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Hydrocarbon exposure and chronic renal disease

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Abstract The study objective was to investigate further the potential role of long-term exposure to hydrocarbons (HCs) in the development of idiopathic chronic glomerulopathy (ICG) using a more refined measurement of HC exposure. A total of 321 pairs of cases and controls, matched by age, gender, and geographical area, were assembled. A detailed questionnaire was blindly administered to cases and controls to collect information on occupational and medical history and sociodemographic data. By integrating quantified measurements of HC exposure from a variety of sources with each subject's occupational history, a lifetime HC exposure score could be estimated and expressed in parts per million (ppm). Cases had an hydrocarbon exposure mean score of 165 ppm (median 48 ppm) as compared to 162 ppm (median 43 ppm) for controls ($P = 0.757$). When using hydrocarbon exposure as a dichotomous variable with a cutoff point at 100 ppm, cases had a higher proportion of exposed than controls, but the difference was not statistically significant at the 0.05 level, even after controlling for possible confounders through logistic regression. Subgroup analyses showed mixed results. In most subgroups differences between cases and controls tended to become significant when hydrocarbon was used as a dichotomous variable. Results from this study do not sufficiently support the hypothesized association of HC exposure and ICG in general. Subgroup analyses need further investigations. Efforts to generate accurate estimates of lifetime HC exposure should be emphasized for future investigations.

Keywords Hydrocarbons · Glomerulopathy · Nephrotoxicity · Occupational health

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Introduction

Occupational exposure to hydrocarbons has for many years been suggested as a potential etiological factor in chronic renal disease [2, 3, 6, 8, 13, 15, 18, 19, 23–25]. Nephrotoxins may generally be grouped into two basic categories: inorganic chemicals, mainly heavy metals, and organic chemicals such as hydrocarbons. The nephrotoxicity of heavy metals such as lead, cadmium, mercury, and arsenic has long been recognized [4, 9, 21, 22]. Hydrocarbons have not been definitively implicated as a cause of chronic renal disease. As recommended in several review articles and journal editorials [1, 5, 7, 9, 12, 14, 21], the lack of consistency from previous studies calls for further investigations.

Major case-control studies investigating the relationship between hydrocarbon exposure and chronic nephropathy have encountered to varying degrees several methodological problems such as sample size requirement, control group selection, interview procedures, questionnaire content, assessment of hydrocarbon exposure, and the ability to measure and control possible confounders.

The purpose of this case-control study was to further investigate the role of chronic exposure to hydrocarbons in the development of idiopathic chronic glomerulopathy with a particular intent to obtain the most accurate estimate of the lifetime hydrocarbon exposure for both cases and controls.

Materials and methods

Case selection

Cases were obtained from hospitals in the metropolitan areas of Oklahoma City and Tulsa, Oklahoma. All the major hospitals and nephrology clinics were contacted. To improve the completeness of recruitment of cases the study nephrologist and investigator (C.K.) provided an additional list of nephrologists in Oklahoma City and Tulsa. Participating hospitals, nephrology clinics, and nephrologists were requested to provide a complete listing of their patients whose diagnoses had an ICD code (*International Classification of Diseases*) pertinent to chronic renal disease, between 1 January 1985 and 31 December 1992. Some small clinics did not participate in the study. The likelihood of missing a significant number of patients with chronic renal disease from these clinics is very small. The recruitment process showed that small hospitals (among those that participated) had few patients with chronic renal disease. Usually patients with such a serious condition are referred to larger, better equipped hospitals. Also the review of medical records at nephrologists' private offices or clinics demonstrated that virtually all of their patients had duplicate files at the hospitals.

The case selection was aimed at retaining adult patients, 18–79 years of age, whose primary diagnosis could be labeled *idiopathic chronic glomerulopathy* or ICG. As shown in Table 1, this broad diagnostic group included three subgroups of patients: (1) biopsy-confirmed ICG patients with or without end-stage renal disease ($n = 249$); (2) patients with a clinical diagnosis of ICG without biopsy, based on the presence of an explained, persistent proteinuria exceeding 2g/24 h ($n = 20$); and (3) patients presenting with end-stage renal disease with unknown primary disease and without any more specific renal diagnosis ($n = 52$). The last subgroup was included based on the fact that glomerulopathies are the most common cause of end-stage renal disease. Chronic renal disease secondary to other disease such as diabetes, hypertension, congestive heart failure, amyloidosis, multiple myeloma, systemic lupus erythematosys, urinary obstruction, and renal artery stenosis, and conditions with well-established genetic components such as polycystic kidney or hereditary nephritis (Alport's syndrome) were excluded. Clearly acute conditions (usually resolved) were excluded, too.

