Occupational Solvent Exposure and Parkinson's Disease: Evidence from PPMI Online

Hector R. Martinez
Instituto de Neurologia y Neurocirugia, Centro Medico Zambrano Hellion, TecSalud
Francisco A. Luna-Rangel
Tecnologico de Monterrey, Escuela de Medicina y Ciencias de la Salud Monterrey
O. Gutierrez-Treviño
Instituto de Neurologia y Neurocirugia, Centro Medico Zambrano Hellion, TecSalud
Cecilio Armengol-Garcia
Instituto de Neurologia y Neurocirugia, Centro Medico Zambrano Hellion, TecSalud
Juan Manuel Luna
Instituto de Neurologia y Neurocirugia, Centro Medico Zambrano Hellion, TecSalud
Beatriz Perez Martinez
Instituto de Neurologia y Neurocirugia, Centro Medico Zambrano Hellion, TecSalud
Jose A. Figueroa-Sanchez
dr.figueroa@tec.mx

Tecnologico de Monterrey, Escuela de Medicina y Ciencias de la Salud Monterrey

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Abstract

Environmental exposures have been increasingly implicated in PD, yet the role of occupational solvents remains incompletely defined. We examined the association between lifetime occupational solvent exposure and PD in 31,656 participants from the PPMI Online cohort. Exposure to 13 organic solvents was assessed by self-report of ≥ 100 days of occupational use. Multivariable logistic regression models adjusted for demographic and clinical factors, with false discovery rate correction, were used to estimate associations. Several solvents were associated with higher odds of PD, including TCE (aOR 1.73, 95% CI 1.48–2.02), kerosene (1.72, 1.51–1.96), and carbon tetrachloride (1.67, 1.40–1.98), with additional associations for mineral spirits, methyl ethyl ketone, toluene, and jet fuel. Increasing exposure burden was associated with progressively higher odds of PD, with a 17% increase per additional solvent. These findings support an association between occupational solvent exposure and PD and highlight the need for longitudinal studies to clarify underlying mechanisms.

Introduction

In recent decades, Parkinson's disease (PD) has emerged as the fastest-growing neurological disorder worldwide. According to the Global Burden of Disease estimates, the global prevalence of PD increased by 274% between 1990 and 2021, underscoring a striking and sustained upward trajectory(1,2). Although this rise has been observed across multiple regions, East Asia particularly China reported the highest age-standardized prevalence in 2021, with projections suggesting a continued increase through at least 2046.

Several factors have been proposed to account for this trend. Population aging remains a central driver, as longer life expectancy increases the number of individuals living to ages at which neurodegenerative diseases become more prevalent. Improved diagnostic recognition and heightened clinical awareness have also contributed to case ascertainment.Additionally, Declining tobacco use may also have contributed to the rising burden of PD(3,4).

Environmental exposures have long been implicated in the pathogenesis of Parkinson's disease, with pesticide use and ambient air pollution consistently associated with increased risk in epidemiologic studies(5). Beyond these well-characterized factors, additional environmental toxicants including heavy metals and industrial chemicals may also contribute to disease susceptibility(6,7). Trichloroethylene (TCE), in particular, has been consistently implicated as a risk factor for Parkinson's disease(8,9).However, other industrial solvents remain understudied, highlighting the need for systematic evaluation of multiple occupational exposures in large cohorts(10).

The objective of this study was therefore to evaluate occupational solvent exposures among individuals with PD, with particular emphasis on chemicals commonly encountered in industrial settings. We aimed to examine whether occupational exposure to a broader range of industrial solvents is associated with Parkinson's disease.

Methods

Study population

Data for the present analysis were retrieved on February 14, 2026 from the Parkinson's Progression Markers Initiative (PPMI) Online study, a longitudinal, multicenter observational cohort that collects participant-reported health data from individuals with and without Parkinson's disease. The PPMI dataset is publicly available through the official study portal (RRID: SCR_006431)(11).

