

**Updated Criteria for the Diagnostic Procedure for Parkinson's Disease Dementia on
Level I and their Validity in Deep Brain Stimulation Cohort**

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Introduction

Parkinson's disease (PD) is a neurodegenerative disorder characterized by a gradual and progressive onset of motor symptoms, including rigidity, bradykinesia, and resting tremors, which eventually extend to both motor and non-motor impairments ([Postuma et al., 2015](#)). Beyond these hallmark features, cognitive decline is a critical aspect of the disease trajectory, culminating in Parkinson's disease dementia (PDD) in a substantial subset of patients ([Meireles & Massano, 2012](#)).

The diagnostic criteria for PDD ([Dubois et al., 2007](#)), first formalized in 2006, were heavily influenced by frameworks established for Alzheimer's disease (AD) owing to the absence of PD-specific biomarkers that could facilitate a biologically grounded diagnostic system. Although these criteria provided a valuable initial foundation, they lacked the specificity required to capture the distinct pathophysiological and cognitive features of PD-related dementia ([Emre et al., 2007](#); [Yamashita et al., 2023](#)).

An important feature of the original criteria was the provision of an algorithm that allowed for flexibility in test selection ([Dubois et al., 2007](#)). Specifically, clinicians could choose between months reversed or seven backwards for attention assessment, lexical fluency or clock drawing for executive function evaluation, MMSE pentagons for visuospatial ability, and three-word recall for memory assessment. The availability of multiple operationalization options for PDD enhances the ability to examine the psychometric properties of the construct. Agreement across different criteria allows for the parallel computation of inter-rater reliability, which, in turn, facilitates the calculation of construct validity ([Conway et al., 1995](#)) further strengthening the diagnostic framework for PDD.

Subsequent advancements have introduced the concept of Parkinson's disease mild cognitive impairment (PD-MCI), refining the understanding of cognitive dysfunction in PD ([Litvan et al., 2012](#)). The PD-MCI criteria, published in 2012, have propelled progress in diverse areas, enabling improvements in clinical characterization, identification of genetic correlates, therapeutic interventions, clinical trial design, and the assessment of progression

risk to PDD ([Aarsland et al., 2021](#); [Hoogland et al., 2017, 2019](#)).

Currently, efforts are focused on refining the PDD diagnostic framework to improve its consistency and applicability in both research and clinical contexts across multiple centers ([Kulisevsky et al., 2024](#)). Our prior investigations have underscored the importance of employing rigorous psychometric methodologies to differentiate PD-MCI from PDD, particularly given that a diagnosis of PDD remains a contraindication for deep brain stimulation (DBS) ([Bezdicek et al., 2016](#); [Deuschl et al., 2006](#)).

The present study evaluates the diagnostic concordance between the original Level I PDD criteria, as established by the Movement Disorder Society (MDS) Task Force ([Dubois et al., 2007](#); [Emre et al., 2007](#)) and criteria inspired by the recent call for change ([Kulisevsky et al., 2024](#)) within a PD cohort selected for DBS. Furthermore, both sets of criteria are compared to PDD diagnosed on Level II. The study aims to address following research objectives (RO): 1) to estimate the prevalence of PDD among patients considered for DBS (RO1), 2) to assess variability in PDD diagnosis depending on the diagnostic criteria applied (RO2), 3) to evaluate the diagnostic concordance between different sets of PDD criteria (RO3), and 4) to identify diagnostic components that contribute to variability in PDD classification across criteria (RO4). By addressing these objectives, this study seeks to validate the revised PDD criteria and evaluate their relevance in the context of DBS eligibility, thereby contributing to the refinement of cognitive assessment protocols in PD.

Methods

Participants

This study retrospectively analyzed clinical data from a cohort of patients with PD considered for treatment via DBS of subthalamic nucleus at the General University Hospital in Prague. All patients were diagnosed with idiopathic PD by a movement disorder specialist according to the MDS Clinical Diagnostic Criteria for PD ([Postuma et al., 2015](#)). Clinical records spanning August 2014 to February 2025 were examined. All participants underwent neuropsychological evaluation conducted by a trained clinical psychologist as part of standard preoperative cognitive assessments for DBS eligibility at the General University Hospital in Prague.

Ethical approval for the study protocol was obtained from the Ethics Committee of the General University Hospital in Prague. Informed consent was secured from all patients prior to their neuropsychological assessments, in adherence to ethical research guidelines.

