(RG3). That is, each of the RGs examined the independent (or direct) effect of one predictor on one binary outcome (PDID, PDND). For RG3, the sex item was excluded from the calculation of overall risk scores on the full MoPaRDS. We ascertained whether prediction patterns varied across sex using prevailing statistical conventions regarding the interpretation and comparison of independent area under the ROC curves (AUC).[19] %ULHI $&9 WKHRU\ ZLGHO\ XVHG PHWULF IRU HYDOXDWLQJ GLDJQRVWLF RU SUHGLFWLYH PRGHOV WKDW GLVFULPLQDWH EHWZHHQ WZR RXWFRPHV model.[20] Values are interpreted such that 0.5 represent chance, 0.5–0.69 represents poor discrimination, 0.7–0.79 represents acceptable discrimination, 0.8–0.89 represents excellent discrimination, and 0.9–1.0 represents outstanding discrimination. We determined the optimal cut-off points for a screen positive result on the MoPaRDS using ROC curves with coordinate points (RG4). We modeled the nature (i.e., extent of between-person differences in within-person changes) and rate (i.e., average number of risk characteristics accumulated per wave) of change in overall risk scores using growth curve analyses (RG5; see[21,22] and Appendix A for further details). Analyses were performed using SPSS 26 (IBM SPSS Statistics, Armonk, 1HZ <RUN 86$ 86$ 86$ 86$ 6RIWZDUH XVHG

RESULTS

Demographic and Clinical Characteristics

Detailed demographic and clinical characteristics of the initially non-demented study sample disaggregated by the two derived baseline groups (PDND, non-demented at Wave 3; PDID, demented at Wave 3) are presented in Table 2.

RG1: Predictive Validity of Individual MoPaRDS Items and Education

We used baseline data to test whether each of the constituent MoPaRDS item independently discriminated PDID from PDND. Detailed results are presented in Appendix B. Findings showed that the two groups were discriminated by three of the eight MoPaRDS items: age (odds ratio [OR] = 5.92; 95% confidence interval [CI] = 1.37–25.62; p = .02); MCI (OR = 37.89; 95% CI = 6.64–216.27; p < .001); and orthostatic hypotension (OR = 7.67; 95% CI = 1.77–33.18; p = .01). There was a trend for falls and/or freezing (OR = 3.50; 95% CI = 0.88–13.88; p = .08). Education did not independently discriminate PDID from PDND (OR = 0.70; 95% CI = 0.20–2.55; p = .59). We FRQGXFWHG DGGLWLRQDO DQDO\VHV WR FKHFN WKH UREXVWQHVV RI WKHVH ILQGLQJV that education did not independently discriminate the groups when (a) tested as a continuous variable (OR = 0.92; 95% CI = 0.73–1.15; p = .46); (b) dichotomized using a cut-point of 12 years of formal education (n below this threshold = 7; OR = 0.97; 95% CI = 0.16–5.69; p = .97); and (c) dichotomized using a cut-point of >1SD below the mean (11 years of formal education; n below this threshold = 5; OR = 1.72; 95% CI = 0.26–11.62; p = .58).

RG2: Predictive Validity of Overall Risk Scores

Detailed results are represented in Table 3. Overall findings showed that full scale risk scores on the eight-item MoPaRDS discriminated the PDID and PDND groups at baseline (OR = 2.45; 95% CI = 1.37–4.38; p = .002). The ROC curve is depicted in Figure 1 (AUC = 0.81; 95% CI = 0.68–0.93; p = .001). Education did not improve predictive validity (median split OR = 1.94; 95% CI = 1.20–3.13; p = .007; AUC = 0.77; 95% CI = 0.63–0.91; p = .004) and was therefore not consid-ered in subsequent analyses.

