NIH Public Access
Author Manuscript
Am J Ind Med. Author manuscript; available in PMC 2012 March 12.

Published in final edited form as:
Am J Ind Med. 2010 March ; 53(3): 217–223. doi:10.1002/ajim.20788.

Occupational Factors and Risk of Parkinson's Disease: A Population-Based Case–Control Study

Jordan A. Firestone, MD, PhD, MPH[1,2,3,*], **Jessica I. Lundin, MPH**[3], **Karen M. Powers, BS**[3], **Terri Smith-Weller, MN, COHN**[3], **Gary M. Franklin, MD, MPH**[1,3], **Phillip D. Swanson, MD, PhD**[1], **W.T. Longstreth Jr., MD, MPH**[1,4], and **Harvey Checkoway, PhD**[3,4]

[1]Department of Neurology, University of Washington, Seattle, Washington

[2]Department of Medicine, University of Washington, Seattle, Washington

[3]Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, Washington

[4]Department of Epidemiology, University of Washington, Seattle, Washington

Abstract

Background—Parkinson's disease (PD) has been associated with various workplace factors, but the evidence is inconsistent.

Objective—To estimate the risk of PD associated with various jobs and workplace exposures.

Methods—We conducted a population-based, case–control study of 404 incident PD cases and 526 age and sex-matched controls, collecting self-reported work histories including job titles and exposures to various industrial toxicants. Relative risks of PD from these exposures were estimated with odds ratios (OR) and 95% confidence intervals (CI) using logistic regression.

Results—Risk was not significantly affected by farming work, by metal work, or by exposure to pesticides, metals, or solvents.

Conclusions—These findings do not provide support for the hypothesis that workplace factors affect the risk of PD.

Keywords

risk factors in epidemiology; Parkinson's disease/parkinsonism; toxicology; occupational; human

INTRODUCTION

Parkinson's disease (PD) has been associated with workplace factors classified by occupational titles or by exposures to various industrial toxicants. Previous studies have reported elevated risks associated with agricultural work [Hertzman et al., 1990; Tuchsen and Jensen, 2000; Park et al., 2005] and with teaching and health care [Tsui et al., 1999; Park et al., 2005], and reduced risk with service occupations [Kirkey et al., 2001]. Elevated risks have also been reported for exposures to heavy metals [Gorell et al., 1999], solvents [McDonnell et al., 2003], and pesticides [Seidler et al., 1996; Gorell et al., 1999; Ascherio et al., 2006; Frigerio et al., 2006; Kamel et al., 2007]. Despite experimental evidence

© 2009 Wiley-Liss, Inc.

*Correspondence to: J.A. Firestone, University of Washington, Box 359739, Seattle, WA 98104-2499. jfire@u.washington.edu.

The authors report no conflicts of interest.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

supporting the biological plausibility of various associations, the epidemiological evidence linking PD with occupational exposures has been inconsistent.

In a population-based, case–control study of incident PD in western Washington State, we reviewed subjects' work histories and estimated the risks for PD related to various workplace factors. We previously reported associations with pesticide exposures based on an interim analysis [Firestone et al., 2005]. Here, based on a larger study sample, we update those results and report associations with other occupational titles and exposures to various industrial toxicants.

