

SELF-REPORTED OCCUPATIONAL RISK FACTORS FOR PROSTATE CANCER. A. Sass-Kortsak,* J. Purdham, N. Lightfoot, N. Kreiger and G. Buchan (Department of Public Health Sciences, University of Toronto, Toronto, Canada M5T 1R4)

Various occupational exposures have been inconsistently associated with prostate cancer risk, including cadmium, other metallic dusts, pesticides and polycyclic aromatic hydrocarbons (PAH's) from combustion products, lubricating oils and greases, asphalt and diesel exhaust. These exposures were evaluated in a case-control study conducted in northeastern Ontario, Canada. Cases, aged 45 to 84 years, were identified through the Ontario Cancer Registry with a first primary cancer of the prostate diagnosis, during the period of January 1995 to December 1998. Male population controls were age frequency-matched to cases. Cases and controls received a mailed questionnaire, which was either self-completed or interviewer-administered by telephone and which included, for a specific list of materials, self-assessment of exposures (ever/never, frequency and level). 756 cases (73% response) and 1634 controls (53% response) were included in logistic regression analyses. Based on self-assessments, statistically significantly elevated risks for prostate cancer were found for ever exposed to diesel exhaust (age-adjusted odds ratio (OR)=1.22, 95% confidence interval (CI)=1.02-1.46), pesticides (OR = 1.28, 95% CI = 1.01-1.63) and PAH's from multiple source (OR = 1.28 95% CI = 1.04-1.59). Interestingly, exposure to noise (OR = 1.35, 95% CI = 1.05-1.74) and sunlight (OR = 1.32, 95% CI = 1.10-1.58) were also associated with an elevated risk of prostate cancer. Exposures to dusts, metal compounds, combustion products, asphalt fumes, lubricating oils and greases, welding fumes, fertilizers, PCB's and asbestos were not associated with an increased risk of prostate cancer. Quantifying cumulative exposures by reported intensity and frequency did not alter the results.

HCG MEASUREMENT IN THE DETECTION OF EARLY PREGNANCY LOSS (EPL). J. F. O'Connor (Columbia University, Irving Center for Clinical Research, New York, NY 10032)

Urinary human chorionic gonadotropin hCG is employed as the biomarker in epidemiological investigations of EPL. However, increased knowledge of hCG metabolism has complicated its measurement. Foremost, hCG is not specific to pregnancy. It is produced in the pituitary of both non-pregnant women and in men, and in lower quantities, in many if not all types of malignancy. It is thought to be secreted throughout the menstrual cycle. Consequently, the cycle window within which the hCG measurement is made of paramount importance. Another concern is the number of urinary isoforms of hCG, namely the intact hCG molecule, both with and without the carboxyl terminal peptide (CTP) present, hCG free beta subunit, a hyperglycosylated form of hCG (in early pregnancy), nicked hCG and hCG beta core fragment. Evidence indicates that the measurement of nicked hCG is not informative, because of its very limited presence in early pregnancy. The measurement of the putative hyperglycosylated form of hCG may be much more important. However, its inclusion in EPL determinations at present is premature, since its molecular identity remains to be determined and a suitable standard is lacking. The practical choice of hCG urinary analytes thus reduces to three, the intact hCG molecule, free hCG beta subunit and hCG beta core fragment. An assay has been developed which will detect these three analytes simultaneously. The downside of this approach may be some loss of specificity, as this assay configuration has the greatest potential for cross reacting with immunologically related urinary gonadotropin fragments, particularly hLH beta core fragment. Perhaps the best compromise, in the absence of a more specific early pregnancy marker, is to screen all samples with this assay and confirm questionable cases with specific analyses for the individual constituents.

