THE GERONTOLOGICAL SOCIETY OF AMERICA®

Journals of Gerontology: Medical Sciences
cite as: *J Gerontol A Biol Sci Med Sci*, 2021, Vol. 76, No. 7, 1340–1345
doi:10.1093/gerona/glab042
Advance Access publication February 25, 2021

OXFORD

Research Article

Frequency of Parkinsonism and Parkinson Disease in African Americans in the Chicago Community

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Received: June 16, 2020; Editorial Decision Date: January 17, 2021

Decision Editor: Anne B. Newman, MD, MPH, FGSA

Abstract

Background: There is paucity of data about African American (AA) patients with Parkinson's disease (PD) and parkinsonism which may precede PD in older adults. Prior studies suggest that there are lower rates of PD in the AA population, with more cognitive impairment in AA with PD. This study aimed to investigate differences in PD, parkinsonism, and cognition between White and AA populations in 3 longitudinal epidemiologic cohort studies of aging.

Methods: This study examined parkinsonism, PD frequency, and cognition of community-dwelling older individuals in 3 longitudinal epidemiologic cohort studies. Parkinsonism was based on an exam utilizing the modified Unified Parkinson's Disease Rating Scale performed by a nurse. PD was based on self-report, medications used for treatment of PD, and examination findings. Cognition was assessed using 19 performance-based tests that assess 5 cognitive domains.

Results: AA participants were less likely to have parkinsonism compared to Whites, even with age and gender differences. Frequency of PD was not significant between groups. AA were more likely to have lower cognitive scores as compared to Whites. AA were less likely to have parkinsonism even with controlling for cognitive differences between groups.

Conclusions: Parkinsonian signs are present among AA in the community at lower rates than in White individuals. Cognitive profiles of AA and Whites with parkinsonism may be different, suggesting differing contributions of pathology to cognitive decline and parkinsonism between groups. Additional research is needed to understand the progression of parkinsonism to PD, as well as to understanding the cognitive differences in AA with parkinsonism.

Keywords: African American, Health Disparities, Parkinson's disease, Parkinsonism

Parkinson's disease (PD) is a progressive neurodegenerative disorder affecting 1 million people in the United States (1) and is characterized by bradykinesia, rigidity, postural instability, and tremor. Parkinsonism has been defined as exhibiting signs in 1 or more of the 4 domains of PD, but without meeting the diagnostic criteria for PD and is associated with poor health outcomes in older adults (2,3). While the nosology of parkinsonism and PD is still debated, identification of parkinsonism in the community may identify adults at risk for developing PD (4). One study examining pathology of older adults with features of parkinsonism showed significant reductions in dopaminergic neurons and terminals in the substantia nigra and putamen that was intermediate between those with no motor deficits and those with diagnosed PD (4). However, more recent work has also shown heterogeneity in parkinsonism patients, with pathological contributions from PD, cerebrovascular disease, and cerebral amyloid angiopathy correlating with premorbid motor features (5). Our current understanding of PD and parkinsonism is disproportionately based on

studying White populations, leading to a gap in knowledge about the frequency and clinical characteristics in individuals of African descent.

The cumulative incidence of PD in African Americans (AA) has been estimated at 23 per 100 000 compared to 54 per 100 000 in White Americans (WA) (6). A study reporting prevalence of PD in AA and WA found that WA had a substantially higher incidence and prevalence of PD (6); however, this is not consistent between studies, with Schoenberg and colleagues reporting equal prevalence between AA and WA in Copiah County, Mississippi (7). Significant geographic variation has been reported, with a higher concentration of PD patients in the Midwest and Northeast regions (2–10 times more) (6). The prevalence and incidence of PD also tended to be greater in urban counties compared to rural ones. It is not known whether the lower prevalence/incidence among AA is due to ascertainment bias owing to health disparities (8) or differences in disease phenotype (9).