METHODS

Subjects

Our methods have been previously described [Firestone et al., 2005]. Newly diagnosed, idiopathic PD cases were identified between 1992 and 2006 at the Group Health Cooperative (GHC) and the University of Washington. To ensure complete ascertainment, subjects were identified using provider referrals or computerized databases containing diagnostic coding and pharmacy information. A panel of neurologists (W.T.L., P.D.S., and G.M.F.) confirmed PD diagnoses by medical chart review, requiring at least two of the four cardinal signs of PD (bradykinesia, resting tremor, cogwheel rigidity, and postural reflex impairment), one of which had to be bradykinesia or resting tremor. Exclusion criteria were use of certain medications whose adverse effects may include parkinsonism (e.g., haloperidol or metoclopramide) during the 12 months preceding symptom onset; prior history of multiple cerebrovascular events; or another explanation for parkinsonism (e.g., brain injury, brain tumor, or encephalitis). Control subjects were randomly selected GHC enrollees with no history of PD or other progressive neurological disorders. Controls were frequency-matched to cases by sex and age. The participation rate for eligible cases was 70% (provider-referred 80%; computer-identified 66%) and for eligible controls was 60%. Human Subjects committees at the University of Washington and the GHC Center for Health Studies reviewed and approved the study, and all participants provided written, informed consent.

Exposures

Exposure information was gathered during structured interviews, all conducted face-to-face by the same nurse practitioner. Though perfect blinding was impossible, given the often apparent manifestations of PD, the interviewer was not informed of the subjects' case–control status in order to minimize potential interviewer bias. To minimize recall bias, subjects were blinded to the study hypotheses. Subjects reported their demographic, medical, and smoking histories, as well as a description of each job held for more than 6 months and workplace exposures to various industrial toxicants identified from a checklist. Jobs were classified using codes from the Dictionary of Occupational Titles (DOT) [US Department of Labor, 1991], and toxicant exposures were grouped by chemical class (i.e., solvents, metals, pesticides). Durations were determined as the inclusive time span between first and last year of employment or exposure.

Analyses

We estimated risks associated with the major DOT divisions and with occupational titles and toxicant exposures of a priori interest. We assessed associations separately for men and women, as their work activities are often dissimilar. Relative risks were estimated by odds ratios (OR) and 95% confidence intervals (CI) from unconditional logistic regression models constructed using SPSS statistical software version 10.0.7 2000 (Chicago, IL). In this

ED_013302_00009367-00002

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

document, the terms "risk" or "risk estimate" are intended to be synonymous with "odds ratio." Statistical significance was set a priori at $\alpha = 0.05$ using two-tailed tests.

Our primary analyses modeled exposures as ever/never dichotomous variables. We also considered cumulative job durations as ordinal variables, choosing the 10-year mark (i.e., never, <10 and ≥10 years) as a convenient and somewhat intuitive boundary for short versus long-term employment. For exposures to metals, we also determined risk estimates using a 20-year cutoff (i.e., never, <20 and ≥20 years) in order to facilitate comparison with a previous report [Gorell et al., 1999]. These analyses did not reveal meaningful trends; we report herein the results from dichotomous analyses.

In addition to estimating risks from self-reported toxicant exposures, we considered risks for probable exposure based on an industrial hygiene assessment of subjects' work histories. Although that assessment increased the number of subjects categorized as exposed, the results did not differ substantially from those based on self-reported exposures; we report herein the results from self-report.

Statistical models included the potential confounders of age, ethnicity (non-Hispanic Caucasian vs. other), and smoking status (cumulative pack-years). The crude and adjusted risk estimates were nearly identical; we reported only adjusted results. For many categories, the small number of subjects precluded meaningful risk estimates, so we only estimated risks for categories with at least four exposed subjects between comparison groups.

RESULTS

Study Population

The demographic characteristics of the study population are summarized in Table I. The study population included 404 PD cases and 526 unrelated controls. The possibility of selection bias could not be assessed, as privacy issues prevented access to detailed information about non-participants. However, participation rates were high (70% of eligible cases; 60% of eligible controls). The time spent on each interview (mean =60 min) did not differ significantly between groups, decreasing the likelihood of interview bias. The education and ethnicity of the study population are similar to those of the source population. The only significant group difference was a lower prevalence of cigarette smoking among cases, as previously reported [Checkoway et al., 2002].

Occupational Titles

For the major DOT divisions (Table II), no significant associations were seen.

