MAGNETIC FIELD EXPOSURES AND BRAIN TUMOUR INFLAMMATORY MARKERS AND EXPOSURE TO AIR POLLUTANTS AMONG WORKERS IN A SWEDISH PULP AND PAPER MILL

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Objectives To investigate whether brain tumour or leukaemia risks are related to occupational exposure to low-frequency magnetic fields.

Method Brain tumour and leukaemia risks experienced by 73 051 UK electricity supply industry workers were investigated for the period 1973–2010. All employees were hired in the period 1952–1982 and were employed for at least six months with some employment in the period 1973–1982. Detailed calculations had been performed to assess exposures to magnetic fields. Poisson regression was used to calculate relative risks (rate ratios) of developing a brain tumour (or glioma or meningioma) or leukaemia (or its sub-types) for categories of lifetime, distant (lagged) and recent (lugged) exposure.

Results Findings for gliomas, all brain tumours combined, and all leukaemia were unexceptional; risks were close to (or below) unity for all exposure categories. There were no significant dose-response effects shown for meningioma, but there was some evidence of elevated risks in the three highest exposure categories for distant exposures. There were no significant dose-response effects shown for the main leukaemia sub-types, but there was a significant positive trend for acute lymphocytic leukaemia (ALL). National comparisons indicated that the limited associations shown for meningioma and ALL were based, in the main, on unusually low risks in the lowest exposure category.

Conclusions The findings are consistent with the hypotheses that both distant and recent magnetic field exposures are not causally related to gliomas or to the main leukaemia sub-types. The limited positive findings for meningioma and ALL may be chance findings; national comparisons argue against a causal interpretation.

Objectives To study the relationship between inhalation of airborne particles in a pulp and paper mill and markers of inflammation and coagulation in blood.

Method Personal sampling of inhalable dust was performed for 72 subjects working in a Swedish pulp and paper mill. Stationary measurements were used to study different particle size fractions including respirable dust, PM10, PM2.5, the particle surface area and particle number concentrations of ultrafine particles. Markers of inflammation such as interleukins (IL-1β, IL-6, IL-8, and IL-10), C-reactive protein (CRP), serum amyloid A (SAA), and fibrinogen and markers of coagulation such as factor VIII, von Willebrand factor (vWF), plasminogen activator inhibitor (PAI-1), and D-dimer were determined before the first shift after a work free period of normally five days and after the first, second and third shift.

Results The average 8hr-TWA level of inhalable dust in was 0.30 mg/m3, range 0.005–3.3 mg/m3. The proxies for 8hr-TWAs of respirable dust was 0.045 mg/m3, PM10 0.17 mg/m3 and PM2.5 0.08 mg/m3. No significant increase of markers of inflammation or coagulation in blood during the working week was noted after a non-exposure period of five days. In a multiple regression analysis adjustments were made for sex, age, smoking, BMI, and blood group. Significant positive correlations were found between several particle exposure metrics and CRP, SAA and fibrinogen taken pre- and post-shift day 1, suggesting a dose-effect relationship.

Conclusions These relations between particle exposure and inflammatory markers may indicate an increased risk of cardiovascular disease.

Objectives An earlier investigation found increased bladder cancer incidence among workers at a rubber chemical manufacturing plant that used o-toluidine, aniline and nitrobenzene. The cohort was expanded to include additional workers (N = 1873) and updated through 2007 to assess bladder cancer with improved exposure characterisation.

Method Work histories were updated and exposure categories and ranks were developed for o-toluidine, aniline and nitrobenzene combined. Incident cancers were identified by linkage to six state cancer registries. Residency in time-dependent cancer...
registry catchment areas was determined. Standardised incidence ratios (SIR) and standardised rate ratios for bladder cancer were calculated by exposure category and cumulative rank quartiles for different lag periods. Cox regression was used to model bladder cancer incidence with estimated cumulative rank, adjusting for confounders. Indirect methods were used to control for smoking.

Results Excess bladder cancer was observed compared to the New York State population (SIR=2.87, 95% CI 2.02–3.96), with higher elevations among workers definitely exposed (moderate/high) (SIR=3.90, 95% CI 2.57–5.68) and in the highest cumulative rank quartile (SIR=6.13, 95% CI 2.80–11.6, 10-year lag). Bladder cancer rates increased significantly with estimated cumulative rank (10-year lag). Smoking only accounted for an estimated 8% elevation in bladder cancer incidence. Other occupational exposures were not associated with excess bladder cancer after adjusting for smoking.

