Predictors of Survival in Patients With Parkinson Disease

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**Objective:** To determine the life expectancy of patients with Parkinson disease (PD) in the United States and identify demographic, geographic, and clinical factors that influence survival.

**Design:** Retrospective cohort study of 138,000 Medicare beneficiaries with incident PD who were identified in 2002 and followed up through 2008.

**Main Outcome Measures:** Confounder-adjusted 6-year risk of death as influenced by 3 groups of factors: (1) race, sex, and age at diagnosis; (2) geography and environmental factors; and (3) clinical conditions. We examined hospitalization diagnoses in patients with terminal PD and compared PD mortality with that of other common diseases.

**Results:** Thirty-five percent of patients with PD lived more than 6 years. Sex and race significantly predicted survival; patients who were female (HR [hazard ratio], 0.74; 95% CI, 0.73-0.75), Hispanic (HR, 0.72; 95% CI, 0.65-0.80), or Asian (HR, 0.86; 95% CI, 0.82-0.91) had a lower adjusted risk of death than white men. Dementia, diagnosed in 69.6% of cases and most often in African American patients (78.2%) and women (71.5%), was associated with a greater likelihood of death (HR, 1.72; 95% CI, 1.69-1.75). Parkinson disease mortality was greater than that of many common life-threatening diseases. Patients with terminal PD were hospitalized frequently for cardiovascular disease (18.5%) and infection (20.9%) but rarely for PD (1.0%). Regional survival rates were similar but patients with PD living in urban high industrial metal emission areas had a slightly higher adjusted risk of death (HR, 1.19; 95% CI, 1.10-1.29).

**Conclusions:** Demographic and clinical factors impact PD survival. Dementia is highly prevalent in patients with PD and is associated with a significant increase in mortality. More research is needed to understand whether environmental exposures influence PD course or survival.

Experience shorter life spans, what illnesses dominate in terminal PD, and how PD mortality compares with other illnesses of elderly individuals. Dementia is a known comorbidity in PD, but its pervasiveness and impact on mortality across demographic subpopulations in PD is unknown. Therefore, we investigated the reasons for hospitalization in the last year of life in patients with PD, produced comparative mortality data for PD, and determined the impact of dementia on PD mortality.

Environmental exposures, most notably to pesticides, have been associated with increased risk of PD in rural areas.16,17 Lead and manganese have emerged as potential urban environmental risk factors for PD, evidenced by recent findings of increased lead deposition in the long bones of patients with PD compared with those of control subjects (odds ratio [OR], 3.21; 95% CI, 1.17-8.83)18 and our report that PD incidence in urban areas is greater where emissions of manganese are highest (risk ratio, 1.78; 95% CI, 1.54-2.07) using a national, nonmobile, neurologist-confirmed cohort.19 However, it is unknown what the potential impact of postsymptom-onset exposures to pesticides or metals could have on disease course or survival. In an exploratory aim, we investigated whether PD survival varies by geography (eg, state, region of the country, urban/rural classification) and then examined the variation in survival by local metal emission using our previously identified urban high- and low-emission areas.

METHODS

This study was approved by the Human Studies Committee at Washington University School of Medicine, St Louis, Missouri.

STUDY POPULATION

Medicare is a government-mandated insurance program used by 98% of Americans 65 years and older. We searched all Medicare outpatient and physician claims data beginning from 2002. Beneficiaries with claims for PD and International Statistical Classification of Diseases, Ninth Revision code 332 or 332.0 were extracted, and incident cases were identified using previously published methods.20,21 Those who also had diagnostic claims for secondary parkinsonism (code 332.1) or other degenerative diseases of the basal ganglia (code 333.0), regardless of the order of diagnoses, were excluded from further analysis.

DEMOGRAPHIC AND CLINICAL DATA

Medicare Beneficiary Annual Summary Files contain individual-level demographic, health service use, and survival data, as well as clinical data on 21 common conditions, identified using International Statistical Classification of Diseases, Ninth Revision-based algorithms.22 We used the clinical data from these files to calculate an age-weighted modified Charlson comorbidity index score for each PD case.23 These data were also used to determine whether a PD case had been diagnosed with Alzheimer dementia/ senile dementia or related disorders, according to the Centers for Medicare & Medicaid Services Chronic Condition Warehouse algorithm22 (hereafter referred to as dementia) between 1997 (the first year available) and 2008.