We tested whether the combination of the three MoPaRDS items that were independent predictors would similarly discriminate PDID from PDND. The three items were age, MCI, and orthostatic hypotension. Findings showed that overall risk scores on this brief configuration discriminated the two groups (OR = 5.72; 95% CI = 2.18–15.03; p < .001). See Figure 1 for the ROC curve (AUC = 0.88; 95% CI = 0.75–1.0; p < .001). Because there was a trend for falls and/or freezing, we also examined predictive validity of a four-item configuration. The AUC of this scale (0.88; 95% CI = 0.78–0.98; p < .001) was equivalent to that of the three-item configuration and was therefore not considered further.

RG3: Se Stratification Analyses

Results for the eight-item MoPaRDS showed that AUC for females and males were quite different and resided in differ-ent interpretive categories. For females, AUC was 0.91 (p = .002), indicating outstanding discrimination. For males, AUC was 0.74 (p = .08), indicating acceptable—but not excellent or outstanding—discrimination. These results, together with the overall pattern of findings reported in Table 3, suggest that the eight-item MoPaRDS performed better for females. Nota-bly, the three-item MoPaRDS performed well and similarly for females and males (see Table 3 for detailed results). For females and males, the AUC was 0.88 and 0.91, respectively.

RG4: Optimal Cut-Off Points for Screen Positive

The optimal cut-off on the eight-item MoPaRDS, as indicated by the extreme upper-left point of the ROC curve (see Figure 1), was a total score ≥ 4. A screen positive distinguished PDID and PDND groups (positive predictive value [PPV] = 50%; negative predictive value [NPV] = 85.7%) with an acceptable level of accuracy (AUC = 0.71; 95% CI = 0.55–0.88; p = .02).

The optimal cut-off on the three-item MoPaRDS was a total score ≥ 2 (see Figure 1). A screen positive (PPV = 63.2%; NPV = 93.1%) discriminated PDID from PDND with a good level of accuracy (AUC = 0.83; 95% CI = 0.69–0.96; p < .001). Complete results for both configurations are reported in Table 3.

RG5: Risk Score Change Tra ectories

Growth curve analyses[21,22] indicated that a random intercept, fixed slope model provided the best fit to longitudinal data for both the eight- and three-item configuration of the MoPaRDS (for model comparisons and fit indices see Appendix C). These results permit three conclusions: (a) baseline overall risk scores statistically differed across participants (8-item: $\hat{\sigma}^2$ = 1.65, p < .001; 3-item: $\hat{\sigma}^2$ = 0.70, p < .001); (b) overall risk scores increased over time (i.e., statistically differed from 0; 8-item: M increase per wave = 0.30, p = .002; 3-item:

TABLE 3.
Research Goals 2–4: Binary logistic regression models for overall risk scores on baseline configurations of the MoPaRDS discriminating PDID from PDND[a]

MoPaRDS Configuration	R^2	*OR*	*PAC*	*Sens*	*Spec*	*PPV*	*NPV*	*LR*	*LR-*	*AUC*
8-item	0.30	2.45[g]	75.0%	42.9%	88.2%	60.0%	78.9%	3.6	0.7	0.81[f]
8-item + education	0.26	1.94[f]	70.8%	21.4%	91.2%	50.0%	73.8%	2.4	0.9	0.77[f]
3-item	0.53	5.72[e]	85.4%	57.1%	97.1%	88.9%	84.6%	19.7	0.4	0.88[e]
4-item	0.52	4.22[e]	81.3%	64.3%	88.2%	69.2%	85.7%	5.4	0.4	0.88[e]
8-item stratified by sex[b]										
Females	0.61	3.81[g]	81.0%	75.0%	84.6%	75.0%	84.6%	4.9	0.3	0.91[f]
Males	0.19	2.04[h]	81.5%	16.7%	100%	100%	80.8%	--	0.8	0.74[h]
3-item stratified by sex										
Females	0.60	5.52[g]	85.7%	75.0%	92.3%	85.7%	85.7	9.7	0.3	0.88[f]
Males	0.57	11.64[f]	85.2%	50.0%	95.2%	75.0%	87.0%	10.4	0.5	0.91[f]
8-item total ≥ 4[c]	0.20	6.00[g]	70.8%	71.4%	70.6%	50.0%	85.7%	2.4	0.4	0.71[g]
3-item total ≥ 2[d]	0.45	23.14[e]	81.3%	85.7%	79.4%	63.2%	93.1%	4.2	0.2	0.83[e]