Neuropsychological Assessment

Cognitive performance was evaluated at both Level I (abbreviated assessment) and Level II (comprehensive assessment) according to the standard MDS neuropsychology battery for Parkinson's Disease Mild Cognitive Impairment (PD-MCI) Bezdicek et al. (2017). Level I was assessed using the Mini-Mental State Examination (MMSE) (Folstein et al., 1975; Stepankova et al., 2015) and the Montreal Cognitive Assessment (MoCA) (Kopecek et al., 2016; Nasreddine et al., 2005), both of which provide measures of global cognitive functioning. The neuropsychological assessment at Level II covered five cognitive domains, each evaluated through specific tests as follows: attention and working memory assessed using Trail Making Test Part A (TMT-A) (Bezdicek et al., 2012; Reitan, 2004), Prague Stroop Test – Dots (PST-D) (?), Letter Number Sequencing (LNS) (Wechsler, 1997), WAIS Digit Span Backward (WAIS DSB) (Wechsler, 1997), and WAIS Corsi Block Backward (WAIS CB) (Wechsler, 1997); executive function evaluated via the Tower of London (ToL) (“Specific Impairments of Planning,” 1982), Categorical Verbal Fluency (CF) (Benton et al., 1989), Trail Making Test Part B (TMT-B) (Reitan, 2004), subtests from the Prague Stroop Test – Words (PST-W) and Colors (PST-C) (Bezdiček et al., 2021); language measured with the WAIS Similarities subtest (Wechsler, 1997) and the Boston Naming Test (BNT-60) (Kaplan et al., 1983); memory examined using the Rey Auditory Verbal Learning Test (RAVLT) (Frydrychová et al., 2018; Rey, 1964) for immediate and delayed recall (including List B and recognition trials), and the Brief Visual Memory Test–Revised (BVMTR) (Benedict, 1997; Havlík et al., 2020), including delayed recall and forced choice recognition; visuospatial function assessed through the Judgment of Line Orientation Test (JoLO) (Benton et al., 1983), Clock Drawing Test (CLOX) (Royall et al., 1998), and the Grooved Pegboard Test (GPT) (Klove, 1963).

In addition to the core cognitive assessments, tasks such as the Clock Drawing Test (CDT) and Letter Fluency tasks were included to capture domain-specific impairments. The

classification of Parkinson's Disease Dementia (PDD) based on Level I criteria was determined using established scoring thresholds from Dubois et al. (Dubois et al., 2007) with corresponding MoCA equivalents.

To assess functional impairment, the Functional Activities Questionnaire (FAQ)(Bezdiček et al., 2011; Pfeffer et al., 1982) was administered. Additionally, neuropsychiatric status was evaluated using the Beck Depression Inventory-II (BDI) (Beck et al., 1996; Ciharova et al., 2020) and State-Trait Anxiety Inventory (STAI) (Mullner et al., 1980; Spielberger et al., 1983). Psychotic symptoms were assessed through structured psychiatric interviews conducted by a trained psychiatrist.

Theoretical and Empirical Estimands

Following the framework proposed by Lundberg et al. (2021), this section connects our research objectives and their corresponding theoretical (i.e., targets of inference) and empirical (i.e., data-driven) estimands. The theoretical estimand refers to a unit-specific quantity defined over a target population and represents the ideal quantity that would address the research question under optimal conditions—such as access to complete population data or perfect experimental control. In contrast, the empirical estimand corresponds to the quantity that is actually computable using the available dataset, given real-world constraints. In our study, the target population for all theoretical estimands is defined as individuals diagnosed with Parkinson's disease (PD) who are potential candidates for deep brain stimulation (DBS) treatment.

The theoretical estimand corresponding to the RO1 is the true prevalence of probable PDD in this population. Empirically, this is estimated by calculating the proportion of patients classified as having probable PDD according to each of the diagnostic criteria under consideration.

For the RO2, the theoretical estimand is the variance in prevalence attributable to the diagnostic process itself—that is, the extent to which different sets of criteria yield differing prevalence estimates for the same population. This variability is empirically quantified by comparing the distribution of PDD classifications across criteria within the cohort.

The RO3 concerns diagnostic concordance. Here, the theoretical estimand is a set of

population-level contingency tables comparing PDD classifications assigned by each pair of diagnostic criteria, along with derived metrics such as sensitivity, specificity, and kappa coefficients. The corresponding empirical estimands are represented by matrices generated through pairwise Receiver Operating Characteristic (ROC) curve analyses that evaluate the discriminatory performance of each diagnostic set.

Finally, for the RO4, the theoretical estimand is defined as the set of diagnostic components whose variation systematically alters the probability of a probable PDD diagnosis. This aspect of the study is exploratory in nature. Empirically, we assess the contribution of each diagnostic feature by examining how variations in operational definitions (e.g., domain-specific thresholds, criteria for functional impairment) influence the empirical estimands derived for the first three objectives. This allows us to identify the diagnostic elements most responsible for inter-criterion discrepancies. We approach this RO from an exploratory point of view and evaluate the importance of each PDD criteria component by observing change in empirical estimands for RO1-3 when stratified by different operationalization decisions.

