Cancer incidence and mortality among underground and surface goldminers in Western Australia

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Background: In a cohort of goldminers, we estimated cancer mortality and incidence, for both surface and underground workers, and we examined the hypothesis that (underground) mining may be protective against prostate cancer.

Methods: Standardised mortality and incidence ratios (SMRs and SIRs) and 95% confidence intervals (CI) were calculated to compare cancer mortality and incidence of former goldminers with that of the general male population. Internal comparisons on duration of underground work were examined using Cox regression.

Results: During 52,608 person-years of follow-up among 2294 goldminers, 1922 deaths were observed. For any cancer, mortality was increased for the total group of miners (SMR = 1.27, 95% CI 1.16–1.39). In the Cox models, lung cancer mortality and incidence were particularly increased among underground miners, even after adjustment for smoking. The SMR for prostate cancer suggested a lower risk for underground miners, whereas incidence of prostate cancer was significantly increased (SIR = 1.31, 95% CI 1.07–1.60) among underground miners.

Conclusion: Overall cancer mortality and incidence was higher among Western Australian goldminers compared with the general male population, particularly for underground mining. This study does not support the hypothesis that miners have a decreased risk of prostate cancer.

Miners have been identified with a lower risk of prostate cancer in recent publications (Girschik et al, 2010; Walsh et al, 2012). The effect appeared to be more pronounced for underground miners. The potential explanations that were put forward by Girschik et al (2010) for the group of miners as a whole were a healthy worker effect or the higher level of physical activity among miners compared with the general population. While the first is unlikely for prostate cancer, the latter would not result in different risks for surface or underground miners. A third explanation, the ‘melatonin hypothesis’, was postulated that would explain lower risk for underground miners. Both surface and underground miners commonly do night shifts, but the lack of ambient light exposure among underground miners may result in increased levels of melatonin (Girschik et al, 2010). This hypothesis was supported by findings in a German cohort of uranium miners (Walsh et al, 2012).

On the other hand, the numbers of years worked in hard rock underground mines have been associated with an excess lung cancer risk (Hodgson and Jones, 1990; Chau et al, 1993; De Klerk et al, 1995). Underground mining is characterised by high exposures to diesel engine exhaust (Pronk et al, 2009) and silica exposure has been particularly high in the Kalgoorlie underground goldmines (Steenland et al, 2001). Both these exposures have been shown to increase the risk of lung cancer (Steenland et al, 2001;
Asbestos fibres have been found in Kalgoorlie goldmines (Lee et al., 1999), whereas exposure to other potential lung carcinogens (radon and arsenic) appeared to be low (Armstrong et al., 1979).

In contrast to lung cancer, men diagnosed with prostate cancer are more likely to die from another cause (Epstein et al., 2012). As the cause of death as reported on death certificates is used to estimate mortality ratios, these ratios might not be adequate to describe altered risks for prostate cancer. Standardised incidence ratios (SIR) based on numbers from cancer registries should therefore give a more precise estimate.

Our objectives were to estimate lung and prostate cancer mortality and incidence, for both surface and underground miners, and to examine the hypothesis that (underground) mining may be protective against prostate cancer.

**METHODS**

Kalgoorlie is a Western Australian (WA) mining town, 550 km east of the capital city of Perth. A cohort of 2294 male goldminers was established from surveys of respiratory symptoms, smoking and lung function performed in Kalgoorlie (1961–1975). Detailed information was collected on smoking and medical history. Updated smoking details were obtained from 250 subjects via an additional questionnaire in 2000. Full employment details were recorded on miners’ health record cards (De Klerk and Musk, 1998). Miners were required to have annual chest X-rays as long as they were employed as miners. Dates of employment at each mine and job descriptions were recorded at each presentation. On the basis of this information, the number of months worked underground for each job was assigned. Fourteen subjects were deleted from the cohort because no employment data could be obtained.