Direct comparison of AA and WA with PD shows greater disability and disease severity in AA (10). It has been reported that Unified Parkinson's Disease Rating Scale (UPDRS) motor scores for AA average 35 (*SD* ± 15) compared to WA at 28 (*SD* ± 14) (10). This difference persists after adjusting for age, cognitive function, and years since diagnosis. Hoehn and Yahr stage in AA with PD has been reported to be higher as compared to WA with PD (2.5 vs 2.0, $p < .01$) (11). Several factors are proposed to account for a more severe PD phenotype, including less likelihood of receiving care (12), barriers in access to neurologic care, and methodological confounds associated with inconsistent diagnostic criteria or ascertainment bias (13). AA are half as likely to be diagnosed with PD as WA and 4 times less likely to receive treatment for PD (7,8). A tertiary movement disorders center in the Northeast reported AA patients represented only 6.1% of the total patients with PD (10). Across all measures of disease severity and disability, significant differences in PD are seen at lower socioeconomic levels (10), where AA are disproportionately represented.

There have been some suggestions that there may be phenotypic differences between WA and AA with PD. One study examining Medicare data reported that AA with PD had higher rates of dementia than WA (14). Cognitive impairment in PD has a well-studied profile including impairments in attention, executive function, processing speed (15). The cognitive profile of parkinsonism and PD mild cognitive impairment (MCI) and dementia has not been well studied in the AA population even with some evidence that it may be more prevalent in AA with PD (14).

The purpose of this study was to examine the frequencies and characteristics of AA with PD or parkinsonism in community-based cohorts in the Chicago area. The Minority Aging Research Study (MARS) (16), the Rush Memory and Aging Project (MAP), and the Religious Orders Study (ROS) (17) are all community-based, longitudinal cohort studies that identify risk factors for the development of Alzheimer's disease and cognitive decline. All 3 studies have a common core of data, so the participant outcomes can be compared. Each participant receives an annual exam, which includes a modified UPDRS and a full cognitive evaluation with a standardized battery of tests (16,17). Most information that is currently available in the literature regarding AA with PD and parkinsonism utilizes retrospective data through chart review or uses surrogate markers for diagnosis such as medication use or diagnostic codes (6,18). Our current study aimed to fill the gap in knowledge concerning parkinsonism and its relationship to cognitive profile, by utilizing data collected from 3 large cohorts of community-dwelling AA participants in ongoing longitudinal studies examining risk factors for the development of Alzheimer's disease and cognitive impairment.

Method

We investigated the presence of parkinsonian signs, diagnosis of PD, and cognition in 3 community-based cohort studies. Studies were approved by the Rush University Institutional Review Board.

This study included participants in ongoing community-based epidemiologic cohort studies: MARS (16), MAP, and ROS (17). All studies were designed to study the natural history and neuropathological findings of dementia and cognitive impairment in community-dwelling individuals. MARS is completely AA. Participants are recruited through churches, senior buildings, and clubs and organizations that cater to older AA (16). MAP participants include mostly non-Latino Whites and about 10% AA and Latinos; participants are recruited from retirement communities, churches, and senior buildings in the northeastern Illinois region (17). ROS recruits Catholic nuns, priests, and brothers from more than 40 groups around the United States (17). The recruitment and evaluation for all 3 cohorts is by the same team. All participants in these studies were evaluated at home. Common comorbidities are well represented since disabled individuals are not required to travel for study visits. Because the studies share a common core of data, data were merged to examine parkinsonian signs across race. Demographic information was collected on each participant (age, sex, education, race, and income). Participants underwent a structured clinical evaluation, including a modified UPDRS (19) and cognitive testing. The modified UPDRS was developed in order to allow nonphysicians to administer an examination that could assess signs of parkinsonism and is similar to the UPDRS with all portions of the examination graded on a numerical scale with higher values correlating with more severe parkinsonian signs. Some motor domains were changed in order to avoid ambiguity so that a nonphysician can more easily administer the assessment. Twenty-six items assess 4 parkinsonian signs including tremor, bradykinesia, rigidity, and gait. Interrater reliability has been demonstrated between nurses and movement disorders trained neurologists in a prior publication (19). Due to the addition and modification of variables in the creation of the modified UPDRS from the UPDRS part III, the total possible score on the modified UPDRS is 127. A previously validated parkinsonism category was utilized which was based on the number of the 4 parkinsonian signs present with modified UPDRS assessment. A nurse examined each participant at each visit and a physician made the diagnosis of parkinsonism if the participant met criteria by examining the modified UPDRS score. A parkinsonian sign was present if 2 or more of its items were scored as a mild abnormality. Parkinsonism was present if at least 2 of the 4 parkinsonian signs were present (20). Data were analyzed from the last visit, and participants with incomplete data for modified UPDRS scores were excluded from analysis. In order to analyze the modified UPDRS score as a continuous measure, the points from all domains were added for a final score in each participant. In addition to each annual modified UPDRS assessment, individuals were asked whether they had ever been told by a doctor, nurse, or therapist that they had parkinsonism or PD, and whether they were currently taking any medications for the condition, including levodopa or a dopamine agonist. A diagnosis of PD cannot be made in the participants during the study by examination only as they are examined by a nurse and not a neurologist during their annual exam, so clinical PD was then determined based on the combination