Operationalization of Parkinson's Disease Dementia

In this study, we applied three distinct sets of diagnostic criteria for **probable**^[1] PDD at Level I. The first set was based on the original framework ([Dubois et al., 2007](#)), which utilized the Mini-Mental State Examination (MMSE) as a global cognitive screening tool, supplemented by assessments of attention, executive function, visuospatial abilities, and memory. The second set of criteria was drawn from the recent call for change of dementia diagnostic guidelines ([Kulisevsky et al., 2024](#)), which advocate for more sensitive cognitive domain assessments in the context of Parkinson's disease (PD). This updated approach incorporated specific items from the Montreal Cognitive Assessment (MoCA) to better detect PD-related dementia. The third approach applied the Czech version of the shortened Montreal Cognitive Assessment (sMoCA) ([Bezdicek et al., 2020](#)), a time-efficient modification designed to ascertain whether equivalent cognitive impairments could be reliably identified using a reduced testing protocol. Lastly, the fourth approach followed the Level II battery protocol, which is commonly used in the evaluation of PD-MCI. **The Level II methodology,**

including the use of a regression-based normative scoring approach, has been detailed in a prior study (Bezdicek et al., 2017). Refer to Table 1¹ to a summary of the components and scoring thresholds of each diagnostic criterion. All non-cognitive criteria of probable PDD (i.e., diagnosis of PD that developed before dementia and absence of Major Depression, delirium or other abnormalities that obscure diagnosis were established by an independent neurological and psychiatric assessments and held true for all patients in the sample).

«««< HEAD [^1]: JM: We should unite the terminology. Most importantly, I reckon, we should differentiate between “probable PDD” (what is implied by meeting all the criteria within an operationalization) and “PDD” (the latent state of patient’s cognition); and to use consistently the following trifecta - “criterion,” “operationalization” and their “component”. =====²: JM: We should unite the terminology. Most importantly, I reckon, we should differentiate between “probable PDD” (what is implied by meeting all the criteria within an operationalization) and “PDD” (the latent state of patient’s cognition); and to use consistently the following trifecta - “criterion,” “operationalization” and their “component”. »»»»>

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[Insert Table 1 here]

For each of these diagnostic approaches, we applied two operationalization strategies based on deficits in Instrumental Activities of Daily Living (IADL). First, we utilized FAQ item 9, which approximates the pill questionnaire from the original criteria (Dubois et al., 2007) employing a cut-off score of 2 points or higher. Second, we applied the entire Functional Activities Questionnaire (FAQ) as suggested in the call for change (Kulisevsky et al., 2024), employing a cut-off score of 7 points or higher based on Czech normative data (Bezďček et al., 2011). These methodologies resulted in a total of 66 operationalizations, which were distributed across different diagnostic criteria: 4 MMSE-based, 60 MoCA-based, 2 sMoCA-based, and 0 based on the Level II battery³.

¹ JM: Placeholder until we make the table proper.

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Statistical Analyses

For sample description, we summarized continuous variables using mean, standard deviation, median, minimum, and maximum values. Categorical variables were summarized by the number of patients in each category. To address study objectives, we started by repeatedly assigning each patient the diagnosis of probable PDD based on each PDD operationalization listed in Table 1 resulting in a 204 (patients) \times 66 (operationalizations) matrix where each cell indicates whether a patient (row) meets criteria for probable PDD according to an operationalization (column). PDD prevalence estimates were then computed as $\frac{N_{PDD}}{N_{total}}$ separately for each operationalization to address RO1.

To address RO2-4, a set of two class cross-tabulations with associated statistics was computed for each pair of operationalizations via the `confusionMatrix()` function from the R package *caret* (Kuhn, 2008). For each pair of operationalizations, the analysis was repeated twice such that each variables of the pair served once as the reference and once as the predictor. Following measures were used to evaluate pairwise concordance between different operationalizations of PDD criteria: 1) Cohen's κ with its 95% confidence interval (CI) computed via the `cohen.kappa()` function from the R package *psych* (William Revelle, 2024); 2) Accuracy (i.e., the proportion of correct predictions, both true positives and true negatives, among the total number of cases) with its 95% CI; 3) Sensitivity/Recall (i.e., the proportion of true positives); and 4) Specificity (i.e., the proportion of true negatives).⁴

Finally, the No Information Rate (NIR) was calculated for each pair of operationalizations. NIR is the accuracy that could be obtained by always predicting the majority class and in our case it is equivalent to the complement of the PDD prevalence estimate according to the reference operationalization. Accuracy of prediction was subsequently compared to the NIR via a one-sided Exact Binomial Test as implemented by the

⁴ Unlike Cohen's κ , Accuracy, Sensitivity and Specificity are not symmetrical, i.e., their value depend on which variable is considered reference and which is considered predictor. Consequently, we report these values twice for each pair of operationalizations. Note that the Sensitivity of a reference/predictor pair corresponds to the Positive Predictive Value if their roles were reversed. The same relationship holds true between the Specificity and the Negative Predictive Value.