Of the 17 373 records reviewed, 1780 were initially retained by the research abstractor team. Following a closer screening by the study nephrologist and after removing duplicates, 496 patients were determined as meeting the inclusion criteria described above. Of these, 161 were not available for interview: 51 had died, 40 refused to participate in the study, and 70 had moved out of the state or could not be located until the conclusion of the recruitment process. As a result a total of 335 cases were identified and interviewed.

Control selection

A general population-based control group was selected using a variation of the random-digit dialing (RDD) technique described by Waksberg [20]. A population control was matched to each case by age ($\pm$ 5 years), gender, and geographical area as determined by the case telephone number. According to the 1990 census figures, about 91% of Oklahoman households have a telephone. The RDD method may be used to effectively draw a sample from this population. On average, 42 phone numbers were called for each case; four of them resulted in refusal, three of which were refusals before it could be determined whether a qualified control lived at that residence, and only one (actually 1.3 on average, data not shown) was a refusal by a qualified control. For the 335 cases identified, 321 matched controls were obtained. The remaining 14 unmatched cases were dropped when it became apparent that locating a control for them would be impractical.

The questionnaire

The questionnaire was administered by the research staff at a location specified by the subject for his/her convenience, usually in the subject's home. The interviewers were intensively trained by the project coordinator and were given instructions with specific attention to question reading, clarifying, pace, marking, and other related procedures. The industrial hygienists on the project gave instructions as to the ascertainment of occupational history. The interviewers were blind to the subject's status (whether case or control).

Data collected through the questionnaire included medical history, medication and pain reliever use, exposure to radiation either through diagnostic or therapeutic procedures, a complete lifetime occupational history (with all job titles, names of employers, locations of jobs, responsibilities, whether full or part time, years employed and any chemical materials to which they may have been exposed), questions concerning chemical exposure outside the work place (such as hobbies), an industrial checklist, smoking history (including cigarettes, cigars, pipe tobacco, chewing tobacco, and snuff), beverage history (including consumption of coffee, tea, diet soft drinks, beer, wine, and alcohol), use of artificial sweeteners, family history of kidney problems, demographics (birth date, family size, marital status, education, and race), and interviewer's observations (respondent's condition and cooperation, estimated accuracy of information gathered).

Hydrocarbon exposure assessment

The project industrial hygiene team was led by two Certified Industrial Hygienists (CIH), both of whom hold earned doctorates in environmental health and have more than 45 years of combined experience, most of which occurred in the geographical area of the study. An extensive and detailed description of the procedure used to assess lifetime hydrocarbon exposure has been provided elsewhere by the same research team [11]. Briefly, hydrocarbon exposure was determined for each subject through the following steps: (1) development of a database of subjects' occupational histories, (2) formation of an expert panel of industrial hygienists to provide hydrocarbon exposure estimates, (3) conduction of literature survey focused on hydrocarbon exposures, (4) extraction of appropriate data from the Occupational Safety and Health Administration (OSHA) sampling database, (5) derivation of final exposure estimates for each industry/occupation category, and finally (6) determination of lifetime exposure estimates for each subject. The project industrial hygiene team was not informed of the subject's status (whether case or control) before exposure estimates were derived.

The lifetime occupational histories taken from the interview were coded using the *Standard Industrial Classification Manual* (SIC manual) and the *Standard Occupational Classification Manual* (SOC manual) [16, 17]. After taking into consideration the decade in which the job was held and by eliminating duplicates, 3159 unique SIC/SOC/decade combination were developed. A panel of expert industrial hygienists was assembled for the purpose of generating hydrocarbon exposure estimates for each of the SIC/SOC/decade combination. Estimates obtained from the expert

panel were supplemented when possible with values obtained from the literature and data from the OSHA Computerized Information System (OCIS). Thus hydrocarbon exposure estimates were determined for each of the SIC/SOC/decade combinations from estimates developed by the expert panel, quantitative and qualitative exposure data obtained from the literature, and values from the OSHA OCIS database.

