

Rapid Cognitive Screening of Patients With Substance Use Disorders

Marc L. Copersino
Harvard Medical School and McLean Hospital

William Fals-Stewart
University of Rochester

Garrett Fitzmaurice
Harvard Medical School, McLean Hospital, and Harvard
School of Public Health

David J. Schretlen
Johns Hopkins University

Jody Sokoloff
McLean Hospital

Roger D. Weiss
Harvard Medical School and McLean Hospital

To date, there has not been a time-efficient and resource-conscious way to identify cognitive impairment in patients with substance use disorders (SUDs). In this study, we assessed the validity, accuracy, and clinical utility of a brief (10-min) screening instrument, the Montreal Cognitive Assessment (MoCA), in identifying cognitive impairment among patients with SUDs. The Neuropsychological Assessment Battery—Screening Module, a 45-min battery with known sensitivity to the mild to moderate deficits observed in patients with SUDs, was used as the reference criterion for determining agreement, rates of correct and incorrect decision classifications, and criterion-related validity for the MoCA. Classification accuracy of the MoCA, based on receiver operating characteristic (ROC) analysis, was strong, with an area under the ROC curve of 0.86, 95% confidence interval [0.75, 0.97]. The MoCA also showed acceptable sensitivity (83.3%) and specificity (72.9%) for the identification of cognitive impairment. Using a cutoff of 25 on the MoCA, the overall agreement was 75.0%; chance-corrected agreement (kappa) was 41.9%. These findings indicate that the MoCA provides a time-efficient and resource-conscious way to identify patients with SUDs and neuropsychological impairment, thus addressing a critical need in the addiction treatment research community.

Keywords: cognitive impairment, substance use disorders, brief cognitive screening instrument, receiver operating characteristic analysis

Cognitive impairment in patients with substance use disorders (SUDs) contributes to poorer treatment outcomes, including decreased treatment retention (Aharonovich et al., 2006; Aharonovich, Nunes, & Hasin, 2003; Donovan, Kivlahan, Kadden, & Hill, 2001; Fals-Stewart, 1993; Fals-Stewart & Schafer, 1992) and less abstinence from substances of abuse (Aharonovich et al., 2006). Cognitive dysfunction has also been shown to have a negative impact on therapeutic mechanisms of change (Bates, Pawlak, Tonigan, & Buckman, 2006). For example, it is associated with less treatment adherence (Bates et al., 2006), less treatment

engagement (Katz et al., 2005), less readiness to change (Blume, Schmalzing, & Marlatt, 2005), lower self-efficacy (Bates et al., 2006), decreased insight (Horner, Harvey, & Denier, 1999; Shelton & Parsons, 1987), increased denial of addiction (Rinn, Desai, Rosenblatt, & Gastfriend, 2002), and greater reflection impulsivity (Clark, Robbins, Ersche, & Sahakian, 2006). In addition, cognitive impairment among people with alcoholism has been shown to have a negative impact on drink refusal skill acquisition and aftercare treatment attendance (Smith & McCrady, 1991).

Marc L. Copersino, Department of Psychiatry, Harvard Medical School, and Division of Alcohol and Drug Use, McLean Hospital; William Fals-Stewart, School of Nursing, University of Rochester; Garrett Fitzmaurice, Department of Psychiatry, Harvard Medical School, Division of Alcohol and Drug Abuse, McLean Hospital, and department of Biostatistics, Harvard School of Public Health; David J. Schretlen, Department of Psychiatry and Behavioral Science, Johns Hopkins University; Jody Sokoloff, Division of Drug and Alcohol Use, McLean Hospital; and Roger D. Weiss, Department of Psychiatry, Harvard Medical School, and Division of Drug and Alcohol Use, McLean Hospital.

Supported by NIDA grant 1 R03 DA024126-01 (Copersino) and 5K24DA022288-02 (Weiss). The authors thank the patients

and staff at the McLean Hospital Alcohol and Drug Abuse Day Treatment Program, especially Judith Faberman, LICSW, Dr. Ruth Reibstein, Jerry Gregoire, and Bruce Pratt; and Dr. Mary A. Kautz of the Division of Clinical Neuroscience and Behavioral Research, National Institute on Drug Abuse. The authors also thank Dr. Ziad Nasreddine of the Université de Sherbrooke and McGill University for permission to use the Montreal Cognitive Assessment.

Correspondence concerning this article should be addressed to Marc L. Copersino, McLean Hospital and Harvard Medical School, Alcohol and Drug Abuse Treatment Program, 115 Mill Street, Belmont, MA 02478. E-mail: mcopersino@mclean.harvard.edu

Estimates regarding the prevalence of cognitive impairment in patients with SUDs vary widely and range from about 30% to 80% (Bates & Convit, 1999; Grant, Adams, Carlin, & Rennick, 1977; Meek, Clark, & Solana, 1989; National Institute on Drug Abuse, 2003; O'Malley, Adamse, Heaton, & Gawin, 1992; Parsons & Nixon, 1993; Rourke & Loberg, 1996). These deficits may range from the relatively subtle temporary effects of cannabis use (Bolla, Brown, Eldreth, Tate, & Cadet, 2002; Hart, van Gorp, Haney, Foltin, & Fischman, 2001; Pope, 2002; Pope et al., 2003; Pope, Gruber, & Yurgelun-Todd, 2001; Solowij et al., 2002) to the moderate executive control deficits observed in chronic cocaine users even after several months of abstinence (Di Sclafani, Tolou-Shams, Price, & Fein, 2002; O'Malley et al., 1992; Strickland et al., 1993; Woicik et al., 2009). Although these estimates do not include people with alcoholism who develop permanent cognitive deficits such as Wernicke-Korsakoff syndrome, they do include the enduring visuospatial information-processing deficits observed in people without dementia and with alcohol use disorders (Schandler, Clegg, Thomas, & Cohen, 1996).

