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NESTED CASE-CONTROL STUDY OF OCCUPATIONAL CHEMICAL EXPOSURES AND PROSTATE CANCER AMONG WORKERS USING A JOB EXPOSURE MATRIX. \*A Krishnadasan, N Kennedy, Y Zhao, B Ritz (University of California, Los Angeles, CA)

To date, consensus about the effects of occupational chemical exposures on prostate cancer is lacking. Many previous studies were limited by the use of mortality data and inadequate assessment of chemical exposures. We conducted a nested case-control study to examine the association between occupational chemical exposures and prostate cancer incidence among 2,167 workers employed between 1950 and 1992 at a nuclear and rocket engine-testing facility in California. We obtained incidence data from the California Cancer Registry and seven other state cancer registries. Data from company records were used to retrospectively construct a job exposure matrix for occupational chemical exposures, including hydrazine, trichloroethylene (TCE), polycyclic aromatic hydrocarbons, benzene, and mineral oil. Furthermore, we conducted mail and telephone surveys among a subset of living workers to collect information on lifestyle factors. Conditional logistic regression was used to estimate associations. With adjustment for socioeconomic status (SES), occupational physical activity, and exposure to the other chemicals evaluated, the rate ratio estimate for low/moderate exposure to TCE was 1.32 (95% CI: 0.81 to 2.14), and 2.14 (95% CI: 1.17 to 3.89) for high TCE exposure. Furthermore, the association between TCE and prostate cancer was dose dependent (p-value for trend = 0.016). Among 338 workers who participated in the survey, we found that those who were highly exposed to TCE were less likely to be highly educated, and to have a family history of prostate cancer. Our results suggest that occupational TCE exposure increased the risk of developing prostate cancers in our worker population. Although we could not directly control for them in our analysis, our findings support the results of previous studies which identified risk factors that should be controlled for in future studies of this relationship.

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MORTALITY IN POLYCHLORINATED BIPHENYL EXPOSED ELECTRICAL CAPACITOR MANUFACTURING WORKERS. M Prince, \*A Ruder, M Hein, M Waters, E Whelan, N Nilsen, E Ward, T Schnorr, P Laber, K Davis-King (National Institute for Occupational Safety and Health, Cincinnati, OH 45226)

Polychlorinated biphenyl (PCB) exposure has been associated with cancer risk. Vital status for 14,458 workers with  $\geq 90$  days potential PCB exposure 1939-77 at two capacitor manufacturing plants was verified through 1998 to examine cancer mortality and excess liver and rectal cancer reported in a subcohort. Standardized mortality ratios (SMRs, U.S. rates) and Poisson regression rate ratios (RRs) were assayed; a job exposure matrix estimated PCB exposure. Liver and prostate cancer mortality was not elevated (liver, 21 deaths, SMR 0.9, 95% confidence interval (CI) 0.6-1.4; prostate, 34 deaths, SMR 1.0, CI 0.7-1.5), but rose with low to high cumulative exposure (liver RRs: 1.7, CI 0.3-10.3; 3.0, CI 0.6-15.4; 4.2, CI 0.9-19.8, p for trend 0.07; prostate RRs: 1.5, CI 0.4-5.6; 2.8, CI 0.8-9.6; 6.1, CI 2.0-18.2, p for trend 0.0001). Stomach cancer mortality among men (n = 24, SMR 1.5, CI 1.0-2.3) rose with cumulative exposure. Mortality from myeloma (n = 28, SMR 1.9, CI 1.2-2.7), intestinal cancer among women (n = 67, SMR 1.3, CI 1.0-1.7), and rectal cancer (n = 21, SMR 1.1, CI 0.7-1.7) were elevated but not clearly related to exposure. Ovarian cancer mortality, elevated among long term workers, rose with cumulative exposure. This study cannot associate intestinal or rectal cancer with PCB exposure but supports prior reports of elevated liver cancer mortality, is the first occupational cohort study to show a strong exposure-response relationship between PCBs and prostate cancer mortality, and suggests that myeloma, ovarian cancer, and male stomach cancer mortality may be related to PCB exposure.