of examination, self-report, and medications used for treatment of PD (21). Cognition was assessed using 19 performance-based tests that assess 5 cognitive domains episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability (perceptual orientation) (22). To minimize floor and ceiling effects, composite measures were used in analyses. For each test, raw scores were standardized using the baseline mean and standard deviation across the cohorts. The *z*-scores were then averaged across all tests to obtain summary measures representing global cognition and the 5 domains. Psychometric properties of the individual tests and composites have been previously described (23). Because our interest was evaluating AA participants, we examined only the AA and the non-Latino White participants as a comparison group.

Statistical Analysis

Univariate analysis with *t* test, Mann–Whitney *U* test, and chi-square test were performed to compare AA and WA participants, including the modified UPDRS score. Multivariate regression analysis was performed to determine the impact of demographic features on the modified UPDRS score and the various cognitive scores. Stepwise linear regression analysis was performed in order to analyze the impact of demographic features and cognitive scores on parkinsonism diagnosis. The modified UPDRS score was not a normal distribution, so a log transformation was used in the regression analysis.

Results

The community-based sample consisted of 1272 AA and 3335 WA (Table 1). The AA group was significantly younger, had fewer years of education, and lower baseline income (Table 1). There were more WA than AA with a clinical diagnosis of PD; however, this was not statistically significant (Table 1, $p = .23$). Frequency of parkinsonism and total modified UPDRS was significantly higher in WA compared to AA (Table 1, $p < .0001$). Although age was significantly higher in the WA groups, there was no effect of age on WA as compared to AA on modified UPDRS score or parkinsonism diagnosis ($p = .10$, $p = .99$). Regression analysis showed that older age and lower baseline income were all associated with higher modified UPDRS scores (Table 2, $p < .0001$). Cognitive scores were significantly different between the 2 groups, with AA more likely to have lower scores on all cognitive performance-based tests, but with the lowest scores in global cognition, working memory, and perceptual orientation as compared to WA (Table 3). AA race, males, lower education, older age, higher income, and PD diagnosis were all associated with lower scores (Table 3). On stepwise logistic regression with parkinsonism as the outcome, AA were still less likely than WA to have parkinsonism even when controlling for the lower cognitive measures (Table 4).

Discussion

We assessed parkinsonian signs and clinical diagnoses of PD in 3 community-based studies of older AA and WA to determine whether there are racial differences in the frequency of parkinsonism. Frequency of parkinsonism using a number of techniques was significantly higher among WA. Further, there were fewer AA participants with PD as compared to WA; however, this was not statistically significant. Taken together, these studies support previous evidence that AA in the community have fewer parkinsonian signs than WA. AA with parkinsonism also appear to have differing cognitive profiles than White participants, which is, at this time, unexplained. Differing pathology in the form of Alzheimer's disease or vascular disease could be affecting AA as compared with WA who may experience more PD pathology. This might explain higher rates of parkinsonism in the WA and differing cognitive profiles between the groups. This needs to be further explored.