PPMI Online includes participants from two main sources: individuals previously enrolled in the original PPMI study who were invited to continue follow-up through the online platform, and additional participants recruited directly into the online cohort, including persons with PD and family members of existing participants. According to the study protocol, recruitment is currently limited to the United States and has been conducted primarily through outreach initiatives supported by the Michael J. Fox Foundation. Eligibility for participants with PD includes age ≥ 18 years, residence in the United States, and a prior diagnosis of PD. The platform captures a broad range of self-reported information, including demographic characteristics, medical history, family history, activities of daily living, and PD-related symptoms.

We conducted an analytical cross-sectional study using de-identified, publicly available PPMI Online data. The study was designed and reported in accordance with the STROBE Statement for observational studies. Data were integrated from several PPMI modules, including Participant Enrollment Status, Chemical Exposure, Socioeconomic Status, Age at Parkinson's Disease Diagnosis, the Geriatric Depression Scale (GDS), and MDS-UPDRS Parts I and II.

Outcome definition

Outcome definition
PD status was determined from the cohort classification recorded in the Participant Enrollment Status module. Participants classified within PD cohorts were considered cases, whereas those classified as healthy controls were considered non-PD participants. In the online PPMI dataset, PD status is based on participant-reported diagnosis and cohort assignment rather than clinician-confirmed diagnostic evaluation. Individuals assigned to other cohort categories were excluded. Participants from the SWEDD and Prodromal PD cohorts were excluded due to incomplete chemical exposure data and because these groups do not represent confirmed Parkinson's disease, thereby minimizing potential misclassification bias. A binary outcome variable representing PD status was subsequently created for regression modeling.

The PPMI Online cohort includes both participants from the original clinically characterized PPMI study and individuals enrolled directly through the online platform. In the latter group, PD diagnosis is self-reported, although the platform records whether the diagnosis was made by a non-neurologist physician,

a general neurologist, or a movement disorders specialist. To preserve statistical power and maximize sample size, all participants reporting PD were retained regardless of the reporting clinician. This approach allowed inclusion of both clinically characterized cases and a broader community-based population, thereby increasing statistical power while improving generalizability.

Exposure assessment

Occupational solvent exposure was derived from the Chemical Exposure module. Thirteen individual organic solvents reflecting lifetime occupational exposure were evaluated, including trichloroethylene, carbon tetrachloride, kerosene, jet fuel, methyl ethyl ketone, mineral spirits, toluene, xylene, perchloroethylene, Stoddard solvent, n-hexane, methylene chloride, and other solvents. In addition, three response categories (none, unsure, and no response) were included, yielding a total of 16 exposure variables analyzed. For each solvent, participants were asked the following standardized question: “In lifetime, used [solvent] 100 days or more at work.” Variable definitions and coding were verified using the PPMI Codebook and the online PPMI Data Dictionary available through the study repository. This threshold was used to capture sustained occupational contact rather than incidental exposure and to reduce the likelihood of exposure misclassification. Participants indicating “none” were considered unexposed, whereas “unsure” and “no answer” responses were retained as separate information-related categories rather than treated as confirmed exposures.

Covariates

Baseline clinical characteristics were defined using the earliest available survey record for each participant. Depressive symptoms were assessed with the 15-item Geriatric Depression Scale (GDS-15), with total scores calculated according to standard scoring procedures. Non-motor and motor experiences of daily living were evaluated using MDS-UPDRS Part I and Part II, respectively, with total scores computed as the sum of individual item responses. Age at PD diagnosis was obtained from the corresponding module and was available only for participants with PD. As age data were not collected for control participants in the relevant questionnaire module, age could not be included as a covariate in the regression models.

Demographic covariates extracted from the Socioeconomic Status module included biological sex, years of education, and household income, the latter treated as an ordinal proxy for socioeconomic status. Household income was categorized into six ordered brackets: <$20,000; $20,000–34,999; $35,000–49,999; $50,000–74,999; $75,000–99,999; and >$100,000. Responses of “prefer not to answer” were retained as a separate category to preserve information and avoid misclassification.

