Occupational and Other Exposures Associated with Male End-Stage Renal Disease: A Case/Control Study

N. Kyle Steenland, PhD, Michael J. Thun, MD, MPH, C. William Ferguson, BA, and Friedrich K. Port, MD

Abstract: We conducted a case-control study of 325 men ages 30–69 who were diagnosed with end-stage renal disease (ESRD) between 1976 and 1984, and resided in four urban areas of Michigan in 1984. Cases were selected from the Michigan Kidney Registry and excluded men with diabetic, congenital, and obstructive nephropathies. Controls were selected by random-digit dialing and were pair-matched to cases for age, race, and area of residence. Telephone interviews were conducted with 69 percent of eligible cases and 79 percent of eligible controls. Risk of ESRD was significantly related to phenacetin or acetaminophen consumption (odds ratio(OR) = 2.66), moonshine consumption (OR = 2.43), a family history of renal disease (OR = 9.30); and regular occupational exposures to solvents (OR = 1.51) or silica (OR = 1.67). Particular occupational exposures with elevated risk were solvents used as cleaning agents and degreasers (OR = 2.50) silica exposure in foundries or brick factories (OR = 1.92), and silica exposure during sandblasting (OR = 3.83). Little or no trend of increased risk with duration of exposure was found for these occupational exposures, with the exception of silica in sandblasting. Limitations of these data include representativeness of cases, possible overreporting by cases, and misclassification of exposures inherent in self-reports. (*Am J Public Health* 1990; 80:153–159.)

Introduction

End-stage renal disease (ESRD) is a major public health problem in the United States. In 1985, over 120,000 Americans suffered from ESRD, and each year approximately 30,000 new cases arise. Cases require dialysis or transplant, and the current annual cost exceeds $2 billion a year, almost entirely paid for by the federal government.[1] Yet very little is known about the causes of ESRD. A minority of ESRD cases are characterized etiologically (e.g., lead nephropathy, analgesic nephropathy, diabetic nephropathy).[2] Many cases are described histologically (e.g., glomerulonephritis, interstitial nephritis). Others are described as "hypertensive nephrosclerosis," with no determination of whether the kidney damage preceded or followed the hypertension.

Acute exposures to heavy metals can cause acute renal disease or dysfunction. There is also some indication that occupational exposures to metals, solvents, and silica may play a role in chronic renal disease.[2] Evidence indicates excess renal disease among workers exposed chronically to uranium,[3] cadmium,[4] and lead.[5–8] In addition, animal studies have shown that high exposures to carbon tetrachloride or perchloroethylene can also cause acute kidney damage.[9] In recent years, case-control studies have indicated that chronic glomerulonephritis is associated with occupational exposure to hydrocarbons.[10–15] Evidence of silica-related renal disease is limited to case reports[16,17] of acute renal failure following high exposures to silica.

To further investigate the hypothesis that occupational exposures might be associated with end-stage renal disease, we have conducted a case-control study of males diagnosed with ESRD between 1976–1984 in four urban areas of Michigan.

Methods

Cases

Patients were identified from the Michigan Kidney Registry,[18,19] which registers all new cases of ESRD in Michigan, collects demographic data, and records the diagnosis provided by the attending physician at the time of the ESRD diagnosis. Eligible cases were all men diagnosed between 1976 and 1984 with selected diagnoses of ESRD, ages 30–69 at diagnosis, and living in four urban areas in southeastern Michigan in 1984. Cases were restricted to those still living because of the unreliability of next-of-kin for reporting detailed exposure history. Diagnoses excluded were diabetic nephropathy, polycystic kidney disease, heroin nephropathy, lupus nephropathy, nephropathy due to malignancy, Alport's syndrome, unspecified chronic renal failure, nephropathy due to obstruction, and a variety of less common nephropathies grouped as "other." These diagnoses were excluded because most have known nonoccupational causes. The diagnoses included were glomerulonephritis, hypertensive kidney disease (nephrosclerosis), and interstitial kidney disease (including pyelonephritis, lead nephropathy and other unspecified interstitial disease). These diagnosis included represented about 65 percent of the ESRD patients in Michigan.