Reasons for the disparity in parkinsonism between AA and WA in these cohorts unknown. The AA participants in the community had significantly lower scores on modified UPDRS, but there was a significant difference in the age of WA and AA, with AA being younger on average, and frequency of PD and parkinsonism are known to increase with age. However, the regression analysis did

Table 1. Demographics of the Community-Based Sample

	AA, $N = 1272$	White, $N = 3335$	*p* Value
Age at visit, mean (*SD*)[1]	79 (6.88)	84.23 (7.39)	<.0001
Sex, *n* (%)			<.0001
Women	1007 (79.17)	2402 (72.02)	
Men	265 (20.83)	933 (27.98)	
Education, median (IQR)[1]	14 (5)	16 (5)	<.0001
Baseline income level, *n* (%)[1706]			<.0001
$0 to $4999	12 (1.05)	38 (2.16)	
$5000 to $9999	76 (6.64)	60 (3.41)	
$10 000 to $14 999	137 (11.98)	113 (6.43)	
$15 000 to $19 999	105 (9.18)	131 (7.46)	
$20 000 to $24 999	121 (10.58)	133 (7.57)	
$25 000 to $29 999	92 (8.04)	131 (7.46)	
$30 000 to $34 999	114 (9.97)	172 (9.79)	
$35 000 to $49 999	161 (14.07)	302 (17.19)	
$50 000 to $74 999	171 (14.95)	315 (17.93)	
$75 000 and over	155 (13.55)	362 (20.6)	
Modified UPDRS score, median (IQR)[1884]	32 (15)	41 (22)	<.0001
Clinical Parkinson's disease, *n* (%)[247]	5 (0.44)	25 (0.78)	.23
Parkinsonism, *n* (%)[1556]	115 (16.72)	913 (38.64)	<.0001

Notes: Superscripts are number of missing data. AA = African American; IQR = interquartile range; modified UPDRS = modified Unified Parkinson's Disease Rating Scale; *SD* = standard deviation.

not show that age was a significant factor in difference in parkinsonism between the groups. Finally, nurses administered the modified UPDRS. Although the scale has been validated and scores show good agreement between nurses and movement disorders neurologists, parkinsonism may have been overestimated due to modified UPDRS scores reflecting factors such as arthritis, stroke, etc. Given that WA were older, they may have had more medical comorbidities which would increase their scores compared to the AA participants. A movement disorders neurologist may be able to better distinguish parkinsonism from other ailments causing slowness or difficulty walking. Parkinsonism may have been overestimated in general because we did not exclude antipsychotic use or atypical parkinsonism; however, this cannot be deduced from the modified UPDRS. However, the modified UPDRS is an easy way to screen for parkinsonism in the community. Sex was also not found to be a factor in parkinsonism or PD diagnosis. With PD, this could be due to low numbers of PD diagnosis or due to the cohorts being majority female. With parkinsonism, this is consistent with prior publications (3), and could be due to parkinsonism not being solely due to PD pathology, and thus might be less likely to have higher prevalence in males.

PD diagnosis was low in both groups, and while lower in the AA group, it was not statistically significant. The rate of clinical PD was lower in these groups than in the general population. Most of the current literature suggests that there is a lower prevalence of PD in the AA population, but this was not the case in these cohorts (6). The diagnosis in these cohorts is partially obtained through self-report, so it is possible there was bias in determining clinical PD as

Table 2. Regression of Modified UPDRS Score (on the log scale) and Demographic Variables

Variable	Estimate (*SE*)	*p* Value
Race (White vs AA)	0.11 (0.019)	<.0001
Gender (men vs women)	0.026 (0.019)	.172
Education	−0.003 (0.003)	.223
Age at visit	0.014 (0.001)	<.0001
Baseline income	−0.019 (0.004)	<.0001
Age × race (White vs AA)	0.004 (0.003)	.10

Notes: R^2 = 0.19. White, older age, and lower income were associated with higher score. AA = African American; *SE* = standard error; UPDRS = Unified Parkinson's Disease Rating Scale.

Table 4. Regression of Parkinsonism on Demographic and Cognitive Variables

Variable	OR (95% CI)	*p* Value
Race (White vs AA)	2.58 (1.89, 3.52)	<.0001
Education	1.05 (1.01, 1.09)	.03
Age at visit	1.04 (1.02, 1.06)	.0003
Baseline income	0.93 (0.89, 0.98)	.01
Perceptual speed	0.58 (0.50, 0.68)	<.0001
Working memory	0.77 (0.64, 0.93)	.01

Notes: All demographic and cognitive variables were included in the regression model, but only significant variables are shown. AA = African American; CI = confidence interval; OR = odds ratio.