Specialized treatment enhancements aimed at cognitively impaired patients with SUDs, such as cognitive rehabilitation (Fals-Stewart & Lucente, 1994; Goldstein, Haas, Shemansky, Barnett, & Salmon-Cox, 2005; Grohman & Fals-Stewart, 2003; Grohman, Fals-Stewart, & Donnelly, 2006) and accommodation (Czuchry & Dansereau, 2003; Czuchry, Dansereau, Dees, & Simpson, 1995; Dansereau, Dees, Chatham, Boatler, & Simpson, 1993; Dansereau, Joe, & Simpson, 1995; Newbern, Dansereau, Czuchry, & Simpson, 2005) have shown some success, but this is still an area in its infancy, and further research is needed. One of the primary challenges in developing treatments and enhancements for cognitively impaired patients with SUDs is a lack of knowledge about which patients should be targeted for specialized interventions (National Institute on Drug Abuse, 2003).

Unfortunately, neuropsychological assessment is typically not an aspect of patient evaluation in substance abuse treatment programs because it is prohibitively time and resource consuming. Moreover, studies have shown that cognitively impaired patients with SUDs cannot be adequately identified by drug counselors via clinical impression (Fals-Stewart, 1997) or through self-report (Horner et al., 1999; Shelton & Parsons, 1987). If accounting for and addressing the presence of cognitive deficits among substance-abusing patients involves, as a first step, identifying those with neuropsychological impairment, treatment providers and researchers alike need a practical neurocognitive assessment approach for patients with SUDs that is both accurate and comparatively less labor intensive.

Montreal Cognitive Assessment

The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) is a brief (10-min) cognitive screening instrument that was developed in a geriatric population to be sensitive to mild cognitive impairment. Although not a clearly defined syndrome, mild cognitive impairment is

regarded as cognitive dysfunction in excess of normal age-related decline that does not interfere notably with activities of daily living and is often undetectable via standard mental status examination. The MoCA has been shown to be sensitive to subtle cognitive deficits in a variety of populations. In comparison to the Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975), the MoCA was shown to be more sensitive to early detection of cognitive decline in people with asymptomatic cerebrovascular disease (i.e., without signs of cerebrovascular disease but having one or more risk factors; Popovic, Seric, & Demarin, 2007). In part because of the findings of Popovic et al. (2007), the National Institute of Neurological Disorders and Stroke has recommended use of the MoCA over the Mini-Mental State Examination as part of a brief, minimal dataset for identifying people in the early stages of cognitive impairment related to vascular factors (Hachinski et al., 2006). For example, the MoCA is recommended for identifying subtle changes in cognitive performance resulting from silent stroke. The MoCA has also been found to be more sensitive than the Mini-Mental State Examination in detecting cognitive impairment in patients with Parkinson's disease (Nazem et al., 2009; Zadikoff et al., 2008). Moreover, on the basis of its agreement with a lengthier neuropsychological battery, data support that the MoCA is reliable and valid as a screening test for detection of early or mild cognitive impairment in Parkinson's disease (Gill, Freshman, Blender, & Ravina, 2008).

The purpose of this study was to assess the validity, accuracy, and clinical utility of the MoCA in identifying cognitive impairment among patients with SUDs in a clinical research setting. We accomplished this through the following steps: (a) assessment of the validity of the screener through its strength of agreement with an accepted standard criterion measure; (b) assessment of its classification accuracy by generating a confusion matrix and deriving measures of sensitivity, specificity, and area under the receiver operating characteristic (ROC) curve; and (c) assessment of its clinical utility via a combination of qualitative assessment of patient acceptability and practical considerations.

Method

Participants

Participants were 60 adult patients receiving treatment at the McLean Hospital Alcohol and Drug Abuse Treatment Program (ADATP), in Belmont, Massachusetts, with at least one current substance use dependence diagnosis (*Diagnostic and Statistical Manual of Mental Disorders*), 4th ed. [*DSM-IV*] criteria; American Psychiatric Association, 1994). Study participants were recruited through the ADATP Partial Hospital and Residential Programs. To be eligible for the study, participants had to meet the following inclusion criteria: (a) recent admission to either the Partial Hospital or the Residential Program at the McLean Hospital ADATP, (b) any non-nicotine *DSM-IV* substance dependence disorder, (c) abstinence from all drugs of abuse other

than nicotine for at least 7 days, and (d) ages 18–65. The inclusion criteria were designed to be as broad as possible to maintain ecological validity of the study findings without compromising internal validity of the neuropsychological measures. Exclusion criteria included acute intoxication or withdrawal and any medical illness or psychiatric condition (including dementia) that in our view would interfere with provision of consent or valid self-report or otherwise compromise participation in research.

On average, participants were 38.3 years old (*SD* = 13.2) and had completed 15.0 years of education (*SD* = 2.4). Participants were predominantly Caucasian (95%; *n* = 57) and unemployed during the past month (70%; *n* = 42). About half of the participants were male (52%; *n* = 31), and half were never married (52%; *n* = 31).

