

hair dyeings. The results were not driven by personal hair dye use, or smoking (key subjects additionally evaluated for cotinine). Analysis of a randomly chosen hair waving product confirmed the presence of o- and m-toluidine.

Conclusions Our observations indicate that hairdressers are currently exposed to an established (o-toluidine), and a suspected (m-toluidine), human carcinogen from permanent hair dyes (including light colours) and unexpectedly also from hair waving.

0255 **BACK SURGERY IN RELATION TO OCCUPATIONAL LIFTING. A COHORT STUDY BASED ON THE MUSCULOSKELETAL RESEARCH DATABASE AT THE DANISH RAMAZZINI CENTRE**

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Objectives Controversies have long existed on causes for low back pain, and the role of occupational mechanical exposures, e.g. lifting has been debated for several decades. The aim of this study was to investigate if lifting is a risk factor for low back surgery.

Method The study is based on data from the Musculoskeletal Research Database at the Danish Ramazzini Centre, comprising nine previous studies on musculoskeletal symptoms in working populations, performed from 1993 to 2005. The study was limited to participants aged 18–65, yielding 39 258 individuals, 22 669 women (58%) and 16 589 men (42%). Mean age at baseline was 42.9 years. Occupational mechanical exposures were assessed by a job exposure matrix linking job title to expert ratings of e.g. lifting, which was divided into three groups based on daily lifting: 0 kg (representing minimal exposure), 1–1000 kg, and >1000 kg. Cases of first time surgery for herniated lumbar disc (n = 1025) or lumbar fusion (n = 447) until 2012 were identified in the Danish National Patient Register. In preliminary analyses, risk estimates were obtained by logistic regression analysis, adjusting for age, gender, and study.

Results An exposure response relationship was seen for herniated lumbar disc: OR=1.2 (95% CI 1.0–1.4) for 1–1000 kg/day, and OR=2.2 (1.9–2.6) for >1000 kg/day. For lumbar fusion: OR=1.5 (1.2–2.0) for 1–1000 kg/day, and OR=2.8 (2.4–3.5) for >1000 kg/day.

Conclusions Lifting was associated with later operations for both herniated lumbar disc and lumbar fusion. In further analyses, life style factors and occupational psychosocial exposures will be addressed.

0256 **HOLES IN THE BURDEN ESTIMATES – SOME CAN BE FILLED, SOME MAYBE NOT**

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Objectives To consider the data shortcomings and methodological decisions involved in current burden of disease studies and the potential for these to be overcome and/or standardised.

Method Most burden of disease estimates require considerable assumptions or methodological decisions about factors concerning exposure, the appropriate relative risk to match with the exposure, and/or the size of the exposed population. These assumptions usually arise from a lack of data and could be largely overcome by the provision of better data. It is reasonable to expect that for some areas these data will improve with time, but for other areas the required data will probably never be available.

Other assumptions or methodological approaches vary depending primarily on theoretical considerations that are arguable and unlikely to ever be definitively solved by better data availability. Modelling may sometimes be of use but may not always be appropriate or practical and is still likely to involve some assumptions.

Results For example, some countries have reasonable estimates of asbestos exposure and some have good data on at least one asbestos-related outcome (mesothelioma incidence/mortality). How can this information be validly used for burden estimates where such data are poor?

Conclusions It is helpful to consider the extent to which burden estimates vary depending on the assumptions and methodologies involved when assessing the validity of estimates and their usefulness. Consideration of the potential for future improvements in data and better understanding of theoretical aspects should be an important input into the planning of future burden of disease work.

0257 **MORTALITY AND MORBIDITY HEALTH IMPACT ASSESSMENT OF EXPECTED EXPOSURE TO PM10 DUE TO THE MAJOR CONSTRUCTION SITE FOR A LARGE INTERNATIONAL EXHIBITION**

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Objectives To assess the short-term impact of expected exposure to PM10 due to a major construction site (Jan 2013–Jan 2015) on the health of the population residing in the seven towns nearby (N = 235 000).

Method Estimates of PM10 short-term effects on all-cause and cause-specific mortality and on selected causes of hospital admissions were estimated for a pre-construction period (2007–2011) using Poisson regression models. Expected PM10 concentrations at ground level were estimated applying the ISCST3 Gaussian dispersion model to forecast PM10 emission rates due to the site. Mean counts of the 2007–2011 deaths and hospitalizations were taken to estimate the expected numbers of health events. The 2013–2015 impact was evaluated in terms of numbers of attributable deaths and hospitalizations during the construction site progress, under several counterfactual scenarios.

Results Between 2013 and 2015, PM10 levels exceeding the mean PM10 pre-construction concentrations would be responsible for 0.54 attributable deaths (0.13 cardiovascular and 0.04 respiratory) and for 0.14 cardiac, 0.05 cerebrovascular and 0.51 hospital admissions. If considering the EU limit of 40 µg/m³, PM10 levels would be responsible for 11.06 attributable deaths (2.69 cardiovascular and 0.90 respiratory) and for 2.81 cardiac, 1.17 cerebrovascular and 10.89 respiratory hospital admissions.