The summary diagnoses in the Michigan Kidney Registry may not be accurate, since many of ESRD patients present with advanced disease, and only an estimated 5 percent or less of ESRD patients have been biopsied. For example, some physicians may have classified a case as nephrosclerotic on the basis of high blood pressure at the time of diagnosis, without further data indicating that the high blood pressure preceded the kidney disease. While the diagnoses available to the Registry were sufficient to exclude certain well-defined entities such as diabetic nephropathy, the diagnoses of the cases which were included (nephrosclerosis, glomerulonephritis, and interstitial nephritis) were not clearly differentiated one from another, and were therefore analyzed together.

The Michigan Kidney Registry identified 612 eligible cases. Contact was initially made by mail, with phone calls to nonrespondents. Eighty-seven of these men had died prior to any attempt to contact them, and 14 died after consenting to be interviewed but prior to being interviewed; another 26 men were found upon interview to be ineligible because they had kidney disease subsequent to diabetes or prolonged heroin use; and 14 men could not participate either because they were in prison or were mentally incompetent. Of the remaining 471 men, we interviewed 325 (69 percent). More nonre-

From the National Institute for Occupational Safety and Health (Steenland, Thun) and the Michigan Kidney Registry (Ferguson, Port). Address reprint requests to N. Kyle Steenland, PhD, NIOSH, R13, Robert A. Taft Laboratories, 4676 Columbia Parkway, Cincinnati, OH 45226–1998. This paper, submitted to the Journal February 6, 1989, was revised and accepted for publication June 26, 1989.

spondents than respondents were Black (67 percent vs 44 percent) and were living in inner Detroit (61 percent vs 40 percent).

Controls

Controls were chosen from the general population in the four urban areas of the study, via random-digit dialing. Controls were pair-matched to cases on age (within five years), race (White versus other), sex, and residential area based on telephone prefix, grouped in 15 geographical areas covering the entire study area. Men who had chronic kidney disease were excluded.

Random digit-dialing was conducted according to a modified Waksberg method[20] from February to October 1985. A potential control identified in calling the randomized number was allowed to match any case of the same age and race in the same geographical area. If numbers did not answer, repeated phone calls were made varying the time of day and day of week prior to abandoning the phone number.

A total of 3,962 numbers were called (with an average 1.95 calls per number). Of these, 61 percent (2,430) were working residential numbers. Twenty-three percent (567) of those who answered at a residential phone refused to provide initial information about males in the household. Once a household did provide such initial information and proved to have an eligible male, 79 percent (325/410) agreed to be interviewed. We compared the 325 controls (respondents) with the 85 individuals who were eligible but refused the interview (refusals). Potential controls who refused differed from respondents regarding race (55 percent Black versus 44 percent), and residence (60 percent from inner Detroit, versus 40 percent).

Interviews

Telephone interviews lasting 30–45 elicited demographic, medical history, and occupational histories. Cases were interviewed from November 1983 through December 1985, while controls were interviewed from March 1985 to January 1987. Interviewers knew which respondents were cases from the answers to a variety of questions. Neither cases nor controls were aware of the hypotheses of the study.

Respondents were asked about regular moonshine use, regular pain pill use, family history of serious kidney disease, years of education, smoking, lead poisoning, injected antibiotics, and environmental exposure to metals and solvents. Exposures were truncated at the year of diagnosis as end-stage for cases and their matched controls. Phenacetin or acetaminophen use was determined by a review of answers to a question about the use of pain pills on a regular basis (more than one pill per week for two years or more). Brands of analgesics were classified by whether or not they contained phenacetin or acetaminophen in the early 1970s, based on data provided by Dr. Dale Sandler at the National Institute of Environmental Health (personal communication). Acetaminophen was grouped with phenacetin because it is a metabolite of phenacetin and may also be associated with renal disease.[21]

Family history of renal disease was considered positive if kidney disease (excluding stones and cancer) occurred among parents, children, or siblings, and was not known to have occurred subsequent to diabetes or trauma.

Each job held for more than six months since age 18 was classified for occupation and industry using 1980 US Census codes.[22] For each job, questions were asked regarding regular exposure to solvents, metal particles, metal fumes, mercury, oil and gasoline, and ammonia. Ammonia was included as a check, since it was not thought to be related to kidney disease. For each material, details of the exposure (e.g., hours per week, process description) were recorded. Work history for cases and their matched controls was considered only up until the year of the diagnosis as end-stage for the case.