Procedures and Instruments

After providing written informed consent, participants completed all study measures at a single time point, requiring approximately 2.5 hr. Cognitive measures included the MoCA (Nasreddine et al., 2005), the Neuropsychological Assessment Battery—Screening Module (NAB–SM; Stern & White, 2003), and the National Adult Reading Test—Revised (Blair & Spreen, 1989). Administration of the MoCA and NAB–SM were counterbalanced to preclude order effects. SUD diagnoses were made using the *DSM–IV* checklist for SUD (Wu et al., 2009); all other Axis I diagnoses were made using the Structured Clinical Interview for *DSM–IV–TR*—Patient Edition (First, Spitzer, Gibbon, &

Williams, 2002). Quantity and frequency of drug and alcohol use during past 30 days, 90 days, and 1 year were measured using the timeline followback method (Sobell & Sobell, 1992). Anxiety was assessed using the Hamilton Anxiety Scale (Hamilton, 1959, 1960), and depression was assessed using the Quick Inventory of Depressive Symptomatology (Rush et al., 2003). Participants were paid \$50 (in the form of gift cards) for completing all study assessments.

MoCA Assessment

The MoCA samples behavior across 14 performance tasks that engage multiple cognitive domains including attention, language, visuospatial, executive, and memory (see Table 1). The time taken to administer the MoCA is approximately 10 min. The total possible score is 30 points (31 if the patient is age 12 or younger), and a score of 26 or greater is classified as normal, that is, without evidence of cognitive impairment (Nasreddine et al., 2005).

Neuropsychological Assessment Battery—Screening Module

The NAB–SM (Stern & White, 2003) assesses cognitive functioning across five domains: attention, language, memory, visuospatial, and executive (see Table 1). Administration time is approximately 45 min. The NAB–SM has been recommended for use with patients with SUDs because of its sensitivity (.81) and specificity (.92) in classifying pa-

Table 1
Cognitive Domains Assessed by the Montreal Cognitive Assessment and the Neuropsychological Assessment Battery—Screening Module

Domain	Montreal Cognitive Assessment	Neuropsychological Assessment Battery—Screening Module
Attention	Orientation Digits Forward ^a Digits Backward ^b Serial Sevens ^c	Orientation Digits Forward ^a Digits Backward ^b Numbers and Letters ^d
Language	Picture Naming	Picture Naming Auditory comprehension
Memory	Memory for words (immediate and delayed recall) Sentence repetition	Story Learning (immediate and delayed recall) Shape learning
Spatial	Cube drawing Clock drawing	Visual discrimination ^e Design cConstruction
Executive	Alternating Trail Making ^f Verbal fluency (word generation) Abstraction ^g	Mazes Word generation

Note. From *Neuropsychological Assessment Battery: Administration, Scoring, and Interpretation Manual* (p. 6), by R. A. Stern and T. White, 2003, Lutz, FL: Psychological Assessment Resources. Adapted and reproduced by special permission of the Publisher, Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Neuropsychological Assessment Battery by Robert A. Stern, PhD and Travis White, PhD, Copyright 2001, 2003 by PAR, Inc. Further reproduction is prohibited without permission from PAR, Inc. Tests are presented according to cognitive domain, not in order of test administration.

^a Repetition of orally presented digits. ^b Reversal of orally presented digits. ^c Serial subtraction by sevens, beginning with 100-7. ^d Two timed tasks involving letter cancellation and letter cancellation plus serial addition; provides measure of psychomotor speed. ^e Visual match-to-target paradigm. ^f Connecting circled letters to numbers in a progressive and alternating pattern. ^g Analogies.

tients with present or absent cognitive impairment in this population (Grohman & Fals-Stewart, 2004). The NAB–SM was used as the reference criterion for determining agreement and rates of correct and incorrect decision classifications for the MoCA. Decision classifications using the NAB–SM were based on the dichotomous Total Screening Index. The Total Screening Index is a composite measure of the five cognitive domain scores and is a standardized score representing the examinee’s overall test performance. Total Screening Index values less than or equal to 84 (corresponding to a score of more than 1 standard deviation, or 15 points, below the mean of 100) are indicative of cognitive impairment. Test norms were demographically corrected relative to a standardization sample ($N = 1,448$) of neurologically healthy individuals of the same age, sex, and educational level (Stern & White, 2003).

Statistical Analyses

We assessed the accuracy, validity, and clinical utility of the MoCA in identifying cognitively impaired patients with SUDs in a clinical research setting. Accuracy and validity of the MoCA were evaluated statistically, and clinical utility was assessed via practical considerations and through qualitative assessment of patient acceptability. Clinical accuracy was evaluated using a decision theory approach. Using the NAB–SM as the reference criterion, we performed analyses to assess the MoCA’s sensitivity and specificity in accurately detecting cognitive impairment. We conducted ROC analysis to assess the quality of the screener at a range of possible cutoff values, and we evaluated the MoCA’s criterion-related validity through its overall agreement with the NAB–SM. We used the kappa statistic for dichotomous data (presence or absence of cognitive impairment) to measure chance-corrected agreement. Patient acceptability was assessed through qualitative assessment of two Likert-type

questions (“How demanding was this test?” and “How unpleasant was this test?”), rated on a scale ranging from 1 (*not at all*) to 5 (*extremely*).