Statistical analysis

Descriptive statistics were summarized according to PD status. Continuous variables are presented as mean ± standard deviation, and categorical variables as counts and percentages. Between-group differences were assessed using Student's t-test for continuous variables and the chi-square test for categorical variables. Associations between occupational solvent exposure and PD were first examined using unadjusted binary logistic regression to estimate crude odds ratios (ORs) and 95% confidence intervals (CIs).

Multivariable binary logistic regression models were then used to estimate adjusted odds ratios (aORs) and 95% CIs while controlling for sex, years of education, household income, caffeine intake, smoking status, and history of traumatic brain injury (TBI) with loss of consciousness. Because occupational solvents may co-occur and display correlated exposure patterns, each solvent was analyzed in a separate regression model to estimate its association with PD while minimizing multicollinearity across exposure variables.

To account for multiple comparisons, p-values from the adjusted models were corrected using the Benjamini–Hochberg false discovery rate (FDR) procedure. To examine cumulative exposure effects, a solvent exposure burden variable was created by summing the number of individual solvent exposures reported by each participant and categorizing the result as 0, 1, 2, or ≥ 3 solvents. Its association with PD was evaluated using multivariable logistic regression, and a trend analysis was performed by modeling exposure burden as an ordinal variable. Additional analyses incorporated residential setting to assess whether associations were consistent across rural and non-rural environments. E-values were also calculated to assess the robustness of the observed associations to potential unmeasured confounding, representing the minimum strength of association that an unmeasured confounder would need to have with both the exposure and the outcome to fully account for the observed effect estimates.

Model assumptions were assessed using variance inflation factors and Hosmer–Lemeshow goodness-of-fit testing. All statistical tests were two-sided, with $p < 0.05$ considered statistically significant. Analyses were conducted in R version 4.4.2.

Results

Participant characteristics

A total of 31,656 participants were included in the analysis, comprising 8,795 individuals with PD and 22,861 healthy controls. Among participants with PD, 56.6% were male, compared with 33.3% among controls. Educational attainment was similar between groups, with both cohorts reporting a mean of approximately 16 years of education. Household income, analyzed as an ordinal variable, was slightly lower among participants with PD compared with controls.

The cohort was predominantly White, accounting for approximately 90% of both PD cases and controls. The mean age at PD diagnosis was 63.28 ± 10.21 years; age was not available for control participants in

the relevant questionnaire module. Smoking status, history of traumatic brain injury with loss of consciousness, and caffeine intake were also evaluated as covariates (Table 1).

Association between solvent exposure and Parkinson disease

After adjustment for sex, education, household income, caffeine intake, smoking status, and history of traumatic brain injury with loss of consciousness, several occupational solvent exposures were associated with increased odds of PD. The strongest associations were observed for trichloroethylene (aOR 1.73, 95% CI 1.48–2.02), kerosene (aOR 1.72, 95% CI 1.51–1.96), and carbon tetrachloride (aOR 1.67, 95% CI 1.40–1.98).

Additional associations were identified for mineral spirits (aOR 1.42, 95% CI 1.30–1.56), methyl ethyl ketone (aOR 1.35, 95% CI 1.17–1.56), jet fuel (aOR 1.28, 95% CI 1.07–1.52), and toluene (aOR 1.27, 95% CI 1.11–1.46). In contrast, xylene, perchloroethylene, methylene chloride, Stoddard solvent, and n-hexane were not significantly associated with PD. Participants reporting no occupational solvent exposure had lower odds of PD (aOR 0.65, 95% CI 0.61–0.70) (aOR 0.65, 95% CI 0.61–0.70) (Table 2). The main results are visually summarized in Fig. 1.[Insert Table 2 here] [Insert Fig. 1 here]

Multiple comparison adjustment and sensitivity to residential context

After applying false discovery rate correction using the Benjamini–Hochberg method, the associations for kerosene, trichloroethylene, carbon tetrachloride, mineral spirits, methyl ethyl ketone, toluene, and jet fuel remained statistically significant ($q < 0.05$). Models incorporating place of residence (rural vs. non-rural) yielded similar effect estimates, with no meaningful change in magnitude or statistical significance.