`binom.test()` R stats function. Reference/predictor pairs associated with $p < .05$ were considered to show significantly better accuracy than NIR. In other word, for reference/predictor pairs associated with $p < .05$, we conclude that knowing the probable PDD status according to the predictor operationalization helps to estimate the probable PDD status according to the reference operationalization and the two opeartionalizations thus show substantial concordance.

Data wrangling and visualizations were done in the *tidyverse* package (Wickham et al., 2019) and tables were formatted in the *gt* package (Iannone et al., 2024). All analyses were conducted with the R (version 4.4.1) software environment for statistical computing (R Core Team, 2024). The software code supporting this article is available at <https://github.com/josefmana/DemCr1t.git>.⁵

Results

Sample Description

A total of 204 patients were included. Demographic and clinical characteristics of the sample are summarized in Table 2. ...⁶

[Insert Table 2 here]

Prevalence Estimates

Operationalization-wise prevalence estimates are presented in Table 3. On average, estimated prevalence was 6.00% (SD = 3.45, Md = 3.94, range 2.00-16.75). Notably, the prevalence estimate was substantially lower when FAQ item 9 was used as a criterion of IADL deficit (M = 3.09%, SD = 0.48, range 2.00-3.94) compared to using total FAQ score criterion (M = 8.90%, SD = 2.56, range 3.50-16.75) as demonstrated also in Figure 1.⁷

[Insert Table 3 and Figure 1⁸ here]

⁵ JM: Do not forget to make it public before submitted!

⁶ JM: Give some brief impressions from the Table 2 here. E.g., evaluate sex proportion, age, (we are missing education for some reason) etc. with general PD population (e.g., from some meta-analysis). Maybe say a word or two about mean cognitive profile or its spread.

⁷ JM: For oneself - work on the code here, it ain't good.

⁸ JM: For thyself - add description.

Criteria Concordance

Results of the analyses of prediction Accuracy, Cohen's κ , Sensitivity and Specificity are presented in Figure 2, Figure A1, Figure A2 and Figure A3 respectively. Numerical results are available ...⁹

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[Insert Figure 2¹⁰ here]

Discussion

This study evaluated the applicability and validity of diagnostic frameworks for diagnosing probable PDD within a cohort of patients considered for deep brain stimulation (DBS). Our results demonstrate that diagnostic outcomes are markedly influenced by the chosen type of operationalization, particularly in relation to the assessment of the cognitive domains and IADLs.

Variability in Prevalence Estimates

One of the key findings of this study was the broad range of estimated PDD prevalence depending on the operationalization strategy. As seen in Table 3, prevalence estimates varied from 2.00% to 16.75%, with the highest being derived from a combination of the sMoCA and the total FAQ score criterion. When only FAQ item 9 was used to determine IADL deficits, the prevalence estimates were significantly lower ($M = 3.09\%$, $SD = 0.48$), underscoring significant influence of functional assessment choice on diagnostic outcomes.

Cognitive and Clinical Context

The relatively low prevalence of PDD across operationalizations may also reflect the relatively preserved cognitive profile of the DBS cohort, as evident in Table 2. The mean MoCA ($M = 24.07$, $SD = 3.48$) and MMSE ($M = 26.69$, $SD = 2.22$) scores suggest that the majority of patients were functioning at a globally intact level. This likely reflects the

⁹ JM: ... as some kind of Supplementary Table, ideally html but Excel file would work as well.

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pre-selection of cognitively preserved individuals for DBS, in line with standard eligibility criteria (?).

Additionally, memory performance metrics, such as RAVLT delayed recall ($M = 7.16/15$), and BVMTR delayed recall ($M = 7.89/12$), also possibly indicate generally mild impairments in the domain crucial for distinguishing PD-MCI from PDD. This aligns with previous literature suggesting that Level I criteria may overestimate dementia prevalence in populations with only subtle deficits if not carefully calibrated ([Aarsland et al., 2021](#); [Bezdicek et al., 2016](#)).

