

Validation of Conversion Between Mini-Mental State Examination and Montreal Cognitive Assessment

Michael Lawton, MPhil, MSc,^{1*} Meike Kasten, MD,² Margaret T. May, PhD, MSc, MA,¹ Brit Mollenhauer, MD,^{3,4} Martina Schaumburg, MSc,³ Inga Liepelt-Scarfone, PhD,⁵ Walter Maetzler, MD,^{5,6} Eva-Juliane Vollstedt, MD,² Michele T.M. Hu, MBBS, FRCP, PhD,^{7,8} Daniela Berg, MD^{5,6} and Yoav Ben-Shlomo, MB, BS, PhD, FFPH¹

¹School of Social and Community Medicine, University of Bristol, Bristol, United Kingdom ²Department of Psychiatry and Psychotherapy and Institute of Neurogenetics, University of Lübeck, Lübeck, Germany ³Paracelsus-Elena-Klinik, Kassel, Germany ⁴Departments of Neurosurgery and Neuropathology, University Medical Center Göttingen, Germany ⁵German Center for Neurodegenerative Diseases (DZNE), Tuebingen, Germany ⁶Department of Neurodegeneration, Hertie Institute for Clinical Brain Research (HIH), University of Tuebingen, Tuebingen, Germany ⁷Nuffield Department of Clinical Neurosciences, Division of Clinical Neurology, University of Oxford, United Kingdom ⁸Oxford Parkinson's Disease Center, University of Oxford, Oxford, United Kingdom

ABSTRACT

Introduction: Harmonizing data across cohorts is important for validating findings or combining data in meta-analyses. We replicate and validate a previous conversion of MoCA to MMSE in PD.

Methods: We used five studies with 1,161 PD individuals and 2,091 observations measured with both the MoCA and MMSE. We compared a previously published conversion table using equipercentile equating with log-linear smoothing to our internally derived scores.

Results: Both conversions found good agreement within and across the studies when comparing true and converted MMSE (mean difference: 0.05; standard deviation: 1.84; median difference: 0; interquartile range: -1 to 1, using internal conversion).

Conclusions: These results show that one can get a reliable and valid conversion between two commonly used measures of cognition in PD studies. These approaches need to be applied to other scales and domains to enable large-scale collaborative analyses across multiple PD cohorts. © 2016 International Parkinson and Movement Disorder Society

Key Words: Parkinson's disease, Mini-Mental State Examination, Montreal Cognitive Assessment, equating

There are many studies of individuals with Parkinson's disease (PD), although often data are limited by

selected samples and small sample sizes meaning that analyses are underpowered. Researchers often seek to validate previous research in a new data set or combine data in a meta-analysis, but comparing and pooling findings may be problematic if different measures have been used. A common nonmotor feature of PD is cognitive impairment, and two of the most popular screening tools are the Mini-Mental State Examination (MMSE)¹ and the Montreal Cognitive Assessment (MoCA).² There are well-established methods for scale conversion³ and a previous study⁴ has applied these to convert the MoCA to the MMSE in patients with PD. The validity of this conversion has been evaluated in a small sample of 139 subjects with PD, with a narrow distribution of MoCA scores, which suggested it was reasonably good.⁵ We present both a replication of the methods of van Steenoven and colleagues and a validation of their conversion chart in a much larger independent sample including individuals with a wider range of MoCA and MMSE scores.

Patients and Methods Study Population

We used data from five studies that are a part of the Joint Programme Neurodegenerative Disease (JPND) consortium who have data collected on both the MMSE and MoCA in patients with PD. In all cases, the MoCA scores were adjusted by adding 1 point (to a maximum of 30) for all those with 12 years or less of education. Brief details are provided below.

*Correspondence to: Mr. Michael Lawton, School of Social and Community Medicine, University of Bristol, Office G.04, Canynge Hall, 39 Whatley Road, Bristol, BS8 2PS, United Kingdom; E-mail: michael.lawton@bristol.ac.uk

Funding agencies: Meike Kasten has received funding for the EPIPARK study from the German Research Foundation (KA3179/2-1). DeNoPa was supported by unrestricted grants by the Paracelsus-Klinike Germany and from TEVA Pharma as well as funding from GE Healthcare and the Deutsche Parkinsonvereinigung. The Oxford Discovery study was funded by the Monument Trust Discovery Award from Parkinson's UK; and supported by the National Institute for Health Research (NIHR) Oxford Biomedical Research Center based at Oxford University Hospitals NHS Trust and University of Oxford, and the Dementias and Neurodegenerative Diseases Research Network (DeNDRoN). The ABC-PD study is supported by Janssen Research & Development, a division of Janssen Pharmaceutica NV. Baseline assessment of the MODEP study has been funded by Abbott. Further funding for the MODEP study has been provided by TEVA Pharma GmbH and UCB Deutschland.