Results

On average, each participant met criteria for 1.4 ($SD = 0.70$; range = 1–3) substance dependence diagnoses. The most common diagnosis was alcohol dependence (65%; $n = 39$), followed by dependence on opioids (32%; $n = 19$), cocaine (17%; $n = 10$), cannabis (12%; $n = 7$), benzodiazepine (10%; $n = 6$), and amphetamine (8%; $n = 5$). Primacy of SUD diagnoses was not determined. Of participants, 41% met criteria for any co-occurring *DSM–IV* Axis I disorder. The most common co-occurring disorder was bipolar affective disorder (17%; $n = 10$), followed by posttraumatic stress disorder (13%; $n = 8$), generalized anxiety disorder (12%; $n = 7$), panic disorder (8%; $n = 5$), and major depressive disorder (5%; $n = 3$) and social phobia (5%; $n = 3$).

Of the participants, 38% were classified as impaired on the basis of the MoCA, and 20% were classified as impaired on the basis of the NAB–SM (see Table 2). Across the five cognitive domain scores that make up the NAB–SM total composite score, the proportion of participants classified as impaired ranged from a high of 37% for the attentional domain to a low of 12% for the language domain. We based the MoCA’s classification accuracy on ROC analysis, which showed an area under the ROC curve equal to 0.86, 95% confidence interval (CI) [0.75, 0.97] (see Figure 1). Areas under the ROC curve for the five cognitive domain scores making up the NAB–SM total composite ranged from 0.73 to 0.92 and included the following scores: attention = 0.73, 95% CI [0.60, 0.86]; language = 0.92, 95% CI [0.80, 1.00]; memory = 0.76, 95% CI [0.61, 0.90]; spatial = 0.77, 95% CI [0.62, 0.93]; and executive = 0.80, 95% CI [0.64, 0.96]. The MoCA also showed acceptable sensitivity (83.3%)

Table 2
Montreal Cognitive Assessment and Neuropsychological Assessment Battery—Screening Module Total and Subdomain Scores and Descriptive Cognitive and Mood Severity Results for 60 Adult Patients Receiving Treatment for Substance Dependence

Measure	<i>M</i>	<i>SD</i>	Min	Max	% (<i>n</i>) classified as impaired
Montreal Cognitive Assessment score	25.6	3.2	12.0	30.0	38.3 (23)
NAB-SM					
Total standard score	95.0	14.8	51.0	129.0	20.0 (12)
Attention domain standard score	89.4	16.9	49.0	129.0	36.7 (22)
Language domain standard score	106.9	17.1	45.0	134.0	11.7 (7)
Memory domain standard score	95.1	13.4	69.0	129.0	21.7 (13)
Spatial domain standard score	98.9	16.1	57.0	127.0	18.3 (11)
Executive Functions standard score	93.4	14.6	60.0	127.0	21.7 (13)
Estimated Full Scale IQ ^a	110.3	9.3	85.7	124.7	—
Estimated Verbal Scale IQ ^a	108.8	10.7	80.1	125.1	—
Estimated Performance Scale IQ ^a	113.6	6.0	96.7	119.4	—
Hamilton Anxiety Rating Scale	8.5	5.2	0	22	—
Quick Inventory of Depressive Symptomatology	13.1	6.2	1	31	—

Note. NAB–SM = Neuropsychological Assessment Battery—Screening Module.
^a Based on the results of the National Adult Reading Test.

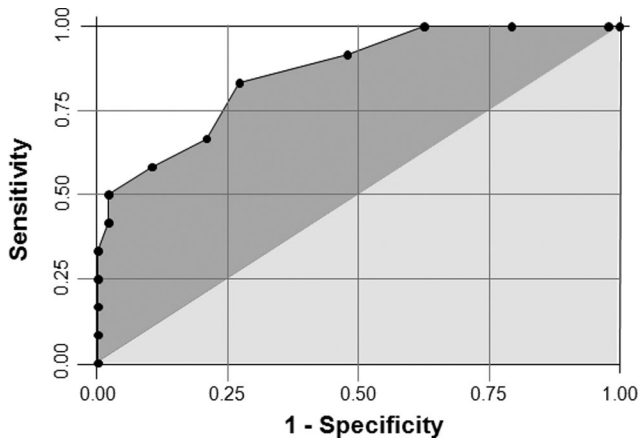


Figure 1. Sensitivity and specificity of the Montreal Cognitive Assessment in classifying 60 patients as cognitively impaired versus unimpaired. The area under the receiver operating characteristic curve = 0.86, SE = 0.06, asymptotic normal 95% confidence interval [0.75, 0.97].

and specificity (72.9%) to identify cognitive impairment. Using a cutoff of 25 or lower on the MoCA, the overall agreement was 75.0%; chance-corrected agreement (kappa) was 41.9%. The quality of the screener across a range of other possible cutoff values was also assessed via ROC analysis (see Table 3). A visual inspection of the trade-off between sensitivity and specificity across cutpoint values shows that 25 is the optimal cutpoint for a sample with SUDs patients.

Assessment of patient acceptability yielded the following results. Of the participants, 27% found the MoCA to be “not at all demanding,” 61% found it “somewhat” or “fairly” demanding, and 12% found it “rather” or “very” demanding; 55% found the MoCA “not at all unpleasant,” 40% found it “somewhat” or “fairly” unpleasant, and 5% found it “rather” or “very” unpleasant.

Discussion

The purpose of this study was to examine the accuracy, validity, and clinical utility of a brief cognitive screening instrument in identifying cognitive impairment in patients with SUDs in a clinical research setting. Results generally support its appropriate and practical use in this population. On the basis of its agreement with a reference criterion, the MoCA showed evidence of criterion-related validity and good accuracy in correctly classifying cognitive impairment cases and noncases.