We reviewed the completed interviews without knowledge of case-control status and with the assistance of NIOSH (National Institute for Occupational Safety and Health) industrial hygienists. For positive responses to questions about metal fumes, metal particles, solvents, and silica, we created sub-categories by type of solvent, metal, etc (Table 2). When an individual had been unable to recall the number of hours per week during which he was exposed, we arbitrarily assigned five hours a week for infrequent exposure, 20 hours a week for frequent but not constant exposure, and 40 hours a week for constant exposure. In a few cases, a self-reported exposure to a substance was clearly wrong due to misclassification by the respondent, and we corrected the response (e.g., self-reported exposure to solvents such as "lime" or "pesticides"). In other cases, we called a respondent back for clarification.

Analysis

Odds ratios (OR) were estimated from conditional logistic regression.[23] Odds ratios for exposure were adjusted for confounding by nonoccupational variables and other occupational variables. Dummy variables (missing versus not exposed) were added to the model to account for those instances in which study subjects were missing data for lifestyle variables. When subjects were missing data on exposure, they were called again to resolve the problem. No cases or controls were lost at the analysis stage due to missing data.

We compared the occupational history of cases and controls in several ways:

- We considered a history of having ever been exposed to specific substances (e.g., all solvents, or solvents used as degreasers).
- We estimated cumulative exposure by multiplying the number of hours per week a man was exposed by the number of years in the job for each job, and summing over all jobs.
- Finally, for solvents, we used an exposure matrix developed by Shalat and others[24] to identify jobs likely to have involved high exposure to solvent (see Appendix). Cases and controls were then compared regarding work in these jobs. Analyses were also run which discounted any exposures (lifestyle or work history) which occurred in the five years preceding the date of diagnosis of the case.

Results

Table 1 gives the frequency of a number of lifestyle variables among (unmatched) cases and controls. After controlling for confounding by years of education and date of birth, phenacetin/acetaminophen use and moonshine use were both significant predictors of kidney disease, with odds ratios of 2.66, (95% confidence intervals 1.04, 6.82) and OR 2.42 (95% CI 1.10, 5.36), respectively. Cumulative measures of exposure for these variables showed positive but nonsignificant trends of increased risk with increased duration of use. The findings for family history were the most remarkable, with cases much more likely than controls (OR 9.30, 95% CI 7.99, 10.82) to have a close family member (parent,

TABLE 1—Demographic Characteristics of Study Population

Characteristics	Cases (n = 325)	Controls (n = 325)
Average years of education	11.7	12.5
Number non-White*	143	143
Number living in inner Detroit	130	130
Number living in outer Detroit	114	114
Number living in Flint, Saginaw, or Lansing	81	81
Some college	106	129
Average date of birth	1930.0	1930.5
Family kidney disease	37	7
Regular moonshine use	31	10
Regular use of phenacetin/acetaminophen	22	7
Current cigarette smokers**	143	151
Former cigarette smokers	119	91
Average years smoked, current smokers	35.8	34.4

*93% of non-Whites were Black.
**Includes men who quit within one year prior to their diagnosis.

child, sibling) with kidney disease (excluding kidney stones and kidney disease subsequent to trauma or diabetes). When any exposure within five years of the date of diagnosis of the case and his matched control was ignored, the results for moonshine consumption were unchanged, while the odds ratio for phenacetin/acetaminophen fell slightly from 2.66 to 2.47 (0.86, 7.12).

Controls were more likely ever to have worked in jobs as professionals, managers, or technicians (56 percent vs 46 percent). The most common industry for both cases and controls was the automobile industry, in which 51 percent of cases and controls had worked. The next most common was construction (17 percent), followed by foundries (9 percent), and hospitals (6 percent).

Table 2 presents the results of a logistic regression for occupational variables. Adjusted odds ratios were significantly elevated for exposure to solvents used as degreasers and silica used in foundries or in sandblasting. An elevated odds ratio was also observed for exposure to lead. No interaction terms were significant between any exposure variables and any lifestyle variables or other exposure variables.

Most men exposed to solvents used as degreasers worked in metal manufacturing industries (Census codes 271–370, 47 cases, 22 controls). Within this sector, the principal industry was auto manufacturing (32 cases, 17 controls). About equal numbers of cases and controls were exposed in dry cleaning shops (six cases, five controls). The subcategories of silica with elevated risk were exposure in foundries, brick making, or sandblasting, which were the categories likely to have involved the highest exposures.