[a]We verified in follow-up analyses that the MoPaRDS continued to discriminate the two baseline groups when the MCI item was removed from (a) the full configuration (OR = 1.87, $p = 0.27$; AUC = 0.71, $p = 0.21$) and (b) the abbreviated configuration (OR = 4.78, $p = .003$; AUC = 0.79, $p = .002$).
[b]Sex was removed from the MoPaRDS for these analyses (i.e., maximum score = 7).
[c]Participants were stratified into two groups according to whether their overall risk score on the eight-item MoPaRDS fell at or below this cut-off at baseline.
[d]Participants were stratified into two groups according to whether their overall risk score on the three-item MoPaRDS fell at or below this cut-off at baseline.
[e] p value < .001.
[f] p value < .01.
[g] p value < .05.
[h] p value < .10.
MoPaRDS = Montreal Parkinson Risk of Dementia Scale; PDND = Parkinson's Disease with No Dementia; PDID = Parkinson's Disease with Incipient Dementia; R^2 = Nagelkerke R^2; OR = odds ratio; PAC = percentage accuracy in classification; Sens = sensitivity; Spec = specificity; PPV = positive predictive value; NPV = negative predictive value; LR^+ = positive likelihood ratio; LR- = negative likelihood ratio; AUC = area under the receiver operating characteristic curve.

