smoking indirectly adjusted standardised mortality ratio (SMR) according to the method of ‘smoking adjustment factor (SAF)’ was presented if the RSE was not statistically significant (i.e., no multiplicative interaction).

**Results** The RSE for total deaths and the deaths from non-malignant respiratory diseases (NMDR) was 0.75 (95% CI: 0.62–0.91) and 0.59 (95% CI: 0.46–0.78); however, it was not statistically significant for other specific causes of death. Smoking indirectly adjusted SMR for oesophagus cancer, lung cancer, chronic obstructive pulmonary diseases, silicosis, pulmonary tuberculosis and pulmonary heart disease was 1.08 (95% CI: 0.72–1.64), 2.41 (95% CI: 1.16–4.59), 2.07 (95% CI: 1.78–2.41), 4.11 (95% CI: 2.10–7.82), 4.99 (95% CI: 3.80–6.54), and 4.09 (95% CI: 3.25–5.64).

**Conclusions** This historical cohort study demonstrated a significant multiplicative interaction between smoking and silicosis on the mortality of total deaths and deaths from NMRD; however, smoking was more likely to play a role of confounding in an increased mortality from other major causes among Chinese silicotic workers.

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**Abstracts**

**101 OCCUPATIONAL EXPOSURE TO TETRACHLOROETHYLENE AND THE RISK OF BLADDER CANCER: A META-ANALYSIS**


**Objectives** The risk of bladder cancer in persons occupationally exposed to tetrachloroethylene was summarised using a meta-analysis.

**Methods** Studies were identified from a PubMed literature search including the terms “drycleaner, dry-cleaning, occupation, tetrachloroethylene, bladder cancer, bladder carcinoma, urothelial carcinoma” in various combinations. We included studies that reported a risk estimate specifically for tetrachloroethylene or employment as a “dry cleaner” based on historical information indicating that a large percentage of dry cleaners were exposed to tetrachloroethylene but not to other occupational bladder carcinogens. We excluded studies that reported results for “dry-cleaners and laundry workers” (the latter group has not been exposed to tetrachloroethylene), PMR analyses (risk estimates are potentially biased), and overlapping publications. Publication bias was assessed using funnel plots. All statistical analyses were performed using STATA.

**Results** Twelve studies were included in the meta-analysis (8 case-control studies, 4 cohort studies) resulting in a meta-RR of 1.24 (95% CI, 1.12–1.37). The meta-RR was 1.20 (95% CI, 1.07–1.34) for case-control studies (all adjusted for smoking) and 1.44 (95% CI, 1.13–1.84) among the cohort studies (none adjusted for smoking). When we restricted the analysis to the studies reporting the meta-RR for specific exposure to tetrachloroethylene the meta-RR was 1.18 (95% CI: 1.05–1.33; 3 studies) and for dry cleaners it was 1.46 (95% CI: 1.17–1.83; 6 studies). A jack knife analysis omitting individual studies demonstrated that there was no overreliance of the overall meta-RR on any one study. There was no evidence of publication bias. One of the included studies assessed exposure-response data and reported an increase in odds ratios with increasing cumulative exposure among men.

**Conclusions** Occupational exposure to tetrachloroethylene is associated with a moderate, significantly increased risk of bladder cancer. Excesses occurred in cohort and case-control studies. The excesses in case-control studies could not be explained by tobacco use.

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**102 OCCUPATIONAL ASBESTOS EXPOSURE AND RISK OF PLEURAL MESOTHELIOMA, LUNG AND LARYNGEAL CANCER IN THE PROSPECTIVE NETHERLANDS COHORT STUDY**


**Objectives** Although asbestos research has been ongoing for decades, there are remaining questions regarding cancer risk associated with low exposure and cancer subtypes, the influence of potential confounders, and the interaction between asbestos and smoking. We addressed these questions by studying the association between occupational asbestos exposure and pleural mesothelioma, lung and laryngeal cancer in the prospective population-based Netherlands Cohort Study (NLCS).

**Methods** The NLCS includes 58279 men aged 55–69 years at enrollment in 1986. Based on job history information obtained from a self-administered questionnaire, asbestos exposure was estimated by linkage to job-exposure matrices. After 17.3 years of follow-up, 132 cases of pleural mesothelioma, 2324 cases of lung cancer, and 166 cases of laryngeal cancer were available for analysis.