Dose-response relationship

To evaluate cumulative exposure, a solvent exposure burden variable was constructed based on the number of distinct solvents reported. A graded increase in the odds of PD was observed with increasing exposure burden. Compared with individuals reporting no solvent exposure, the odds of PD increased for exposure to one solvent (aOR 1.37, 95% CI 1.24–1.51), two solvents (aOR 1.33, 95% CI 1.16–1.54), and three or more solvents (aOR 1.55, 95% CI 1.39–1.73). When modeled as an ordinal variable, each additional exposure category was associated with a 17% increase in the odds of PD (OR 1.17, 95% CI 1.13–1.21; $p = 2.09 \times 10^{-21}$).

Sensitivity analyses

E-value analyses suggested that the main associations were moderately robust to potential unmeasured confounding. The largest E-values were observed for kerosene, trichloroethylene, and carbon

tetrachloride, indicating that an unmeasured confounder would require risk ratios of approximately 2.1–2.8 with both exposure and outcome to fully account for the observed associations.

Model diagnostics

Model diagnostics indicated low multicollinearity among covariates (variance inflation factors ≈ 1). Model discrimination was modest (AUC = 0.65), consistent with epidemiologic models designed to evaluate associations rather than predictive performance.

Discussion

Our analysis identified several occupational solvent exposures associated with increased odds of PD after adjustment for major known risk and protective factors, including nicotine use, sex, TBI, and caffeine consumption.The large sample size and inclusion of multiple covariates allowed more precise estimation of associations and a more comprehensive evaluation of occupational solvent exposures across multiple compounds.

Among the solvents examined, carbon tetrachloride showed one of the strongest associations with PD. This chlorinated solvent has long been recognized as a neurotoxic compound, and cases of parkinsonism associated with chronic inhalation of carbon tetrachloride have been reported since the late 1970s(12,13). Experimental studies have also demonstrated that carbon tetrachloride can increase markers of oxidative and nitrative stress, activate microglia, and disrupt mitochondrial function (14–16). Due to its lipophilic nature, the compound distributes readily across biological tissues, including the central nervous system. Historically, carbon tetrachloride has been widely used in the production of chlorofluorocarbons, as well as in industrial cleaning products, anesthetic agents, and household applications(17,18). Occupational exposure has been reported among electricians, mechanics including aviation mechanics painters, dry-cleaning workers, and other industrial workers. Notably, a discordant twin study conducted by Goldman et al. reported an odds ratio of approximately 2.3 for industrial solvent exposure and PD, which is comparable to the magnitude observed in our analysis. Although causal inference cannot be established from observational data alone, the concordance between our findings and previous epidemiological evidence supports the hypothesis that carbon tetrachloride exposure may contribute to PD risk(15,19).

Similarly, TCE was associated with increased odds of PD in our analysis. This finding is consistent with a growing body of literature implicating TCE as a potential environmental risk factor for Parkinson's disease(20). TCE is a highly lipophilic compound capable of crossing the blood–brain barrier and has been shown in experimental models to reduce mitochondrial complex I activity and induce selective loss of dopaminergic neurons in the substantia nigra pars compacta, a hallmark pathological feature of PD(21–27).

Kerosene exposure also showed a significant association with PD in our analysis. This finding is particularly relevant given the widespread use of kerosene worldwide, where it serves as a fuel source,

lubricant, heating agent, cooking fuel, and lighting source in many regions. Acute ingestion or inhalation of kerosene is well known to produce neurological symptoms, most commonly impaired consciousness (28–31). However, evidence regarding the potential long-term neurodegenerative effects of chronic exposure remains limited. The scarcity of longitudinal epidemiological studies and mechanistic in vitro investigations represents an important gap in the literature, highlighting the need for further research to clarify whether chronic exposure to kerosene may contribute to neurodegenerative processes.