Relevant conflicts of interest/financial disclosures: Nothing to report. Full financial disclosures and author roles may be found in the online version of this article.

Received: 28 August 2015; **Revised:** 28 October 2015; **Accepted:** 28 October 2015

Published online 10 February 2016 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.26498

© 2016 International Parkinson and Movement Disorder Society
This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

The MODEP cohort consists of incident PD ($n = 22$), prevalent PD ($n = 18$), and controls ($n = 24$) with up to 8 visits.⁶ We restricted this analysis to the incident and prevalent PD arms. The ABC-PD study is an ongoing cross-sectional study of PD with 91 individuals included in this analysis. The De Novo Parkinson (DeNoPa) study is an untreated incidence PD cohort ($n = 159$) with a control arm ($n = 110$).^{7,8} This cohort has data at baseline and on one follow-up visit. We included 123 PD patients after excluding individuals who were found to have other neurological diseases at follow-up. The EPIPARK cohort has 112 PD patients and 543 controls with data on up to three visits.⁹ We restricted this analysis to the PD patients. The Oxford Discovery cohort has data on 958 PD patients, 293 controls, and 180 at risk of PD (as of January 2015)¹⁰ with data on up to three visits per individual. We included only the 795 PD patients who were recently diagnosed (within 3.5 years) and with clinical probability of PD $\geq 90\%$.

The analyzed data included only the PD subjects for direct comparison with the original van Steenoven and colleagues article.⁴ In studies where longitudinal data were available, we used data from every visit.

Ethics

MODEP received ethical approval no. 46/2010BO1 of the Medical Ethical Board of the University of Tuebingen. ABC-PD received ethical approval no. 686/2013BO1 of the Medical Ethical Board of the University of Tuebingen. For DeNoPa, institutional review board approval was obtained from the Ethikkommission der Hessischen Landesärztekammer in Frankfurt, Germany (FF89/2008) on 26 March 2009. EPIPARK received ethical approval no. 09-069. Oxford Discovery received ethical approval reference no. 10/H0505/71 from the NRES committee, South Central, Berkshire Ethics Committee.

Statistical Analysis

To convert MoCA score to MMSE score, we used exactly the same equipercentile method with log-linear smoothing that is described in the van Steenoven and colleagues article⁴ (see another work³ for methodological details). This method matches scores on the two tests by their percentile ranks after smoothing the distribution. The analysis was performed in the R statistical software (R Foundation for Statistical Computing), using the *equate* library.¹¹ We then validated our MoCA to MMSE conversion and the proposed MoCA to MMSE conversion from the van Steenoven and colleagues article within each of our five cohorts and also across the five cohorts. This was carried out by calculating the difference (Δ) between the true and equivalent MMSE and reporting the mean, standard deviation (SD), median, and interquartile range (IQR) of this Δ along with the root mean squared error

(RMSE). Smaller (in terms of absolute value) mean, SD, median, IQR, and RMSE denotes a more accurate conversion from MoCA to MMSE. We also calculated the intraclass correlation and the percentage of observations which were within ± 2 points of the true and equivalent MMSE to enable comparison with a previous validation.⁵

Results

We analyzed data from 1,161 individuals contributing 1,112 observations at baseline and 979 observations at follow-up visits. (MODEP: 40 individuals with 39 observations at baseline, 40 at the second visit, 32 at the third and fourth visits, 25 at the fifth visit, 22 at the sixth visit, 26 at the seventh visit, and 20 at the eighth visit, giving 236 observations in total. The ABC-PD study contributed 91 observations from 91 individuals. The DeNoPa cohort had 123 individuals with 93 observations at baseline and 121 at the second visit, giving 214 observations. The EPIPARK cohort had 112 individuals contributing 111 observations at baseline. The Oxford Discovery cohort had 795 individuals, of which 778 had baseline observations, 468 with visit 2 and 193 with visit 3 data, giving 1,439 observations in total.)

