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Environmental exposure to cadmium and risk of cancer: a prospective population-based study

Provided they were non-smokers, lung cancer cases with an environmental exposure leading to daily urinary excretion levels of more than 24 nmol of cadmium could argue that cadmium was the most likely explanation for the cancer. Cadmium exposure maps for the UK are publicly available.

Cadmium is a well-known carcinogen (IARC 1993), tends to accumulate in body tissues (elimination half life ~ 20 years, targeting bones and kidneys) and is everywhere in the environment, at low levels. Hot spots occur naturally and around mineral works.

Cadmium dust inhalation at work is subject to exposure standards: 8 hour exposures should average less than 0.025 mg/m^3 (measured as the amount of cadmium in cadmium dust and cadmium compounds).

Apart from lung cancer, it is proposed that excess cadmium exposure leads to renal dysfunction and osteoporosis.

This prospective study followed a cohort of people with known environmental exposure to cadmium for approximately 17 years (521 residents of a high cadmium area; 473, low-exposure area). The aim was measure lung cancer risk.

The following figure illustrates the regions of Belgium involved in the study. It shows how soil concentration correlates (weakly) with cadmium exposure and the pattern of lung cancer incidence.

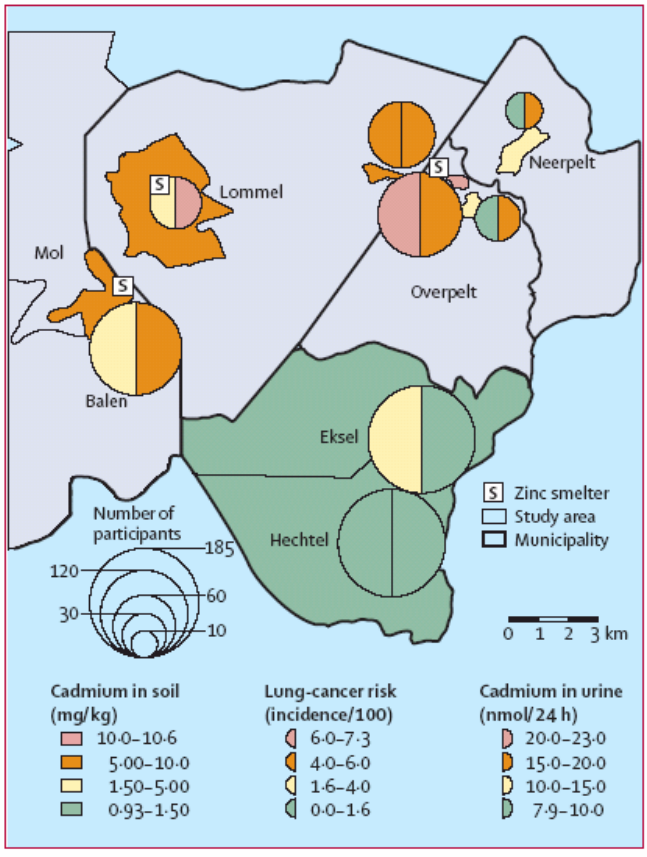


Figure 1: Geographical association between risk of lung cancer, 24-h urinary cadmium excretion, and cadmium concentration in soil of participants' gardens

Occupational exposures were obtained by contacting the occupational physician for those workers with a history of work at zinc refining plants, it was noted that men in the low cadmium exposure area often worked in coal mining. Questionnaire at baseline: lifestyle, past and current residence, possible exposure to cadmium at work, **smoking habits**, and previous medical history. Urine and blood samples

were taken at baseline. Soil samples were taken from 307 gardens at depths of up to 25 cm. Outcomes were taken from GP records.

In residential areas close to the smelting works, cadmium soil concentrations were 8 mg/kg, and in the control area, 0.8 mg/kg. Blood cadmium levels were 11·9 (11·2–12·8) in the high exposure area and 10·5 (5·4–11·8) nmol/L in the control area. Total urinary cadmium excretion (per day) was 12·3 (11·5–13·2) in the high exposure area and 7·7 (7·3–8·2) in the control area. Measures of exposure were statistically significantly different in high and low exposure areas.