For the final estimate of lifetime hydrocarbon exposure for each subject, hydrocarbon exposure estimates for each SIC/SOC/decade combination were cross-matched with the SIC/SOC/decade and length of employment for each job reported by each subject. For each subject, the length of employment (in years) in each decade for each job was multiplied by that job's hydrocarbon exposure estimate in parts per million (ppm). The individual job dose estimates were then summed for each subject to generate a lifetime hydrocarbon exposure estimate.

Statistical procedures

With respect to statistical analyses, hydrocarbon exposure was treated both as a continuous variable (exposure score in ppm as measured in the study) and as a dichotomous variable with a cutoff point set at 100 ppm based on industrial hygienists' opinion and the distribution of exposure score. Results from both approaches are presented.

When hydrocarbon exposure was treated as a continuous variable, the Wilcoxon Signed Rank Test was used to compare cases and controls for overall and subgroup analyses. When it was broken down into a dichotomous variable, the Mantel-Haenszel method for estimating odds ratio (with 95% CI) for matched sample and McNemar test (to determine the *P*-value) were used to compare cases and controls. To compare demographic data and other health-related characteristics (besides hydrocarbon exposure), paired *t* test, Mantel-Haenszel odds ratio (OR) for matched sample, and McNemar test were used as appropriate. A logistic regression for matched sample was performed as well, primarily to control for potential confounders such as race, education, consumption of soda, consumption of coffee, and use of artificial sweeteners.

Results

Three hundred twenty-one pairs of cases and matched controls were obtained and their data analyzed. Results are presented in three steps. First, demographic and other health-related data are presented in order to characterize the sample as a whole, and to examine the comparability between cases and controls. Then, the actual comparisons of hydrocarbon exposure between cases and controls (including subgroup analyses) are presented. Finally, information on additional, potential chemical exposure (occupational or not) is provided.

Renal biopsies were used for diagnosis determination in 249 of the 321 cases. Two hundred twenty-seven diagnoses were *classified* and 94 were *unclassified*. As shown in Table 1, unclassified diagnoses included unclassified glomerulonephritis (biopsy confirmed as such), clinically diagnosed ICG when no biopsy was available, and clinically diagnosed end-stage renal disease with unknown primary disease and no biopsy available. Classified diagnoses were all other diagnoses as confirmed by biopsy.

Table 1 Diagnoses and hydrocarbon exposure

Diagnostic subgroups	No. (%) of cases	Mean (median) exposure score[a]
A. Biopsy confirmed ICG[b]		
Focal glomerular sclerosis	76 (23.7)	111 (37)
Membranous GMN	57 (17.8)	259 (50)
IgA nephropathy	38 (11.8)	124 (33)
Minimal change disease	19 (5.9)	54 (6)
Mesangial proliferative GMN	14 (4.4)	97 (36)
Crescentic GMN	9 (2.8)	92 (27)
Focal proliferative	6 (1.9)	117 (117)
Memoranoproliferative GMN	4 (1.2)	77 (1)
Diffuse proliferative	3 (0.9)	111 (15)
Fibrillary	1 (0.3)	74 (74)
Unclassified GMN[c] (biopsy confirmed as such)	22 (6.9)	195 (132)
B. Clinically diagnosed ICG (no renal biopsy available)[c]	20 (6.2)	157 (149)
C. Clinically diagnosed end-stage renal disease (no renal biopsy available)[c,d]	52 (16.2)	247 (108)

[a] Mean and median score in ppm
[b] Regardless of clinical stage of the disease (end-stage or not)
[c] Referred to as unclassified diagnoses in the text
[d] With unknown primary disease

There were more males than females (58% vs 42%) in the entire sample. Forty-two percent of all subjects were over 50 years of age. The case sample included about 7% fewer whites than did the control sample [OR = 0.59 (95%CI 0.36–0.97)]. Fewer cases had completed more than 12 years of education [CR = 0.58 (95%CI 0.39–0.87). The distributions of marital status and family size did not differ significantly between cases and controls.