Our analysis also identified an association between PD and mineral spirits, a petroleum-derived solvent mixture composed primarily of medium-chain aliphatic hydrocarbons such as heptane, octane, nonane, and decane, along with smaller amounts of aromatic hydrocarbons including toluene, xylene, and ethylbenzene. These solvents are widely used in industry as paint thinners, degreasing agents, and cleaning solvents. In contrast to our findings, a recent Finnish case–control study reported no significant association between mineral spirit exposure and PD(32). Differences in exposure assessment may partly explain these contrasting results. The Finnish study relied on the FINJEM job-exposure matrix, which assigns average exposure levels to occupational categories and may introduce aggregation bias and non-differential exposure misclassification. Such misclassification generally biases results toward the null. In contrast, our analysis used participant-reported occupational exposure histories with a ≥ 100-day exposure threshold, which may better capture individual variability in solvent exposure not reflected in job-exposure matrices.

Within this group of petroleum-derived solvents, toluene was analyzed separately and also demonstrated a positive association with PD. Due to their high lipophilicity, aromatic hydrocarbons such as toluene readily cross the blood brain barrier and can disrupt neuronal homeostasis through multiple mechanisms, including oxidative stress, mitochondrial dysfunction, lipid peroxidation, and neuroinflammatory activation. These processes may contribute to dopaminergic neuronal vulnerability through mechanisms involving protein misfolding, aggregation, and pro-inflammatory cytokine release(33,34).

Although jet fuel is composed predominantly of kerosene, it is chemically distinct due to the presence of additional additives such as corrosion inhibitors, anti-icing agents, biocides, and thermal stabilizers. These compounds may alter volatility, toxicity, and biological metabolism. Kerosene and jet fuel exposures frequently occur in different occupational contexts, potentially reflecting distinct exposure intensities and co-exposure profiles. In the present study, these exposures were analyzed separately because the chemical exposure questionnaire assessed them as distinct occupational exposures. While direct evidence linking jet fuel exposure to neurodegenerative disease remains limited, the neurotoxicity of hydrocarbon mixtures suggests plausible mechanisms that warrant further investigation. One potential pathway involves activation of the aryl hydrocarbon receptor (AHR). Experimental evidence suggests that AHR signaling may influence microglial activation and the ubiquitin proteasome system, including regulation of parkin and α-synuclein, two key proteins implicated in Parkinson’s disease pathogenesis.

To address the issue of multiple comparisons across the evaluated chemical exposures, we applied a false discovery rate correction using the Benjamini–Hochberg procedure. After adjustment, several solvent exposures remained significantly associated with PD, including carbon tetrachloride,TCE, kerosene, methyl ethyl ketone, mineral spirits, toluene, and jet fuel. This finding suggests that the observed associations are unlikely to be explained solely by multiple testing.

Furthermore, the exposure burden analysis revealed a pattern suggestive of a dose-response relationship. Participants exposed to a greater number of solvents exhibited progressively higher odds of PD compared with those reporting no exposure. The trend observed across exposure categories may reflect a cumulative effect of multiple solvent exposures, consistent with the potential additive or synergistic neurotoxic effects of hydrocarbon mixtures.

Additional models incorporating place of residence (rural versus urban) yielded nearly identical effect estimates, indicating that the associations were not materially influenced by residential context. This finding suggests that the observed relationships are unlikely to be explained by broader environmental differences between rural and urban populations.