Excluding men with known occupational exposure to cadmium and correcting for age, gender and smoking history, the risk of lung cancer increased by 60% for every doubling of personal exposure level and 40% for every doubling of soil exposure level. Overall, the hazard ratio for lung cancer in high vs. low exposure areas was 3.6 (95% CI = 1.0 to 12.7). This risk increased when cadmium exposed workers were included in the analysis. Cadmium exposed workers had a risk ratio of 3.2 (95% CI = 1.0 to 10.8).

The numbers of people who developed lung cancer was quite small (19) and measurements of arsenic exposure were less precise than the measures of exposure to cadmium.

Rates of emission from the zinc works have fallen by a factor of 1000 since the study began. The rate of change of soil cadmium content in this research project was not commented upon but emission rates have been high since 1888. It is generally agreed that cadmium is only very slowly lost from soils.

Comment

Risk ratios of this magnitude (HR ~ 1.6) should not be problematic for liability insurers unless it was argued that exposure to risk was equivalent to material contribution. The interaction between smoking and cadmium exposure was not examined in detail in this report but there was evidence that smoking was the dominant risk factor and, that cadmium increased the risk for smokers.

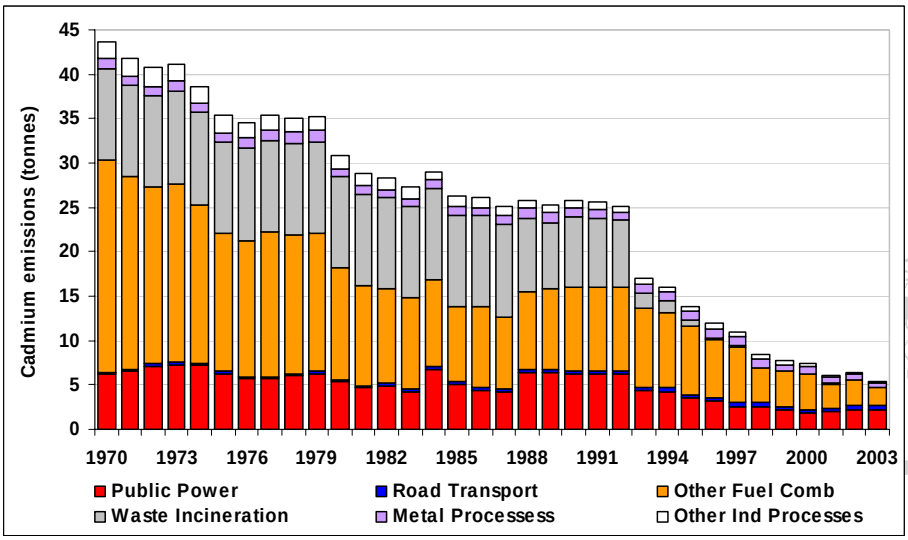
The number of people living in the high exposure areas was 9480. 6 lung cancers would be expected over the period of study, 18 were observed. According to the analysis, 12 of these could be due to cadmium in the environment and 12 due to smoking (some overlap is expected but the paper did not report on how much).

Cigarette smoking is a route of exposure to cadmium, the variation of urinary cadmium level with smoking status was not reported. According to DEFRA; the average daily intake of cadmium for humans is estimated as 0.15µg from air and 1µg from water. Smoking a packet of 20 cigarettes can lead to the inhalation of around 2-4µg of cadmium, but levels vary.

It is highly probable that environmental exposures were increased by the operations of the smelting works and that this led to significantly higher exposures in residents who did not work at the smelting works. Urine analysis could be used to measure the risk to individuals. Lung cancer victims, with no smoking history and with daily urinary excretion levels of more than 24 nmol could be of interest to claims lawyers. Date of knowledge estimated at 1993, but zinc manufacturers would be expected to be more than generally knowledgeable.

The most use of cadmium is in nickel/cadmium batteries. It is also used as a sacrificial coating, as a pigment and was once used as a stabiliser for PVC (until 2001). Directive 86/278 limits the cadmium content of soil and sludge used in agriculture (1-3 mg/kg dry matter and 20-40 mg/kg dry matter respectively).

Sources of environmental cadmium in the UK have been measured as follows (CSF/03/68):



Pollutant emission maps are available from www.naei.org.uk