The NAB–SM served as the reference criterion, and we used it for determining agreement and rates of correct and incorrect decision classifications. The NAB–SM and MoCA similarly sample a broad range of cognitive domains. This study’s results showed good agreement between the MoCA and the five NAB–SM cognitive subdomains, including attention, language, memory, spatial, and executive. Thus, among the processes sampled across the NAB–SM cognitive subdomains, none are disproportionately weighted in the MoCA. However, unlike the NAB–SM, the MoCA does not include performance tasks related to psychomotor speed or visual learning and delayed recognition. Also, the NAB–SM assesses verbal memory through learning and delayed recall of verbally presented narrative, whereas the MoCA assesses verbal memory through immediate and delayed recall of five unrelated words. How these differences might affect overall test performance is not apparent.

On the basis of patient acceptability and other practical considerations, the MoCA has good clinical utility. Assessment of patient acceptability indicated that patients in general did not find the MoCA particularly unpleasant or demanding. It also provides an accurate and valid screening measure that is easy to use, time efficient, and resource conscious. This makes conducting cognitive assessment with patients with SUDs more practical for treatment settings and providers, such that patients who screen positive

Table 3
Detailed Report of Sensitivity and Specificity of Montreal Cognitive Assessment in Classifying 60 Patients as Cognitively Impaired Versus Unimpaired

Cutpoint	Sensitivity (%)	Specificity (%)	Correctly classified (%)	LR+	LR–
30	100.00	0.00	20.00	1.0000	—
29	100.00	2.08	21.67	1.0213	0.0000
28	100.00	20.83	36.67	1.2632	0.0000
27	100.00	37.50	50.00	1.6000	0.0000
26	91.67	52.08	60.00	1.9130	0.1600
25	83.33	72.92	75.00	3.0769	0.2286
24	66.67	79.17	76.67	3.2000	0.4211
23	58.33	89.58	83.33	5.6000	0.4651
22	50.00	97.92	88.33	24.0001	0.5106
21	41.67	97.92	86.67	20.0000	0.5957
20	33.33	100.00	86.67	—	0.6667
19	25.00	100.00	85.00	—	0.7500
18	16.67	100.00	83.33	—	0.8333
17	8.33	100.00	81.67	—	0.9167
16	0.00	100.00	80.00	—	1.0000

Note. Bold row indicates cutpoint selected as optimal. LR+ = positive likelihood ratio; LR– = negative likelihood ratio.

may be referred to more comprehensive evaluation. Moreover, the MoCA, including protocol sheet, instructions for administration, and scoring criteria, is available at no cost from the test developer (<http://www.mocatest.org/>). The MoCA may be used, reproduced, and distributed without permission for clinical and educational noncommercial purposes. For noncommercially funded research, it may be used with prior written permission. If used for commercially funded research, prior written permission and a licensing agreement are required. In contrast, the NAB-SM's list price is \$825, plus the cost of additional screening module record forms (\$94) and response booklets (\$52) per every 25 administrations. Purchase of the NAB-SM is restricted to professionals who meet competency-based qualification guidelines and who have completed the registration and qualification process attesting to their eligibility on the basis of training, education, and experience.

In comparison to the 10-min administration time required for the MoCA, the NAB-SM takes approximately 45 min to complete. Moreover, hand scoring the NAB-SM can take 30 min or longer. Scoring software is available (i.e., NAB Software Portfolio) that can reduce the time needed to score the NAB-SM, but it requires a PC-based computer with a CD-ROM drive for installation and an Internet connection or telephone for software activation. In comparison to the MoCA, administration of the NAB-SM also requires significantly more space—that is, a larger working surface with sufficient space to spread out testing materials including puzzle pieces and the stimulus book.

Finally, in addition to English, the MoCA has been translated into 22 languages. Multiple language versions of the MoCA have shown high sensitivity for screening patients with mild cognitive impairment, including the Korean (Lee et al., 2008), Arabic (Rahman & El Gaafary, 2009), and Chinese (Wen, Zhang, Niu, & Li, 2008) language versions.

There are several strengths of this investigation. The natural heterogeneity of the sample is a strength because it demonstrates the MoCA's validity for standard clinical practice. In other words, the ecological validity of the study findings is maximized without compromising the internal validity of the neuropsychological measures. Another strength is that the criterion measure has specifically been recommended for use with patients with SUDs because of its strong sensitivity and specificity in classifying patients with present or absent cognitive impairment in this population. The most common neuropsychological batteries typically require several hours of administration time, scoring, and interpretation (Rabin, Barr, & Burton, 2005). The 45-min administration time and computerized scoring of the NAB-SM enabled a more comprehensive evaluation across cognitive domains while conferring two additional benefits: (a) it enabled testing in a single day and (b) it avoided test fatigue that almost invariably results from lengthy neuropsychological batteries. Another strength of this study is the counterbalanced presentation of the MoCA and NAB-SM, which avoids possible test order effects.

A limitation of this study is the unknown influence of abstinence duration on overall prevalence of cognitive im-

pairment. An exclusion criterion for this study was known substance use within 7 days before study participation. This is consistent with the recommendations made by some investigators who suggest a duration of at least 1 week between admission to treatment and testing (Miller, 1985; Parsons & Farr, 1981). The rationale for this recommendation is that some cognitive recovery generally follows abatement of intoxication and acute abstinence effects. As a result, rates of detection among newly admitted patients may be artifactually inflated because of the effects of residual intoxication or withdrawal. However, because the goal of the study was not to study prevalence of cognitive impairment but rather the concordance between the MoCA and the NAB-SM, the latter should be relatively insensitive to any potential inflation.