To investigate terminal health experiences in PD, we extracted inpatient clinical summary data and health care service use data from the 2005 Medicare Beneficiary Annual Sum-
mary Files for patients with PD who died between January 1, 2006, and March 30, 2007. Last year of life hospitalization, skilled nursing facility, and hospice use rates were calculated. To identify specific illnesses that factor into the death of a person with PD, we extracted the first 10 (all that were available in this data set) diagnosis-related group codes for each hospitalization that occurred in the year leading to death. Diagnosis-related group codes are used nationwide to indicate the clinical reason for each admission to Medicare. These were grouped by organ system or disease process: (1) cardiovascular, (2) infection, (3) pulmonary, (4) malignancy, (5) gastrointestinal, (6) cerebrovascular, (7) musculoskeletal, (8) psychiatric, and (9) other. Hospitalizations for degenerative nervous system disorders were assigned to the category of Parkinson disease. Finally, clinical and survival data for the entire aged Medicare population were extracted from these files to compare PD mortality with that of other common diseases among elderly individuals.

GEOGRAPHIC FACTORS

Residential information was used to assign each PD case to (1) a state/US territory, (2) a census region (Northeast, Midwest, South, or West), and (3) a US Department of Agriculture Rural-Urban Continuum category.24,25 To investigate the impact of passive environmental metal exposure on PD survival in urban areas, we applied our previously developed county-level metal exposure classification derived from Environmental Protection Agency Toxic Release Inventory data. A geographic information system was used to spatially link geographic data to the residential, demographic, and clinical data, allowing quantitative analysis of survival by state and region and between urban areas with high or low metal release.

DESCRIPTIVE ANALYSES

The race, sex, age, and geographic distributions of the study population were determined. We compared the cumulative incidence of dementia by race, age, and sex. For investigation of terminal PD health care consumption, we report the rate of use of last-year-of-life hospital, skilled nursing facility, and hospice services for patients who died in 2006 or the first quarter of 2007. We also identified the clinical condition categories associated with hospitalization in this terminal PD cohort. To provide comparative mortality data in patients with PD, we compared crude mortality rates and adjusted survival between patients with incident PD and the entire Medicare beneficiary population, as well as beneficiaries with (1) myocardial infarction, (2) colorectal cancer, (3) congestive heart failure, (4) ischemic heart disease, (5) chronic obstructive pulmonary disease, (6) Alzheimer dementia, (7) hip fracture, and (8) stroke/transient ischemic attack.

SURVIVAL ANALYSES

Survival status was determined from 2002 to 2008. Cox proportional hazards models estimated the risk of death associated with demographic, clinical, and geographic variables. The time-to-event variable was from the beginning of the study period to the date of death (measured in months). Surviving cases were censored at the end of 2008.

Our primary model examined the risk of death according to race (white, African American, Hispanic, or Asian), age at diagnosis, and sex. A second model included the diagnosis of dementia as a variable of interest. Separately, we investigated survival according to geographic variables: (1) state, (2) census region, and (3) rurality. We then compared survival between patients with PD residing in urban counties with the low-
Finally, we compared the survival rates of elderly patients with Parkinson disease (PD) with that of patients with common life-threatening diseases, adjusting the model for race, age, sex, and comorbidity index.

### Statistical Analyses

Standard methods were used to produce odds ratios or Cox proportional hazard coefficients with 95% confidence intervals. Statistical analyses were performed using SPSS versions 16 and 17 (IBM SPSS).

### Results

#### Subject Demographics

We identified 138,728 Medicare beneficiaries with incident PD, 99.3% of whom were identified by International Statistical Classification of Diseases, Ninth Revision code 332.0. White individuals comprised 90.6% of the cohort; the remaining were African American (6.1%), Hispanic (2.2%), and Asian (1.1%). The age-adjusted incidence of PD in our cohort was greater in men (537.36 per 100,000) than in women (367.70 per 100,000), consistent with previous studies (Table 1).

Dementia had been diagnosed in 69.6% of our study population by the end of the study period. Thirty-one percent (n = 43,621) of PD cases had documented clinical evidence of dementia or cognitive impairment prior to PD diagnosis. The highest frequency of dementia was found in African American individuals (78.2%) followed by Hispanic individuals (73.1%). White and Asian subjects with PD had lower, similar rates of dementia (69.0% and 66.8%, respectively). Seventy-one percent of women with incident PD were diagnosed with dementia within 6 years of diagnosis, compared with 67.5% of men. Dementia frequency increased with age (Table 2).