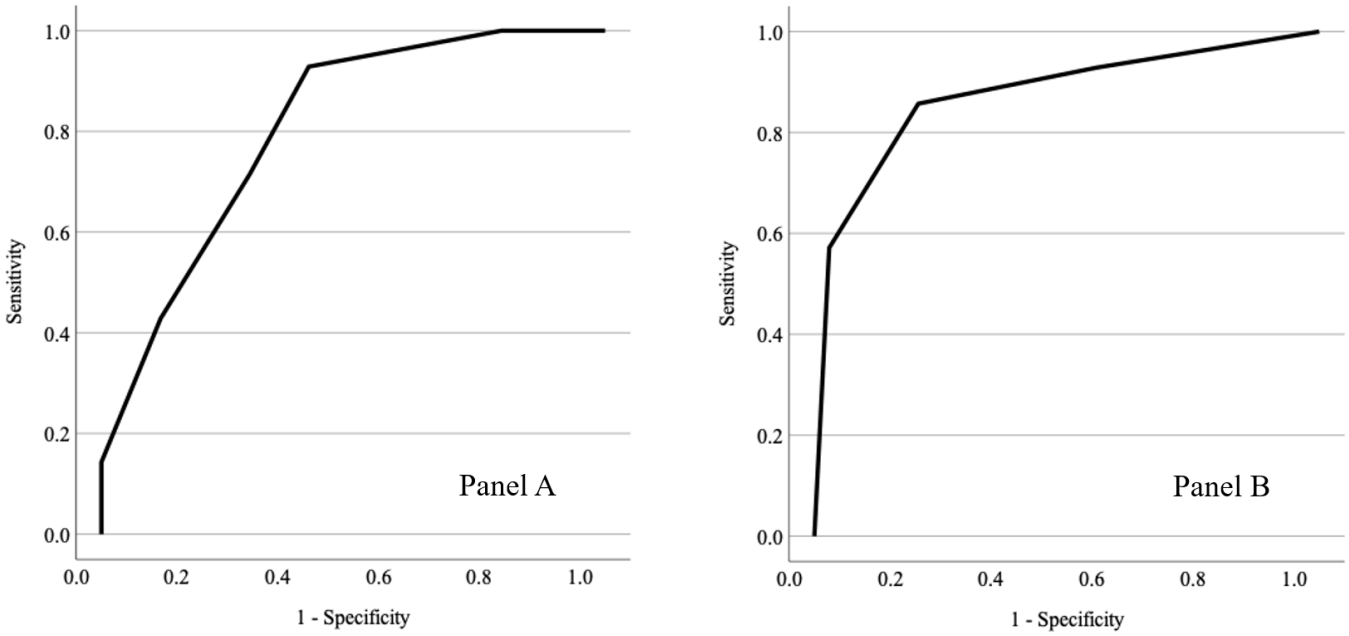

FIGURE 1. Receiver operating characteristic curve (ROC) for baseline scores on the eight-item (Panel A) and three-item (Panel B) Montreal Parkinson Risk of Dementia Scale (MoPaRDS) discriminating Parkinson's Disease with Incipient Dementia (PDID) from Parkinson's Disease with No Dementia (PDND). For the eight-item MoPaRDS, area under the ROC curve (AUC) is 0.81 (95% confidence interval [CI] = 0.68–0.93; $p = .001$). For the three-item MoPaRDS, AUC is 0.88 (95% CI = 0.68–0.93; $p = .001$).

M increase per wave = 0.23, p < .001); and (c) the rate at which scores increased did not vary across participants (i.e., participants tended to accumulate deficits at the same rate). Collectively, these results indicate that overall risk scores on both configurations of the MoPaRDS generally increased across the study duration, although the rate of increase was slightly slower for the three-item version. Change trajectories are presented in Figure 2.

DISCUSSION

The MoPaRDS[5] was developed as an inexpensive and non-invasive dementia screening tool for identifying at-risk PD patients in routine care settings. The eight constituent items represent demographic, motor, and non-motor features that are regularly evaluated in office-based care settings. Because fluid biomarkers, neuroimaging, and genetic features are not