Implications for DBS Eligibility and Clinical Practice

Our findings also bear direct implications for clinical decision-making, especially in the context of surgical candidacy for DBS. Given that PDD remains a contraindication for DBS, the observed diagnostic instability could result in disparate treatment decisions based solely on which cognitive criteria are employed. Thus, harmonization of assessment procedures and operational cutoffs is essential to ensure equitable access to surgical treatment while maintaining diagnostic rigor.

Moreover, the relatively high variability in diagnostic outputs calls for the incorporation of multidimensional tools, such as comprehensive neuropsychological batteries (Level II) or standardized regression-based normative approaches, as confirmatory steps in ambiguous cases. Such practices may mitigate false positives and enhance the robustness of Level I screening.

Constraints on generalizability

This study is limited by its retrospective nature and the homogenous DBS candidate cohort, which may reduce generalizability to more or less cognitively impaired PD populations. Moreover, the use of a Czech-specific normative framework may limit cross-cultural applicability, though it reflects the clinical reality of local practices (?).

Future Directions

Future research should focus on prospective validation of updated Level I criteria against longitudinal functional outcomes and neurobiological markers. Additionally, the development of adaptive diagnostic algorithms that can integrate performance across

cognitive, functional, and psychiatric domains may enhance diagnostic sensitivity while reducing the risk of over-classification.

Conclusions

This study systematically evaluated various Level I diagnostic criteria for PDD and the operationalizations in a DBS candidate cohort. Our findings underscore the significant variability in PDD prevalence estimates depending on both the selected cognitive tests and the thresholds applied to define functional impairment. The divergence observed across operationalizations demonstrates the sensitivity of diagnostic outcomes to seemingly minor methodological choices.

While the call for a change (Kulisevsky et al., 2024) criteria offer enhanced sensitivity by leveraging MoCA-based components and broader IADL assessments, the use must be cautiously calibrated to prevent over-diagnosis in populations with mild or borderline cognitive deficits. Conversely, overly conservative criteria, such as reliance on pill questionnaire (i.e. FAQ item 9 equivalence) may fail to detect meaningful functional decline and thus under-identify true cases of probable PDD.

Ultimately, this study contributes to the improving landscape of PDD diagnostics by offering empirical evidence for the refinement of Level I criteria and reinforcing the value of psychometric rigor in clinical neuropsychology. Future work should extend this validation to longitudinal trajectories and integrate neurobiological correlates, ensuring that cognitive criteria remain both scientifically grounded and clinically actionable.

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Table 1

group	type	glob	glob_t	atte	atte_t	exec	exec_t	memo	memo_t
mmse	mmse1	mmse	26	mmse_7	4	cloc	2	mmse_3words	3
mmse	mmse2	mmse	26	mmse_7	4	cloc	2	mmse_3words	3
mmse	mmse3	mmse	26	mmse_7	4	vf_s	10	mmse_3words	3
mmse	mmse4	mmse	26	mmse_7	4	vf_s	10	mmse_3words	3
moca	moca1	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	1
moca	moca2	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	1
moca	moca3	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	1
moca	moca4	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	1
moca	moca5	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	2
moca	moca6	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	2
moca	moca7	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	2
moca	moca8	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	2
moca	moca9	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	3
moca	moca10	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	3
moca	moca11	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	3
moca	moca12	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	3
moca	moca13	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	4
moca	moca14	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	4
moca	moca15	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	4
moca	moca16	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	4
moca	moca17	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	5
moca	moca18	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	5
moca	moca19	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	5
moca	moca20	moca_total	26	moca_7	3	moca_cloc	2	moca_5words	5
moca	moca21	moca_total	26	moca_7	3	vf_k	11	moca_5words	1
moca	moca22	moca_total	26	moca_7	3	vf_k	11	moca_5words	1
moca	moca23	moca_total	26	moca_7	3	vf_k	11	moca_5words	1
moca	moca24	moca_total	26	moca_7	3	vf_k	11	moca_5words	1
moca	moca25	moca_total	26	moca_7	3	vf_k	11	moca_5words	2
moca	moca26	moca_total	26	moca_7	3	vf_k	11	moca_5words	2

Table 2

Table 2 Sample description. Demographic, clinical and cognitive characteristics of the sample.