The cohort was linked to WA mortality records up to 31 December 2009. Expected numbers of death were estimated using age- and period-specific cancer mortality rates calculated for the WA male population in 5-year periods from 1970 to 2009. Mortality rates for the period 1970–1974 were used to calculate expected rates from the person-years accumulated for the first period of 1961–1969 as period-specific rates were not available for those years. Vital status of 329 individuals (46 surface-only workers (12%); 283 ever-underground miners (15%)) was unknown in December 2009. These miners were treated as alive until last observed date and then censored. The cohort was also linked to the WA cancer registry, with incidence data available from 1982 until 31 December 2011. Data linkage was approved by the UWA human research ethics committee.

Standardised mortality ratios (SMR) and SIRs for any cancer, lung and prostate cancer were calculated as the ratio of the observed events to expected events using Stata v12.0 (StatCorp LP, College station, TX, USA).

Cox proportional hazard models were fitted to examine the risk of working underground on cancer within the cohort, using PROC PHREG in SAS v9.3 software (SAS Institute Inc., Cary, NC, USA). Hazard ratios (HR) and corresponding 95% confidence intervals (CI) adjusted for year of birth were calculated for the whole cohort. Duration of work underground was modelled as a time-dependent variable. Models for lung and any cancer were additionally adjusted for smoking duration (in years). The number of months worked underground on cancer within the cohort, using PROC PHREG in SAS v9.3 software.

| Cause of death       | Full cohort (n = 2294) 52 608 person-years | Surface only (n = 400) 9828 person-years | Ever underground (n = 1894) 42 780 person-years |
|----------------------|-------------------------------------------|----------------------------------------|--------------------------------------------------|
|                      | n            | SMR | 95% CI         | n          | SMR | 95% CI          | n          | SMR | 95% CI          |
| Any cause            | 1922         | 1.38 | 1.32–1.44      | 345        | 1.35 | 1.22–1.50       | 1577       | 1.42 | 1.35–1.49       |
| Any cancer           | 455          | 1.27 | 1.16–1.39      | 76         | 1.06 | 0.84–1.32       | 379        | 1.32 | 1.20–1.46       |
| Lung cancer          | 182          | 1.64 | 1.42–1.90      | 20         | 0.91 | 0.59–1.41       | 162        | 1.82 | 1.56–2.13       |
| Prostate cancer      | 37           | 0.94 | 0.68–1.30      | 11         | 1.44 | 0.82–2.54       | 25         | 0.81 | 0.55–1.20       |

Abbreviations: CI = confidence intervals; SMR = standardised mortality ratio.
underground. Cancer mortality and incidence were increased. For lung cancer, this increase was particularly seen among underground miners. The SMR for prostate cancer pointed towards a lower risk for underground miners, but the incidence data showed a significantly increased incidence among underground miners.

The overall increased mortality in the gold miners’ cohort is, besides cancers, mainly attributable to pneumoconiosis (SMR = 11.2, 95% CI 8.4–14.8) (De Klerk and Musk, 1998). The increased mortality from cancer among miners appeared to be mainly driven by lung cancer, as the SMR for any cancer dropped to 1.10 (95% CI 0.98–1.24) when lung cancer was excluded (data not shown).

The finding of an increased risk of lung cancer among miners—especially among underground miners—supports previous findings in this cohort (De Klerk et al, 1995; De Klerk and Musk, 1998). Increased risks of lung cancer in relation to duration of underground hard rock mining have also been described in other cohorts. Hodgson and Jones (1990) observed an overall SMR of 1.58 for lung cancer with a significant linear trend for years worked underground among tin miners (Hodgson and Jones, 1990). In a French iron mine, significantly increased mortality from lung cancer was observed with SMRs increasing by duration worked underground: 1.81 for 1–19 years, 4.23 for 20–29 years and 6.25 for 30+ years (Chau et al, 1993).

Smoking may explain part of the observed differences in lung cancer risk, as more underground miners were smokers. However, our Cox regression models were adjusted for smoking duration and showed a significantly increased risk for underground miners compared with surface-only. The main causative agents suggested for lung cancer risk in underground mines are diesel engine exhaust and respirable silica (Steenland et al, 2001; Attfield et al, 2012). Further analyses, for example, by looking on a job level, might explain the association with these exposures better in this cohort.

