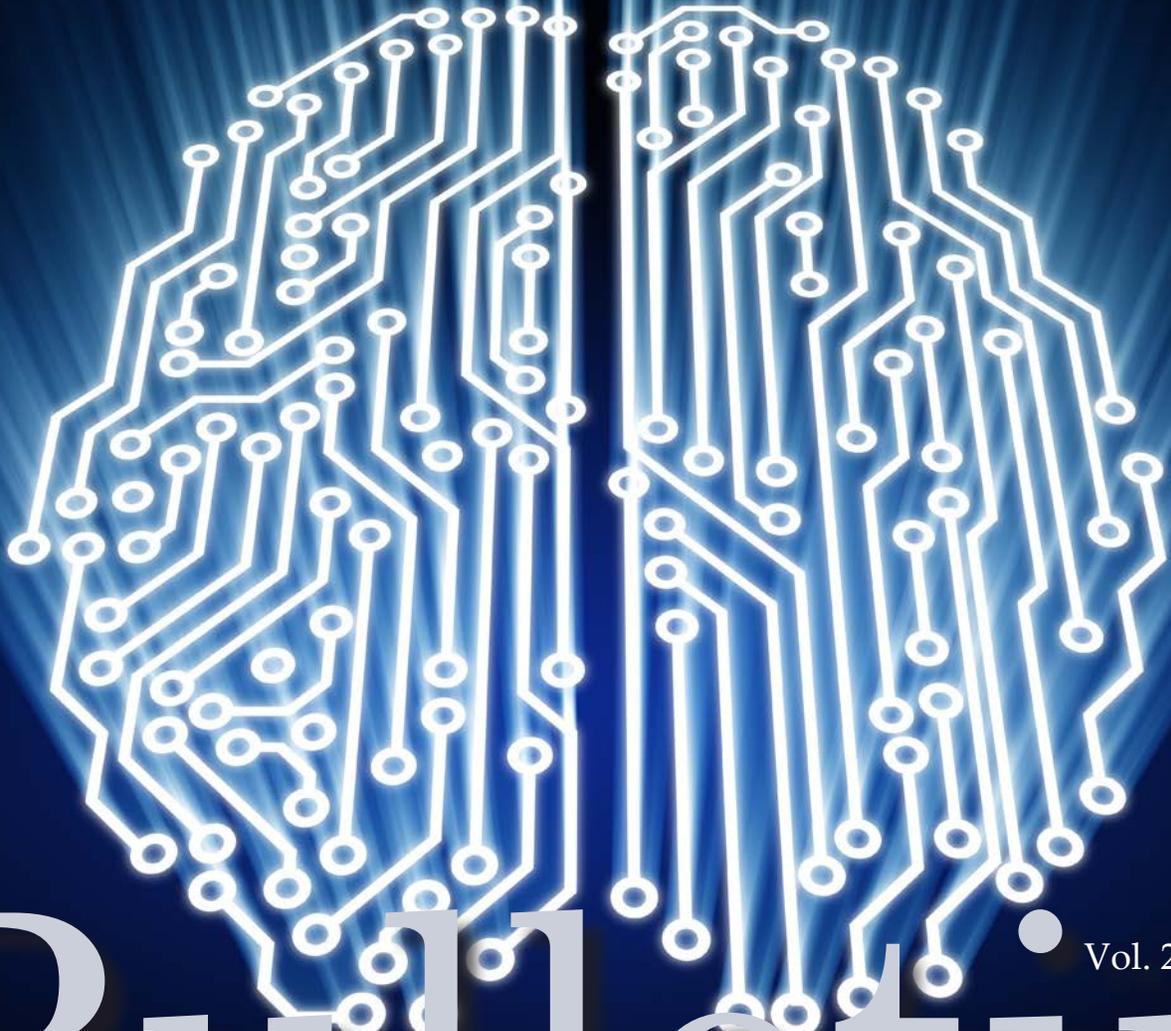


NATIONAL ACADEMY OF NEUROPSYCHOLOGY



Vol. 29 No. 2

Bulletin

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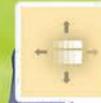
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Editor's Corner



Peter Arnett, Ph.D.,
NAN Bulletin Editor

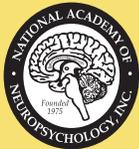
As the Editor of the *NAN Bulletin*, I am happy to present to you our latest issue, with a focus on secondary influences on neurocognitive test performance. Secondary influences are the result of something associated with brain injury or disease besides the specific areas of the brain affected. These can be contrasted to primary influences, which are the direct result of the extent and location of damage to the brain. In the Professional Issues section of this *NAN Bulletin*, three experts in the field address core issues of interest in the area of secondary influences that are relevant to practitioners. These include sleep, response expectancies, and depression. All of these factors can have a significant impact on cognitive test performance, something practitioners should be keenly aware of as they proceed with their clinical evaluations. As with other recent issues, to enhance translation of the research reviewed to clinical practice, each article in this section includes several clinical take home points.

The Student Corner section of the Bulletin includes a discussion by a current doctoral student from Australia about her experience as a graduate student at her university there. Readers will find this to be very interesting, as it provides a nice cross-cultural window into her experience there. In the Journal Section, a recent article published in the *Journal of the International Neuropsychological Society* is reviewed that focuses on neuropsychological functioning in veterans with PTSD and its impact on cognitive functioning, as well as performance validity, comorbidities, and functional outcomes. This article dovetails nicely with the secondary influences theme of this issue. Finally, we have again included a Special Topics section in this issue that includes two pieces of practical significance. One article presents a pilot study on the potential usefulness of webinars in the educational process for neuropsychologists, and the other reviews a recent FDA workshop on medical devices involving cognitive assessment (e.g., computerized cognitive batteries) and non-invasive brain stimulation (e.g., transcranial magnetic stimulation) that readers should find of great interest.

Of note, Dr. John Randolph has continued to serve as Associate Editor of the *Bulletin*, and was instrumental in working with me on completing this issue. We also appreciate the continued help from the members of the NAN Publications Committee, and welcome the new chair of this committee, Dr. Lee Ashendorf, who provided valuable input on the contributions to this issue.

Peter Arnett, Ph.D.,
Professor & Director of the Neuropsychology of Sports Concussion and MS Programs at
Penn State University
NAN Bulletin Editor

Opinions expressed by the authors and advertisers do not necessarily reflect the position of the National Academy of Neuropsychology.



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Evaluate Attention Disorders and Neurological Functioning Across the Lifespan.



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My Experience as a Graduate Clinical Neuropsychology Student in Australia

Coco Bernard
Monash University (Australia)

Hello *NAN Bulletin* readers, I am a recent (almost) graduate of the Doctorate Clinical Neuropsychology program at Monash University in Melbourne, Australia, and have been asked to share with you some of my experiences as a graduate clinical neuropsychologist student in Australia.