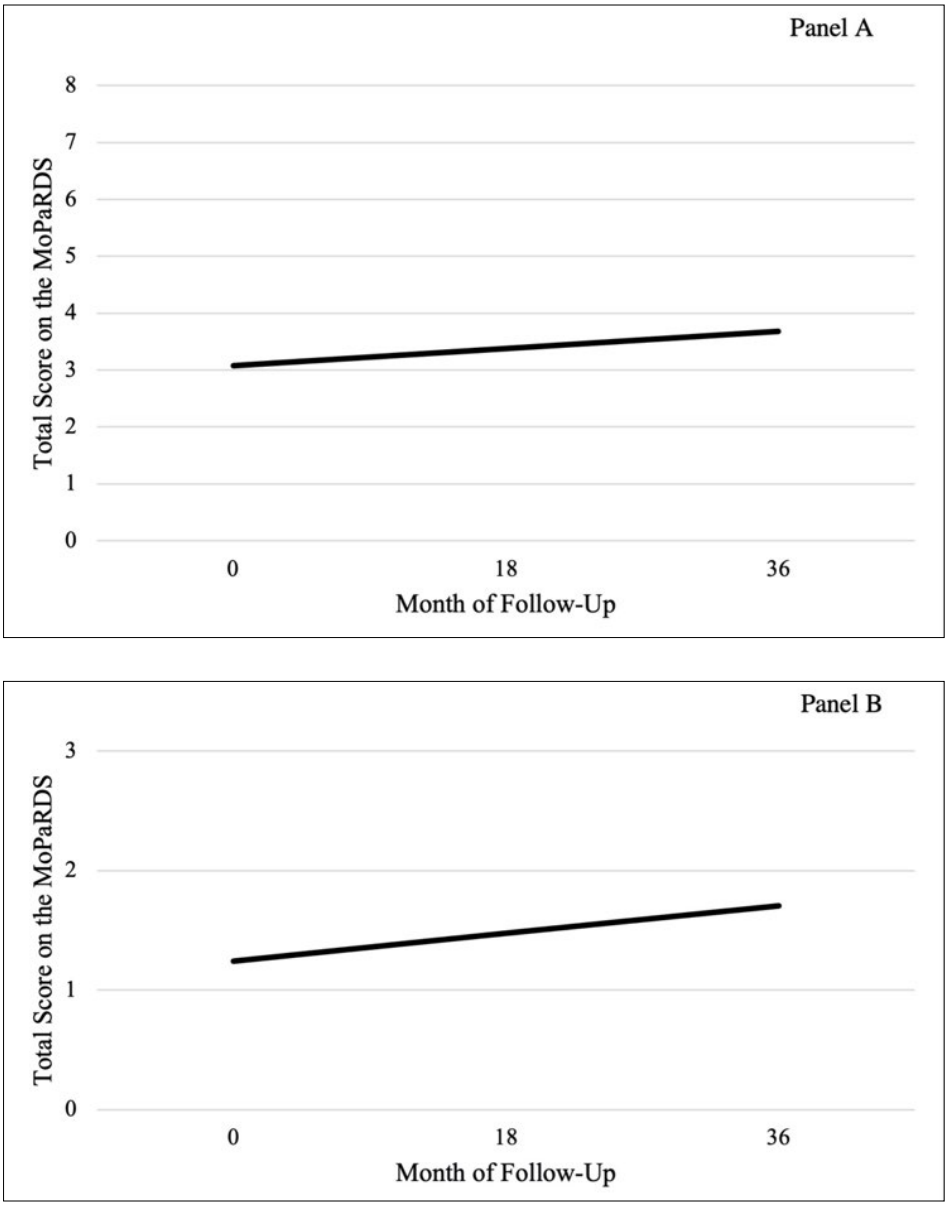

FIGURE 2. Predicted growth curve model for overall risk scores on the eight-item (Panel A) and three-item (Panel B) Montreal Parkinson Risk of Dementia Scale (MoPaRDS). Baseline overall risk scores on the eight-item MoPaRDS statistically differed across participants ($\hat{\sigma}^2 = 1.65$, $p < .001$). Overall risk scores on the eight-item MoPaRDS increased across the three waves (*M* increase per wave = 0.30, $p = .002$). Baseline overall risk scores on the three-item MoPaRDS statistically differed across participants ($\hat{\sigma}^2 = 0.70$, $p < .001$). Overall risk scores on the three-item MoPaRDS increased across the three waves (*M* increase per wave = 0.23, $p < .001$).

required, overall risk scores can be compiled within a single office visit. This follow-up validation study is the first to our knowledge to (a) address important research directions noted in the original report (i.e., integrate education into overall risk scores and evaluate longitudinal change trajectories); and (b) examine whether the MoPaRDS performs screening robustly across minor variations in constituent items and PD cohorts varying in age (geriatric), longitudinal follow-up (shorter), and geographic location (Western Canada). We assembled three waves of data for a well-characterized[6-13] geriatric PD cohort, including 48 patients who were non-demented at baseline, a subset of which (29%) had converted three years later to PDD.

A principal finding was that, for a geriatric PD cohort, the full eight-item MoPaRDS discriminated PDID from PDND (three years prior to dementia) at a level (AUC = 0.81) that was similar to that reported for the international cohort examined in the Dawson and colleagues article[5] (AUC = 0.88). These results support the key interpretations that the full MoPaRDS (a) can be clinically useful in older non-demented PD patients; and (b) is robust for minor item variations which are likely to appear across active clinics. In addition, results of the planned item-level analyses led us to a follow-up examination of a three-item configuration. In the present dataset, we observed that this brief version of the MoPaRDS also discriminated the PDID and PDND groups at an excellent level (AUC = 0.88). The three items in the brief configuration have been separately linked with concurrent PDD in other reports.[5,7,23] However, this configuration was not tested in the original report,[5] and we are unaware of any previous study describing such a brief MoPaRDS-derived screening tool to identify at-risk geriatric PD patients three years prior to the onset of clinically detectable dementia.