With respect to personal medical history, the case sample included significantly more subjects with reports of high cholesterol, arteriosclerosis, fast heartbeats, angina, gout, gallstones, and frequent headaches than did the control sample. Controls had significantly greater number of people with sinus condition. The history of prescription and nonprescription drug use did not show any significant differences between cases and controls [10]. Other self-reported medical conditions (data not shown) were not statistically different between cases and controls. With respect to family medical history no significant differences were noticed, although more cases (12%) apparently had siblings with chronic renal problems than did controls (7%). Information on medical history should be interpreted with caution since it was self-reported by the subjects, not obtained through a systematic review of medical records.

Regarding certain health habit measures, based on average use and duration of use, controls had a greater intake of coffee ($P = 0.016$) and artificial sweeteners ($P = 0.047$). The consumption of diet soda was higher for controls, but the difference was only suggestive

Table 2 Hydrocarbon exposure in cases and controls

A. *Using hydrocarbon exposure as a continuous variable* (score are expressed in ppm; p value = 0.757 for Wilcoxon Signed Rank Test)

	Mean	SD	Median	(Range)
Cases	165	298	48	(0–2599)
Controls	162	283	43	(0–1790)

B. *Using hydrocarbon exposure as a dichotomous variable* (score $\geqslant 100$ ppm = exposed; score < 100 ppm = nonexposed; percentages are based on the total of 321 matched pairs; OR for matched sample = 1.39, 95% CI 0.94–2.04; McNemar $\chi^2 = 2.75, P = 0.097$

	Cases	
	Exposed	Nonexposed
Controls		
Exposed	71 (22%)	44 (14%)
Nonexposed	61 (19%)	145 (45%)

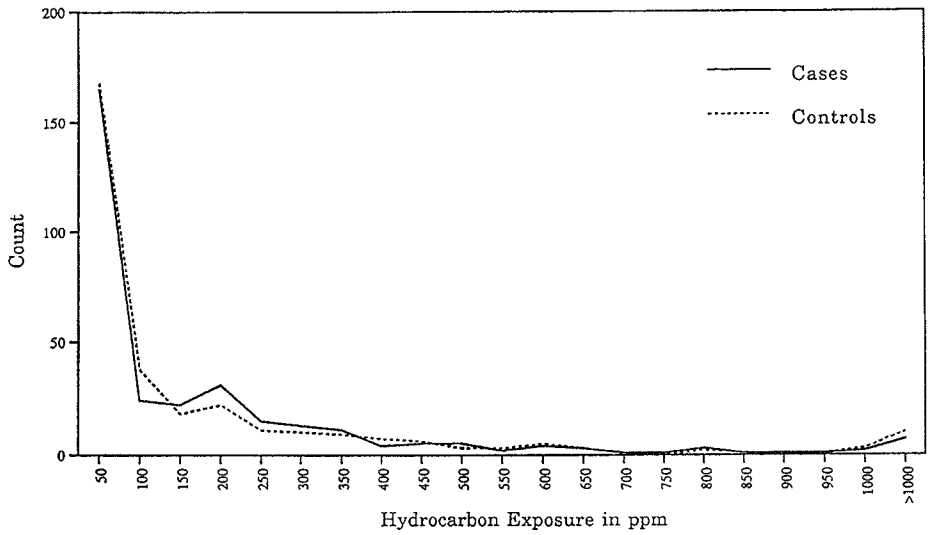

Fig. 1 Distribution of hydrocarbon exposure

($P = 0.07$). There was no significant differences between cases and controls for smoking history and consumption of tea and alcoholic beverages.

As for the overall comparison of lifetime hydrocarbon exposure, which was the focus of the study no significant difference was found between cases and controls. Cases had a hydrocarbon exposure mean score of 165 ppm (median 48 ppm) as compared to 162 ppm (median 43 ppm) for controls ($P = 0.757$) (Table 2). Using hydrocarbon exposure as a dichotomous variable (exposed if score $\geqslant 100$ ppm vs nonexposed if score < 100 ppm), the case sample had a higher proportion of exposed persons than the control sample (132/321 vs 115/321, see also Table 2), but this difference was not statistically significant at the 0.5 level (OR = 1.39, 95%CI 0.94–2.04, and McNemar chi square = 2.75, $P = 0.097$). The frequency distributions of exposure scores by increment of 50 ppm as depicted on Fig. 1 were not statistically different between cases and controls either. After controlling for possible confounders such as race, eduction, and consumption of coffee, diet soda, and artifiical sweetners, through logistic regression for matched samples no significant difference was found for the overall comparison between cases and controls (Table 3).