The relationship between duration of abstinence and cognitive impairment, furthermore, is unclear. Paradoxically, cognitive performance may actually deteriorate slightly over the first few weeks of abstinence before gradually improving. For example, a recent study showed a gradual worsening in most neuropsychological categories, such that cocaine-dependent people with a positive urine drug screen for cocaine (typically indicating use within the past 72 hr) perform better on a broad range of neuropsychological measures in comparison to cocaine-dependent individuals with a negative screen (Woicik et al., 2009). These findings are consistent with those of a previous study showing that the scope of neuropsychological deficit among currently abstinent cocaine-dependent people actually increased from 72 hr to 14 days (Berry et al., 1993). These findings suggest that the influence of abstinence duration on cognitive performance may not be linear. However, it is unknown how exclusive such "nonlinear" effects of abstinence duration may be to predominantly heavy psychostimulant users. Therefore, there may be little relevance to the present study given that only 17% of patients met criteria for cocaine dependence and 8% met criteria for amphetamine or methamphetamine dependence.

Conclusion

A body of evidence is emerging showing that cognitive impairment in patients with SUDs has a significant and negative impact on treatment outcomes and therapeutic mechanisms of change. Specialized treatments and enhancements aimed at improving outcomes for SUD patients have shown some success, but this area is still in its infancy and further research is needed. One of the main challenges associated with developing treatments for cognitively impaired patients with SUDs is uncertainty about which patients to target for specialized interventions. To date, the search for a brief cognitive screening instrument sensitive to the mild to moderate impairment observed in patients with SUDs has been unsuccessful. These findings show that the MoCA addresses a critical need in the addiction treatment research community by providing a quick and accurate screening instrument that can expedite the progression of research in this area.

References

- Aharonovich, E., Hasin, D. S., Brooks, A. C., Liu, X., Bisaga, A., & Nunes, E. V. (2006). Cognitive deficits predict low treatment retention in cocaine dependent patients. *Drug and Alcohol Dependence, 81*, 313–322.
- Aharonovich, E., Nunes, E., & Hasin, D. (2003). Cognitive impairment, retention and abstinence among cocaine abusers in cognitive-behavioral treatment. *Drug and Alcohol Dependence, 71*, 207–211.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bates, M. E., & Convit, A. (1999). Neuropsychology and neuroimaging of alcohol and illicit drug abuse. In A. Calev (Ed.), *The assessment of neuropsychological functions in psychiatric disorders* (pp. 373–445). Washington, DC: American Psychiatric Publishing.
- Bates, M. E., Pawlak, A. P., Tonigan, J. S., & Buckman, J. F. (2006). Cognitive impairment influences drinking outcome by altering therapeutic mechanisms of change. *Psychology of Addictive Behaviors, 20*, 241–253.
- Berry, J., van Gorp, W. G., Herzberg, D. S., Hinkin, C., Boone, K., Steinman, L., & Wilkins, J. N. (1993). Neuropsychological deficits in abstinent cocaine abusers: Preliminary findings after two weeks of abstinence. *Drug and Alcohol Dependence, 32*, 231–237.
- Blair, J. R., & Spreen, O. (1989). Predicting premorbid IQ: A revision of the National Adult Reading Test. *Clinical Neuropsychologist, 3*, 129–136.
- Blume, A. W., Schmalig, K. B., & Marlatt, G. A. (2005). Memory, executive cognitive function, and readiness to change drinking behavior. *Addictive Behaviors, 30*, 301–314.
- Bolla, K. I., Brown, K., Eldreth, D., Tate, K., & Cadet, J. L. (2002). Dose-related neurocognitive effects of marijuana use. *Neurology, 59*, 1337–1343.
- Clark, L., Robbins, T. W., Ersche, K. D., & Sahakian, B. J. (2006). Reflection impulsivity in current and former substance users. *Biological Psychiatry, 60*, 515–522.
- Czuchry, M., & Dansereau, D. F. (2003). Cognitive skills training: Impact on drug abuse counseling and readiness for treatment. *American Journal of Drug and Alcohol Abuse, 29*, 1–18.
- Czuchry, M., Dansereau, D. F., Dees, S. M., & Simpson, D. D. (1995). The use of node-link mapping in drug abuse counseling: The role of attentional factors. *Journal of Psychoactive Drugs, 27*, 161–166.