I think it's safe to say that no matter where you are based in the world, the journey to becoming a neuropsychologist is akin to a long, windy, and at times rocky Californian road. Nonetheless, as I approach the home stretch and reflect on this time, it is a journey I have thoroughly enjoyed. To be part of such an exciting and progressive scientific field that has a direct impact on the everyday clinical care of our patients is inspiring. Everyone I have met in this field, whether here in Australia, or at international conferences, is genuinely passionate about their work as a researcher, clinician, or scientist-practitioner. As a student, this fosters a rich learning environment that has continued to drive my thirst for knowledge and to seek experiences across a breadth of research and clinical settings. Whilst this is not something I reflect upon daily or even weekly, particular moments remind me to appreciate this (i.e. largely my friends talking about their jobs as providing merely a means to live and not offering much beyond that). So that said, I will briefly outline the process to becoming a neuropsychologist in Australia.

The pathway to becoming a neuropsychologist in Australia can be divided into three consecutive stages; a three year undergraduate degree (with psychology major), a 'fourth year' which can be undertaken either through an Honours or Graduate Diploma, and a post-graduate training program (Masters, Master/PhD Combined, or a Doctorate or 'DPsych'). As such, it is a minimum six year (Masters) or eight year (Combined or Doctoral degree) process, if undertaken full-time. Psychology is extremely popular in Australia, and following the basic laws of supply and demand, progressing through each stage is competitive.

Currently, there are six post-graduate psychology training programs across Australia that offer the neuropsychology specialty, and is not uncommon for students to travel interstate to complete all or part of their degree. The programs are predominately based in Melbourne with one program in each of Sydney, Perth and Brisbane. Somewhere between 6 and 20 students will graduate from each program annually. Upon completion of the post-graduate degree, you gain registration as a General Psychologist. You are then required to undertake one year (Doctorate) or two years (Masters) of clinical practice – fulfilling a range of supervision/CD requirements – before you gain 'endorsement' as a Clinical Neuropsychologist.

So you've finally graduated!?! What next? Well first... you drink several bottles of champagne, put your thesis in a dark hole at the back of your bookshelf, and perhaps sleep for a solid 20 hours. Then, it is time to find a job.

Your first real job...that will actually pay you money! What a great feeling. In Australia, neuropsychologists are employed across a wide range of pediatric, adult, and older adult settings. They can work in acute inpatient hospital settings, sub-acute and rehabilitation facilities (inpatient and outpatient), community clinics, education settings, private practice (and medico-legal settings), and even within managerial or governance based roles. In addition, many hold con-joint research positions within universities or other institutions.

Landing the first position is probably the most challenging, and most students will take on temporary (e.g. maternity leave/leave of absence replacement) or part-time work to get their 'foot in the door'. From conversations I have had with new graduates, this on average takes anywhere from 3 – 6 months. New graduates in Australia are encouraged to work in a diverse range of areas to begin with, so they can continue to gain experience whilst they figure out their 'niche' and hone their skills. There has also been a recent push for neuropsychologists in Australia to develop more skills and confidence in delivering targeted intervention and ongoing therapy, so as to increase our 'employability' and utilise our (incredible) skills more broadly across different areas of the health care sector.

So as I progress from a piece of furniture in the university student room to a new graduate, I have also come to appreciate the importance of a supportive student culture. Given we have three training programs in Melbourne, we have been fortunate to be part of a great student culture driven by various student bodies, but importantly also facilitated and heavily supported by more experienced professions in the field. There are both state and national student bodies that operate within the national College of Clinical Neuropsychologists (equivalent to NAN), as well as local university-run groups (Neuropsychology Students' Society) and student committees that operate within discipline specific bodies (e.g. Australian Society for the Study of Brain Impairment). These groups are constantly hosting seminars, lectures, workshops and social events that are either free or heavily discounted, and provide a great place for the student neuropsychology community to congregate, share stories, and make connections with key neuropsychology figures in Australia. Personally, these sessions really complement our coursework, research, and clinical experiences and have been a real highlight for me.

I have thoroughly enjoyed my 8 years training (well... technically 10) to become a neuropsychologist in Australia and enter the next phase with anticipation and excitement. Please feel free to get in contact with me if you want any more details about Neuropsychological training in Australia, or even just to have a chat. I check my email way too frequently, so you are bound to get a quick response. My best contact is cocobernard@yahoo.com.



Coco Bernard is about to submit her thesis, following which she will have completed the Doctorate of Clinical Neuropsychology from Monash University, Australia. In addition to her studies, she works as a part-time researcher within the Monash Epworth Rehabilitation Research Centre, conducting follow up assessments with adults following traumatic brain injury. She is also the Mid-Year Programs Representative within the International Neuropsychological Society Student Liaison Committee, where her role has been to organize student events at the past two mid-year meetings in Sydney (2015) and London (2016), and has recently commenced planning for the upcoming INS 50th Anniversary meeting in Cape Town (July 5 – 8th, 2017).



HONE-In

Health Outcomes and Neuropsychology Efficacy Initiative

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Neuropsychologists are increasingly being asked to provide evidence of effectiveness to support reimbursement for neuropsychological services, yet this information is not always easily accessible to neuropsychology practitioners. In response to this challenge, the National Academy of Neuropsychology (NAN) authorized its Legislative Action and Advocacy Committee (LAAC) to launch an initiative that would help NAN membership respond to these practice challenges. The result was the Health Outcomes and Neuropsychology Efficacy Initiative (HONE-In).

The primary goal of HONE-In is to assist NAN membership in any effort to demonstrate the value of neuropsychological services through cost effectiveness and/or cost savings.

HONE-In Phase I Sample Article Summary

BRAIN INJURY, CONCUSSION, REHABILITATION

The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury.

Population: Traumatic brain injury, Inpatient rehabilitation

Categories: Outcome prediction

Authors: Hanks RA, Millis SR, Ricker JH, Giacino JT, Nakase-Richardson R, Frol AB, Novack TA, Kalmar K, Sherer M, Gordon WA.

Date: 2008

Title: The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury

Type: Journal article

Citation: Hanks, R. A., Millis, S. R., Ricker, J. H., Giacino, J. T., Nakase-Richardson, R., Frol, A. B., et al. (2008). The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury. *Archives Of Physical Medicine And Rehabilitation*, 89(5), 950-957.

Utility: Prospective study of predictive validity of NP assessment during subacute brain injury rehab, including pts in PTA, within ~ 1 month of injury. Brief NP assessment predicted handicap, functional outcome, supervision needs, employability in adults w/ TBI at 1 year. Adding NP increased predictive power over injury severity and early functional status (with exceptions – SWLS and FIM Motor). Including those w/ PTA did not diminish predictive validity. Findings important given trend toward shorter rehab stays, strengthens argument for role of NP testing during acute rehab.

Synopsis and review of: Wrocklage, Schweinsburg, Krystal et al. (2016).
Neuropsychological functioning in veterans with posttraumatic stress disorder:
Associations with performance validity, comorbidities, and functional outcomes.
Journal of the International Neuropsychological Society, 22, 399-411.