	N	Md	Min-max	M	S
Demographics					
Sex of Participants (Male, Female)	126 (62%)	-	-	-	-
Age of Participants (in Years)	203	60	34-73	58.96	8.3
Clinical					
Type of PD: Akinetic-rigid, Tremor-dominant; Axial	32/107	-	-	-	-
Hoehn Yahr stage (range 0-5)	2/8/66/33/14/2	-	-	-	-
PD duration (in years)	162	10	1-25	10.71	4.3
asym_park	66 (51%)	-	-	-	-
L-DOPA (in mg)	138	1607	0-4138	1697.58	670
UPDRS III off state	135	35	10-81	37.43	13.5
UPDRS III on state	135	14	2-45	15.97	8.3
MMSE					
MMSE (range 0-30)	203	27	15-30	26.69	2.3
MMSE 7 (range 0-5)	1/2/8/20/34/139	-	-	-	-
VF S (number of words per minute)	202	15	1-34	14.95	5.3
Clock Drawing (range 0-2)	26/91/86	-	-	-	-
MMSE pentagons (range 0-1)	187 (92%)	-	-	-	-
MMSE (range 0-3)	5/14/60/124	-	-	-	-
MoCA					
MoCA (range 0-30)	203	24	9-30	24.07	3.4
sMoCA (range 0-16)	203	11	1-16	11.26	2.3
MoCA 7 (range 0-3)	1/2/29/171	-	-	-	-
VF K (number of words per minute)	204	16	0-29	15.50	5.3
MoCA (range 0-3)	24/83/96	-	-	-	-
MoCA (range 0-1)	164 (81%)	-	-	-	-

Table 3

Table 3 Estimates of prevalence. Estimates of the prevalence of probable PD-D in the sample.

				Impaired cog
Global deficit	Attention	Executive function		
sMoCA (range 0-16) < 13	-	-		
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 2		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	MoCA (range 0-3) < 3		MoC
MoCA (range 0-30) < 26	MoCA 7 (range 0-3) < 3	VF K (number of words per minute) < 11		MoC