There were no strongly positive trends of increased risk with increased duration of exposure for any exposure variable, with the exception of silica used in sandblasting. The odds ratio for exposure to silica in sandblasting for 2.5 years for 40 hours a week (cumulative duration = 100, an average exposure) was 2.74 (.69, 9.97).

We also considered employment in jobs considered to have had high solvent exposure (see Appendix). There were no differences between cases and controls regarding either ever having worked in these jobs (31 cases, 30 controls) or the length of time having worked in these jobs (5.3 years for cases, 6.8 for controls).

The above results for occupational variables were changed little when all exposures in the five years preceding the date of diagnosis of the case (and matched control) were ignored. For example, the odds ratio for ever having been exposed to solvents increased from 1.51 to 1.68 (1.13, 2.48), while the odds ratio for ever having been exposed to silica increased from 1.67 to 1.81 (1.09, 3.02). The coefficients for cumulative duration of exposure also were affected only slightly (for solvents, the coefficient changed from −.0000095 to .00009, while for silica it changed from −.0039 to −.0004).

Discussion

This study suffered from several limitations inherent in an interview study of ESRD patients. First, patients are often

TABLE 2—Adjusted Logistic Regression Results for Occupational Variables

Exposure	Number Cases Exposed	Number Exposed	Odds Ratio* (95% CI) Ever-Never Exposed*
All solvents	124	82	1.51 (1.03, 2.22)
Solvents used in paints and glues	38	34	1.01 (.58, 1.74)
Solvents used as cleaning agents or degreasers	94	40	2.50 (1.56, 3.95)
Solvents used in other processes	17	13	1.05 (.44, 2.48)
Metal fumes	139	94	1.17 (.77, 1.80)
Lead (soldering and other)	32	16	1.73 (.82, 3.65)
Iron/steel	109	72	1.15 (.73, 1.80)
Welding fumes	51	44	0.75 (.44, 1.28)
Metal particles	119	96	0.97 (.58, 1.48)
Oil and gas	135	129	0.74 (.64, 1.51)
Gas and diesel fuel	74	65	0.98 (.49, 1.06)
Motor and fuel oil	68	49	1.13 (.69, 1.84)
Cooling fluids	26	40	0.50 (.27, .92)
Silica	87	54	1.67 (1.02, 2.74)
Cement and sand	17	18	0.78 (.34, 1.78)
Brick and foundry	57	31	1.92 (1.06, 3.46)
Other silica	16	12	1.08 (.42, 2.77)
Sandblasting	9	3	3.83 (.97, 15.19)
Ammonia	33	19	1.31 (.66, 2.60)

*Models included variables for date of birth, years of education, regular use of moonshine, and family history of kidney disease, metal fumes, solvents, and silica.

quite ill and difficult to interview. This may contribute to a high refusal rate. Second, 482 men had been diagnosed between 1976–84 and would have been eligible for our study but had died by 1984, so that our study was of a population of survivors. A demographic analysis of those who died before they could enter the study indicated they differed little on race or residence, but were approximately five years older than men who were studied. A bias in our results could exist if these decedents differed substantially in their occupational exposures with those who survived to be studied. It is possible, for example, that those who died were of lower socioeconomic status and had different exposures than those who survived, although it is not clear that this would either increase or decrease odds ratios. We did not attempt to use of surrogate interviews by next-of-kin because next-of-kin could not report the decedent's detailed occupational histories. Third, it is difficult to study separate diagnostic groups among ESRD patients. We did not have confidence that the summary diagnosis in the Registry was sufficiently accurate to allow us to divide our data into the principal diagnostic groups (glomerulonephritis, nephrosclerosis, pyelonephritis) for diagnosis-specific analyses. Many of the above difficulties could be overcome by studying individuals with less severe kidney disease, prior to end-stage. With little data available to date, it is not clear whether occupational associations are more or less apparent in patients with end-stage versus less severe disease.