Review by John Randolph, Ph.D., Geisel School of Medicine at Dartmouth

Study Rationale:

Neuropsychological research has found that those with PTSD can experience episodic memory, attentional, executive functioning, and processing speed deficits. However, previous work has been inconsistent regarding the scope and magnitude of cognitive dysfunction in individuals with PTSD. Factors that have not always been accounted for in earlier studies include psychiatric comorbidity (including comorbid substance use disorders, depression, and ADHD), history of TBI, and performance validity. Further, PTSD “caseness” has at times been established via self-report questionnaire data, rather than through a formal diagnostic interview. As Wrocklage et al. indicate, any of these factors could impact cognition, thereby calling into question prior research that has not taken secondary factors into account. Finally, the functional significance of cognitive dysfunction in PTSD has rarely been examined.

Overarching Goal:

The authors sought to improve upon prior work by using the Clinician Administered PTSD Scale (CAPS), a “gold standard” diagnostic interview for PTSD, administering multiple performance validity measures, and examining functional outcomes and quality of life related to cognitive dysfunction in veterans with PTSD.

Methods:

Study participants included 44 veterans with CAPS-diagnosed PTSD and 40 demographically matched combat-exposed veterans (TC, or trauma comparison group). Participants with a history of moderate to severe TBI, ADHD, learning disorder, psychotic disorder, or bipolar disorder, and/or current benzodiazepine use were excluded from the study. The authors assessed combat exposure, depression, military-related concussions, neuropsychological functioning (including performance validity), work performance, and quality of life. Four PTSD group participants were excluded from primary analyses due to insufficient effort on at least one performance validity test. A subsample of the overall PTSD group was employed and completed the work performance measure.

Results:

MANOVAs were conducted that revealed differences between PTSD and TC participants regarding processing speed and executive functioning but not attention/working memory, verbal/language functioning, visuoconstruction, or episodic memory. Specifically, veterans with PTSD showed worse performance on WAIS-IV Coding and Symbol Search, DKEFS Trails Number Sequencing, and DKEFS Color-Word Inhibition.

Subsequent MANCOVAs indicated that neither depression nor history of mild TBI accounted for effects of PTSD on processing speed or executive functioning. Current substance use disorders did impact executive functioning, although PTSD remained associated with executive dysfunction after controlling for this variable. Current or lifetime substance use disorders were not related to processing speed deficits.

The authors then examined relationships between cognition and specific PTSD symptom clusters across the entire sample. These analyses revealed that executive functioning was negatively correlated with the CAPS Total score, as well as with emotional numbing and hyperarousal CAPS factors. Processing speed was associated with the CAPS hyperarousal factor, but not other factors or the total score.

Regarding analyses related to perceptions of daily functioning, processing speed and executive functioning were positively correlated with physical health-related quality of life. The CAPS Total score negatively correlated with mental health quality of life and “presenteeism”—being physically present at work but not being engaged at work.

Conclusions:

In summary, the authors found that participants with PTSD showed information processing speed and executive function deficits “of a medium magnitude” in their well-characterized sample relative to trauma controls. They argued that their findings were consistent not only with a recent meta-analysis in this area but also with proposed fronto-limbic/inhibitory control dysfunction in PTSD.

The results were divergent from other studies in the literature, particularly regarding the lack of episodic memory and attentional deficits in their PTSD group. They noted that this may relate to their strict exclusion criteria, assessment of performance validity, young mean sample age (35.2 years), use of a formal diagnostic interview for PTSD, and lack of assessment of prose memory. As the authors note, their sample was of moderate size, and informant reports were not included, potentially limiting some of their conclusions. They also had a small sample that completed the work questionnaire (less than half of the PTSD group), suggesting that their related findings should be viewed as tentative.

This provocative study suggests that cognitive dysfunction in PTSD is present and can impact multiple outcomes in daily life. While questions remain in this area, this work suggests that clinicians should be mindful not only of the possibility of cognitive impairment in veterans and others with PTSD, but

that such impairment can influence both quality of life and functional status. More generally, these findings highlight the importance of secondary factors in understanding cognition in neuropsychiatric populations.



Dr. John Randolph is a board-certified clinical neuropsychologist in independent practice and Adjunct Assistant Professor of Psychiatry at the Geisel School of Medicine at Dartmouth. He earned his Ph.D. in Clinical Psychology (Neuropsychology specialization) from Washington State University, and completed clinical and research fellowships in Neuropsychology and Neuroimaging at the Geisel School of Medicine at Dartmouth. His research has focused on metacognition, executive functioning, cognitive and neuroimaging aspects of multiple sclerosis, and contributors to cognitive health, and he has received grant funding from the National MS Society and NIH. He is Past President of the New Hampshire Psychological Association, a National Academy of Neuropsychology Fellow, past recipient of the NAN Early Career Service Award, and editor of the recent book, *Positive Neuropsychology: Evidence-Based Perspectives on Promoting Cognitive Health*.



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The Role of Sleep in Neuropsychological Assessment

Jessica Zamzow, Ph.D.

University of California, Los Angeles Semel Institute for Neuroscience and Human Behavior

Maria Schultheis, Ph.D.

Drexel University, Department of Psychology and the School of Biomedical Engineering, Science and Health Systems

The Role of Sleep in Neuropsychological Assessment

Insufficient sleep is very common in modern society, in which stress, social and occupational demands, caffeine and alcohol consumption, psychiatric disorders, medical conditions, and sleep disorders contribute to about 35 to 40% of U.S. adults obtaining less than the recommended 7 to 8 hours of sleep per night.¹ Poor sleep quality and sleep disorders are especially prevalent in those populations often seen by neuropsychologists, due to the role of the central nervous system (CNS) in sleep/wake regulation and the effects of medical and mental health co-morbidities on sleep. A large proportion of individuals diagnosed with epilepsy (40-51%), Alzheimer's disease (40-60%), parkinsonism (approximately 60%), multiple sclerosis (MS; over 50%), Attention Deficit Hyperactivity Disorder (ADHD; up to 70%) traumatic brain injury (TBI; 46-72%), and stroke (20-50%) experience significant sleep difficulties.² This is particularly relevant for neuropsychologists given that short sleep duration, poor sleep quality, and the effects of sleep disorders can all negatively impact cognitive functioning. This can make it difficult to differentiate between potentially transient and treatable deficits related to sleep versus impairments related to a neuropsychological disorder. In order to help differentiate these effects, neuropsychologists must be knowledgeable about these relationships and routinely consider the role of sleep in clinical assessment and research.

Sleep deprivation has the largest effects on attention, vigilance, processing speed, and working memory, with smaller, less reliable effects on short-term memory, verbal fluency, language and executive functioning.³ This is consistent with functional neuroimaging research showing decreased activation in fronto-parietal attentional systems following sleep deprivation.⁴ Of note, there is individual variability in both sleep need and resiliency to the cognitive effects of sleep loss.

Long sleep duration is also associated with cognitive impairments, particularly in older adults.⁵ The nature of the relationship between long sleep and cognitive difficulties is not well understood. In some cases, excessive sleepiness may arise secondary to a pathological processes (e.g., inflammation) or an underlying sleep disorder (e.g., sleep apnea), both of which may independently contribute to cognitive decline.