Table 3. Regression of Cognitive Scores and Demographic Variables

Variable	Global Cognition	Perceptual Orientation	Perceptual Speed	Semantic Memory	Working Memory	Episodic Memory
White vs AA						
Estimate (*SE*)	0.248 (0.034)	0.487 (0.036)	0.294 (0.043)	0.317 (0.043)	0.227 (0.036)	0.18 (0.041)
p Value	<.0001	<.0001	<.0001	<.0001	<.0001	<.0001
Gender (M/F)						
Estimate (*SE*)	−0.213 (0.035)	0.144 (0.038)	−0.300 (0.045)	−0.167 (0.045)	−0.105 (0.038)	−0.377 (0.043)
p Value	<.0001	.0002	<.0001	.0002	.005	<.0001
Education						
Estimate (*SE*)	0.041 (0.005)	0.051 (0.005)	0.059 (0.006)	0.043 (0.006)	0.052 (0.005)	0.03 (0.006)
p Value	<.0001	<.0001	<.0001	<.0001	<.0001	<.0001
Age						
Estimate (*SE*)	−0.047 (0.007)	−0.026 (0.002)	−0.057 (0.003)	−0.049 (0.003)	−0.022 (0.002)	−0.046 (0.003)
p Value	<.0001	<.0001	<.0001	<.0001	<.0001	<.0001
Baseline income						
Estimate (*SE*)	0.048 (0.006)	0.048 (0.007)	0.055 (0.008)	0.056 (0.008)	0.036 (0.007)	0.044 (0.008)
p Value	<.0001	<.0001	<.0001	<.0001	<.0001	<.0001
PD (Y/N)						
Estimate (*SE*)	−0.869 (0.182)	−0.472 (0.197)	−0.472 (0.197)	−1.26 (0.217)	−1.185 (0.185)	−0.848 (0.221)
p Value	<.0001	.017	.017	<.0001	<.0001	.0001
R^2	0.21	0.19	0.23	0.18	0.12	0.17

Notes: AA = African American; PD = Parkinson's disease; *SE* = standard error.

some may not report their diagnosis. PD also may be present more frequently in both cohorts but is undiagnosed; however, it is not possible to diagnose PD during the visits as the nurses are unable to give this diagnosis using the modified UPDRS.

The lower modified UPDRS scores in the AA group are also contradictory to what has been seen in the literature with regards to AA with PD. Most studies report that AA with PD are more severe at presentation than WA; however, the AA in these cohorts have milder motor scores. It may be that the health disparities that exist in the AA population create decrease in access to care, so AA with PD have poor access to specialized care and thus have later diagnosis than WA with PD (12). This may also be a difficult comparison as many participants with parkinsonism may never go on to develop PD. As parkinsonism is not a characteristically well-defined disorder and there are no standardized diagnostic criteria used clinically, it is difficult to assess how many patients with parkinsonism will go on to develop clinical PD. At this time, there is no information about how often parkinsonism patients convert to clinical PD, although it appears that the rate of development of parkinsonism in the older adults is high, with prior publications in these cohorts documenting 53.9% of the participants developing parkinsonism after 8.5 years (24). In that same study, postmortem analysis in these individuals showed that either nigral cell body loss, Lewy bodies, or the combination of both was seen in only 25% of those with parkinsonism (24). However, those who had a prior diagnosis of PD showed faster increased in modified UPDRS scores than those without clinical PD. In a smaller study in this cohort, the amount of PD pathology correlated with the degree of motor pathology, but these numbers were much smaller (4). In another study looking at patients with parkinsonism and excluding those patients with diagnosed PD, only 39% of parkinsonian patients had nigral body pathology, with either Lewy bodies or nigral cell loss on autopsy (21). Prior work in non-Latino Whites has shown that mild parkinsonism in older adults can occur without the presence of PD pathology (3). Further pathology in parkinsonism patients has shown cerebral amyloid angiopathy and vascular disease which suggests there may be other factors contributing to the motor features of parkinsonism (5). Genetic data on parkinsonism in the ROS and MAP cohorts have also been analyzed, with some evidence showing alleles that have been linked to increased PD risk also being associated with parkinsonism in these groups (25). However, these data thus far have not been analyzed in AA. A hindrance to many of studies is the small amount of pathological and genetic data we have on AA patients with PD or parkinsonism, as many of these studies have majority of data from non-Latino Whites. Further research is needed in this area.