In an effort to validate the present novel results, we used shared data from the original 2018 study[5] (N = 607) to test whether the new three-item version predicted PDD at a comparable level to (a) that observed the present study; and (b) the full MoPaRDS in the validation data.[5] Both findings were strikingly supportive of validation. Specifically, in the validation data (a) the three-item MoPaRDS reliably predicted PDD (AUC = 0.84); and (b) this value was similar to the original full-MoPaRDS prediction (AUC = 0.88). A content inspection of the three-item version indicated that it represented a concentration of the dementia-intensive items (especially age and MCI). Because the shorter version accomplished three-year PDD prediction without representation of PD-related items in both data sets, we recommend further research testing the generalizability of this configuration in other geriatric samples. Moreover, because the third item was orthostatic hypotension, we suggest additional research on its role in elevating PDD risk.[23]

Regarding sex, the full MoPaRDS in the 2018 study[5] reported somewhat higher predictive validity for males (AUCmales = 0.92; AUCfemales = 0.81), whereas we observed that the full MoPaRDS performed substantially better for females (AUCmales = 0.91; AUCfemales = 0.74). The reasons for these divergent patterns are unclear,[24] but may reflect the comparatively older age of the current cohort.[25] This aging-related possibility merits future examination. Increased understanding of sex differences in PDD—and the early prediction of elevated risk—may allow clinicians to tailor timely intervention and prevention strategies more precisely. Importantly, the three-item MoPaRDS showed high and nearly equivalent performance for females (AUC = 0.88) and males (AUC = 0.91), suggesting that this brief version may be similarly applicable for both sexes in generally older adults.

Notably, our cut-off score results on the eight-item MoPaRDS were convergent with the original study.[5] Specifically, we found that an overall risk score ≥ 4 was the optimal cut-off for a screen positive result, yielding 71.4% sensitivity and 70.6% specificity (AUC = 0.71). The previous study also adopted this cut-off and reported 77.1% sensitivity and 87.2% specificity (AUC = 0.83). Although these findings confirm the viability of applying the full scale to a geriatric PD cohort, our findings with the three-item MoPaRDS should also be mentioned. The optimal cut-off was an overall risk score ≥ 2, which yielded 85.7% sensitivity and 79.4% specificity (AUC = 0.83), a comparable set of results.

The previous study[5] identified education as a potentially useful component of the MoPaRDS that was unavailable in the international data set. In our study, education was examined in several operational variations but did not independently or in combination demonstrate predictive validity for PDD. Although education may be associated with cognitive reserve in PD, which in turn is correlated with motor and cognitive performance,[26] cognitive reserve might affect dementia risk differentially in asymptomatic aging as compared to people already aging with PD, a substantial dementia risk factor.[27] Because relatively few studies have examined education-PDD associations (including related measures such as continuing late life learning),[18] we support future work exploring whether this factor increases prediction accuracy of the MoPaRDS.

A novel contribution of the present study is that we used longitudinal growth curve modelling techniques[21,22] to demonstrate that overall risk scores on both the full and brief MoPaRDS configurations increased over the three waves. The results of these longitudinal analyses are noteworthy, given that some items do not change over time (sex) or may be more likely to appear earlier in the clinical course of PD (orthostatic hypotension). To our knowledge, no prior studies have examined actual change trajectories of the MoPaRDS. Consequently, we propose this as a priority area of continued research attention. Findings from this line of investigation may support integrating this screening tool into routine care settings in order to monitor transitional changes in PDD risk. Early identification of at-risk individuals may allow for targeted interventions that mitigate or offset subsequent risk for PDD and the associated downstream negative effects.[2,3]