### Survival According to Patient Characteristics and Clinical Conditions

Sixty-four percent of patients with PD died during the 6-year study. African American individuals had the highest crude death rate (66.4%) followed closely by white individuals (64.6%). Hispanic individuals had a lower frequency of death (55.4%); Asian individuals had the lowest death rate (50.8%). Adjusting for age, sex, comorbidity index, socioeconomic deprivation, and treating physician specialty with white individuals as the reference group, African American individuals had a slightly higher risk of death (hazard ratio [HR], 1.05; 95% CI, 1.02–1.08), but Hispanic individuals had a 14% lower adjusted likelihood of death (HR, 0.86; 95% CI, 0.82–0.91) and Asian individuals had a 27% lower adjusted likelihood of death (HR, 0.72; 95% CI, 0.65–0.80). Women with PD had a 26% lower risk of death than men (HR, 0.74; 95% CI, 0.73–0.75). Survival decreased with age. Patients with PD with dementia had a lower survival rate (28.1%) than those without dementia (53.9%). Patients with dementia and PD were 72% more likely to die during the study (HR, 1.72; 95% CI, 1.69–1.75) (Table 3).
SURVIVAL ACCORDING TO GEOGRAPHIC VARIABLES

Crude death rates and adjusted likelihood of death were similar across states, between regions of the country, and by rural/urban classification (Table 4). However, adjusted PD death risk was 19% higher in those living in urban areas with high industrial manganese output at the time of diagnosis (HR, 1.19; 95% CI, 1.10-1.29) when compared with the adjusted risk in low-manganese counties. A sensitivity analysis using only neurologist-diagnosed cases reduced the magnitude but not the direction of this finding. There was no increase in the risk of death associated with residence in an urban county with high industrial lead emission.

COMPARATIVE MORTALITY

Beneficiaries with incident PD had crude mortality rates comparable with Medicare beneficiaries with incident diagnoses of acute myocardial infarction (65.8%), hip fracture (66.6%), and Alzheimer dementia (68.2%). As di-

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**Table 3. Parkinson Disease Death According to Subject Characteristics and Disease Complications**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted Death Rate, No. (%)</th>
<th>Unadjusted Risk of Death</th>
<th>HR (95% CI)</th>
<th>Adjusted 6-y Risk of Deatha</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>43,871 (67.1)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>45,437 (62.0)</td>
<td>0.87 (0.85-0.88)</td>
<td>0.74 (0.73-0.75)</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>81,203 (64.6)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>56,559 (66.4)</td>
<td>1.05 (1.02-1.08)</td>
<td>1.05 (1.02-1.08)</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>16,811 (55.4)</td>
<td>0.82 (0.77-0.86)</td>
<td>0.86 (0.82-0.91)</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>7,655 (50.8)</td>
<td>0.68 (0.63-0.73)</td>
<td>0.72 (0.65-0.80)</td>
<td></td>
</tr>
<tr>
<td>Age, yb</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>67-69</td>
<td>36,14 (37.9)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>70-74</td>
<td>10,996 (47.0)</td>
<td>1.32 (1.27-1.37)</td>
<td>1.31 (1.26-1.37)</td>
<td></td>
</tr>
<tr>
<td>75-79</td>
<td>19,644 (58.1)</td>
<td>1.81 (1.75-1.87)</td>
<td>1.81 (1.74-1.88)</td>
<td></td>
</tr>
<tr>
<td>80-84</td>
<td>24,435 (69.4)</td>
<td>2.47 (2.39-2.56)</td>
<td>2.49 (2.40-2.59)</td>
<td></td>
</tr>
<tr>
<td>≥85</td>
<td>30,619 (83.2)</td>
<td>3.82 (3.69-3.95)</td>
<td>3.9 (3.79-4.01)</td>
<td></td>
</tr>
<tr>
<td>Disease complications</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No dementia</td>
<td>19,874 (47.1)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>Dementia</td>
<td>69,434 (71.9)</td>
<td>1.85 (1.82-1.87)</td>
<td>1.72 (1.69-1.75)</td>
<td></td>
</tr>
</tbody>
</table>