- Dansereau, D. F., Dees, D. M., Chatham, L. R., Boatler, J. F., & Simpson, D. D. (1993). *Mapping new roads to recovery: Cognitive enhancements to counseling*. Fort Worth: Texas Christian University, Institute of Behavioral Research. Retrieved from <http://www.ibr.tcu.edu/pubs/trtmanual/mapping.html>
- Dansereau, D. F., Joe, G. W., & Simpson, D. D. (1995). Attentional difficulties and the effectiveness of a visual representation strategy for counseling drug-addicted clients. *International Journal of the Addictions, 30*, 371–386.
- Di Sclafani, V., Tolou-Shams, M., Price, L. J., & Fein, G. (2002). Neuropsychological performance of individuals dependent on crack-cocaine, or crack-cocaine and alcohol, at 6 weeks and 6 months of abstinence. *Drug and Alcohol Dependence, 66*, 161–171.
- Donovan, D. M., Kivlahan, D. R., Kadden, R. M., & Hill, D. (2001). Cognitive impairment as a patient-treatment matching hypothesis. In R. Longabaugh & P. W. Wirtz (Eds.), *Project MATCH hypothesis: Results and causal chain analyses* (Vol. 8, pp. 62–81). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.
- Fals-Stewart, W. (1993). Neurocognitive defects and their impact of substance abuse treatment. *Journal of Addictions and Offender Counseling, 13*, 46–57.
- Fals-Stewart, W. (1997). Ability of counselors to detect cognitive impairment among substance-abusing patients: An examination of diagnostic efficiency. *Experimental and Clinical Psychopharmacology, 5*, 39–50.
- Fals-Stewart, W., & Lucente, S. (1994). The effect of cognitive rehabilitation on neuropsychological status of patients in drug abuse treatment who display neurocognitive impairment. *Rehabilitation Psychology, 39*, 75–94.
- Fals-Stewart, W., & Schafer, J. (1992). The relationship between length of stay in drug-free therapeutic communities and neurocognitive functioning. *Journal of Clinical Psychology, 48*, 539–543.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research version, patient edition (SCID-IP)*. New York: Biometrics Research, New York State Psychiatric Institute.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). “Mini-Mental State”: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.
- Gill, D. J., Freshman, A., Blender, J. A., & Ravina, B. (2008). The Montreal Cognitive Assessment as a screening tool for cognitive impairment in Parkinson’s disease. *Movement Disorders, 23*, 1043–1046.
- Goldstein, G., Haas, G. L., Shemansky, W. J., Barnett, B., & Salmon-Cox, S. (2005). Rehabilitation during alcohol detoxification in comorbid neuropsychiatric patients. *Journal of Rehabilitation Research and Development, 42*, 225–234.
- Grant, I., Adams, K. M., Carlin, A. S., & Rennick, P. M. (1977). Neuropsychological deficit in polydrug users: A preliminary report of the findings of the collaborative neuropsychological study of polydrug users. *Drug and Alcohol Dependence, 2*, 91–108.
- Grohman, K., & Fals-Stewart, W. (2003). Computer-assisted cognitive rehabilitation with substance-abusing patients: Effects on treatment response. *Journal of Cognitive Rehabilitation, 21*, 2–9.
- Grohman, K., & Fals-Stewart, W. (2004). The detection of cognitive impairment among substance-abusing patients: The accuracy of the Neuropsychological Assessment Battery—Screening module. *Experimental and Clinical Psychopharmacology, 12*, 200–207.
- Grohman, K., Fals-Stewart, W., & Donnelly, K. (2006). Improving treatment response of cognitively impaired veterans with neuropsychological rehabilitation. *Brain and Cognition, 60*, 203–204.
- Hachinski, V., Iadecola, C., Petersen, R. C., Breteler, M. M., Nyenhuis, D. L., Black, S. E., . . . & Leblanc, G. G. (2006). National Institute of Neurological Disorders and Stroke—Canadian Stroke Network vascular cognitive impairment harmonization standards. *Stroke, 37*, 2220–2241.
- Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology, 32*, 50–55.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology and Neurosurgical Psychiatry, 23*, 56–62.
- Hart, C. L., van Gorp, W., Haney, M., Foltin, R. W., & Fischman, M. W. (2001). Effects of acute smoked marijuana on complex cognitive performance. *Neuropsychopharmacology, 25*, 757–765.
- Horner, M. D., Harvey, R. T., & Denier, C. A. (1999). Self-report and objective measures of cognitive deficit in patients entering substance abuse treatment. *Psychiatry Research, 86*, 155–161.