**Results** Overall, occupational asbestos exposure was associated with an increased risk of mesothelioma, lung and laryngeal cancer, also for relatively low exposure. Correcting for potential confounders as age, smoking, alcohol, and several occupational carcinogens hardly influenced these results. Associations with lung cancer subtypes were generally comparable to overall lung cancer, except for adenocarcinoma (HR ever versus never exposed = 1.43, 1.52, 1.49 and 0.94 for small cell, large cell, squamous cell and adenocarcinoma respectively). Adenocarcinoma showed only a weak positive association at higher exposure levels for long duration. For laryngeal cancer, associations were usually stronger for supraglottis cancer (HR = 2.48, 95% CI: 1.33–4.65) than glottis cancer (HR = 1.12, 95% CI: 0.74–1.69). There was no statistically significant additive or multiplicative interaction between asbestos and smoking for any of the cancers.

**Conclusions** The well-established associations between asbestos and smoking for any of the cancers. Adenocarcinoma may only show a weak positive association at higher exposure levels for long duration. For laryngeal cancer, associations were usually stronger for supraglottis cancer (HR = 2.48, 95% CI: 1.33–4.65) than glottis cancer (HR = 1.12, 95% CI: 0.74–1.69). There was no statistically significant additive or multiplicative interaction between asbestos and smoking for any of the cancers.

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**103 INTERACTIONS BETWEEN OCCUPATIONAL EXPOSURES TO EXTREMELY LOW FREQUENCY MAGNETIC FIELD AND CHEMICALS FOR BRAIN TUMOUR RISK IN THE INTEROCOCC STUDY**

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**Objectives** Although asbestos research has been ongoing for decades, there are remaining questions regarding cancer risk associated with low exposure and cancer subtypes, the influence of potential confounders, and the interaction between asbestos and smoking. We addressed these questions by studying the association between occupational asbestos exposure and pleural mesothelioma, lung and laryngeal cancer in the prospective population-based Netherlands Cohort Study (NLCS).

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**Conclusions** The well-established associations between asbestos and smoking for any of the cancers. Adenocarcinoma may only show a weak positive association at higher exposure levels for long duration. For laryngeal cancer, associations were usually stronger for supraglottis cancer (HR = 2.48, 95% CI: 1.33–4.65) than glottis cancer (HR = 1.12, 95% CI: 0.74–1.69). There was no statistically significant additive or multiplicative interaction between asbestos and smoking for any of the cancers.

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ASSOCIATION BETWEEN OCCUPATIONAL EXPOSURE TO ENGINE EMISSIONS AND LUNG CANCER

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Objective To analyse the associations between life-time occupational exposures to diesel, leaded and unleaded gasoline engine emissions and lung cancer.

Methods Our case-control study enrolled 1503 lung cancer cases and 1198 population controls between 1996 and 2001 in Montreal. Occupational exposure to diesel, leaded and unleaded gasoline engine emissions was assessed using a combination of subject-reported job and tasks history and expert assessment. Exposure status “Ever or never exposed”, duration, frequency and concentration of exposure based on qualitative assessment were assigned to each participant. Lifetime average exposure and cumulative exposure were derived as semi-quantitative indices.

Results In models including only one type of engine emission at a time, only diesel showed an association with lung cancer (OR for average exposure: 1.19, 95% CI: 1.03, 1.37). When restricting the analysis to participants exposed to one of the three types of engine emissions exclusively, cumulative exposure to leaded gasoline, unleaded gasoline, and diesel engine emissions were associated with odds ratios of lung cancer of 2.11 (95% CI: 1.25, 3.56), 0.66 (95% CI: 0.25, 1.72) and 1.09 (95% CI: 0.95, 1.24), respectively. The inclusion of the three types of engine emissions in one model, using either multivariate logistic or generalised additive logistic regression, yielded similar estimates to those obtained for segments of the population with increased exposure to only one type of engine emission.

Conclusion Exposure to diesel and unleaded gasoline engine emissions confounded the association of leaded gasoline engine emissions and lung cancer. Increases in cumulative exposure to leaded gasoline were associated with an increased risk of lung cancer.

POOLED ANALYSIS OF TWO CASE-CONTROL STUDIES OF POLYCYCLIC AROMATIC HYDROCARBONS AND RISK OF LUNG CANCER

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Objective To examine the effects of occupational exposure to PAHs arising from different combustion products on risk of lung cancer.

Methods Data was provided from two case-control studies conducted in Montreal. Study 1 (1979–1986) included 857 men with lung cancer, 533 controls from electoral lists, and 1346 controls with other cancers. Study 2 (1996–2001) included 738 men with lung cancer and 899 controls selected from the electoral list. Occupational histories were obtained and lifetime chemical exposure was derived by hygienists for benzo(a)pyrene and profiles of PAHs, according to source (wood, petroleum, coal, or other, which included rubber, plastic, and food). Data were pooled across studies.

Results Prevalence of any PAH was 68% in the Study 1 population and 53% in Study 2. Adjusting for confounding due to smoking and common demographics, exposure to any PAH