We note several methodological strengths and limitations. First, among the former, we tested our research questions using longitudinal data for an extensively characterized[6–13] geriatric cohort of initially non-demented PD patients. Second, participants completed a comprehensive testing battery at all

three waves and (a) were missing no baseline data across the constituent MoPaRDS items or education; and (b) were evaluated for both MCI[6,28,29] and PDD[6,8,10] using the prevailing clinical diagnostic criteria. Third, we verified that the new empirically indicated three-item MoPaRDS also accurately predicted PDD in the validation cohort.[5] Among study limitations is, first, the relatively small sample size. However, we note that our study was sufficiently powered to obtain reliable estimates in binary logistic regression analyses[30] and growth curve analyses,[21] and has been successful in previous biomarker prediction studies.[6–13] Second, in the present data, only three of eight individual MoPaRDS items discriminated PDID from PDND. We note that this pattern is consistent with an attribution related to the older age of this cohort and the concurrent possibility that geriatric PD patients might present alternative dementia risk profiles given age-associated comorbidities, such as white matter disease[31] and Alzheimer's co-pathology.[32] This possibility could be tested in future studies.

CONCLUSIONS

Overall, our results suggest that the recently established eight-item MoPaRDS is also a promising tool for rapidly screening for dementia risk in a geriatric PD cohort. We tested and confirmed that an abbreviated configuration (a) performed well and similarly across sex; and (b) reliably predicted PDD in the validation cohort[5] of a comparatively younger international PD patient group. Because the risk factors comprising the three-item version are not specific to PD, this abbreviated screening tool may be of relevance to other aging cohorts and has potential for broader application in busy clinical-geriatric settings. We verified that education (as variously defined) did not increase predictive validity of the MoPaRDS and that risk scores on both configurations increased over time. These results have implications not only for research purposes, but also for clinical guidance and decision-making. Early detection of PDD risk (in this case up to three years prior to diagnosis) may encourage closer medication monitoring, surveillance for medical conditions that exacerbate dementia risk, and advance care planning.[33–35] Future work could examine the generalizability of these findings across a longer duration of follow-up and in larger clinical cohorts with sample characteristics that complement the present study. In the interim, the MoPaRDS can be used judiciously in clinical geriatric settings as informative, but not definitive, regarding dementia risk in persons diagnosed with PD.

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CONFLICT OF INTEREST DISCLOSURES

We have read and understood the Canadian Geriatrics Journal's policy on conflicts of interest disclosure and declare the following interests: R. B. Postuma has received grants and personal fees from Fonds de la Recherche en Sante, the Canadian Institute of Health Research, Parkinson Canada, the Weston-Garfield Foundation, the Michael J. Fox Foundation, the Webster Foundation, and personal fees from Takeda, Roche/Prothena, Teva Neurosciences, Novartis, Biogen, Boehringer Ingelheim, Theranexus, Merck, Abbvie, Jannsen, Curasen, and Inception Sciences outside the conduct and reporting of the submitted work. The remaining authors declare no competing interests.

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APPENDIX A. Further information on growth curve analyses

Growth Curve Analyses

We established the functional form of change for the Montreal Parkinson Risk of Dementia Scale (MoPaRDS) using growth curve models.[21,22] We identified the best-fitting model by testing the following four models in sequence: (a) fixed intercept (no slope) model, which assumes that there is no variability between or within persons; (b) random intercept (no slope) model, which allows for variability across persons in overall level but assumes no intraindividual change; (c) random intercept, fixed slope model, which allows for variability across persons in level but assumes that each person changes at the same rate; and (d) random intercept, random slope model, which allows for variability across persons in both level and change.[22] We compared each successive model using the chi-square difference test. This statistic is interpreted such that positive values indicate that the less restrictive model provides a better fit to the data relative to the preceding, more restrictive model.

Model Fit

The extent to which each model provided a good fit to the data was evaluated using the following standard indices: (a) comparative fit index for which ≥ .95 was judged a good fit and between .90 and .94 was judged an adequate fit; (b) root mean square error of approximation, for which ≤ .05 would be judged as good and between .06 and .08 would be judged adequate; and (c) chi-square for which a good fit would produce a non-statistically significant result (i.e., $p > .05$; indicates that the data do not statistically differ from model-based estimates).