Cognition was also assessed in the cohorts. Clinical PD was associated with lower global cognition and lower scores in all the cognitive domains except for perceptual organization. Overall, AA scored lower on all the cognitive scores than the WA, but with the most significant differences seen in working memory and perceptual orientation. However, when controlling for the cognitive variables, AA were still less likely to have parkinsonism. AA may be more likely to have other neurodegenerative illness such as Alzheimer's disease that is less likely to cause parkinsonism as compared to WA who may be more likely to have cognitive deficits associated with parkinsonism and PD. Another factor may be that lower cognitive scores can occur due to racial disparities in cognitive test performance. This has been well documented and is thought to be due to a number of social and cultural factors that differ between AA and WA (26). Cognitive changes may be better examined as changed over time, as opposed to a single assessment, in order to get a better understanding of the cognitive decline occurring in conjunction with parkinsonism features in these participants (23). Whether or how racial differences in cognition affect parkinsonism is unclear and should be investigated in future studies.

In conclusion, our study shows that parkinsonian signs are less frequent in Chicago community-dwelling AA, and AA with parkinsonism may have more cognitive abnormalities than WA with parkinsonism. Although parkinsonism encompasses more than just PD, this study is similar to other studies that show lower rates of PD in the AA community (6). Further studies are needed to characterize PD and parkinsonism in AA in order to identify the reasons for the discrepancy in the diagnostic rates in these patients. Identifying AA with parkinsonism is the first step to ensure access and quality of care. With better identification, a large study can be undertaken to establish whether phenotypic differences exist between AA and WA patients with PD.

Funding

Funding for the study includes the Parkinson Disease Foundation and National Institutes of Health grants RF1AG22018, P30AG10161, R01AG17917, R01AG15819, R01NS78009, and R01AG56352. J.M.S. was supported by Huffington Foundation and a Career Award for Medical Scientists from the Burroughs Wellcome Fund.

Conflict of Interest

Dr. M.B. has received research support from Abbvie, Biogen, and Parkinson Foundation. Dr. L.M.S. has received research support from National Institutes of Health, Michael J. Fox Foundation, The Rosalyn Newman Foundation, Eugenia and Michael Brin, Biotie and Acorda Pharmaceuticals. Drs. D.R., B.O., J.M.S., A.S.B., D.A.B., and L.L.B. have no disclosures. Dr. D.A.H. has received research support from NINDS, Parkinson Foundation, Anti-Aging Foundation, Shapiro Foundation, Abbvie, Pfizer, Biogen, and Neurocrine. We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

Acknowledgments

We thank the participants of this study.

Author Contributions

Dr. M.B.: Acquisition of data, interpretation of data, drafting and revising for important intellectual content, final approval of the version to be submitted. Dr. L.M.S.: Conception and design of the study, analysis and interpretation of data, revising the article, final approval of the version to be submitted. Dr. D.R.: Final approval of the version to be submitted. Dr. B.O.: Analysis of the data, revising the draft for important intellectual content, final approval of the version to be submitted. Dr. J.M.S.: Interpretation of the data, revising the draft for important intellectual content, final approval of the version to be submitted. Dr. A.S.B.: Interpretation of data, revising the draft for important intellectual content, final approval of the version to be submitted. Dr. D.A.B.:

Acquisition of data, interpretation of data, revising the draft for important intellectual content, final approval of the version to be submitted. Dr. L.L.B.: Acquisition of data, interpretation of data, revising the draft for important intellectual content, final approval of the version to be submitted. Dr. D.A.H.: Conception and design of the study, acquisition of data, analysis and interpretation of data, drafting and revising for important intellectual content, final approval of the version to be submitted.

Statement of Ethics

Participants have given written informed consent to participate in these studies. The study protocol has been approved by the Rush University Medical Center Institutional Review Board.

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