For farming and related occupations (Table III), although none of the estimates was statistically significant, the trend paralleled the predicted intensity of pesticide exposures (i.e., pesticide worker >crop farmer >animal farmer), and risk estimates were generally higher for women than men. For metal working occupations (Table III), risk estimates for most categories were decreased, though not significantly. For health care occupations (Table III), non-significant, increased risk estimates were seen for men who worked as "physicians" (OR=6.1; CI=0.65, 56.26) and for women who worked as "medical or dental technicians" (OR=2.8; CI=0.82, 9.29). For teaching occupations (Table III), risk estimates for men did not differ substantially between primary, secondary, and college level, whereas for women, risk estimates for teaching at the secondary or college level were reduced, though not significantly. For other, miscellaneous occupations (Table III), risk estimates were not significant.

ED_013302_00009367-00003

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Industrial Toxicants

For workplace exposures to industrial toxicants of a priori interest (Table IV), none of the estimates was statistically significant. In women, the risk estimate was increased for "pesticides" (OR=3.9; CI=0.39, 39.4), though the number of exposed subjects was quite small. The risk estimate was also increased for women exposed to "solvents" (OR=1.7; CI=0.98, 3.04). In men, none of the risk estimates were increased. The risk estimate for exposure to both lead and copper for more than 20 years (OR=1.4; CI=0.45, 4.34) was only marginally higher than for exposure to either metal alone or in combination, though again the number exposed was quite small. Although, risk estimates were generally higher for women than for men, direct comparisons were not always possible as these exposures were largely segregated by gender. For occupational exposures to specific pesticides (Table V), the only increased risk estimate was for men exposed to parathion, the most potent of the organophosphates reported. None of the estimates was statistically significant.

DISCUSSION

In this relatively large, population-based, case–control study of incident PD from western Washington State, our findings do not provide strong support for the hypothesis that workplace factors affect the risk of PD. The fact that our results replicate the commonly observed inverse association between smoking and PD provides some reassurance of internal validity [Checkoway et al., 2002; Ritz et al., 2007]. Although some of our findings are consistent with previous reports, we found no statistically significant associations, so risk estimates must be interpreted cautiously.

Our results (Table II) are consistent with a previous report which examined major divisions of the DOT and identified decreased risk from "service" work [Kirkey et al., 2001]. The explanation for this observation remains uncertain, as classifications based on occupational titles capture a wide range of work activities and potential exposures and there is no apparent biological mechanism to link service work and PD.

Agricultural exposures are among the workplace factors most consistently reported to be associated with PD [Hertzman et al., 1990; Seidler et al., 1996; Gorell et al., 1999; Tuchsen and Jensen, 2000; Baldi et al., 2003; Park et al., 2005; Ascherio et al., 2006; Kamel et al., 2007]. Although several studies have indicated pesticides may be the causal factor, specific agents have not been consistently identified [Priyadarshi et al., 2000]. Specific pesticides of a priori interest, based on previously reported associations, include organophosphate insecticides such as parathion, malathion, and diazinon [Bhatt et al., 1999; Arima et al., 2003; Firestone et al., 2005], whose mechanism of action specifically targets the nervous system; organochlorines such as DDT [Seidler et al., 1996], whose lipid solubility and biopersistence intensify and extend exposures to the nervous system; herbicides such as paraquat [Hertzman et al., 1990], whose chemical structure mimics that of the parkinsonian neurotoxin MPTP; and 2,4-D [Kamel et al., 2007], whose reputation is notorious as one of the primary constituents in Agent Orange. Our results (Table V) do not provide strong support for the hypothesis that exposure to any of these pesticides affect the risk of PD. Although the data do suggest increased risk for parathion, a potent organophosphate that is no longer used in United States, that risk estimate is not significant.