The MoCA adjusted scores ranged from 8 to 30 in our five cohorts with a median of 26 and an IQR range of 23 to 28. The mean MoCA was 25.0 and the SD 3.5. MMSE scores ranged from 13 to 30 with a median of 28 and an IQR of 27 to 29. The mean was 27.6 and the SD 2.3. These results indicate that MoCA may be better able to differentiate the range of cognitive function and is less prone to ceiling effects.

Table 1 shows that the conversion using the method proposed by van Steenoven and colleagues and our own conversion are remarkably similar with the equivalent MMSE only differing by 1 in 11 of 26 cases (ignoring where the MoCA was 4 or lower). However, it should be noted that we were both extrapolating to MoCA scores below 8 (and below 10 in the van Steenoven and colleagues article), so one must be cautious at the very low end of the distribution.

Table 2 shows the difference (both within and across cohorts) between the true and equivalent MMSE for the van Steenoven and colleagues article conversion and our own conversion. The median difference was 0 (IQR -1 to 1) for the van Steenoven and colleagues article across all studies and the median difference was also 0 within each study, except for the MODEP cohort where the median difference was -1 . However, even within the MODEP cohort, the IQR was -1 to 0 , still showing that at least 50% of the results are very close. The median difference was also 0 (IQR, -1 to 1) using our own internal conversion across all the studies. The median

TABLE 1. Conversion from MoCA to MMSE using the equipercentile method with log-linear smoothing using our datasets and compared to that from van Steenoven and colleagues

MoCA Total Adjusted	Equivalent MMSE Total (From the van Steenoven Article)	Equivalent MMSE Total (Internal Data Conversion)
1	6	1
2	9	2
3	11	4
4	12	10
5	13	13
6	14	14
7	15	15
8	15	16
9	16	17
10	17	18
11	18	18
12	18	19
13	19	20
14	20	20
15	21	21
16	22	22
17	22	22
18	23	23
19	24	24
20	25	24
21	26	25
22	26	26
23	27	26
24	28	27
25	28	28
26	29	28
27	29	29
28	30	29
29	30	30
30	30	30

MoCA was adjusted for the years of education. Scores that are in the shaded boxes are derived from extrapolated data.

difference was also 0 within each study, except for the EPIPARK cohort where the median difference was 1.

Across all cohorts, the RMSE was remarkably similar comparing the van Steenoven conversion to our own conversion, 1.88 compared to 1.84, respectively. Comparing within the cohorts, the van Steenoven conversion worked slightly better within the ABC-PD

study, the DeNoPa cohort, and the EPIPARK cohort (lower RMSE and smaller mean differences), whereas our own conversion worked slightly better within the MODEP and Oxford Discovery cohorts.

The van Steenoven conversion demonstrated an intraclass correlation coefficient of 0.66 (95% confidence interval [CI]: 0.64–0.69) between the true and equivalent MMSE. For the van Steenoven conversion, 11.1% of MMSE equivalent scores were more than 2 points higher than true MMSE, 83.8% were within 2 points, and 5.1% were more than 2 points lower. Our own conversion showed an intraclass correlation coefficient of 0.66 (95% CI: 0.64–0.68), with 8.1% of MMSE equivalent scores were more than 2 points higher than true MMSE, 83.2% were within 2 points, and 8.7% were more than 2 points lower.

Discussion

Our replication of the analysis resulted in a very similar conversion table to the one in the van Steenoven and colleagues, even though our sample is 10 times larger (n = 2,091, compared to n = 197). Validation of the conversion table from the van Steenoven and colleagues article shows that it has very good characteristics, with 0 median and small IQR of the difference and a RMSE, which is almost as good as our own internal conversion across all cohorts. An internal validation will almost always be better than an external validation, which further demonstrates the validity of the van Steenoven conversion. These findings are similar to a previously reported smaller validation study.⁵

Our sample had a wider range of MoCA and MMSE performance than a previous publication (lowest MoCA value 8 points, as compared to 17 points), which greatly enhances the generalizability of this conversion table. However, we still did not have any participants with a MoCA less than 8. This limitation is probably not of high clinical relevance given that it is unusual to recruit subjects with such poor performance into research. For example, in the UK, ethical committees would prohibit recruitment of subjects who could not consent into research unless it was a therapeutic trial that may have patient benefit.

