls matched 7:1 on sex and age | Quantitative estimates of cumulative respirable silica dust exposure (mg-days/$m^3$) based on company work histories and industrial hygiene samples modelled using foundry type, time period, production, and job title. | **Cumulative silica dust exposures:** ***Unlagged*** 0 – 0.001 (mg-days/$m^3$) 0.001 – 1.0 > 1.0 | RR 1.0 1.6 (0.54 – 4.6) 2.6 (0.67 – 9.2) | No adjustment for smoking or other confounders | Small study. Main focus of study was on exposure assessment |

**Table 2.2. Case–control studies of crystalline silica and lung cancer**

| Reference, study location and period | Organ site (ICD code) | Characteristics of cases | Characteristics of controls | Exposure assessment | Exposure categories | Relative risk (95% CI)* OR | Adjustment for potential confounders | Comments |
|---|---|---|---|---|---|---|---|---|
| *Pooled analysis* | | | | | | | | |
| Steenland et al. (2001b) United States, Finland, China, South Africa, Australia | Lung | 992 M & F cases nested within a pooled analysis of 10 cohorts of diatomaceous earth, granite, sand and gravel, and pottery workers, and tungsten, tin, and gold miners. 65 980 individuals and 15 171 deaths. Some contributing cohorts had extended follow-up beyond original publication | 100 controls per case matched on race, sex, date of birth (5 years) and study. Controls had to have survived case. Matching accounted for different country-specific background lung cancer rates. | Quantitative estimates of cumulative respirable silica exposure based on based on company work histories and industrial hygiene data.; median = 4.27 mg- yr/$m^3$. Methods differed by study | **Cumulative silica dust exposure:** ***Unlagged*** < 0.04 (mg-yr/$m^3$) 0.04 – 2.0 2.0 – 5.4 5.4 – 12.8 ≥ 12.8 *15 year lag* Log Continuous ln(mg-yr/$m^3$+1) | 1.0 1.0 (0.85–1.3) 1.3 (1.1–1.7) 1.5 (1.2–1.9) 1.6 (1.3–2.1) Coefficient = 0.06 2 ($P$ = 0.0001) ($P$ = 0.34 for heterogeneity across cohorts) | No adjustment for smoking or other confounders | Categorical 15 year lagged results similar to unlagged. Heterogeneity was evident except for log cumulative exposure lagged 15 years. Smoking noted not to be a major confounder in 5 of the studies. No difference when mines with potential confounders omitted. Results for mines and nonmines similar. Relationship with average intensity but not as strong as for cumulative exposure |

* specify *p*-value if no confidence interval indicated
• Study location includes city or region, and country.
• ICD codes to be given only for some cancers, incl. upper aerodigestive tract, colorectal, uro-genitary, leukaemia, lymphoma. ICD-9, unless otherwise specified.
• Characteristics of cases: number (men, women) – source: hospital/registry/death certificate – age range – response rate – histological confirmation (%).
• Characteristics of controls: number (men, women) – source: hospital/registry/death certificate – response rate – matching to cases (age range to be given if different from that of cases).
• Exposure assessment: e.g. mailed questionnaire – structured interview – job exposure matrix – biomarker.
• RR: consider the most valid point estimate (e.g. adjusted)
• Comments, if relevant for the interpretation of the study, such as: ethnicity – type of diseases for hospital controls – proportion of next-of-kin/proxies interviewed – stratified results/interaction