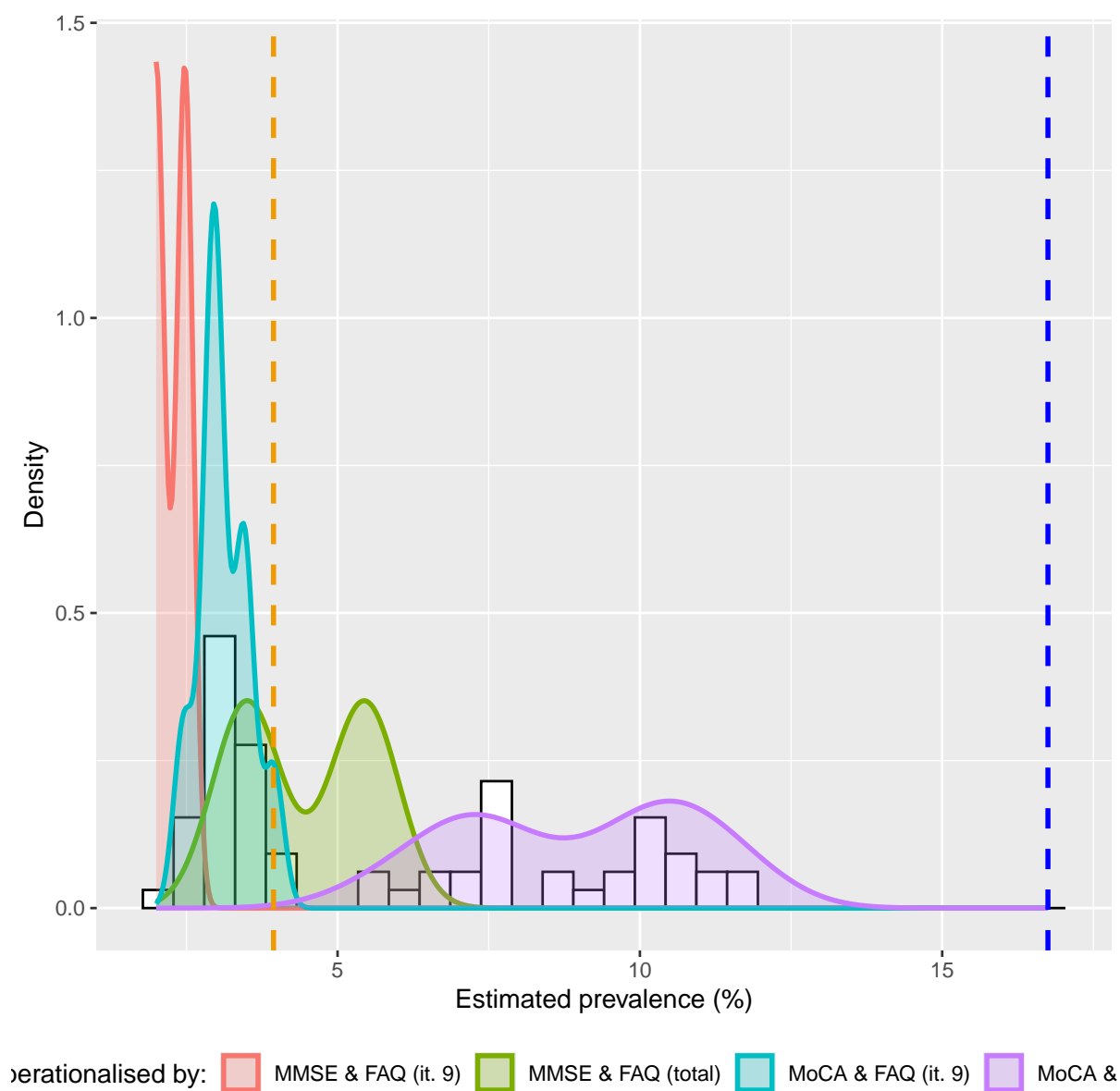
Figure 1

Figure 1 is a heatmap illustrating the relationship between Reference variables (rows) and Predictor variables (columns). The Reference variables are listed on the left, and the Predictor variables are listed at the bottom. The heatmap is color-coded: red for high values, yellow for medium values, and green for low values. The top-left quadrant (Reference vs. Predictor) shows a strong positive correlation (red). The bottom-right quadrant (Predictor vs. Predictor) shows a strong negative correlation (green). The top-right and bottom-left quadrants show weak or no correlation (yellow).