Subgroup analyses comparing cases with controls showed mixed results (Table 4). The difference in hydrocarbon exposure, when treated as a dichotomous variable, was found to be significant between cases and controls in those with unclassified diagnosis ($P = 0.009$), in male subjects ($P = 0.042$), in subjects over 30 years of age ($P = 0.049$), and in those with a creatinine level $\geqslant 4.0$ mg/dl ($P = 0.016$). Yet, the statistical significance disappeared when hydrocarbon exposure was used as a continuous variable (in ppm), except for those subjects with unclassified diagnosis ($P = 0.043$). When considering only the pool of cases, i.e., the 321 patients with CIG (without comparing with controls), the mean levels of hydrocarbon exposure for different diagnostic categories ranged from 259 ppm

Table 3 Multivariate analysis (logistic regression for matched sample). Number of pairs: 321; outcome: chronic renal disease

Predictors	Coefficient	Std. error	Wald χ^2	*P* value
A. *Using hydrocarbon exposure as a continuous variable scored in ppm*				
Exposure score	− 0.00003	0.00003	0.007	0.934
Race	0.603	0.317	3.614	0.057
Education	− 0.527	0.211	6.230	0.013
Coffee	− 0.029	0.018	2.485	0.115
Soda	− 0.001	0.002	0.154	0.695
Sweetener	− 0.018	0.041	0.191	0.662
B. *Using hydrocarbon as a dichotomous variable (cutoff at 100 ppm)*				
Exposure status	0.253	0.204	1.535	0.215
Race	0.566	0.315	3.240	0.072
Education	− 0.516	0.212	5.904	0.015
Coffee	− 0.028	0.018	2.433	0.119
Soda	− 0.001	0.002	0.150	0.699
Sweetener	− 0.011	0.041	0.076	0.783

(median 50) for membranous glomerulonephritis (GMN) to 54 ppm (median 6) for minimal change glomerulopathy (Table 1 last column).

The interview forms also contained information on additional chemical exposures, including exposure to large chemical spills or leaks, acute exposures resulting in illness or nonoccupational exposure to any chemicals relating to farming, home maintenance, yard work, gardening, hobbies, or painting. More controls (150) than cases (113) were involved in yard or home maintenance ($P = 0.003$). Controls had slightly more exposure to cleaning and household products and pesticides ($P = 0.007$). Controls (72) were more likely than cases (51) to have hobbies involving solvents ($P = 0.033$). No differences were seen with respect to exposure to large chemical spills, inhalation or skin contact episodes, incidence of acute exposure leading to illness, being raised on a farm, driving a tractor, home painting, or type of paint used. The industrial checklist of 30 occupations, intended to refresh the subject's memory of employment in industries with potential for hydrocarbon exposure, which was used in the assessment of hydrocarbon exposure by the project industrial hygiene team, did not reveal any differences between cases and controls.

Discussion

That no significant association between long-term hydrocarbon exposure and ICG was found in this study was surprising since most previous case-control studies have shown some kind of relationship. The lack of consistency between studies indicates that the role of low-level, long-term hydrocarbon exposure in the development of ICG should be reconsidered. Differences in methodology may not entirely explain the discrepancy between our results and those reported in other studies.

The present study was intended to measure more accurately the actual level of lifetime exposure of hydrocarbons, expressed in parts per million (ppm),

Table 4 Selected subgroup analyses

Subgroup	Mean (median)	*P*-value[a]	No. exposed (%)[b]	OR (95%)	*P*-value[c]
A. *Male subjects* ($n = 187$ pairs)					
Cases	248 ppm (154)		115 (61%)		
		$P = 0.533$		1.60 (1.02–2.51)	$P = 0.042$
Controls	240 ppm (112)		97 (53%)		
B. *Over 30 years* ($n = 252$ pairs)					
Cases	196 ppm (89)		122 (48%)		
		$P = 0.617$		1.51 (1.01–2.23)	$P = 0.049$
Controls	190 ppm (64)		103 (41%)		
C. *Creatinine level* $\geqslant$ 4mg/dl[d] ($n = 111$ pairs)					
Cases	195 ppm (101)		56 (51%)		
		$P = 0.124$		2.40 (1.18–4.90)	$P = 0.016$
Controls	159 ppm (63)		42 (38%)		
D. *Unclassified*[e] ($n = 94$ pairs)					
Cases	216 ppm (122)		49 (52%)		
		$P = 0.043$		2.67 (1.28–2.72)	$P = 0.009$
Controls	159 ppm (43)		34 (36%)		