Finally, we conducted an E-value sensitivity analysis to assess the potential impact of unmeasured confounding. The observed E-values indicate that relatively strong unmeasured confounding would be required to fully explain the main associations, representing the minimum strength of association that an unmeasured confounder would need to have with both the exposure and the outcome to account for the observed effects. Notably, E-values for key solvent exposures, such as kerosene and trichloroethylene, exceeded 2 at the lower bounds of the confidence intervals, suggesting that an unmeasured confounder would need to be associated with both exposure and outcome by a risk ratio of at least this magnitude to attenuate the observed findings. While these results support a degree of robustness, residual confounding cannot be excluded. Okubadejo et al. have proposed the development of a polyexposure score to capture the cumulative impact of multiple environmental risk factors associated with PD. This approach is conceptually aligned with the emerging exposome framework, which emphasizes the role of lifelong cumulative environmental exposures in disease susceptibility. Although these approaches represent an important step toward integrating complex exposure profiles, several environmental determinants of PD remain incompletely understood. Consequently, additional unidentified exposures may still be contributing to the global burden of PD. In this context, participants who responded “unsure” or “no answer” to solvent exposure questions exhibited (aOR 1.98 [95% CI 1.79–2.19] and 3.84 [1.89–8.42], respectively; both $q < 0.001$). While these categories were not interpreted as true exposures and were therefore analyzed separately, they highlight several important methodological considerations. First, they underscore the presence of recall bias inherent to the retrospective design of this study. The “unsure” category, in particular, likely reflects uncertainty or difficulty recalling past occupational exposures, which may be influenced by Parkinson’s disease itself. This supports the notion that self-reported exposure is not independent of disease status, as exposure assessment relies on memory, a domain that may be affected in PD. In this context, the observed associations may reflect differential misclassification of exposure, as individuals with PD may recall or report occupational exposures

differently compared with controls, rather than true exposure effects. Alternatively, these findings may also be consistent with the presence of unmeasured environmental or occupational exposures not captured by the predefined solvent variables, further supporting the need for more comprehensive exposomic approaches in PD research.

The present study has several strengths. The large sample size allowed precise estimation of associations, reflected in relatively narrow confidence intervals. In addition, multivariable adjustment for established PD risk factors, correction for multiple testing, and the evaluation of cumulative exposure burden strengthen the robustness of the findings. Each solvent was analyzed individually, allowing a more granular assessment of compound-specific associations.Several aspects of our findings are consistent with key considerations for causal inference, including biological plausibility, a pattern suggestive of dose–response, and consistency with prior evidence; however, causality cannot be inferred given the observational design.

These findings should be interpreted with caution in light of several limitations inherent to the study design. Because exposure information was self-reported, recall bias cannot be excluded. Participants may have misremembered or inaccurately reported past exposures, potentially leading to either overestimation or underestimation of associations. A notable proportion of participants selected non-response options when answering exposure-related questions, raising the possibility of differential reporting patterns between comparison groups. Such differences may introduce differential exposure misclassification, whereby measurement error is not evenly distributed across groups. In particular, individuals with PD were more likely to report uncertainty (“unsure”) or omit exposure information (“no answer”), which may reflect disease-related differences in recall or reporting rather than true differences in exposure history. These categories were not interpreted as true exposures but rather as indicators of potential information bias, including differential recall and non-random missingness.

At the same time, the questionnaire specifically assessed exposures lasting 100 days or more, which likely reduced the likelihood that incidental or brief exposures were misclassified as meaningful occupational exposure. This threshold may therefore mitigate, at least partially, the impact of recall bias by focusing on more sustained and salient exposure histories. Nevertheless, residual information bias remains an inherent limitation of retrospective exposure assessment.

An additional limitation concerns outcome ascertainment. Although a portion of the cohort originates from the original Parkinson’s Progression Markers Initiative study, another subset of participants was recruited through the Michael J. Fox Foundation online platform. As a result, it cannot be confirmed that standardized diagnostic criteria such as those proposed by the Movement Disorder Society were uniformly applied across all participants. Finally, age could not be included in the adjusted model because age data were not collected for healthy controls within the relevant questionnaire module. Given that age is one of the strongest determinants of Parkinson’s disease risk, its absence represents a key source of potential residual confounding. To partially address this limitation, we conducted an E-value

sensitivity analysis to estimate the strength of association that an unmeasured confounder, such as age, would need to have to fully explain the observed findings.