TABLE 2. Validation of the MoCA to MMSE conversion using both the conversion from the van Steenoven and colleagues article and our own internal conversion

Cohort	N	van Steenoven Article Conversion		Internal Conversion	
		MMSE Total–Equivalent MMSE: Mean (SD); Median (IQR)	RMSE	MMSE Total–Equivalent MMSE: Mean (SD); Median (IQR)	RMSE
MODEP ⁶	236	–0.68 (1.45); –1 (–1, 0)	1.60	–0.29 (1.51); 0 (–1, 1)	1.54
ABC-PD	91	–0.13 (1.74); 0 (–1, 1)	1.74	0.29 (1.80); 0 (–1, 1)	1.82
DeNoPa ^{7,8}	214	0.08 (1.69); 0 (–1, 1)	1.69	0.53 (1.72); 0 (–1, 1)	1.80
EPIPARK ⁹	111	0.32 (2.19); 0 (–1, 2)	2.21	0.74 (2.14); 1 (–1, 2)	2.25
Discovery ¹⁰	1,439	–0.49 (1.86); 0 (–2, 1)	1.92	–0.03 (1.85); 0 (–1, 1)	1.85
All data	2,091	–0.39 (1.83); 0 (–1, 1)	1.88	0.05 (1.84); 0 (–1, 1)	1.84

The equipercentile method has the strengths that it can deal with nonlinearity within scales and that the equated scores will always be within the range of possible scores. However, the method is limited because it can lead to an irregular distribution of scores. An alternative approach to combine data across studies would be to internally standardize data using a Z-score or a T-score¹² method; however, these approaches do not take into account the difference in distributions or variability across the populations; hence, we favor the former approach.

These results provide independent replication and validation of a previous conversion table from MoCA to MMSE. The closeness in scores using either conversion table shows that this approach is useful in converting MoCA scores to MMSE scores and enables harmonization and meta-analysis across cohorts to determine determinants of cognitive impairment or decline in PD cohorts. This work needs to be extended to other domains, such as olfaction, depression, and so on, and to incorporate other methods, such as item response theory, to enable researchers to apply cross-scale conversions. This will greatly enhance the utility of existing research data and facilitate greater collaboration and shared analyses, leading to more robust research findings. ■

Acknowledgment: The authors thank Christina M Lill for critical revision of the manuscript.

References

1. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975;12:189–198.
2. Nasreddine ZS, Phillips NA, Bedirian V, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *J Am Geriatr Soc* 2005;53:695–699.
3. Kolen MJ, Brennan RL. *Test Equating, Scaling, and Linking*. New York: Springer; 2004.
4. van Steenoven I, Aarsland D, Hurtig H, et al. Conversion between Mini-Mental State Examination, Montreal Cognitive Assessment, and Dementia Rating Scale-2 scores in Parkinson's disease. *Mov Disord* 2014;29:1809–1815.
5. Armstrong MJ, Duff-Canning S, Psych C, Kowgier M, Marras C. Independent application of montreal cognitive assessment/mini-mental state examination conversion. *Mov Disord* 2015;30:1710–1711.
6. Maetzler W, Ellerbrock M, Heger T, Sass C, Berg D, Reilmann R. Digitomotography in Parkinson's disease: a cross-sectional and longitudinal study. *PLoS One* 2015;10:e0123914.
7. Mollenhauer B, Trautmann E, Sixel-Döring F, et al. Nonmotor and diagnostic findings in subjects with de novo Parkinson disease of the DeNoPa cohort. *Neurology* 2013;81:1226–1234.
8. Mollenhauer B, Zimmermann J, Sixel-Döring F. Monitoring of thirty marker candidates in early Parkinson's disease as progression markers. *Neurology* 2015 (In revision).
9. Kasten M, Hagenah J, Graf J, et al. Cohort Profile: a population-based cohort to study non-motor symptoms in parkinsonism (EPI-PARK). *Int J Epidemiol* 2013;42:128–128k.
10. Szewczyk-Krolikowski K, Tomlinson P, Nithi K, et al. The influence of age and gender on motor and non-motor features of early Parkinson's disease: initial findings from the Oxford Parkinson Disease Center (OPDC) discovery cohort. *Parkinsonism Relat Disord* 2014;20:99–105.
11. Albano A. *Equate: observed-score linking and equating*. R package. version 2.0-3. Vienna, Austria: R Foundation for Statistical Computing; 2014
12. Tuokko H, Woodward TS. Development and validation of a demographic correction system for neuropsychological measures used in the Canadian Study of Health and Aging. *J Clin Exp Neuropsychol* 1996;18:479–616.