There has been considerable interest in the possible association between PD and occupational exposures to neurotoxic metals in various metal working activities. In particular, exposure to manganese-oxide dust in mining and ore processing has been linked with parkinsonism [McMillan, 1999]. Although it is likely that manganese-related parkinsonism is clinically distinct from PD [Olanow, 2004; Jankovic, 2005; Perl and Olanow, 2007], it has been suggested that PD may be associated with exposure to

ED_013302_00009367-00004

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

manganese in welding fumes [Racette et al., 2005]. Our results (Table III) showing reduced risk in metal workers are consistent with several other studies showing no significant risk of PD from metalworking occupations including welding [Frigerio et al., 2005; Fryzek et al., 2005; Goldman et al., 2005; Park et al., 2005; Fored et al., 2006].

Some metal exposures have been previously associated with PD [Gorell et al., 1999; Coon et al., 2006]. However, our results (Table IV) did not corroborate these findings, as we found no increased risk for lead or manganese exposure or for combined lead and copper exposures, even after 20 years. Though these differences may relate to different study designs, it is likely that the small number of exposed subjects in each of these studies compromises the stability of statistical results.

Solvent exposures have also been associated with PD [Pezzoli et al., 2000; McDonnell et al., 2003]. Although our results (Table IV) suggest increased risk for solvent exposures in women, this was not statistically significant. The small number of subjects in these exposure categories and lack of statistical significance prevents firm conclusions.

Our results (Table III) suggest increased risk from work in teaching and health care, as previously reported from studies in Korea [Park et al., 2005] and Canada [Tsui et al., 1999]. Though these observations may reflect ascertainment bias resulting from better access to care, they would also be consistent with the neuroinflammatory hypothesis of PD pathogenesis [Barcia et al., 2003; McGeer and McGeer, 2004; Whitton, 2007], as workers in these occupations have relatively frequent exposures to infectious agents. However, the lack of association with work as a nurse argues against this hypothesis.

The difference in observed risk between some occupational titles fits with the idea of career self-selection based on early determinants of susceptibility with deterioration of dopaminergic pathways producing subtle, sub-clinical effects long before diagnosis. Thus, for example, we identified decreased risk of PD from jobs with high physical demands, such as "construction and electrical work," and increased risk from jobs with low physical demands, such as "administration, computing, and sales" (Table III).

That the risk estimates for men and women were different for several workplace factors fits with the idea that patterns of work exposures differ between men and women. Thus, for example, risk estimates for men tended to be lower than for women working in farming and related occupations (Table III).

The case–control study design is efficient for studying relatively uncommon diseases such as PD. However, when exposure strata contain few subjects even slight misclassification of diagnosis or exposure may substantially alter observed associations. Misclassification of diagnosis may occur since PD diagnosis relies on clinical criteria whose specificity only approaches 80% [Hughes et al., 1992; Tolosa et al., 2006]. Misclassification of exposure may occur since retrospective exposure assessment does not allow confirmatory environmental monitoring, even though recall bias was limited by blinding the subjects to specific hypotheses. Because diagnostic uncertainty and inaccurate recall are likely to similarly affect cases and controls, the resulting non-differential misclassification may mask true associations. This study's power to detect significant differences is further limited by the small number of subjects in some exposure groups, as this was not an industry-based but rather a population-based study. Fortunately this means that the findings can be reasonably generalized, as the demographic characteristics of our study population are representative of GHC enrollees, who in turn reflect the demographics of western Washington State (Table I).

The growing scientific consensus is that PD is not a single disorder, but instead reflects a common pathological endpoint resulting from the interaction of various environmental and

ED_013302_00009367-00005

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

genetic risk factors. Thus, the risk attributable to any single factor is likely to be small and to differ depending on specific population characteristics. An epidemiological approach to identify etiologic factors with small attributable risk for PD, such as from workplace factors, will require very large study populations. Conclusive studies will likely require inter-disciplinary, multi-center collaborations.

Acknowledgments

This study was funded by the United States National Institute of Environmental Health Sciences, National Institutes of Health, Grants K23ES10756, P42ES04696, R01ES10750, and P30ES07033.