As in many case-control studies, we relied on self-reports to determine exposures. Cases may have had a tendency to overreport exposures compared to controls. We included a variable (ammonia exposure) thought not to be related to renal disease to test such overreporting. Cases did report more exposure to ammonia than did controls, although numbers were small and the difference was not statistically significant. Complicating any assessment of overreporting among cases was the fact that cases were more likely to have had less education than controls, and were more likely to have worked in blue-collar jobs than controls. Hence, they were more likely to have truly been exposed to occupational agents. This increased likelihood of "blue collar" status among cases compared to controls may have been an artifact of the random-dial dialing process. An alternative explanation is that our findings may represent a real phenomenon, whereby people with renal disease are of a lower socioeconomic strata than those without renal disease. While the published literature provides little data to support this thesis, one recent report reached similar conclusions.[21] To control for the observed confounding effects of socioeconomic class in our study, we have included a variable in the model for years of education.

Regarding nonoccupational variables, the effect of moonshine on renal disease is presumably due to contamination by lead.[25] Other studies have indicated that analgesics, particularly phenacetin, may be associated with renal disease (see the consensus report[26]).

Regarding occupational variables, our findings of elevated risk associated with self-reported exposure to solvents (used as cleaning agents or degreasers) and silica (used in foundries or brick manufacturing, and in sandblasting) are consistent with reports of renal disease resulting from acute exposure to these substances. An effect of solvents on chronic renal disease has also been seen in case-control studies.[15]

The lack of trend with duration of exposure to solvents can be seen as weakening the observed association between solvents and ESRD. However, duration of exposure may be a poor surrogate for cumulative dose, if those with short-term exposures also have higher exposures. Similarly, the negative findings for solvents using the pre-defined "high exposure" jobs may be due to the insensitivity of the job-exposure matrix which we used. Few attempts have been made to validate job-exposure matrices (none had been attempted for the one we used), and when such attempts have been made results have not always been encouraging.[27]

The covariate with the strongest association with renal disease in our study was a history among close relatives (parents, children, siblings) of renal disease (other than stones, or kidney disease subsequent to cancer or trauma). Due to the extremely strong association (odds ratio 9.3) observed for this variable, it seems unlikely that this positive association was due to overreporting. Familial patterns are known to exist for a number of relatively rare kidney diseases, but many of these would have been excluded from our case series (e.g., Alport's syndrome, polycystic kidney disease). Family history of hypertension, however, was not examined in our study, and may have contributed to the positive association we observed.

In summary, this study found a number of associations with ESRD which, in our opinion, warrant further investigation. These associations were generally supported by other data in the literature. The epidemiology of chronic renal disease is a relatively new area of investigation with many potential benefits, in light of the high prevalence of renal disease, its high mortality rate, and its high cost of treatment.

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REFERENCES

1. HCFA: End-stage Renal Disease 1985. Baltimore, Maryland: Health Care Finance Administration (HCFA) #03274, 1987.
2. Landrigan P, Goyer R, Clarkson T: The work relatedness of renal disease. Arch Environ Health 1984; 39(3):225–230.
3. Waxweiler R, Roscoe R, Archer V, *et al:* Mortality follow-up through 1977 of the white underground miners cohort examined by the US Public Health Service. *In:* Gomez M (ed): Radiation Hazards in Mining. New York: Society of Mining Engineers, 1981.
4. Friberg L, Elinder C, Kjellstrom T, *et al,* (eds): Cadmium and Health, Vol II. Boca Raton, Florida: CRC Press, 1986.
5. Cooper W, Wong O, Kheifets L: Mortality among employees of lead battery plants and lead-producing plants, 1947–1980. Scand J Work Environ Health 1985; 11:331–345.
6. Selevan S, Landrigan P, Stern F, *et al:* Mortality of lead smelter workers. Am J Epidemiol 1985; 122, 4:673–683.
7. Malcolm D, Barnett H: A mortality study of lead workers. Br J Ind Med 1982; 39:404–410.
8. McMichael A, Johnson H: Long-term mortality profile of heavily exposed lead smelter workers. JOM 1982; 24:378.
9. Zimmerman SW, Norbach DH: Nephrotoxic effects of long-term carbon tetrachloride administration in rats. Arch Pathol 1980; 104(2):94–99.
10. Zimmerman S, *et al:* Hydrocarbon exposure and chronic glomerulonephritis. Lancet Aug. 2, 1975; 2:199–201.
11. Ravnskov U, Forsberg B, Staffan Skerfving: Glomerulonephritis and exposure to organic solvents. Acta Med Scand 1979; 205:575–579.
12. Ravnskov U, Lundstrum S, Norden A: Hydrocarbon exposure and glumerulonephritis. Lancet Nov. 26, 1983; 2:1214–1216.
13. Finn R, Fennerty A, Ahmad R: Hydrocarbon exposure and glomerulonephritis. Clin Nephrol 1980; 14(4):173–175.

