Cancer

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Occupational exposure to silica and lung cancer risk in the Netherlands

The study lends some support to the contention that low levels of exposure to crystalline silica can cause lung cancer. Lung cancer risk was increased at levels of exposure below those likely to cause silicosis. If accurate, the findings imply that the current WEL of 0.1 $mg.m^{-3}$ would not prevent all cases of lung cancer. Risk was detectable only after 25 years of low level exposure. Silicosis status was not reported or corrected for.

Lung cancer is more common among those with diagnosable silicosis. There is some evidence that silica can cause cancer even when exposure is below the level required for silicosis. Cigarette smoking is a potent cause of lung cancer, so all studies of lung cancer should take this into account as thoroughly as possible. Many reports on the association between silica and lung cancer make no correction for the effects of smoking. If so, these reports rely on cases and controls having the same exposure to tobacco smoke and that there is no interaction effect between smoke and silica. Even a small imbalance could lead to erroneous conclusions.

With the outcome in the balance, all new research in this field could help decide if silicosis is a necessary precursor.

The Netherlands Cohort Study (NLCS), began in 1986 and is a large (120,852 men and women aged 55-69) population-based prospective cohort study. It has individual data on many potential confounders, including smoking and asbestos exposure. Silica exposure was deduced from job titles pre 1986. 5,000 subjects were followed up for 11 years by reference to the national cancer register.

In all there were 1,667 male lung cancer cases. The analysis focussed solely on men. On average, cases were older, were more likely to report a family history of lung cancer, were more likely to smoke cigarettes, smoked more cigarettes per day and for more years, had a lower consumption of fruit and vegetables, and were more likely to drink more than 30g alcohol per day.

Cumulative exposure to respirable crystalline silica was higher among cases at 2.3 vs 1.6 mg/m^3.year. 10% of exposed people had a cumulative exposure of ≥4-5 mg/m^3.year.

Lung cancer risk was not significantly related to duration of exposure below 25 years, to mean concentration of exposure, or cumulative exposure. For those exposed longer than 25 years the relative risk was 1.7 (1.1 to 2.4) after adjustment for age and smoking. The effect of adjustment for smoking was 0.2.

Additional adjustment for asbestos exposure increased the risk estimates i.e. the rate of asbestos related lung cancer in the control group was higher than in the silica exposed group. For those with > 25 years exposure to silica the RR was 2.3 (1.1 to 4.9) and cumulative exposure ≥ 3 mg/m^3.years gave RR = 2.0 (1.1 to 3.8).

Comment
There was no obvious cause of bias in the method used.

The current WEL for crystalline silica is 0.1 $mg.m^{-3}$; equivalent to 4.5 $mg.m^{-3}$.years for a working lifetime of 45 years. In 1993 HSE estimated that > 90,000 UK workers were exposed to levels in excess of this.

There was no account taken of silicosis status in this research. At the levels of exposure estimated here silicosis should be rare. Given the importance of this issue it would help if silicosis status was corrected for in this analysis.

The study supports the proposal that crystalline silica is carcinogenic even in the absence of silicosis and smoking and even when the statutory duty of care standard is complied with.