No protective effect of mining on prostate cancer was observed in the current analyses. Although mortality data might have suggested a slight decreased risk for underground mining, incidence data showed a clearly increased risk with a SIR of 1.27 (95% CI 1.07–1.52). No difference was observed between surface and underground miners. As men with prostate cancer are more likely to die from another cause (Epstein et al, 2012), mortality data are probably less accurate than incidence data when studying risk of prostate cancer.

As mines operate 24 × 7, workers are required to do regular night shifts. In 2007, shiftwork involving circadian disruption was classified as being probably carcinogenic to humans (Straif et al, 2007). The circadian system gets disturbed by exposure to light at night, leading to alterations in sleep patterns, suppression of melatonin production and deregulation of circadian genes involved in cancer-related pathways (Stevens et al, 2007). The available studies on prostate cancer and night shift were mainly suggestive of a positive association, but evidence of a causal association is as yet insufficient (Kolstad, 2008; Sigurdardottir et al, 2012).

Table 2. SIR and 95% CI overall and by worker location (1982–2011)

| Location                   | Full cohort (n = 1373) 16 450 person-years | Surface only (n = 223) 2793 person-years | Ever underground (n = 1150) 13 657 person-years |
|----------------------------|-------------------------------------------|------------------------------------------|-------------------------------------------------
| n                          | SIR 95% CI                                | n                          | SIR 95% CI                                | n                          | SIR 95% CI                                |
| Any cancer                 | 477 1.50 1.37–1.64                        | 84 1.33 1.11–1.70                | 376 1.47 1.32–1.62                        |
| Lung cancer                | 110 1.89 1.57–2.28                        | 15 1.33 0.80–2.20                | 95 2.03 1.66–2.48                         |
| Prostate cancer            | 122 1.32 1.10–1.57                        | 25 1.33 0.90–1.97                | 97 1.31 1.07–1.60                         |

Abbreviations: CI = confidence intervals; SMR = standardised incidence ratio.

Table 3. HR and 95% CI for duration worked underground—mortality and incidence

| Location                   | Never underground (reference) | <20 years underground | 20+ years underground | P_{trend}^b |
|----------------------------|-------------------------------|-----------------------|------------------------|-------------|
| n                          | HR^c 95% CI                   | n                     | HR^c 95% CI            | n           | HR^c 95% CI | n          | HR^c 95% CI |
| Mortality                  |                               |                       |                        |             |             |             |             |
| Any cause                  | 345 1.21 1.06–1.37            | 724 1.29 0.98–1.69    | 853 1.31 1.00–1.71     | 0.003       |
| Any cancer                 | 76 1.93 1.17–3.16            | 178 1.93 1.17–3.16    | 201 1.93 1.19–3.15     | 0.115       |
| Lung cancer                | 20 0.63 0.28–1.40            | 74 0.63 0.28–1.40     | 88 0.64 0.30–1.40      | 0.694       |
| Prostate cancer            | 12 1.04 0.80–1.34            | 12 1.04 0.80–1.34     | 13 1.04 0.80–1.34      | 0.344       |
| Incidence                  |                               |                       |                        |             |             |             |             |
| Any cancer                 | 84 1.04 0.80–1.34            | 195 1.04 0.80–1.34    | 198 1.15 0.89–1.49     | 0.996       |
| Lung cancer                | 15 1.09 0.60–2.00            | 39 1.09 0.60–2.00     | 56 1.57 0.88–2.77      | 0.344       |
| Prostate cancer            | 25 1.08 0.67–1.74            | 52 1.08 0.67–1.74     | 45 0.99 0.61–1.63      | 0.987       |

Abbreviations: CI = confidence intervals; HR = hazard ratio. ^aMedian duration of underground work was 240 months (20 years) among ever-underground miners. ^bP-value for trend (continuous duration: months worked underground). ^cConfidence intervals; HR = hazard ratio. ^dSIR = standardised incidence ratio.
may add to the body of evidence for an association between night shifts and prostate cancer. Overall cancer mortality and incidence was higher among Western Australian goldminers compared with the general population, especially for lung cancer and particularly for underground mining. This study does not support the hypothesis that miners have a decreased risk of prostate cancer.

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