References

Arima H, Sobue K, So M, Morishima T, Ando H, Katsuya H. Transient and reversible parkinsonism after acute organophosphate poisoning. J Toxicol Clin Toxicol. 2003; 41:67–70. [PubMed: 12645970]

Ascherio A, Chen H, Weisskopf MG, O'Reilly E, McCullough ML, Calle EE, Schwarzschild MA, Thun MJ. Pesticide exposure and risk for Parkinson's disease. Ann Neurol. 2006; 60:197–203. [PubMed: 16802290]

Baldi I, Lebailly P, Mohammed-Brahim B, Letenneur L, Dartigues JF, Brochard P. Neurodegenerative diseases and exposure to pesticides in the elderly. Am J Epidemiol. 2003; 157:409–414. [PubMed: 12615605]

Barcia C, Fernandez Barreiro A, Poza M, Herrero MT. Parkinson's disease and inflammatory changes. Neurotox Res. 2003; 5:411–418. [PubMed: 14715444]

Bhatt MH, Elias MA, Mankodi AK. Acute and reversible parkinsonism due to organophosphate pesticide intoxication: Five cases. Neurology. 1999; 52:1467–1471. [PubMed: 10227636]

Checkoway H, Powers K, Smith-Weller T, Franklin GM, Longstreth WT Jr, Swanson PD. Parkinson's disease risks associated with cigarette smoking, alcohol consumption, and caffeine intake. Am J Epidemiol. 2002; 155:732–738. [PubMed: 11943691]

Coon S, Stark A, Peterson E, Gloi A, Kortsha G, Pounds J, Chettle D, Gorell J. Whole-body lifetime occupational lead exposure and risk of Parkinson's disease. Environ Health Perspect. 2006; 114:1872–1876. [PubMed: 17185278]

Firestone JA, Smith-Weller T, Franklin G, Swanson P, Longstreth WT Jr, Checkoway H. Pesticides and risk of Parkinson disease: A population-based case-control study. Arch Neurol. 2005; 62:91–95. [PubMed: 15642854]

Fored CM, Fryzek JP, Brandt L, Nise G, Sjogren B, McLaughlin JK, Blot WJ, Ekbom A. Parkinson's disease and other basal ganglia or movement disorders in a large nationwide cohort of Swedish welders. Occup Environ Med. 2006; 63:135–140. [PubMed: 16421393]

Frigerio R, Elbaz A, Sanft KR, Peterson BJ, Bower JH, Ahlskog JE, Grossardt BR, de Andrade M, Maraganore DM, Rocca WA. Education and occupations preceding Parkinson disease: A population-based case-control study. Neurology. 2005; 65:1575–1583. [PubMed: 16301484]

Frigerio R, Sanft KR, Grossardt BR, Peterson BJ, Elbaz A, Bower JH, Ahlskog JE, de Andrade M, Maraganore DM, Rocca WA. Chemical exposures and Parkinson's disease: A population-based case-control study. Mov Disord. 2006; 21:1688–1692. [PubMed: 16773614]

Fryzek JP, Hansen J, Cohen S, Bonde JP, Llambias MT, Kolstad HA, Skytthe A, Lipworth L, Blot WJ, Olsen JH. A cohort study of Parkinson's disease and other neurodegenerative disorders in Danish welders. J Occup Environ Med. 2005; 47:466–472. [PubMed: 15891525]

Goldman SM, Tanner CM, Olanow CW, Watts RL, Field RD, Langston JW. Occupation and parkinsonism in three movement disorders clinics. Neurology. 2005; 65:1430–1435. [PubMed: 16162857]

Gorell JM, Johnson CC, Rybicki BA, Peterson EL, Kortsha GX, Brown GG, Richardson RJ. Occupational exposure to manganese, copper, lead, iron, mercury and zinc and the risk of Parkinson's disease. Neurotoxicology. 1999; 20:239–247. [PubMed: 10385887]