Taken together, these results indicate that occupational exposure to several organic solvents, including carbon tetrachloride, trichloroethylene, kerosene, and mineral spirits, is associated with higher odds of Parkinson's disease, although causality cannot be inferred. The consistency of these associations across multiple analytical approaches, including adjustment for key confounders, correction for multiple comparisons, dose-response analyses, and sensitivity analyses, supports the robustness of the findings.

Despite advances in the understanding of Parkinson's disease pathogenesis, a substantial proportion of its global burden remains unexplained. Environmental exposures, particularly those occurring in occupational settings, may represent underrecognized contributors. These findings underscore the need for further investigation of solvent exposure and other environmental toxicants as potentially modifiable factors. Longitudinal and mechanistic studies will be essential to better characterize these associations and clarify their role in neurodegenerative processes.

Declarations

CRediT authorship contribution statement

HRM: Conceptualization, Investigation, Writing – original draft, Writing – review & editing

FALR: Conceptualization Investigation, Data curation, Formal analysis, Methodology, Writing – original draft

CAG: Data curation, Formal analysis, Methodology, Software, Writing – original draft

OGT: Investigation, Writing – original draft

JML: Investigation, Writing – original draft

BPM: Investigation, Writing – original draft

JAFS: Conceptualization,Investigation, Methodology ,Project administration, Writing – original draft, Writing – review & editing

Declaration of competing interest

The authors declare that they have no competing financial interests or personal relationships that could have influenced the work reported in this paper.

Artificial Intelligence Assisted Language Editing

Grammarly and DeepL were used exclusively for language editing and grammatical refinement. No automated tools were involved in study design, data collection, analysis, or interpretation. All scientific

content and conclusions are the sole responsibility of the authors.

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Data availability

The data that support the findings of this study are available from the Parkinson's Progression Markers Initiative (PPMI) database (www.ppmi-info.org). Access to the dataset is subject to application and approval by the PPMI study administrators.

Code availability

The code used to perform the analyses in this study is available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate:

This study was conducted using de-identified data from the Parkinson's Progression Markers Initiative database. The PPMI study protocol was approved by the institutional review boards at all participating sites. All participants in PPMI provided written informed consent at the time of enrollment. The study was conducted in accordance with the Declaration of Helsinki. As this analysis used publicly available, de-identified data, additional institutional review board approval was not required.

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Tables

Table 1. Baseline characteristics of the analytic cohort.

Comparison of demographic, clinical, and lifestyle variables between participants with Parkinson's disease and healthy controls. Continuous variables are presented as mean ± standard deviation, and categorical variables as counts (%). *P-values* reflect group comparisons between PD and controls.

Table 1. Baseline characteristics of the analytic cohort

Characteristic	PD	Healthy controls	p-value
N	8795	22861	—
Sex, male, n (%)	4977 (56.6)	7618 (33.3)	<0.001
Education (years), mean ± SD	16.53 ± 4.08	16.91 ± 3.80	<0.001
Household income, mean ± SD	4.29 ± 1.48	4.36 ± 1.41	<0.001
Race/ethnicity, n (%)			0.040
White	8019 (91.2)	20716 (90.6)	
Latino	265 (3.0)	823 (3.6)	
Black/African American	47 (0.5)	160 (0.7)	
Asian	123 (1.4)	293 (1.3)	
Other / Unknown	341 (3.9)	869 (3.8)	
GDS-15 score, mean ± SD	4.06 ± 3.64	3.17 ± 3.35	<0.001
MDS-UPDRS Part I score, mean ± SD	9.05 ± 4.79	6.09 ± 4.33	<0.001
MDS-UPDRS Part II score, mean ± SD	10.56 ± 7.70	2.12 ± 3.93	<0.001
Age at PD diagnosis (years), mean ± SD	63.28 ± 10.21	—	—
Smoking status, n (%)			0.104
Never	886 (10.1)	2137 (9.3)	
Ever	1981 (22.5)	5095 (22.3)	
Unknown	5928 (67.4)	15629 (68.4)	
Severe head injury with LOC, n (%)	1518 (17.3)	3282 (14.4)	<0.001
Caffeine intake (drinks/week), mean ± SD	23.16 ± 18.80	24.45 ± 18.98	<0.001

Table 2:Association between occupational solvent exposure and Parkinson's disease.