Sleep Disorders

The relationship between sleep disorders, neuropathology, and cognitive function is complex. Sleep difficulties are highly prevalent among individuals with neurological disorders.⁶ Certain

sleep disorders, such as narcolepsy, REM behavior disorder (RBD), restless leg syndrome (RLS), periodic leg movement disorder (PLMD), central sleep apnea, and circadian rhythm disorders can arise from CNS pathology and may occur secondary to neurological conditions such as parkinsonism, MS, TBI, or stroke. Furthermore, a growing body of literature indicates that sleep disorders can also negatively impact cognitive function.

Insomnia disorder is the most prevalent sleep disorder, characterized by difficulties falling asleep or maintaining satisfactory sleep, despite opportunity for adequate sleep.⁷ Insomnia has been associated with moderate impairments in working memory, episodic memory, problem solving, and selective attention.⁸ RLS, characterized by unpleasant sensations in the lower extremities accompanied by an urge to move one's legs and PLMD, characterized by periodic limb movements during sleep cause frequent awakenings, sleep disturbance, and daytime sleepiness.⁶ Research on the effects of RLS and PLMD on cognitive function is limited and mixed, but there is evidence that RLS and PLMD are associated with cognitive decrements in executive function, likely due to the effects of chronic sleep loss and fragmentation.⁹

Circadian rhythm disorders are characterized by a misalignment between an individual's endogenous sleep-wake pattern and their required sleep-wake schedule.⁷ Individuals with advanced or delayed circadian rhythms may perform better on cognitive tasks in the morning or evening, respectively, due to circadian peaks and low points in alertness throughout the day.¹⁰

Sleep apnea is characterized by recurrent episodes of halted or reduced breathing due to airway restriction during sleep.⁶ Neurocognitive impairments in sleep apnea are associated with sleep fragmentation, hypoxemia, vascular burden, and inflammatory processes.^{11,12} Sleep apnea is associated with moderate to large effects on tests of sustained attention, psychomotor speed, working memory, novel problem solving, inhibitory control, and verbal fluency with smaller effects on delayed recall and processing speed.¹³

Narcolepsy is characterized by excessive sleepiness, episodes of irresistible sleep, cataplexy, and/or hypnagogic hallucinations.⁶ Findings of cognitive difficulties in narcolepsy are mixed, but suggest performance deficits in vigilance, attention, and memory, related to degree of sleepiness.¹³

RBD is characterized by complex motor behaviors during dreaming, which is due to a lack of atonia during REM sleep.⁶ Some individuals with idiopathic RBD experience visuo-spatial impairments.¹⁴ Visuo-spatial deficits in idiopathic RBD may signify the prodromal phase of a synucleinopathy, which the majority of individuals with RBD eventually develop.

Assessment

The Pittsburgh Sleep Quality Index (PSQI) questionnaire is commonly used to assess sleep difficulties.¹⁵ The PSQI measures habitual sleep duration, efficiency, quality, causes of sleep disturbances, sleep medication use, and daytime sleepiness. The PSQI also includes an optional informant report of sleep behaviors, such as movement and gasping for air. If poor sleep quality is indicated (PSQI total score > 5), additional assessment may be provided to understand the nature of the individual's sleep difficulties and to screen for potential sleep disorders.¹⁵ In addition to assessment of habitual sleep behavior, brief assessment of sleep the night before testing and state sleepiness is also informative given the effects of acute sleep deprivation and the potential for recovery from habitual sleep loss. Clinicians may wish to consider using the Insomnia Severity Index to assess for symptoms of insomnia disorder, the STOP-BANG questionnaire to assess for risk of sleep apnea, or the Global Sleep Assessment Questionnaire for a brief, global assessment of symptoms of multiple sleep

disorders (i.e., sleep apnea, insomnia, RLS, PLMD, parasomnias, and circadian rhythm disorders). If an individual reports a diagnosis of a sleep disorder, it is prudent to ask about treatment efficacy and compliance to understand the extent to which the sleep disorder may impact the neuropsychological assessment.

Implications

Sleep deprivation and sleep disorders can decrease validity of neuropsychological assessment. Assessment of the nature and severity of sleep difficulties and consideration of the typical pattern of deficits associated with various sleep problems can help the clinician to parse out the effects of sleep on the neuropsychological evaluation.

The literature suggests that many individuals with sleep disorders are undiagnosed. This is particularly concerning given the consequences of untreated sleep disorders on one's cognitive functioning, physical health, quality of life, and mortality. Treatment of sleep disorders may even ameliorate sleep-dependent cognitive deficits.¹¹ Sleep disorders are highly treatable, but proper assessment and diagnosis is important for optimal treatment. Clinicians working with populations at heightened risk for sleep disorders should regularly screen patients for symptoms of sleep disorders and may refer to a sleep specialist for additional assessment, diagnosis, and treatment if a sleep disorder is suspected.

Clinical Take Home Points:

1. Sufficient sleep quality and duration are necessary for optimal cognitive function.
2. Sleep disorders are generally associated with cognitive difficulties, particularly in the domains of attention and vigilance.
3. Clinicians may wish to consider the PSQI, Insomnia Severity, STOP BANG questionnaire, and/or the Global Sleep Assessment Questionnaire in assessing for sleep during a neuropsychological assessment.
4. Sleep disorders can often go undiagnosed, but are also highly treatable. If an undiagnosed sleep disorder is suspected, neuropsychologists may refer to a sleep specialist who can provide comprehensive assessment, diagnosis, and treatment.



Jessica Zamzow received her Ph.D. in clinical psychology from Drexel University in 2016. She is currently a Post-Doctoral Scholar at the University of California, Los Angeles Semel Institute for Neuroscience and Human Behavior. Her current research focuses on the role of sleep and sleep disorders in cognitive function. She and Dr. Maria Schultheis received a grant from

the National Multiple Sclerosis Society, which helped to fund her dissertation research, examining the association between sleep and cognitive function in relapsing-remitting multiple sclerosis.



Maria Teresa Schultheis is a Professor with appointments in the Department of Psychology and the School of Biomedical Engineering, Science and Health Systems. At Drexel University, she serves as the Head of the Department. Dr. Schultheis' clinical and research experience have been focused on the rehabilitation of cognitively impaired populations, including traumatic brain injury, stroke and multiple sclerosis. A main focus of her research is studying the demands of driving following neurological compromise, which includes the development of new virtual reality driving assessment protocols.

Dr. Schultheis' work was recognized early in her career in student awards from the National Academy of Neuropsychology and the Philadelphia Neuropsychological Society. She is also the recipient of the 2007 American Psychological Association Early Career Award for Division 40 (Clinical Neuropsychology) and is a Fellow of APA (Div 40 Society for Clinical Neuropsychology).