ED_013302_00009367-00006

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Hertzman C, Wiens M, Bowering D, Snow B, Calne D. Parkinson's disease: A case-control study of occupational and environmental risk factors. Am J Ind Med. 1990; 17:349–355. [PubMed: 2305814]

Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: A clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry. 1992; 55:181–184. [PubMed: 1564476]

Jankovic J. Searching for a relationship between manganese and welding and Parkinson's disease. Neurology. 2005; 64:2021–2028. [PubMed: 15985567]

Kamel F, Tanner C, Umbach D, Hoppin J, Alavanja M, Blair A, Comyns K, Goldman S, Korell M, Langston J, Ross G, Sandler D. Pesticide exposure and self-reported Parkinson's disease in the agricultural health study. Am J Epidemiol. 2007; 165:364–374. [PubMed: 17116648]

Kirkey KL, Johnson CC, Rybicki BA, Peterson EL, Kortsha GX, Gorell JM. Occupational categories at risk for Parkinson's disease. Am J Ind Med. 2001; 39:564–571. [PubMed: 11385640]

McDonnell L, Maginnis C, Lewis S, Pickering N, Antoniak M, Hubbard R, Lawson I, Britton J. Occupational exposure to solvents and metals and Parkinson's disease. Neurology. 2003; 61:716–717. [PubMed: 12963777]

McGeer PL, McGeer EG. Inflammation and neurodegeneration in Parkinson's disease. Parkinsonism Relat Disord. 2004; 10(suppl 1):S3–S7. [PubMed: 15109580]

McMillan DE. A brief history of the neurobehavioral toxicity of manganese: Some unanswered questions. Neurotoxicology. 1999; 20:499–507. [PubMed: 10385908]

Olanow CW. Manganese-induced parkinsonism and Parkinson's disease. Ann NY Acad Sci. 2004; 1012:209–223. [PubMed: 15105268]

Park J, Yoo CI, Sim CS, Kim HK, Kim JW, Jeon BS, Kim KR, Bang OY, Lee WY, Yi Y, Jung KY, Chung SE, Kim Y. Occupations and Parkinson's disease: A multi-center case-control study in South Korea. Neurotoxicology. 2005; 26:99–105. [PubMed: 15527877]

Perl DP, Olanow CW. The neuropathology of manganese-induced Parkinsonism. J Neuropathol Exp Neurol. 2007; 66:675–682. [PubMed: 17882011]

Pezzoli G, Canesi M, Antonini A, Righini A, Perbellini L, Barichella M, Mariani CB, Tenconi F, Tesei S, Zecchinelli A, Leenders KL. Hydrocarbon exposure and Parkinson's disease. Neurology. 2000; 55:667–673. [PubMed: 10980731]

Priyadarshi A, Khuder SA, Schaub EA, Shrivastava S. A meta-analysis of Parkinson's disease and exposure to pesticides. Neurotoxicology. 2000; 21:435–440. [PubMed: 11022853]

Racette BA, Tabbal SD, Jennings D, Good L, Perlmutter JS, Evanoff B. Prevalence of parkinsonism and relationship to exposure in a large sample of Alabama welders. Neurology. 2005; 64:230–235. [PubMed: 15668418]

Ritz B, Ascherio A, Checkoway H, Marder KS, Nelson LM, Rocca WA, Ross GW, Strickland D, Van Den Eeden SK, Gorell J. Pooled analysis of tobacco use and risk of Parkinson disease. Arch Neurol. 2007; 64:990–997. [PubMed: 17620489]

Seidler A, Hellenbrand W, Robra BP, Vieregge P, Nischan P, Joerg J, Oertel WH, Ulm G, Schneider E. Possible environmental, occupational, and other etiologic factors for Parkinson's disease: A case-control study in Germany. Neurology. 1996; 46:1275–1284. [PubMed: 8628466]