Adjusted odds ratios (ORs) with 95% confidence intervals for the association between specific solvent exposures and Parkinson's disease. P-values and false discovery rate (FDR) q-values are reported to account for multiple comparisons.

Association between occupational solvent exposure and Parkinson's disease

Solvent	Adjusted OR (95% CI)	p-value	FDR q-value
Trichloroethylene	1.73 (1.48–2.02)	<0.001	<0.001
Kerosene	1.72 (1.51–1.96)	<0.001	<0.001
Carbon tetrachloride	1.67 (1.40–1.98)	<0.001	<0.001
Mineral spirits	1.42 (1.30–1.56)	<0.001	<0.001
Methyl ethyl ketone	1.35 (1.17–1.56)	<0.001	<0.001
Jet fuel	1.28 (1.07–1.52)	0.005	0.009
Toluene	1.27 (1.11–1.46)	<0.001	0.001
Perchloroethylene	1.12 (0.85–1.48)	0.419	0.558
Other solvents	1.11 (1.00–1.22)	0.043	0.063
Methylene chloride	1.05 (0.85–1.29)	0.644	0.793
Xylene	1.02 (0.87–1.20)	0.788	0.900
Stoddard solvent	0.99 (0.73–1.33)	0.940	0.940
n-Hexane	0.98 (0.74–1.28)	0.864	0.921

Figures

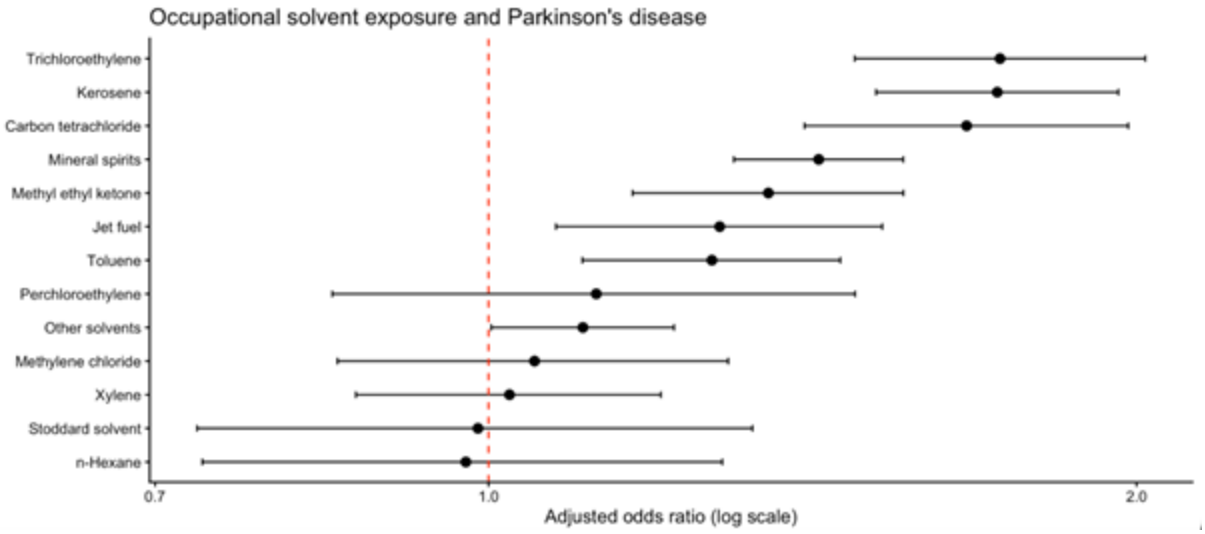

Figure 1

Association between occupational solvent exposure and Parkinson's disease.

Forest plot showing adjusted odds ratios (ORs) and 95% confidence intervals for the association between individual solvent exposures and Parkinson’s disease. The dashed vertical line represents the null value (OR = 1).