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Illness Identity and Its Implications for Neuropsychological Assessment

Julie Suhr, Ph.D.
Ohio University

I have long felt a conflict between my belief that training in clinical psychology and neuropsychology should cover a breadth of psychological and non-psychological material and my feeling that there are simply too many breadth requirements for students to complete training programs in a timely manner. I still err on the side of retaining my research laboratory's long-held motto, which is "keep the psychology in neuropsychology" and feel this applies equally to both the science and practice of clinical neuropsychology. In fact, research in the areas of health psychology, cognitive psychology, and social psychology led me to the development of a conceptual model of illness identity as a way of potentially explaining some aspects of noncredible symptom/history report and noncredible performance on neuropsychological tests. In the following, I will describe aspects of the model and their implications for neuropsychological assessment.

Consistent with the core predictions of the Common Sense Model (CSM) of Illness (1), patients' illness beliefs (such as controllability or chronicity of their illness) have been shown to be related to clinical outcomes in many medical disorders. Recently, researchers have shown that CSM illness beliefs are also related to neuropsychological outcomes (2-5). In addition, work in our laboratory has led us to suggest extensions to CSM that have implications not only for health outcomes, but for assessment of individuals who present for evaluation, due to their influence on patients' presentations of their history, report of their current symptoms and impairments, and their behavior on neuropsychological tests (4-5).

In the original CSM, illness beliefs originate from sources of illness information in one's broader culture. In fact, the original CSM was applied to individuals with actual medical illness; their beliefs about how that illness presented itself in terms of severity, chronicity, controllability, etc. were assumed to emerge from what they learned about their illness from various healthcare and non-healthcare sources. Illness beliefs are not that difficult to develop in the modern world, where access to both accurate and inaccurate medical and health information is instantly accessible on the internet and through pharmaceutical commercials, as well as more the traditional routes of family, friends, and healthcare providers. In fact, for some of the top neuropsychological referral questions (mild traumatic brain injury (TBI)/concussion, Attention Deficit/Hyperactivity Disorder (ADHD), and early signs of dementia), both accurate and inaccurate illness information is readily available. It is our belief that inaccurate sources of illness information may suggest to individuals that high base rate and nonspecific experiences are actually unique symptoms of a particular disorder, which creates the illness identity itself (a belief that one HAS a particular illness), in addition to developing beliefs about how that illness may manifest in their everyday life (associated illness beliefs). Individuals who have adopted an illness identity may present to a neuropsychological evaluation with

a) a strong pre-existing belief in what their diagnosis is ("I think I have X"), b) reports of both past history and current symptoms and experiences in light of the illness identity and related illness beliefs, and c) behavior on neuropsychological tests that is consistent with the illness identity and associated illness beliefs. In effect, exposure to various sources of illness information may have created a distorted illness identity lens through which individuals view their everyday experiences, affecting their overall presentation during the evaluation. It is the neuropsychologist's role to pay attention to whether the individual being assessed is wearing illness identity lenses and the degree to which those lenses affect the interpretability of data gathered during the assessment.

We propose that additional mechanisms can strengthen illness identity lenses, which are more fully explained (and applied specifically to ADHD) in a recent publication (5), but which I will summarize below. We proposed these mechanisms based on research findings in cognitive, social, and health psychology, as well as work conducted by other neuropsychologists, and we simply applied them to the context of the CSM and to neuropsychological presentations.

We refer to the first mechanism as an attentional lens, which leads an individual to selectively focus on information consistent with the illness identity and disregard information inconsistent with the illness identity. Consistent with the well-documented cognitive psychology phenomenon of attribution bias, the attentional lens results in attribution of high base rate experiences to the perceived illness. Classic work by Mittenberg and colleagues (6) demonstrated how the attentional lens can operate within mild TBI, relating not only to overreport of current symptoms but also underreport of symptoms prior to the TBI. Their work illustrates well how an attentional lens can affect not only a patient's report of current functioning and impairment, but report of their history. Our lab has also demonstrated how the attentional lens might operate in self-report of ADHD symptoms and memory concerns in dementia screening. The implications of the attentional lens for clinical practice are likely obvious, in that they point to the major limitation of relying only on self-report of history, current symptoms, and past/current functioning. Patients wearing an attentional lens will report only that which is consistent with the illness identity, and thus the neuropsychologist must work hard to consider all alternative explanations for the same symptoms, as well as ask about presence/absence of symptoms and history that would both confirm AND disconfirm the illness identity.

The second lens that we added to the CSM is the emotional lens, which is a tendency for an individual to interpret something that is benign as being more pathological and thus distressing. For example, an experience of blanking on someone's name is more distressing when viewed through a dementia illness identity than a normal aging identity, and forgetting an appointment is much

more distressing if one is viewing the action through a concussion identity than considering lack of sleep and a busy stressful day as potential sources of this common error. The emotional lens adds additional nonspecific distress-related symptoms (anxiety, worry, depression) that can also get attributed to the illness identity, which further reinforces and expands the illness identity. There are clear implications of the emotional lens for clinical practice. Individuals affected by the emotional lens are likely to report their symptoms at greater severity or as causing more distress and impairment, key criteria for determining whether symptoms are clinically significant/meet diagnostic criteria or whether the individual is in need of treatment or accommodation for the symptoms. Given the clinical implications of the emotional lens, neuropsychologists should use self-report measures that include validity scales assessing for overreport of symptomatology, and consider whether other assessment evidence is congruent or incongruent with the severity of the self-reported symptoms and level of dysfunction.

While many readers may be familiar with the psychological mechanisms underlying the first two lenses, they may be less familiar with the third, which is self-handicapping. Self-handicapping arose out of social psychology research, although even early on Adler described the potential for this psychological mechanism to explain self-reported psychological symptoms. Self-handicapping is the tendency for individuals to provide themselves with premorbid excuses for potential failure experiences, particularly when those experiences are highly valued by the individual (7). For example, in sports psychology, research has shown that athletes have a tendency to provide "handicaps" ("I hurt my ankle yesterday, I didn't get enough rest") prior to participating in a competitive event. Self-handicapping allows an individual to maintain a self-image of general competence and ability, as well as a view of oneself as functioning as best as one can despite a barrier (the handicap). In this way, self-handicapping allows a person to protect their self-esteem in the case of actual failure ("I would have succeeded if only my ankle wasn't injured") or even enhance self-esteem in the case of success ("I won even though I didn't sleep enough"). Researchers have also suggested that self-handicapping is reinforcing by allowing individuals to avoid threatening activities ("I'll have to drop out of competition because of my ankle") and manage their public impression (when others are made aware of the "handicap" through self-report or behavioral presentation). What we and others have argued is that having a psychological (or neuropsychological) illness identity can serve as a self-handicap. Some find self-handicapping paradoxical, in that they can't understand why anyone would want to have a diagnosis. However, research does suggest that, particularly in the context of evaluation, which may pose a more significant blow to one's self esteem if failure is attributed to more central characteristics (intelligence), it is in fact reinforcing to believe that you could do better if only you didn't have a certain diagnosis (such as ADHD, 8-9). The clinical implications of this third lens are similar to the other two lenses (self-reported past and/or current symptoms of increasing frequency, severity, and with higher levels of distress/impairment).