- Katz, E. C., King, S. D., Schwartz, R. P., Weintraub, E., Barksdale, W., Robinson, R., & Brown, B. S. (2005). Cognitive ability as a factor in engagement in drug abuse treatment. *American Journal of Drug and Alcohol Abuse, 31*, 359–369.
- Lee, J. Y., Dong Woo, L., Cho, S. J., Na, D. L., Jeon, H. J., Kim, S.-K., . . . & Cho, M. J. (2008). Brief screening for mild cognitive impairment in elderly outpatient clinic: Validation of the Korean version of the Montreal Cognitive Assessment. *Journal of Geriatric Psychiatry and Neurology, 21*, 104–110.
- Meek, P. S., Clark, H. W., & Solana, V. L. (1989). Neurocognitive impairment: The unrecognized component of dual diagnosis in substance abuse treatment. *Journal of Psychoactive Drugs, 21*, 153–160.
- Miller, L. (1985). Neuropsychological assessment of substance abusers: Review and recommendations. *Journal of Substance Abuse Treatment, 2*, 5–17.
- Nasreddine, Z. S., Phillips, N. A., Bedirian, V., Charbonneau, S., Whitehead, V., Collin, I., . . . & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*, 695–699.
- National Institute on Drug Abuse. (2003). *Developing behavioral treatments for drug abusers with cognitive impairments* (NIDA Workshop Summary). Retrieved from <http://www.drugabuse.gov/whatsnew/meetings/cognitiveimpairment.html>
- Nazem, S., Siderowf, A. D., Duda, J. E., Have, T. T., Colcher, A., Horn, S. S., . . . & Weintraub, D. (2009). Montreal Cognitive Assessment performance in patients with Parkinson's disease with "normal" global cognition according to Mini-Mental State Examination score. *Journal of the American Geriatrics Society, 57*, 304–308.
- Newbern, D., Dansereau, D. F., Czuchry, M., & Simpson, D. D. (2005). Node-link mapping in individual counseling: Treatment impact on clients with ADHD-related behaviors. *Journal of Psychoactive Drugs, 37*, 93–103.
- O'Malley, S., Adamse, M., Heaton, R. K., & Gawin, F. H. (1992). Neuropsychological impairment in chronic cocaine abusers. *American Journal of Drug and Alcohol Abuse, 18*, 131–144.
- Parsons, O. A., & Farr, S. P. (1981). The neuropsychology of alcohol and drug use. In T. J. B. S. B. Filskov (Ed.), *Handbook of clinical neuropsychology* (pp. 320–365). New York: Wiley.
- Parsons, O. A., & Nixon, S. J. (1993). Neurobehavioral sequelae of alcoholism. *Neurologic Clinics, 11*, 205–218.
- Pope, H. G., Jr. (2002). Cannabis, cognition, and residual confounding. *Journal of the American Medical Association, 287*, 1172–1174.
- Pope, H. G., Jr., Gruber, A. J., Hudson, J. I., Cohane, G., Huestis, M. A., & Yurgelun-Todd, D. (2003). Early-onset cannabis use and cognitive deficits: What is the nature of the association? *Drug and Alcohol Dependence, 69*, 303–310.
- Pope, H. G., Jr., Gruber, A. J., & Yurgelun-Todd, D. (2001). Residual neuropsychologic effects of cannabis. *Current Psychiatry Reports, 3*, 507–512.
- Popovic, I. M., Seric, V., & Demarin, V. (2007). Mild cognitive impairment in symptomatic and asymptomatic cerebrovascular disease. *Journal of the Neurological Sciences, 257*(1–2), 185–193.
- Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology, 20*, 33–65.
- Rahman, T. T., & El Gaafary, M. M. (2009). Montreal Cognitive Assessment Arabic version: Reliability and validity prevalence of mild cognitive impairment among elderly attending geriatric clubs in Cairo. *Geriatrics and Gerontology International, 9*, 54–61.
- Rinn, W., Desai, N., Rosenblatt, H., & Gastfriend, D. R. (2002). Addiction denial and cognitive dysfunction: A preliminary investigation. *Journal of Neuropsychiatry and Clinical Neurosciences, 14*, 52–57.
- Rourke, S. B., & Loberg, T. (1996). The neurobehavioral correlates of alcoholism. In K. M. A. I. Grant (Ed.), *Neuropsychological assessment of neuropsychiatric disorders* (pp. 423–485). New York: Oxford University Press.
- Rush, A. J., Trivedi, M. H., Ibrahim, H. M., Carmody, T. J., Arnow, B., Klein, D. N., . . . & Keller, M. B. (2003). The 16-Item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): A psychometric evaluation in patients with chronic major depression. *Biological Psychiatry, 54*, 573–583.
- Schandler, S. L., Clegg, A. D., Thomas, C. S., & Cohen, M. J. (1996). Visuospatial information processing in intoxicated, recently detoxified, and long-term abstinent alcoholics. *Journal of Substance Abuse, 8*, 321–333.
- Shelton, M. D., & Parsons, O. A. (1987). Alcoholics' self-assessment of their neuropsychological functioning in everyday life. *Journal of Clinical Psychology, 43*, 395–403.
- Smith, D. E., & McCrady, B. S. (1991). Cognitive impairment among alcoholics: Impact on drink refusal skill acquisition and treatment outcome. *Addictive Behaviors, 16*, 265–274.
- Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back: A technique for assessing self-reported alcohol consumption. In J. P. A. R. Z. Litten (Ed.), *Measuring alcohol consumption: Psychosocial and biochemical methods* (pp. 41–72). Totowa, NJ: Humana Press.
- Solowij, N., Stephens, R. S., Roffman, R. A., Babor, T., Kadden, R., Miller, M., . . . & Vendetti, J. (2002). Cognitive functioning of long-term heavy cannabis users seeking treatment. *Journal of the American Medical Association, 287*, 1123–1131.
- Stern, R. A., & White, T. (2003). *Neuropsychological Assessment Battery: Administration, scoring, and interpretation manual*. Lutz, FL: Psychological Assessment Resources.
- Strickland, T. L., Mena, I., Villanueva-Meyer, J., Miller, B. L., Cummings, J., Mehringer, C. M., . . . & Myers, H. (1993). Cerebral perfusion and neuropsychological consequences of chronic cocaine use. *Journal of Neuropsychiatry and Clinical Neurosciences, 5*, 419–427.
- Wen, H. B., Zhang, Z. X., Niu, F. S., & Li, L. (2008). [The application of Montreal Cognitive Assessment in urban Chinese residents of Beijing]. *Zhonghua Nei Ke Za Zhi, 47*(1), 36–39.
- Woicik, P. A., Moeller, S. J., Alia-Klein, N., Maloney, T., Lukasik, T. M., Yeliosof, O., . . . & Goldstein, R. Z. (2009). The neuropsychology of cocaine addiction: Recent cocaine use masks impairment. *Neuropsychopharmacology, 34*, 1112–1122.
- Wu, L. T., Pan, J. J., Blazer, D. G., Tai, B., Stitzer, M. L., Brooner, R. K., . . . & Blaine, J. D. (2009). An item response theory modeling of alcohol and marijuana dependences: A National Drug Abuse Treatment Clinical Trials Network study. *Journal of Studies on Alcohol and Drugs, 70*, 414–425.
- Zadikoff, C., Fox, S. H., Tang-Wai, D. F., Thomsen, T., de Bie, R. M., Wadia, P., . . . & Marras, C. (2008). A comparison of the Mini Mental State Exam to the Montreal Cognitive Assessment in identifying cognitive deficits in Parkinson's disease. *Movement Disorders, 23*, 297–299.

Received May 20, 2009

Revision received July 27, 2009

Accepted July 31, 2009 ■