Figure A1

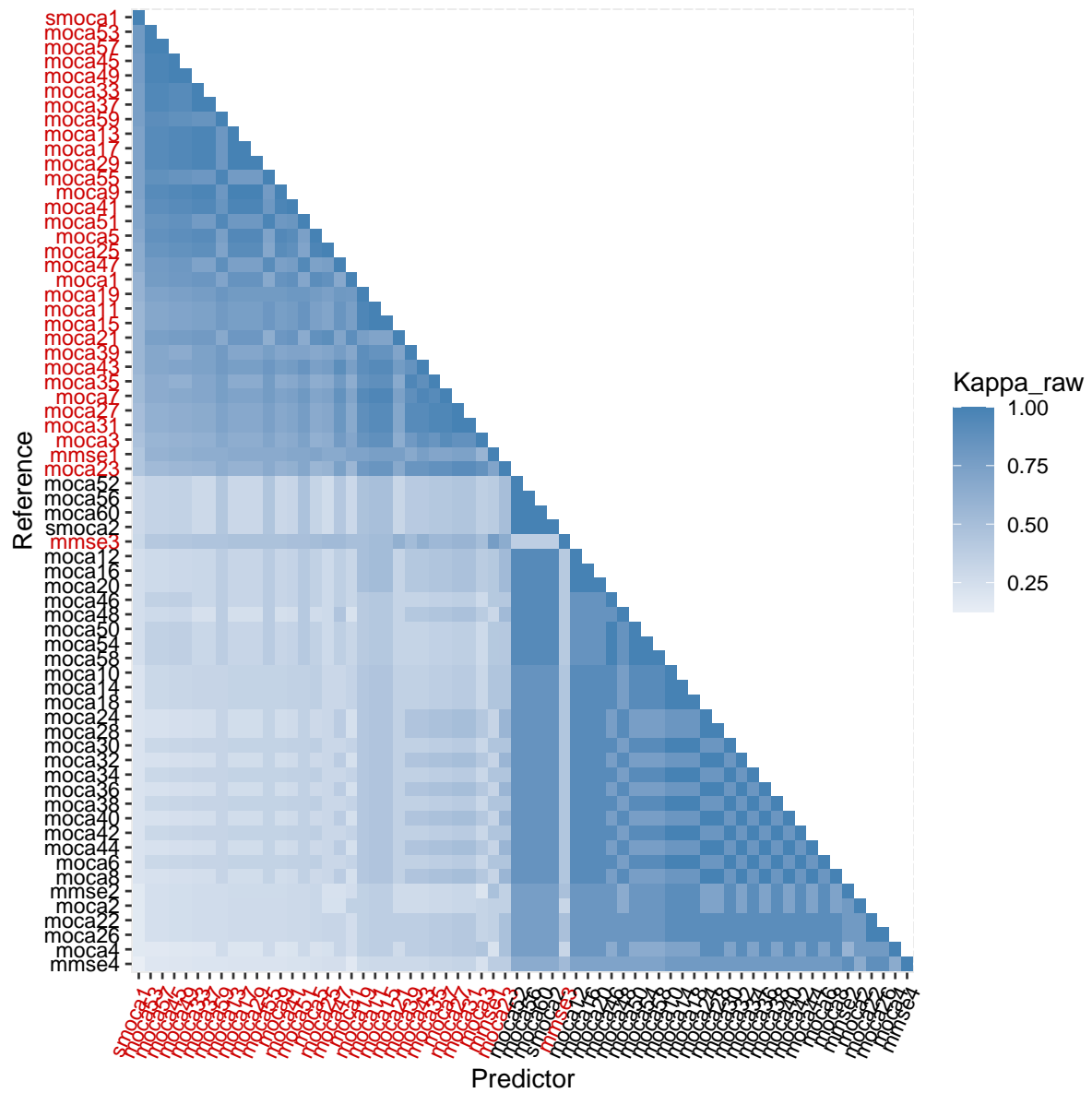


Figure A2

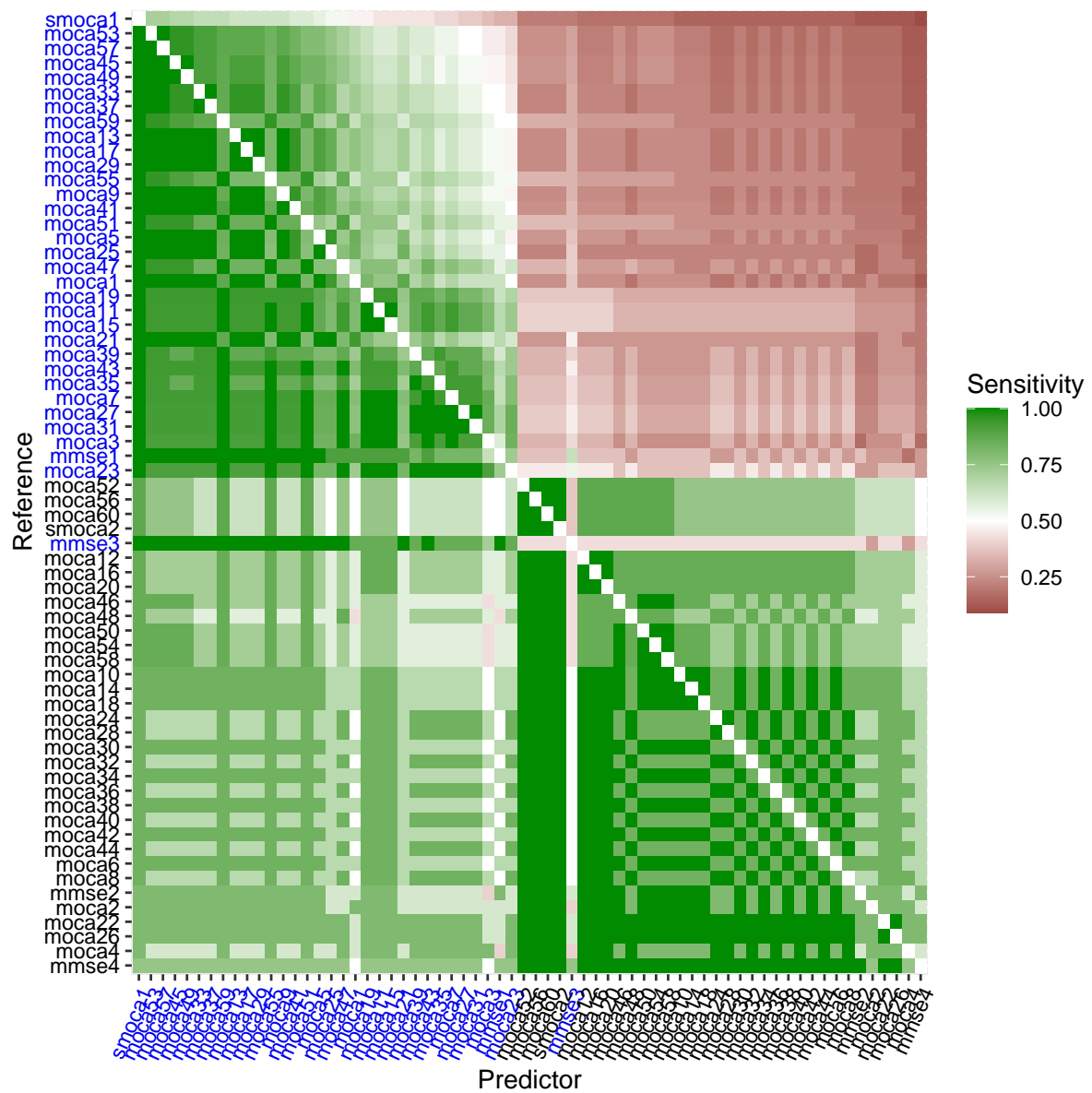


Figure A3