Conclusions Bladder cancer incidence remains elevated in this cohort and significantly associated with estimated cumulative exposure. Results are consistent with earlier findings in this and other cohorts. Despite other concurrent chemical exposures, we consider o-toluidine most likely responsible for the bladder cancer incidence elevation and recommend a reexamination of occupational exposure limits.

0095 MULTIMORBIDITY AND PREVIOUS SICKNESS ABSENCE EPISODES ARE DETERMINANTS OF INCIDENCE AND DURATION OF FUTURE EPISODES

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Objectives While sociodemographic and work-related factors are frequently studied as determinants of sickness absence (SA), health-related determinants have surprisingly received little attention. We examined the effect of multimorbidity and previous SA on the incidence and duration of future SA.

Method A retrospective (2004–2008) cohort of 373,905 workers who underwent a standardised medical evaluation in 2006 from which information on chronic conditions, health-related symptoms and behaviours was used to construct a sex-specific multimorbidity score (MMBS). Information on SA episodes occurring during the two years prior to the examination came from the employment history. We estimated the effect of the MMBS and prior SA on the 2-year incidence and duration of SA post-examination using a Cox model adjusted for age and occupational social class. Effects on SA duration were also adjusted for diagnosis.

Results Men, but not women, showed an effect with a trend of higher SA incidence risk from low (HR=1.06; 95% CI: 1.01–1.11) to high MMBS (HR=1.22; 95% CI: 1.18–1.28). Having five or more prior episodes was related to higher SA incidence risk, both in men (HR=2.19 95% CI: 2.11–2.28) and in women (HR=2.47; 95% CI: 2.35–2.61). Women, but not men, had longer SA duration from low (HR=0.91; 95% CI: 0.83–0.99) to high MMBS (HR=0.88; 95% CI: 0.78–0.99). Having 5 or more prior SA episodes was related to shorter duration in men (HR=1.67; 95% CI: 1.30–2.16) and women (HR=2.12; 95% CI: 1.56–2.89).

Conclusions Multimorbidity increases the risk of higher SA incidence and duration while the effect of prior SA episodes is more complex.

0096 MARGINAL STRUCTURAL MODELS FOR RISK OR PREVALENCE RATIOS AND DIFFERENCES

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Objectives Occupational epidemiologists often analyse binary outcomes in cohort and cross-sectional studies using multivariable logistic regression models, yielding estimates of adjusted odds ratios. When the outcome is common the adjusted odds ratio will not closely approximate the covariate-adjusted risk or prevalence ratio. Consequently, investigators may decide to directly estimate the risk or prevalence ratio using a log-binomial regression model; however, such models tend to be unstable and may not converge.

Method A marginal structural log-binomial model can be used to estimate risk and prevalence ratios and differences. The approach reduces problems with model convergence typical of log-binomial regression by shifting all explanatory variables except the exposures of primary interest from the linear predictor of the outcome regression model to a propensity score model for the exposure. The approach also facilitates evaluation of departures from additivity in the joint effects of two exposures.

Results We illustrate the proposed approach using data from several illustrative occupational studies of common outcomes.

Conclusions The proposed approach facilitates analysis of risk or prevalence ratios and differences in cohort and cross-sectional studies with common outcomes.

0097 MATCHING AND COUNTER-MATCHING ON PROPENSITY SCORES IN NESTED CASE-CONTROL STUDIES

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Objectives Occupational cohort studies often collect information on a primary exposure of interest and a small set of potential confounders. A nested case-control study may be undertaken to collect additional information on covariates that are potential confounders.

Method We describe and illustrate an approach to matched nested case-control studies in such settings. The approach reduces problems of sparse data typical of matched nested case-control studies by reducing the set of matching on explanatory variables to a single dimension defined by a propensity score model for the exposure. We then consider additional uses of propensity score for matching. We first consider varying control: case ratio in matched designs to increase efficiency when exposure prevalence is rare. Next we consider a second propensity score that include additional covariates not in the set of strong confounders selected for matching. Counter matching on this score can further increase efficiency.

Results We illustrate the proposed approach using data from a cohort study of underground miners.

Conclusions The proposed approach can improve efficiency of nested case control designs.

0103 RACIAL AND GENDER DIFFERENCES IN THE RISK OF ISCHAEMIC HEART DISEASE AND THE HEALTHY WORKER SURVIVOR EFFECT AMONG AUTOWORKERS

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0094 Bladder cancer incidence among workers exposed to o-toluidine, aniline and nitrobenzene at a rubber chemical manufacturing plant
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