Occupational health research has identified numerous carcinogens prior to the 1990s. Most occupational carcinogens were first identified through clinical observations and epidemiological studies rather than experimental studies. The most frequently quoted estimate of cancers due to workplace exposures is 4% and was estimated nearly 40 years earlier. There is a lack of current valid estimates at a global scale. There are significant trends in exposure to occupational carcinogens with a reduction of exposed workers and exposure levels in high income countries and increase in prevalence and high exposure levels in newly developed countries. New technologies and changing employment patterns are posing new challenges in the identification and control of occupational carcinogens. Working time and particularly shift work are among the major new areas for research and prevention. Epidemiological research in recent years has had significant difficulty in providing strong evidence on new carcinogens. This has been particularly the case in complex exposure scenarios such as exposure to pesticides. Different phases in epidemiological research can be identified. Case series and later SMR studies dominated in early periods. These were followed by the development of advanced exposure assessment methods and JEMs and their application in both cohort and case-control studies. In recent years studies in the wider area of molecular epidemiology have developed incorporating mechanistic information. Overall, the most productive studies in identifying carcinogens were the early and relative simple SMR studies that were done in a context of high exposures and limited work mobility. Use of classical epidemiological designs and particularly large cohort studies with advanced exposure assessment methods and the combination with new research approaches using powerful tools for exposure assessment, biomarkers and omic technologies will provide new evidence and allow quantitative risk assessment. Conduct of ‘big data’ type studies without advanced exposure assessment methods are unlikely to identify new occupational carcinogens. Occupational cancer research has been seriously underfunded and has been inefficient in promoting prevention of occupational carcinogens globally. This is a consequence of factors both within the occupational health community (repetitive non-innovative research; lack of efficient coordination in the occupational health community) but mostly due to wider factors and particularly the general hostile wider political environment concerning work conditions. Occupational exposure to carcinogens continues being in the 21st century a major cause of preventable cancer and in many parts of the world the prevalence of these exposures is increasing.

Chrysotile asbestos causes an increased risk of mesothelioma (MM), but the extent of this risk and the carcinogenic potency of chrysotile fibers is in discussion. We studied mortality and MM incidence among workers employed at the Balangero mine (Italy), the largest chrysotile mine in Western Europe, active from 1917 to 1985. The cohort included 974 male workers employed for at least 6 months and active on January 1st, 1946 or subsequently hired. Vital status and causes of death were ascertained. Past exposure to asbestos by working area and calendar period was estimated, based on historical measurements of fibre concentrations, and individual cumulative exposure assessed by applying these estimates to the job history of cohort members.

Local reference rates were used to compute expected deaths from selected diseases and expected incident MM cases. Observed to expected ratios were calculated along with 95% confidence intervals.

Mortality was increased for all causes (SMR=1.28; CI95% 1.17–1.40), pleural cancer (SMR=4.30; CI95% 1.58–9.37) and asbestosis (SMR=375.06; CI95% 262.68–519.23). SMRs for lung cancer (SMR=1.14; CI95% 0.81–1.55) and peritoneal cancer (SMR=3.25; CI95% 0.39–11.75) showed a non-statistically significant increase. Six cases of pleural MM were observed and the SIR was 6.3 (CI95% 2.3–13.7). The analysis by duration and latency for pleural cancer showed an increased risk with increasing duration of exposure and the risk flattened out for latency greater than 40 years. Further analyses based on quantitative exposure indices are being conducted to contribute to the debate on chrysotile potency.