

Mortality from obstructive lung diseases among asphalt workers

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Work in the asphalt industry entails exposure to bitumen and coal tar fumes. Asphalt work has been associated with non-malignant respiratory diseases, but causal links have not been established. A historical cohort of asphalt workers from eight countries (Denmark, Finland, France, Germany, the Netherlands, Norway, Sweden, Israel) was assembled by the International Agency for Research on Cancer. The cohort consisted of 79,822 male workers first employed for at least one season between 1913 and 1999 and accumulated 1,287,209 person-years of observation. Mortality from non-malignant respiratory diseases was compared to the general population. Exposure-response relationships were evaluated using a study-specific exposure matrix. The exposure matrix contained semi-quantitative exposure estimates to bitumen fume, coal tar, polycyclic aromatic hydrocarbons (PAHs), silica, asbestos and diesel exhaust for all jobs. In addition, quantitative exposure estimates were available for exposures to bitumen fume and PAH among pavers (the most numerous bitumen-exposed group in the cohort). Bitumen workers had higher mortality rates from chronic bronchitis, emphysema and asthma (obstructive lung diseases) relative to the general population: 143 deaths; standardised mortality ratio: 1.21; 95% confidence interval: 1.02, 1.43. Mortality from obstructive lung diseases was associated with cumulative and average exposures to polycyclic aromatic hydrocarbons (PAHs), mostly originating from coal tar. For example, cumulative exposure above 624 (ng benzo(a)pyrene/m³)*years were associated with three-to-four fold increase in relative risk of obstructive lung diseases (p<0.05). Bitumen fume was weakly associated with mortality from obstructive lung diseases, but its effects could not be distinguished from those of coal tar. For example, in a sub-cohort unexposed to coal tar, exposures to more than 3.73 (mg bitumen fume/m³)*year increased the relative risk of mortality from obstructive lung disease twofold, but the effect was not statistically significant. Our results seem to support the notion that exposures to PAHs, derived primarily from coal tar and possibly bitumen, are determinants for mortality from obstructive lung diseases. This is supported by mechanistic considerations of oxidative stress due to PAHs and their oxygenated derivatives. However, confounding (especially by tobacco smoking) and bias cannot be securely ruled out as an explanation for the observed associations. Our findings imply that the recently implemented ban on the use of coal tar in asphalt in Western Europe can be expected to reduce the risk of work-related obstructive lung diseases among asphalt workers.