Finally, an illness identity and associated illness beliefs operate not only on an individual's self-reported history, current symptoms,

and ratings of impairment, but also set up expectancies for how an individual should function, not only in real life, but also within the context of a neuropsychological evaluation. The original CSM focused on illness beliefs that led an individual to cope with an illness differently, ultimately affecting long-term outcome. For example, having an illness belief that a disease is chronic and uncontrollable is likely to lead to poor coping and maladaptive outcome. As noted before, recent research has also shown that illness beliefs/expectancies as identified by CSM are related to outcome in mild TBI (2,3). However, we have argued that expectancy beliefs also play a role in how patients behave on neuropsychological tests (through mechanisms such as diagnosis threat; 4,5). Response expectancies are automatic emotional, physical, or behavioral reactions to specific situational cues. In the context of a neuropsychological evaluation, an individual with an illness identity has certain expectancies for their own cognitive performance; being administered measures of those cognitive domains may elicit behavior that results in performing in ways consistent with the response expectancies. Reviews of the evidence for response expectancy effects on neuropsychological test performance can be found in (4,10). What has not yet been clearly demonstrated is whether these effects can be identified on performance validity tests; this is an area ripe for additional study. However, at the very least, knowing that non-neurological mechanisms have an impact on neuropsychological test performance points to the need for inclusion of performance validity tests (in addition to symptom validity tests) in all neuropsychological evaluations.

Overall, it is important to note that individuals with strong illness identities not only have intent to convince others that they are experiencing impairments associated with a particular disorder, but also are convinced of this themselves. In this way they are a contrast to patients who are malingering, for whom the deception is other-directed rather than self-directed (11). Nevertheless, if their illness identity leads to a noncredible report of history, current symptoms/impairment, and noncredible behavior on tests, and the neuropsychologist has not tested for the validity, misdiagnosis and inappropriate treatment may result.

Given that the mechanisms associated with development and reinforcement of an illness identity are universal in nature, anyone could develop an illness identity, given the right circumstances. However, research suggests that some individuals may be more vulnerable to the development of an illness identity than others. Health psychology research suggests that individuals with somatization tendencies are more vulnerable to the attentional lens and the emotional lens, and are more vulnerable to response expectancies (see 4 for a review). Nevertheless, the research I have reviewed here suggests these mechanisms can operate in all individuals (including those who actually have the neuropsychological conditions around which they have developed a maladaptive illness identity). Research in this area emphasizes the need for the clinical neuropsychologist to take a comprehensive biopsychosocial approach to interviewing and assessing individuals who present with high base rate, nonspecific symptoms and a diagnostic label that they are already convinced applies to them, and for inclusion of measures of noncredible report and noncredible behavior in every neuropsychological evaluation.

Clinical Take Home Points:

1. Consider carefully whether an illness identity lens is affecting patient report of their past, their current symptoms, and their current distress/impairment.
2. Assess for the validity of self-report.
3. Include assessment beyond self-report to integrate with self-reported history, current symptomatology, and current impairment.
4. Assess for the validity of neuropsychological test results by including performance validity tests in every assessment.
5. Remember that invalidity of self-report or neuropsychological performance may not indicate malingering, but other psychological mechanisms that perhaps can be addressed therapeutically.



Julie Suhr completed a Ph.D. in clinical psychology in 1994 from the University of Iowa. She also completed three years of postdoctoral fellowship in clinical neuropsychology at the University of Iowa Carver College of Medicine Department of Neurology before joining the faculty at Ohio University in 1997, where she is now a Professor of Psychology and Director of Clinical Training for their doctoral program in clinical psychology. She has authored or co-authored over 70 peer reviewed journal articles on topics in various areas of neuropsychological assessment, and also wrote a text on assessment, [Empirically Based](#)

[Assessment: A problem-Solving Approach](#), published in 2015. She also serves as Associate Editor for *Psychological Assessment* and *Journal of Clinical and Experimental Neuropsychology*, and is on the editorial board for *The Clinical Neuropsychologist*. She is a fellow of the American Psychological Association.

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Neuropsychological Deficits in Major Depressive Disorder: A Brief Review

Michael R. Basso, Bradley Reynolds, Lily Lau, Ashley Miller, Eduardo Estevis
University of Tulsa

Dennis Combs
University of Texas at Tyler

Douglas Whiteside
University of Iowa

People diagnosed with major depressive disorder (MDD) manifest diminished functional, vocational, and medical outcomes. These problems occur during acute depressive episodes and in periods of remission, implying that MDD is a chronic syndrome characterized by considerable morbidity. It is further marked by cerebral abnormalities. Reduced brain volumes have been observed in orbital and dorsal lateral frontal cortex, anterior cingulate, hippocampus, insula, amygdala, cerebellum, and basal ganglia structures, with abnormalities involving frontal structures being most pronounced (1, 2). White matter tracts are also degraded, especially those connecting prefrontal regions with limbic and thalamic structures (3). These structural anomalies coincide with functional abnormalities. Hypometabolism occurs in pre-frontal cortex, medial frontal structures, and the basal ganglia, and hypermetabolism appears in anterior subgenual cingulate cortex (2). These abnormalities persist despite symptom remission but to a lesser extent than during acute illness, especially in frontal-striatal and limbic systems (4).

A compelling body of research reveals that MDD results in significant neuropsychological impairment. At least 15 meta-analyses have examined neuropsychological function in people with MDD, thereby providing an empirical summary of findings. Among the first of these studies, Burt et al. (5) found moderate to large effect sizes for differences between patients and healthy individuals on measures of recall and recognition memory. Notably, depressed patients learned and remembered as poorly as schizophrenics, but not as poorly as patients with Alzheimer's disease. Inpatients performed worse than outpatients, and outpatients generally performed normally. Recall was worse for visual than verbal material.

Expanding upon the review by Burt et al. (5), Christensen et al. (6) conducted a meta-analysis of 154 papers. They found moderately large effect sizes, and depressed patients performed worse than controls on measures of executive function, memory, working memory, and speed of information processing. Neuropsychological test difficulty failed to moderate differences between depressed and non-depressed individuals, and recognition memory and recall memory were equally impaired. Christensen et al. further concluded that reduced effort or motivation fails to account for neuropsychological deficits in MDD.

Veiel (7) conducted a broad meta-analysis of 13 studies which excluded elderly patients. Depression achieved moderate effects upon recall and retention of verbal and visual information, and

it had large effects on executive function. Collapsing across neuropsychological domains, 40% of MDD patients were impaired, performing at the 2nd percentile of control subjects.

Two meta-analyses focused upon elderly depressives. Kindermann and Brown (8) studied memory function across 40 studies, finding an average effect size of 0.6. Medication had no impact on memory function. In Herrmann et al.'s (9) meta-analysis of 10 studies, patients whose MDD began prior to age 50 were compared to those who became symptomatic after 50. On measures of executive function and speed of information processing, patients with late-onset depression performed worse than those who became symptomatic during young adulthood. Collectively, these data suggest that cognitive impairment is present in elderly patients with MDD, but those who sustain a first episode during senescence manifest worse dysfunction than those with a longer history of MDD.