KIDNEY DISEASE AND ARTHRITIS AMONG WORKERS EXPOSED TO SILICA. K. Steenland,* W. Sanderson, and G. Calvert (National Institute for Occupational Safety and Health, Cincinnati, OH 45215)

Several studies have linked silica exposure to renal disease (particularly glomerulonephritis) and rheumatoid arthritis, but these associations are still not widely accepted. Approximately 2 million people are occupationally exposed to silica in the U.S. We studied 4620 workers exposed to silica in the industrial sand industry. We found an excess of mortality from acute renal disease (SMR 2.61, 95% CI 1.49-4.24, 16 deaths), chronic renal disease (SMR 1.61 (95% CI 1.13-2.22, 36 deaths), and arthritis (SMR 4.36, 95% CI 2.76-6.54, 23 deaths) based on multiple cause mortality data (any mention on a death certificate). A large excess of mortality from silicosis was also seen (SMR 18.2, 95% CI 10.6-29.1, 17 deaths). Linking the cohort with the U.S. registry of end stage renal disease (ESRD) cases for 1977-1996, we found a two-fold excess of ESRD (rate ratio 1.97, 95% CI 1.25-2.96, 23 cases), concentrated in glomerulonephritis (rate ratio 3.85, 95% CI 1.55-7.93, 7 cases). A job-exposure matrix based on over 4000 industrial hygiene samples was used to estimate silica exposures over time. We found a positive trend between ESRD and cumulative exposure ($p = .01$); the rate ratio for the highest quartile of exposure versus the U.S. population was 3.82 (95% CI 1.24-8.91). A male exposed at the NIOSH Recommended Exposure Level (0.05 mg/m³) is estimated to have a lifetime risk of ESRD of 13%; the background risk for a nonexposed male is 2%. In nested case-control analyses, a positive exposure-response was observed for rheumatoid arthritis mortality ($p = .04$), but not for other types of arthritis. These data represent the largest number of kidney disease cases analyzed to date in a cohort with well-defined silica exposure, and suggest a causal link between silica and kidney disease. The results for arthritis are also suggestive, but based on smaller numbers.

RELIABILITY OF HCG DATA INTERPRETATION. S.-I. Cho,* M.B. Goldman, L. Ryan, C. Chen, A.I. Damokosh, D.C. Christiani, B.L. Lasley, J.F. O'Connor, A.J. Wilcox, and X. Xu (Harvard School of Public Health, Boston, MA 02115)

Urine beta-hCG (human chorionic gonadotropin) is a sensitive biomarker for detecting early pregnancy loss. However, the interpretation of daily hCG data involves investigator's judgment, even with several proposed algorithms to guide the assessment. To examine the reliability of hCG data interpretation, five experienced researchers independently assessed data from 153 menstrual cycles, determining whether each cycle represented no conception, a continuing conception, or a conception loss. The cycles provided to the experts were randomly selected from the data collected from 190 Chinese women participating in a prospective study. The intact hCG heterodimer levels were measured by immunoradiometric assay using a combination of antibodies for a beta subunit epitope (B109) and the intact heterodimer (B204). For each cycle, hCG data were presented as graphs of triplicate assays in a chart including cycle day and menstrual bleeding days. Summary statistics for hCG data from 46 sterilized women were provided to represent baseline values. Pairwise agreement among the assessors for any of the three options ranged from 54 to 86%. All five experts' assessments agreed for 55 cycles (36%), accounting for 12 conception losses and 4 continuing conceptions. At least three experts agreed for 147 cycles (96%), accounting for 28 conception losses and 19 continuing conceptions. The multi-rater kappa was 0.63 for the conception lost category and 0.68 for continuing conceptions, indicating substantial agreement. The main sources of disagreement involved deciding whether there was sufficient information for assessment, interpreting cycle parameters such as cycle length or bleeding event, and interpreting a distinct hCG rise pattern below the baseline value obtained from the sterilized women.

EPIDEMIOLOGY

Volume 151
Number 11
June 1, 2000

Published for The Johns Hopkins University
School of Hygiene and Public Health
by Oxford University Press
Sponsored by the Society for Epidemiologic Research

1 134110 30 SEP 19 151/11
F&T LIBRARY C-21
47336243
OHHS PHS CDC NIOSH
4876 COLUMBIA PARKWAY
CINCINNATI
OH 45226 1922



Society for Epidemiologic Research

ABSTRACTS OF THE 33RD ANNUAL MEETING
SEATTLE, WASHINGTON, JUNE 15-17, 2000