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LEAD, GENETIC SUSCEPTIBILITY AND RISK OF ADULT BRAIN TUMORS. \*P Rajaraman, P A Stewart, J M Samet, B S Schwartz, M S Linet, S H Zahm, N Rothman, M Yeager, H A Fine, P M Black, J Loeffler, W R Shapiro, R G Selker, and P D Inskip (National Cancer Institute, Bethesda, MD)

Although few etiological factors for brain tumors have been identified, limited data suggest that lead may increase the risk of brain tumors, particularly meningioma. The ALAD G177C polymorphism affects the toxicokinetics of lead and may confer genetic susceptibility to lead exposure effects. We examined occupational exposure to lead and risk of brain tumors in a multi-site, hospital-based case-control study of 489 patients with glioma, 197 with meningioma, and 799 controls admitted to the same hospitals for non-malignant conditions. ALAD genotype was assessed for 355 glioma patients, 151 meningioma patients, and 505 controls. Exposure to lead was estimated using a rigorous questionnaire-based exposure assessment strategy incorporating lead measurement and other occupational data. Increased risk of meningioma with occupational lead exposure (estimated by odds ratios and 95% confidence intervals) was most apparent in individuals with the ALAD2 variant allele, for whom risk rose dramatically from 1.1 (0.3, 4.5) to 5.6 (0.7, 45.5) and 12.8 (1.4, 120.8) for estimated cumulative lead exposures of 1-49  $\mu\text{g}/\text{m}^3\text{-yrs}$ ; 50-99  $\mu\text{g}/\text{m}^3\text{-yrs}$  and  $\geq 100$   $\mu\text{g}/\text{m}^3\text{-yrs}$  respectively, compared to unexposed individuals (p-trend = 0.06). This relationship became stronger after excluding occupational lead exposures characterized by a low confidence level, or occurring in the 10 years prior to meningioma diagnosis. Occupational lead exposure was not associated with glioma risk. Lead, a toxic metal that continues to be widely used, appears to be implicated in risk of meningioma in genetically susceptible individuals.

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THE WELFARE STATE AS A CONTEXT FOR CHILDREN'S DEVELOPMENTAL HEALTH: A STUDY OF THE EFFECTS OF UNEMPLOYMENT AND UNEMPLOYMENT PROTECTION. \*A Siddiqi, S V Subramanian, L Berkman, C Hertzman, and I Kawachi (Columbia University, New York, NY, 10027)

We seek to understand national-level policy influences on children's developmental health among the wealthier countries of the world. We investigate the effect of parental unemployment on children's cognitive development (measured by reading literacy) and the role of unemployment protection policies in moderating the effects of parental employment status on reading literacy. Data come from the Organization for Economic Cooperation and Development (OECD) Program for International Student Assessment (PISA). The sample consists of 61, 946 children, nested in 3, 918 schools, among 17 market economies. Preliminary results of multi-level analyses indicate that, after controlling for a range of individual, family, and school co-variables, children with unemployed fathers in all countries have significantly worse reading literacy scores than those of employed fathers ( $\beta = -8.84$ , SE = 2.01). The contextual effect of unemployment protection are not significant after accounting for father's employment status ( $\beta = -18.66$ , SE = 16.24). However, there is a significant negative interaction between unemployment protection and father's unemployment, yielding the unexpected suggestion that, in countries with higher levels of unemployment protection, children with unemployed fathers fare worse, both in relation to children with unemployed fathers in lower protection countries, and in comparison to children with employed fathers ( $\beta = -26.89$ , SE = 8.63). Possible explanations are advanced for this result, including the potential for a "discouraged child effect," arising from the potential association between unemployment protection and higher unemployment rates.



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