14. Churchill D, Fine A, Gault M. Association between hydrocarbon exposure and glomerulonephritis. Nephron 1983; 169–172.
15. Lauwerys R, Bernard A, Viau C, *et al:* Kidney disorders and hematotoxicity from organic solvent exposure. Scan J Work Environ Health 1985; 11 (suppl 1):83–90.
16. Bolton W, Suratt P, Stingill A: Rapidly progressive silica nephropathy. Am J Med 1981; 71:823–828.
17. Giles R, Sturgill B, Suralt P, *et al:* Massive proteinuria and acute renal failure in a patient with acute silicoproteinosis. Am J Med 1978; 64:336–342.
18. Weller J, Port F, Swarz R, *et al:* Analysis of survival of end-stage renal disease patients. Kidney Int 1982; 21:78–83.
19. Weller J, Wu S, Ferguson W, *et al:* End-stage renal disease in Michigan. Am J Nephrol 1985; 5:84–95.
20. Waksberg J: Sampling methods for random digit dialing. J Am Stat Assoc 1978; 73:40–46.
21. Sandler D, Smith J, Weinberg C, *et al:* Analgesic use and chronic renal disease. N Engl J Med 1989; 320(19):1238–1243.
22. US Bureau of the Census: Alphabetical index of industries and occupations. Washington, DC: US Dept of Commerce, May 1981.
23. SAS: Proportional Hazards General Linear Models (PHGLM) Procedure, Supplemental Users Guide. Cary, NC: SAS Inc, 1986.
24. Shalat S: Occupational and other risk factors of Alzheimer disease and related disorders. ScD thesis Harvard University, 1986.
25. Bennett W: Lead nephropathy. Kidney Int 1985; 28:212–220.
26. Robinson R (chairperson, panel): Analgesis-associated kidney disease. JAMA 1984; 251(23):3123–3125.
27. Magnani C, Pannet B, Winter P, *et al:* Application of a job-exposure matrix to national mortality statistics for lung cancer. Br J Ind Med 1988; 45:70–72.

APPENDIX

*US Census Codes for Jobs Considered Likely to Have Had High Exposures to Solvents**

Occupation Code/Occupation	Industry Code	Industry
203 Clinical lab tech	any	any
224 Chemical tech	any	any
455 Pest control	any	any
188 Painter, sculptor	any	any
789 Handpainting, coating, decorating	any	any
579 Painters, constr and maint	any	any
736 Typesetters	any	any
734 Printing machine operators	any	any
759 Painting/paint spray machine opr	any	any
885 Garage servs/gas station attnd	any	any
764 Washing, cleaning, pickling	270–301	Metal
725 Misc. metal/plastic processing machine operator	270–301	Metal
756 Mixing/blending machine opr	181,191,192,190	Chemicals, drugs, paints
	180,210,211,212	Rubbers and plastics
757 Separating filtering mach opr	181,191,192,190, 200,201	Chemicals, drugs, paints Fuel
758 Compressing/Compacting mach opr	180,210,211,212	Rubber, plastics
709 Grinding, abrasive, buffing, polishing	180,210,211,212	Rubber, plastics
705 Milling/planning mach opr	180,210,211,212	Rubber, plastics
777 Misc. machine opr	180,210,211,212	Rubber, plastics
755 Shaping/forming mach opr	180,210,211,212	Rubber, plastics
753 Cementing/gluing mach opr	180,210,211,212	Rubber, plastics
786 Handcuffing/trimming	180,210,211,212	Rubber, plastics
787 Hand molding/casking/forming	180,210,211,212	Rubber, plastics
796 Production inspectors	180,210,211,212	Rubber, plastics
889 Laborers	220,221,222	Leather
745 Shoe machine opr	220,221,222	Leather

*SOURCE: Shalat 1986. Shalat ranked all possible jobs on a scale of 0–3 for both likelihood and intensity of exposure. Jobs here defined as high solvent jobs were those for whom the product of intensity and exposure was five or more.