**Table 4. Parkinson Disease Mortality According to Geographic Variables From 2002 to 2008**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted Death Rate, No. (%)</th>
<th>Unadjusted Risk of Death</th>
<th>HR (95% CI)</th>
<th>Adjusted 6-y Risk of Deatha</th>
</tr>
</thead>
<tbody>
<tr>
<td>Geographic US regionb</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>20,088 (65.1)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>Midwest</td>
<td>22,597 (65.1)</td>
<td>0.99 (0.98-1.01)</td>
<td>1.04 (1.03-1.07)</td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>33,253 (65.5)</td>
<td>1.00 (0.99-1.02)</td>
<td>1.06 (1.05-1.08)</td>
<td></td>
</tr>
<tr>
<td>West</td>
<td>12,316 (60.5)</td>
<td>0.89 (0.88-0.92)</td>
<td>0.93 (0.91-0.96)</td>
<td></td>
</tr>
<tr>
<td>Population densityc</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Completely urban, &gt;1,000,000</td>
<td>68,972 (64.7)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>Completely rural, &lt;2500</td>
<td>55,782 (62.4)</td>
<td>0.89 (0.84-0.95)</td>
<td>0.91 (0.88-0.94)</td>
<td></td>
</tr>
<tr>
<td>Urban heavy metal released</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low manganese, &lt;25th percentile</td>
<td>30,117 (61.9)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>High manganese, &gt;75th percentile</td>
<td>59,862 (66.2)</td>
<td>1.06 (1.03-1.08)</td>
<td>1.19 (1.10-1.29)</td>
<td></td>
</tr>
<tr>
<td>Low lead, &lt;25th percentile</td>
<td>16,632 (62.9)</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td></td>
</tr>
<tr>
<td>High lead, &gt;75th percentile</td>
<td>13,117 (64.4)</td>
<td>1.01 (0.96-1.08)</td>
<td>1.02 (0.98-1.09)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; HR, hazard ratio.

a Adjusted for race, sex, age, dementia status, socioeconomic deprivation score, physician specialty, and age-weighted modified Charlson comorbidity index score.

b To be designated as an incident case, a beneficiary was required to have 2 years of Medicare data with no Parkinson disease claims; therefore, the youngest age possible is 67 years.

c According to the US Department of Agriculture Rural Urban Continuum Code Classification.

d Total on-site release as reported to the Environmental Protection Agency.
played in Table 4, the age-, race-, and sex-adjusted 6-year risk of death among those with PD was nearly 4 times greater than those without PD or other common diseases (OR, 3.87; 95% CI, 3.82-3.93). Additionally, patients with PD had a significantly higher adjusted mortality than those with new diagnoses of colorectal cancer, stroke/transient ischemic attack, ischemic heart disease, or chronic obstructive pulmonary disease (Table 5).

**TERMINAL PD**

We identified 12,897 patients with PD who died in 2006. Hospitalization in the last year of life was very common; 73.5% of patients with terminal PD were hospitalized at least once. Hospice services were used frequently as well, by 69.8% of those who were dying. Skilled nursing facility services were used the least often, by 44.0% of patients with terminal PD.

We identified 44,543 hospitalizations (average, 3.4 per person). Diagnosis-related group data were available for 44,505 of these admissions. The most common reasons for hospitalization in terminal PD were infection (20.9%), cardiovascular disease (18.5%), and noninfectious pulmonary disease (12.8%). Less common reasons for hospitalization included treatment of chronic obstructive pulmonary disease, (13) ischemic heart disease, and (14) hip fracture.

In this nationwide, population-based study, we report the survival of Medicare beneficiaries with incident PD. Our data suggest that PD is associated with a reduced life expectancy compared with common diseases of elderly individuals. Furthermore, we demonstrate that dementia occurs commonly in patients with incident PD 65 years and older; this had the strongest effect on age-adjusted survival among the variables that we studied. Dying patients with PD are hospitalized frequently for cardiovascular and infectious diseases rather than nervous system disorders, and they use hospice care. Our exploratory data raise the question that environmental factors may also influence risk of death after diagnosis.

There are several strengths to this study. This cohort of nearly 140,000 patients with PD provides substantial power to detect variables with large and small effects on survival. We are also able to provide survival data for women and minorities, who are often absent or under-represented in PD epidemiologic studies. Our study design linked otherwise separate clinical, administrative, and environmental data sets to investigate unique combinations of predictors of survival in patients with PD.

The use of the Medicare database for case identification also permitted detailed investigation of dementia/cognitive impairment in PD. Previous estimates of dementia in PD are variable, suggesting a prevalence of 26% to 89% in white individuals. Interestingly, the ethnic pattern of PD-associated dementia in our data set appears to follow that seen in Alzheimer disease, with African American individuals and women being affected more often than whites and men. This may suggest a unique pathophysiologic course of dementia in African American individuals and women with PD, or it may reflect the increased susceptibility of women and African American individuals with PD to develop coexistent Alzheimer dementia. However, given the broad definition of dementia/cognitive impairment used in this study and the difficulties with clinical diagnostic accuracy, clinical-pathologic studies are needed to clarify the pathologic basis of dementia in women and minorities with PD. However, our data highlight the need for prevention or treatment for dementia in patients with PD because of its effect on survival.