Corresponding values above the WHO threshold of 20 $\mu\text{g}/\text{m}^3$ would be 51.73 attributable deaths (12.58 cardiovascular and 4.17 respiratory) 13.60 cardiac, 5.37 cerebrovascular and 49.13 respiratory hospital admissions.

Conclusions The expected exposure appears to have a limited impact on health. Future monitoring of the actual exposure levels during the progress of the works will allow evaluating the accuracy of those estimates.

0259 INTERACTION BETWEEN GENETIC AND OCCUPATIONAL FACTORS IN LUNG CANCER AETIOLOGY. A POPULATION-BASED CASE-CONTROL STUDY

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Objectives Genetic susceptibility in work-related lung cancer aetiology could have an important public health impact. Few studies have previously evaluated this issue, with inconsistent results. We aimed to investigate interactions between exposure to occupational carcinogens and genetic polymorphisms in lung cancer aetiology, adopting a systematic integrated approach.

Method EAGLE, a population-based case-control study, enrolled 2100 lung cancer cases and 2120 controls (Italy, 2002–2005). Lifetime work histories were collected for 4059 subjects and translated into exposure to six occupational carcinogens (asbestos, silica, polycyclic aromatic hydrocarbons, diesel exhausts, chromium, and nickel) using a job-exposure matrix. We selected 23 candidate genes among phase II metabolic genes reported in association with lung cancer susceptibility and/or metabolism of selected carcinogens. 298 tagging single nucleotide polymorphisms (SNPs) were genotyped on 4050 subjects. We tested for interaction within smoking-adjusted logistic regressions where SNPs were modelled individually, by gene group (using gene scores and haplotypes), and by pathways. False discovery rate (FDR) was used to account for multiple testing. Gene expression changes in lung tissues were studied for SNPs-carcinogens significant interactions.

Results As asbestos had the highest impact on lung cancer burden, we restricted interaction tests to this carcinogen. GSTM4 polymorphisms consistently showed positive interactions across different analysis levels, especially by SNP group score (FDR-adjusted p-value for interaction < 0.0001). No significant genetic “signal” by asbestos exposure was found at lung tissue level.

Conclusions GSTM4 polymorphisms may play a role in asbestos-related lung cancer aetiology. These findings are biologically plausible and have never previously been reported; they should therefore be validated in further studies.

0261 ESTIMATING THE BURDEN OF OCCUPATIONAL CANCER TAKING INTO ACCOUNT AGE

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Objectives Estimates of burden of disease are generally based on population attributable fractions (PAFs) calculated for a whole population. However, the age structure of an exposed group has an impact on these estimates, because disease rates vary by age and the exposed population may be younger than the national population in the estimation year.

Method To account for this, PAFs can be calculated by age, and applied separately by age to national incidence data. We have adapted our risk period methodology, which takes account of latency to estimate numbers exposed to a causative agent using Levin's formula for PAF, to estimate a workforce turnover factor by age group, which accounts for the age structure of an exposed population. To estimate age-specific RRs from unit relative risks per year of exposure, the link between age and duration of exposure can be modelled using Monte-Carlo methods.

Results We show the effect of estimating the burden of lung cancer due to occupational exposure to respirable crystalline silica for Britain using PAF estimates which do or do not take age into account. Taking account of age and assuming recruitment between ages 15–44, there were 1188 lung cancer registrations in males in 2010, or 798 without accounting for age, or 636 vs. 804 assuming recruitment between ages 15–24. The extension to using age-specific RRs is demonstrated for occupational asbestos-related lung cancers.

Conclusions Given the above results, and although highly dependent on assumptions made about workforce ages, there is clearly a case to be made to estimate PAFs by age.

0266 MORTALITY IN THE FRENCH COHORT OF NUCLEAR WORKERS MONITORED FOR EXTERNAL RADIATION EXPOSURE

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Objectives Understanding the effect of chronic low dose radiation exposure is crucial for radiation protection. This study analyses mortality of workers monitored for external radiation exposure while employed at three major French nuclear companies.

Method The cohort includes all workers employed at least one year by CEA, AREVA NC or EDF between 1950 and 1994, monitored for radiation exposure and alive on 1 January 1968. The mortality follow-up was to 2004. Vital status and causes of death were obtained from national registries. Standardised mortality ratios were assessed using national rates as the reference.

Results A total of 59 004 workers were followed-up for an average of 25 years. Mean age at end of follow-up was 56 years. Less than 1% of workers were lost to follow-up. 6310 deaths occurred between 1968 and 2004 including 2547 cancer deaths. A strong healthy worker effect was observed (all-cause SMR = 0.61, 95%-CI: 0.60–0.63). Significant excess mortality was observed for pleura cancer (SMR= 1.71, 95%-CI: 1.24–2.30) and for melanoma (SMR= 1.43, 95%-CI: 1.04–1.92), with no significant trend in SMRs for these outcomes across categories of cumulative radiation exposure.

Conclusions This analysis of French nuclear workers confirms a healthy worker effect but also an excess risk of death