Abstracts

MULTIPLE PESTICIDE EXPOSURES AND THE RISK OF LYMPHOMA IN THE EPILYMPH STUDY


Objectives Some industrial chemicals and pesticides might have endocrine disrupting effects. While some pesticides and solvents have been associated with an increased risk of lymphoma, whether this would be the result of their potential endocrine disrupting effect has not been investigated as yet. We explored the role of occupational exposure to endocrine disruptors in lymphoma aetiology.

Methods The EpiLymph study is a multicenter case-control study carried out in six European countries from 1998 to 2004. We evaluated 2,437 controls and 2,013 lymphoma cases and its subtypes. Information on occupational history was collected through face-to-face interviews. We applied a job-exposure matrix (JEM) for endocrine disrupting chemicals to assess occupational exposures (Brouwers et al. 2009). We evaluated exposure to ten chemical groups: polycyclic aromatic hydrocarbons, polychlorinated organic compounds, pesticides, phthalates, solvents, bisphenol-A, alkylphenolic compounds, brominated flame retardants, metals and a miscellaneous group.

Results Prevalence of ever occupationally exposed among controls ranged from 1% (bisphenol-A) to 48% (solvents). Risks for non-Hodgkin lymphoma (NHL) and chronic lymphocytic leukaemia (CLL) were increased with cumulative exposure to endocrine disruptors among men (OR = 1.20 CI95%: 1.04–1.38 and 1.28 CI95%: 1.01–1.61, respectively). However, none of the individual chemical groups evaluated was associated with NHL or follicular lymphoma risk. For other subtypes such as CLL, multiple myeloma, Hodgkin lymphoma and T-cell neoplasms risks were increased with several exposures, including metals.

Objectives To test the association between occupational exposure to trichloroethylene (TCE) and risk of non Hodgkin lymphoma (NHL), we conducted a pooled analysis of four international case-control studies.

Methods Studies were selected which included state-of-the-art retrospective assessment of occupational exposure to TCE and histological information on lymphoma subtype. Overall, the pooled study population included 3788 NHL cases and 4279 controls. Summary indicators of exposure were harmonised across studies. We conducted unconditional logistic regression analysis to test the association between the harmonised TCE exposure estimates and NHL and its major subtypes, adjusting by age, gender, and study.

Results Among the total study population, risk of follicular lymphoma, but not NHL overall or other subtypes, increased by probability (p = 0.02) and intensity level (p = 0.04) of TCE exposure. When the analysis was restricted to subjects most likely exposed to TCE, risk of NHL overall (p = 0.009), follicular lymphoma (p = 0.04), and chronic lymphocytic leukaemia (CLL) (p = 0.01) showed a linear increase by duration of exposure. No heterogeneity in NHL risk associated with high probability of exposure to TCE (all intensity levels combined) was detected.

Conclusion With due caution because of several limitations, our pooled analysis supports the hypothesis of an increased risk of NHL, and particularly of the follicular lymphoma and CLL subtypes, associated with occupational exposure to TCE.