Tolosa E, Wenning G, Poewe W. The diagnosis of Parkinson's disease. Lancet Neurol. 2006; 5:75–86. [PubMed: 16361025]

Tsui JK, Calne DB, Wang Y, Schulzer M, Marion SA. Occupational risk factors in Parkinson's disease. Can J Public Health. 1999; 90:334–337. [PubMed: 10570579]

Tuchsen F, Jensen AA. Agricultural work and the risk of Parkinson's disease in Denmark, 1981–1993. Scand J Work Environ Health. 2000; 26:359–362. [PubMed: 10994803]

US Department of Labor. Dictionary of Occupational Titles. 4. Washington, DC: US Department of Labor, Employment and Training Administration, US Employment Service; 1991. Revised

Whitton PS. Inflammation as a causative factor in the aetiology of Parkinson's disease. Br J Pharmacol. 2007; 150:963–976. [PubMed: 17339843]

ED_013302_00009367-00007

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

TABLE I

Demographic Characteristics of Study Population

	Cases (n = 404)	Controls (n = 526)
Sex (%)		
Male	62.4	62.0
Female	37.6	38.0
Age, median (range)	69 (29–88)	71 (38–85)
Ethnicity (%)		
Non-Hispanic white	93.6	92.0
Other	6.4	8.0
Education (%)		
≤12 years	14.1	19.0
>12 years	85.1	80.2
Smoking		
Pack-years, median (range)	0 (0–143)[a]	5.0 (0–128)
Ever smoker (>100 cigs) (%)	43.1[a]	58.2

[a]The difference in smoking between cases and controls is statistically significant ($P<0.05$).

ED_013302_00009367-00008

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

TABLE II

Risk of PD by Major Divisions of Dictionary of Occupational Titles

	Men				Women			
DOTmajor division	**Cases (252)**	**Controls (326)**	**OR[a]**	**95% CI[a]**	**Cases (152)**	**Controls (200)**	**OR[a]**	**95% CI[a]**
0/1. Professional, Technical, and Managerial	142	162	1.2	0.82,1.62	90	95	1.4	0.90, 2.17
2. Clerical and Sales	84	92	1.2	0.86,1.78	76	110	0.8	0.54,1.28
3. Service	50	76	0.7	0.49,1.13	49	62	1.1	0.67,169
4. Agricultural, Fishery, Forestry, and Related	98	127	1.0	0.70,1.39	27	21	1.7	0.93, 3.26
5. Processing	62	104	0.7	0.49,1.03	14	10	2.1	0.88, 4.96
6. MachineTrades	42	74	0.7	0.44,1.05	4	4	1.6	0.33, 7.40
7. Benchwork	64	78	1.1	0.75,1.63	6	20	0.4	0.16,1.08
8. Structural Work	67	110	0.8	0.52,1.09	8	16	0.8	0.34, 2.06
9. Miscellaneous	32	52	0.8	0.51,1.34	5	6	1.1	0.31, 3.67

[a] Adjusted for age, ethnicity, and smoking; compared to subjects never exposed.