Our methods also allowed us to identify potential areas of improvement in the care of patients with PD by identifying illnesses temporally associated with death. There are several reasons why patients with PD may be hospi-
talized for cardiovascular disease and infection often prior to death. Patients with PD are likely susceptible to urinary tract infections and pneumonia secondary to disease-mediated bladder dysfunction and microaspiration. Cardiovascular disease is the most common cause of hospitalization in Americans older than 65 years, and PD is not thought to confer protection against cardiovascular disease risk factors. Fewer than half of patients with PD are treated by neurologists, indicating that another possible contributor is that the management of PD symptoms consumes the majority of health care encounters. This may lead to less-aggressive screening for or treatment of other common illnesses. Additionally, physicians and patients alike may attribute nonspecific symptoms such as fatigue, weakness, or exercise intolerance to PD when they in fact represent other illnesses such as subacute cardiopulmonary disease. Future studies investigating specific ways by which specialist care reduces PD mortality would be valuable.

Many researchers have investigated the environmental triggers of neurodegeneration in PD, with pesticides and redox-active metals such as lead and manganese implicated in multiple studies. Exposure to a combination of maneb (an herbicide that contains manganese) and paraquat, by virtue of living within 500 m of application sites, has been associated with an increased risk of being diagnosed with PD (HR, 1.75; 95% CI, 1.13-2.73). We recently reported that age-, race-, and sex-adjusted PD incidence is greater in urban counties with the highest industrial release of manganese (risk ratio, 1.78; 95% CI, 1.54-2.07) compared with urban counties with no or low release of this metal, using a nonmobile, neurologist-confirmed cohort. This additional finding of a slightly higher adjusted death risk in urban areas with high manganese output, but not just urban areas of the United States, calls into question whether continued exposure to basal ganglia toxins after symptom onset may accelerate the clinical course of idiopathic PD or be associated with the development of important comorbidities. Future studies with detailed clinical and exposure data are essential to determine whether postsymptom-onset exposures to neurotoxins (including pesticides and metals) are associated with the development of more severe PD clinical features or more rapid progression.

There are several limitations to this study. Differential misdiagnosis is always a potential bias in studies that rely on clinical or administrative data. It is possible that in addition to PD, cases with 1 of the much rarer but more lethal Parkinson plus syndromes may have been included in our cohort. Future studies testing the endurance and robustness of our case definition methods may include prescription data record review (using cases diagnosed after 2003 when the Medicare Prescription Drug, Improvement, and Modernization Act was passed) may be helpful. However, these diseases are rare and unlikely to significantly alter our primary findings. It is possible that dementia in men and white individuals with PD may be underreported, but a recent study suggests that white men tend to see neurologists more often than patients with PD in other demographic groups. Differences in health care-seeking behaviors or health care access could potentially impact our baseline population, clinical, or survival data.

Although we accounted for known demographic, comorbid, and physician factors likely to impact survival, there may be undiscovered confounders that would alter the risk ratios we obtained.

Our environmental data analyses were performed primarily to generate hypotheses for future environmental epidemiologic research and assess for geographic confounders in survival, and they should be interpreted cautiously. We lack historical individual exposure data (community or occupational) that are impossible to obtain retrospectively for an administratively identified cohort over the decades of exposure that may be relevant for an adult-onset neurodegenerative disease. Although specific to manganese, the change in risk was modest and could be explained entirely by unknown confounders. There may be unknown nonneurologic mechanisms by which PD mortality is increased in urban areas with high metal emissions. In spite of these limitations, postsymptom-onset environmental exposures may need to be considered when investigating future neuroprotective therapies in PD as ongoing individual exposures may influence disease course.

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Author Contributions: Dr Willis had full access to and takes full responsibility for the accuracy of the data. Study concept and design: Willis, Evanoff, Perlmutter, and Racette. Acquisition of data: Willis and Racette. Analysis and interpretation of data: Willis, Schootman, Kung, Evanoff, and Perlmutter. Drafting of the manuscript: Willis and Kung. Critical revision of the manuscript for important intellectual content: Schootman, Evanoff, Perlmutter, and Racette. Statistical analysis: Willis, Schootman, and Evanoff. Obtained funding: Willis and Racette. Study supervision: Perlmutter and Racette.

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