It is apparent from these results that prevalence of neuropsychological impairment varies across depressed patients. Relevant to this implication, approximately 25% of patients with MDD display psychotic symptoms (primarily delusions of guilt), and such individuals experience greater morbidity than those without psychotic features. Two meta-analyses have addressed presence of psychotic features as a vulnerability for neuropsychological impairment (10, 11). Presence of psychotic features corresponded with pronounced dysfunction compared to non-psychotic depression, especially on measures of psychomotor speed, executive function, and memory. Moderate effect sizes were observed on measures of working memory and visual-spatial reasoning. Thus, psychotic features connote greater neurocognitive morbidity, and they serve to moderate which depressed patients manifest deficits.

McDermott and Ebmeier (12) examined whether depressive severity predicted cognitive impairment in a meta-analysis of 14 studies. Increasing severity correlated with worsening executive function, psychomotor speed, and verbal memory. Other domains of neurocognitive function failed to correlate with depressive severity. Moreover, the magnitude of effects was modest, and depressive severity accounted for approximately 10% of the variance in neuropsychological function.

Zaninotto et al. (13) conducted a meta-analysis concerning effects of melancholia on neuropsychological function in MDD. Among the nine studies, melancholia was associated with deficits involving executive function, working memory, visual learning,

and processing speed. The effect sizes were generally moderate, and were not better accounted for by age or symptom severity. Hence, melancholia seemed to have a unique impact on neuropsychological function in MDD.

Although these meta-analyses reveal neuropsychological impairment is common in MDD, they provide no indication concerning the durability of cognitive deficits. Lee et al. (14) conducted a meta-analysis involving 13 studies concerning individuals with a first episode of MDD. Medium to small effect sizes between the patients and healthy individuals were observed on measures of executive function, working memory, visual learning, and psychomotor speed. Hence, dysfunction seems to emerge early during the course of the disorder. In Douglas and Porter's (15) meta-analysis of 30 studies, neuropsychological function across time was investigated. As symptoms remitted, verbal memory improved. There was no clear relationship between changes in depressed mood and visual memory. Regarding executive function, working-memory, and psychomotor speed, poor performance persisted despite symptom reduction, even two-years after the initial examination. Notably, however, verbal

and figural fluency improved as depressive symptoms remitted. These aspects of executive function were the sole measures that differentiated patients who responded to treatment from those who retained residual symptoms. Overall, these data imply that some form of residual brain dysfunction remains despite symptom remission.

Most of the aforementioned investigations reveal that MDD yields potent deficits on executive function. Subsequent studies have sought to further clarify effects of MDD on executive function. Henry and Crawford (16) conducted a meta-analysis of 42 studies concerning verbal fluency. Large effects were observed with semantic fluency but modest effects were seen with phonemic fluency. Snyder (17) conducted a meta-analysis of 113 studies concerning executive function in MDD. She found moderate to large effect sizes, with inhibition tasks such as the Stroop having larger effects than concept formation tasks such as the Wisconsin Card Sorting Test. Planning and problem-solving was indexed by the Tower of London, and effects sizes due to depression were small in this domain.

Conclusions

- Neuropsychological dysfunction occurs commonly in people with MDD, with patients tending to perform at least .5 standard deviations below the mean of healthy individuals.
- As summarized in Table 1, impairment is neither uniform across neuropsychological domains nor global. Rather, deficits appear specific to certain domains.

<u>Cognitive Domains Often Impaired in MDD</u>	<u>Cognitive Domains Sometimes Impaired in MDD</u>	<u>Cognitive Domains Infrequently Impaired in MDD</u>
Mental Flexibility	Concept Formation	Intelligence
Semantic Fluency	Phonemic Fluency	Receptive Language
Working Memory	Psychomotor Speed	Object Naming
Speed of Information Processing		Visual Spatial Perception
New-Learning		

- Within domains, impairment is inconsistent. For example, executive function is not uniformly affected. On measures of verbal fluency, phonemic fluency is less vulnerable to MDD than semantic fluency, and mental flexibility tends to be especially diminished.
- Intellect, language, and visual-spatial perception are resilient to MDD.
- Patient characteristics seem to mitigate neurocognitive morbidity. As summarized in Table 2, those who are elderly, psychotic, more severely depressed, melancholic, or admitted to a hospital appear most likely to manifest impairment. In contrast, reduced effort, or endogenous onset of symptoms does not seem to reliably explain cognitive dysfunction.

<u>Factors That Exacerbate Impairment</u>
Inpatient Status
Onset in Senescence
Presence of Psychotic Features
Depressive Severity
Melancholia

Clinical Take Home Points:

1. Ultimately, it is essential to recognize that subjective complaints may reflect objective deficits that merit assessment and intervention.
2. Patients should be screened for these neurocognitive impairments, and treatment plans should take these deficits into consideration.
3. Depressed patients may struggle to incorporate or benefit from psychological treatments, especially those that emphasize abstract concepts pertaining to cognitive-behavioral therapy. Accommodations in therapy may be required for these patients to benefit from treatment.



Michael Basso, Ph.D., ABPP-CN, McFarlin Professor of Psychology at the University of Tulsa. Dr. Basso's research concerns neuropsychological function in people with severe mood disorders or multiple sclerosis.



Ashley Miller earned her Ph.D. in clinical psychology from the University of Tulsa. She is currently completing a two-year postdoctoral fellowship in clinical neuropsychology at the Cleveland Clinic.



Eduardo Estevis is a second year neuropsychology postdoctoral fellow in the Department of Neuro-Oncology, Section of Neuropsychology at the University of Texas MD Anderson Cancer Center.



Lily Lau is a doctoral student in clinical psychology at The University of Tulsa. Her research explores memory remediation techniques in people with mild cognitive impairment and Alzheimer's disease.



Bradley Reynolds is a doctoral student in Clinical Psychology at the University of Tulsa. He is currently serving as an associate director of the True Blue Neighbors Behavioral Health Clinic, a low-income community outpatient service provider. His research investigates the relationships between decision-making, executive functioning, and risk-taking behavior.



Dennis R. Combs is a professor of professor of psychology at the University of Texas at Tyler. He is a licensed psychologist in Texas and specializes in the assessment and treatment of psychosis. Dr. Combs has published 65 papers in the areas of psychosis, neuropsychology, and paranoia.



Douglas Whiteside, Ph.D., ABPP-CN, is a Clinical Professor of Psychiatry at the University of Iowa. His research concerns neuropsychological assessment of performance and symptom validity.