Missing Data

A small number of participants were missing data across a subset of the items used to calculate overall risk scores on the eight- and three-item MoPaRDS at the second (n = 6) or third wave (n = 7). Risk scores for these participants were estimated using full information maximum likelihood. We selected this approach to handling missing data based on literature indicating that generalizations from studies using full information maximum likelihood are superior to those from studies using such approaches to handling missing data as listwise or pairwise deletion.[36]

APPENDIX B. Research Goal 1: Binary logistic regression models for the constituent MoPaRDS items independently discriminating PDID from PDND

Item	*OR*	*p value*	*95% CI*	*AUC*	*p value*	*95% CI*
Male sex	0.46	.46	[0.13–1.64]	0.41	.31	[0.23–0.58]
Age > 70	5.92	.02	[1.38–25.30]	0.70	.03	[0.54–0.86]
Mild cognitive impairment	37.89	< .001	[6.64–216.27]	0.85	< .001	[0.71–0.99]
Bilateral disease onset	2.78	.12	[0.76–10.15]	0.62	.20	[0.44–0.80]
RBD	0.41	.17	[0.12–1.46]	0.39	.24	[0.21–0.57]
Hallucinations	1.23	.87	[0.10–14.78]	0.51	.95	[0.32–0.69]
Falls and/or freezing	3.50	.08	[0.88–13.88]	0.63	.17	[0.44–0.81]
Orthostatic BP drop	7.67	.01	[1.77–33.18]	0.73	.01	[0.57–0.89]
Education < 14 years[a]	0.70	.59	[0.20–2.55]	0.46	.65	[0.28–0.64]

[a] Findings stemming from follow-up analyses exploring (a) continuous education; and (b) alternate cut-points are presented in the Results section.
MoPaRDS = Montreal Parkinson Risk of Dementia Scale; PDID = Parkinson's Disease with Incipient Dementia; PDND = Parkinson's Disease with No Dementia; OR = odds ratio; CI = confidence interval; AUC = area under the receiver operating characteristic curve; RBD = rapid eye movement sleep behaviour disorder; BP = blood pressure drop > 10 mmHg. Each item was scored dichotomously (0 = deficit absent, 1 = deficit present) and tested as an independent predictor in separate binary logistic regression analyses (i.e., we serially tested each item for prediction of the binary outcome PDID, PDND).

APPENDIX C. Research Goal 5: Goodness-of-fit indices fo MoPaRDS growth curve models

Model	*AIC*	*BIC*	*CFI*	*RMSEA*	χ^2 *(df)*	*D*	*Δdf*
8-item MoPaRDS							
Fixed intercept, no slope	500.97	508.46	0	0.49	62.69 (5)[c]	--	--
Random intercept, no slope	452.73	462.09	0.85	0.21	12.45 (4)[d]	50.24[c]	1
Random intercept, fixed slope[a]	446.52	457.75	0.98	0.09	4.24 (3)	8.21[d]	1
Random intercept, random slope[b]	--	--	--	--	--	--	--
3-item MoPaRDS							
Fixed intercept, no slope	389.70	397.18	0	0.50	65.45 (5)[c]	--	--
Random intercept, no slope	339.77	349.12	0.84	0.22	13.54 (4)[d]	51.91[c]	1
Random intercept, fixed slope[a]	329.24	340.46	1.00	0	1.01 (3)	12.53[c]	1
Random intercept, random slope[b]	--	--	--	--	--	--	--

[a]Best fitting model.
[b]This model was not considered due to a not positive definite covariance matrix.
[c]*p* value < .001.
[d]*p* value < .01.
MoPaRDS = Montreal Parkinson Risk of Dementia Scale; χ^2 = chi-square test of model fit; df = degrees of freedom; AIC = Akaike information criterion; BIC = Bayesian information criterion; CFI = comparative fit index; RMSEA = root mean square error of approximation; *D* = chi-square difference test; Δ*df* = change in degrees of freedom.