[a] *P* value for Wilcoxon Signed Rank Test
[b] Exposed if hydrocarbon score $\geqslant$ 100 ppm. Actual 2 × 2 tables for matched sample for each subgroup may be obtained upon request from author
[c] *P* value for McNemar test
[d] Subgroup of cases with creatinine $\geqslant$ 4.0 mg/dl and their matched controls
[e] Subgroup of cases with unclassified diagnoses as defined in the text and their matched controls

a familiar quantitative exposure assessment scale used regularly by industrial hygienists. It incorporated information concerning hours/day, days/week, and number of years on the job with hydrocarbon exposure estimates from occupational data sources. The methodology was quite involved and detailed and has been described in another manuscript by members of the same research team [11]. Previous studies had a cruder assessment scale of long-term hydrocarbon exposure, which may have led to varying degrees of misclassification of the exposure for either cases or controls.

The criteria used to select cases of chronic renal disease are very far from uniform between studies. Heterogeneity of diagnoses represents a serious risk of selection bias. Whether or not the case diagnoses were confirmed by renal biopsies in previous studies is not clear. The present study had 249 case diagnoses (78%) confirmed by a renal biopsy, and 94 diagnoses were grouped into the unclassified category (Table 1). Comparison of studies may be difficult if they have not all used the same subgroups of renal disease. The number of unclassified diagnoses in other studies varied from 60/247 cases [8], 23/37 cases [6] 16/63 cases [25], and 4/50 cases [13], to none or unknown [3, 15, 18].

The possibility of confounding was less likely in this study. A multivariate logistic regression was applied to control for confounders. We included in the model those factors that showed a significant difference between cases and controls (or whose difference was close to the conventional level of significance) at univariate analysis. The adjusted odds ratio was slightly lower than that obtained through overall univariate analysis. This result is not surprising. On the one hand our data showed that hydrocarbon scores were higher for less educated and nonwhite subjects. On the other hand, the case group included a greater number of nonwhite and less educated people. Controlling for education and race would further reduce the difference between cases and controls as to hydrocarbon exposure.

Another problem with renal disease is that renal function may have been decreasing for many years before presenting with significant symptoms. Kidneys have the capacity to perform efficiently even though a large portion of their tissue is being destroyed. Those controls (supposedly healthy subjects) who had very high exposure scores may be experiencing loss of renal function that has not yet reached a stage where symptoms begin to be obvious enough for them to attract medical attention. Renal function studies on controls with high scores would be an interesting follow-up project.

Some limitations to the study should be mentioned. For instance, cases were limited to surviving patients. Those patients who had died by the time of recruitment were no longer available for interview and were excluded. With respect to controls, although the random-digit dialing technique as used in this study has proved relatively efficient, the process has its drawbacks. If we consider the 407 refusals by potential, eligible controls for the 321 controls obtained (on average 1.3 per case, as stated earlier), the response rate for controls would be 44%. Very little (except age and gender) is known about these refusals, restricting comparisons with the actual sample who participated in the study. Such comparisons might help determine whether there is any relevant difference between participant and nonparticipant controls. As stated earlier, another limitation is that data collected through the questionnaire were solely based on self-report. They were not double-checked from other sources such as hospital or employer's records. The reliability of self-reported information may always be questioned.

In summary, this study found no statistically significant association between ICG and hydrocarbon exposure in the overall analysis, independent of the way the hydrocarbon exposure was treated, i.e., as a continuous or a dichotomous variable. Subgroup analyses showed conflicting results, except for the category of unclassified diagnoses, where the difference in hydrocarbon exposure between cases and controls was consistently significant in both approaches. The crudeness of the measurement of hydrocarbon exposure in most studies and the heterogeneity of diagnoses used to define the cases in different studies are among the major problems faced by investigators when assessing the role of long-term hydrocarbon exposure in the development of chronic renal disease. We think that results obtained by using hydrocarbon exposure as a continuous variable in ppm are probably the most informative. Future research should emphasize accurate assessment of lifetime hydrocarbon exposure and the uniformity of diagnoses for inclusion in the case definition.

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