Hydrocarbon Exposure and Glomerulonephritis

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Dear Sir,

Churchill et al. [1] argue convincingly in their review that a cohort study of exposed and unexposed individuals is not possible because of the low incidence of glomerulonephritis. They mention that proteinuria could possibly be chosen as outcome. In a cohort study of 182 individuals, Askergren et al. [2], using a sensitive immunochemical method for measuring albuminuria, found in fact that about 5% of the exposed individuals had a slight but significant increase of urine albumin.

Recall bias is difficult to avoid. Churchill et al. [1] nevertheless conclude that a risk ratio of 3.9, which we found in our study [3], suggests a strong association. I agree, but I would like to explain it further, because the recall bias by many nephrologists has been used as an argument for rejecting the idea of the nephritogenic properties of hydrocarbons.

Recall bias implies that cases for various reasons recall more exposure than controls, or that controls forget previous exposure. In our study [3] almost all exposure of the cases with a score above 10 was occupational, i.e. it occurred in the routine work of painters, gluers, rubber and plastic plant workers, typographers, auto mechanics and similar occupations. It seems improbable that such exposure should have been forgotten by the controls or missed by the interviewers. The occupations mentioned were much more common among the patients with glomerulonephritis than expected (unpublished), which also argues against recall bias as a major disturbance.

The implications of considering glomerulonephritis as an occupational disease are so great, however, that one further well-designed case-control study is desirable, as suggested. It should not include glomerulonephritis secondary to systemic disease. Solvent exposure does not seem to be of importance in SLE nephritis and similar conditions. van der Laan [4], whose cases were drawn from the files of a renal pathology department, did not mention the number of cases with systemic glomerulonephritis in his study. A possible explanation of its negative result could be that a number of such cases were included. The controls should also be chosen carefully. Many diseases are directly or indirectly induced or worsened by exposure to hydrocarbons. Will we get a satisfying answer to this urgent problem before 1992, the centenary of the first report [5] about an association between hydrocarbon exposure and glomerulonephritis ?

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