ED_013302_00009367-00009

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

TABLE III

Risk of Parkinson's Disease by EverWorking in a Given Occupation

	Men				Women			
Occupation	**Cases (n =252)**	**Controls (n =326)**	**OR[a]**	**95% CI[a]**	**Cases (n =152)**	**Controls (n =200)**	**OR[a]**	**95% CI[a]**
Farming and related	81	102	1.0	0.72, 1.49	25	21	1.6	0.85, 3.01
Pesticide worker	8	7	1.53	0.54, 4.35	—	—	—	—
Crop farmer	27	28	1.4	0.76, 2.40	10	6	2.3	0.82, 6.73
Animal farmer	6	15	0.5	0.19, 1.35	2	2	1.4	0.19,10.64
Crop and animal farmer	31	38	1.2	0.71, 2.00	6	2	3.4	0.66,16.98
Dairy farmer	20	34	0.8	0.42, 1.38	1	2	0.6	0.05, 6.75
Orchard grower	7	11	0.9	0.32, 2.27	3	3	1.2	0.24, 6.22
Metal working	81	141	0.7	0.46, 1.94	12	18	1.1	0.50, 2.45
Welding and cutting	25	48	0.6	0.37, 1.07	3	4	1.6	0.34, 7.92
Mining and refining	21	29	1.0	0.55, 1.81	3	2	2.0	0.33,12.40
Fabrication and machining	53	97	0.7	0.47, 1.03	7	14	0.8	0.31, 2.09
Health care	10	5	2.2	0.71, 6.56	23	21	1.3	0.69, 2.51
Physician	4	1	6.1	0.65, 56.26	1	0	—	—
Nurse	2	2	0.9	0.12, 6.83	14	17	1.0	0.45, 2.03
Medical or dental technician	4	2	1.7	0.31, 9.71	9	4	2.8	0.82, 9.29
Teaching	39	39	1.3	0.77, 2.05	23	32	0.8	0.44,1.46
Primary	6	5	1.5	0.44, 5.06	17	17	1.2	0.60, 2.52
Secondary	16	18	1.2	0.57, 2.36	2	8	0.3	0.05,1.27
College	17	16	1.2	0.60, 2.52	4	8	0.5	0.15,1.81
Construction	58	89	0.8	0.53, 1.16	5	9	0.7	0.22, 2.15
Administration, computing, and sales	137	147	1.4	0.98, 1.93	110	136	1.2	0.77,1.99
Military and protective services	46	60	0.9	0.61, 1.45	4	1	8.3	0.80, 85.96

[a] Adjusted for age, ethnicity, and smoking; compared to subjects never exposed.

ED_013302_00009367-00010

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

TABLE IV

Risk of Parkinson's Disease by Exposure to Industrial Toxicants

	Men				Women			
Industrial toxicant	**Cases (n =252)**	**Controls (n =326)**	**OR[a]**	**95% CI[a]**	**Cases (n =152)**	**Controls (n =200)**	**OR[a]**	**95% CI[a]**
Pesticides	12	24	0.6	0.30, 1.29	3	1	3.9	0.39, 39.4
Solvents	122	163	1.0	0.68, 1.34	34	30	1.7	0.98, 3.04
Metals	79	120	0.9	0.60, 1.24	18	20	1.3	0.67, 2.66
Manganese	2	3	1.2	0.19, 7.90	1	2	0.6	0.06, 7.07
Lead	35	64	0.7	0.46, 1.15	4	4	1.5	0.37, 6.44
Copper	23	29	1.1	0.59, 1.95	4	0	—	—
Lead+copper	15	18	1.2	0.59, 2.50	1	0	—	—
Lead+copper (≥20 years)	6	7	1.4	0.45, 4.34	1	0	—	—

[a] Adjusted for age, ethnicity, and smoking; compared to subjects never exposed.

ED_013302_00009367-00011

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

TABLE V

Risk of Parkinson's Disease by Exposures to Specific Pesticides

	Men				Women			
	Cases (n =252)	Controls (n =326)	OR[a]	95% CI[a]	Cases (n =152)	Controls (n =200)	OR[a]	95% CI[a]
Parathion	5	1	5.8	0.66, 50.79	1	0	—	—
Malathion	10	12	1.0	0.39, 2.30	1	0	—	—
Diazinon	7	11	0.8	0.30, 2.15	1	0	—	—
DDT	14	22	0.8	0.40,1.64	1	0	—	—
2,4-D	8	12	0.8	0.30, 2.00	1	0	—	—
Paraquat	2	3	0.9	0.14, 5.43	0	0	—	—

[a] Adjusted for age, ethnicity, and smoking; compared to subjects never exposed.

ED_013302_00009367-00012