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Utility of Using Webinars As An Educational Tool In Neuropsychology: A Preliminary Study Using Pre- and Post Webinar Surveys

Preeti Sunderaraman, Ph.D.
Columbia University Medical Center

Christina Eguizabal Love, M.S.
Florida Institute of Technology

Michelle Madore, Ph.D.
VA Palo Alto Health Care System

Introduction

Webinars are one of the most recent forms of synchronous “real-time” computer-mediated communication (CMC) technologies¹. Other examples of CMC include instant messaging and voice-over-internet protocol (VoIP; e.g., Skype). Advantages of utilizing synchronous CMC tools such as webinars are: (1) Cost effectiveness: Webinars can be used across several internet platforms at minimal cost including Google Hangouts, Anicam-Live, Adobe’s Connect Pro Live (CPL), and Elluminate on computers and handheld devices². The primary requirement to host and view a webinar is access to broadband internet services. As such, travel and its associated costs for participants are considerably lowered³. (2) Common platform: Webinars provide professionals with similar interests with a universal platform to further their learning, irrespective of geographic regions. (3) Community development: Strong learning communities can be created, thus mitigating common barriers such as distance, race, and culture. (4) Ease of use: CMC tools permit the use of multiple mediums to transmit information, such as inclusion of audio, video, hands-on demonstrations, and links to sites, images, and resources. (5) Multiple options to interact: Regarding webinar format, presenters can be at a single site or several locations. Similarly, the audience can view from multiple locations, and can be restricted to those who have been invited for the webinar or can be public. Regarding the level of interaction, filters can be set wherein the audience is allowed to interject verbally during the webinar, or it can be restricted to the audience members typing comments and questions via chat messages during the webinar presentation. Thus, if the level of interaction is two-way and immediate, then instant feedback on learned material or training can be provided⁴. Moreover, audience members can communicate among themselves during the webinar, enhancing collaborations⁵. (6) Convenient broadcasting and reception: Webinars can be recorded, providing an asynchronous CMC where the communication occurs subsequent to viewing the webinar via email or other mechanisms that do not occur in real-time. Such cases allow the audience to access the content if they missed the live presentation. The audience can also be encouraged to post

comments and questions in offline discussions. Thus, webinars can combine both synchronous and asynchronous CME technologies, making it a rather unique tool to educate and train professionals.

Rationale for the study

Webinars have been found to facilitate students’ interaction and satisfaction in online learning programs^{6,7}. Webinars can also be used as a teaching medium for enhancing conceptual learning¹, distance education⁸, coaching patients in optimizing treatment decisions⁹, and training professionals in data collection methods¹⁰. In neuropsychology, webinars are quickly becoming a practical tool for facilitating training. Various organizations, such as the NAN CE series and Division 40’s Ethnic Minority Affairs Sub-Committee (SCN-EMA), are utilizing this approach regularly. However, the effectiveness of imparting information by participating in webinars has not been evaluated. Therefore, the current preliminary study sought to investigate the qualitative and quantitative gains of utilizing webinars for educational purposes in neuropsychology.

Methods

The current webinar was conducted as part of a series of presentations by SCN-EMA. The purpose of the webinar series is to promote diversity training for trainees and professionals who are committed to increasing their skills in cultural competence in neuropsychology. The current webinar was developed in collaboration with the Hispanic Neuropsychological Society (HNS). Event flyers were disseminated via listservs, social media posts, and HNS’s webpage. The webinar was hosted on the Google Hangouts platform, an easily accessible online application. The presentation, *Neuropsychological Assessment of Spanish-Speaking Pediatric Populations*, aired “live” in January 2016. Overall, the goal was to increase understanding of neuropsychology-related health disparities in the Hispanic/Latino population. Questions and comments via the Google Hangouts platform were solicited and addressed following the webinar. The 90-minute presentation was made available on YouTube to allow professionals to access the recording and PowerPoint slides following the presentation.

To examine the effectiveness of using webinar as a tool to achieve the learning objectives, participants were requested to anonymously complete a brief pre- and post-survey consisting of demographic questions and 10 content-focused multiple choice questions.

Results

Ninety individuals completed the pre-webinar survey and 20 completed the post-webinar survey.

Participants consisted of Psychologists/Neuropsychologists (51%), Graduate Students (29%), and other professionals such as Interns and Post-Doctoral Fellows (20%). Participants also had mostly completed or were currently in Ph.D. programs (60%) or Psy.D. programs (31%). Most indicated having minimal (41%) or moderate (30%) exposure to Cross-Cultural Neuropsychology, while 23% indicated having much exposure in this area. Similarly, participants indicated being somewhat confident (43%) or confident (27%) in their ability to provide Cross-Cultural Neuropsychology services, while 21% did not feel confident and 9% felt extremely confident.

Qualitative analysis revealed that, of the 20 individuals that completed the post-webinar survey, 65% felt they learned "Some Useful Information" following the webinar, 25% indicated learning "A Great Deal of Information" and only 10% indicating they had learned "Very Little Information." We conducted a comparative, quantitative analysis for the mean number of items correctly answered pre-webinar vs. post-webinar. Considering the unequal sample sizes and the anonymous nature of the survey, an unpaired samples t-test was conducted. Results indicated a significant increase in the mean number of items correctly answered after viewing the webinar ($M = 6.60$, $SD = 1.39$) as compared to before the webinar ($M = 5.08$, $SD = 1.71$), $t(108) = -3.71$, $p < .001$.

Discussion

Webinars in neuropsychology have recently gained popularity and prominence as an information disseminating tool. From a pedagogical perspective, it becomes critical for the field of neuropsychology to examine the utility of webinars as teaching tools. In this preliminary study, we found that participants correctly answered a significantly higher number of items after viewing the webinar compared to their responses before viewing it. Additionally, a majority endorsed benefitting from the presentation from a moderate to high extent.

Despite these encouraging findings, it is important to note that the majority of participants did not complete the post-webinar survey. We postulate that following the conclusion of the webinar, participants may have experienced fatigue and decreased motivation to complete the survey. Additionally, some participants may not have viewed the entire webinar presentation, or may have viewed the webinar intermittently while being engaged in another activity. Others may have viewed the full presentation, but may not have been confident about their learning, or may not have benefitted from the webinar. It is also possible that some

participants may have completed the webinar survey only after viewing it. Therefore, preventing participants' attrition may be one of the challenges of designing a repeated measures survey study based on online webinars.

The benefits of conducting a webinar outweigh such costs. A specific advantage includes broadcasting the webinar to a wider local and international audience, and developing specialized webinar topics. In regards to neuropsychology, many professionals can benefit from webinars. For example, webinars can be tailored based on level of training (graduate students, post-doctoral fellows, early career psychologist), setting (research, clinical, VA, academic, medical), developmental stage (young adults, women, older adults), and by specialized topics (pediatrics, geriatrics, cross-cultural, diversity, specific disease processes, assessment practices). Finally, using webinars for Continuing Education (CE) credits can benefit neuropsychologists who desire to advance their knowledge without compromising their busy schedules.

Despite the numerous merits, some caveats should be considered:

1. Broadcasting a webinar is highly reliant on efficient technology, including internet connection and electricity. Fortunately, the economic costs of disruptions are negligible and can be offset by restarting the presentation or pre-recording the webinar at a more convenient time.
2. Recorded webinars can be made available to professionals through social media sites for later viewing. However, there is a risk of placing copyrighted material or information about testing procedures in the public domain, which then can be misused or misconstrued. One way to circumvent such a risk would be to make the webinar accessible to individuals upon request, or to embed the webinars in professional society's websites to which access can be obtained only after members of the profession log into the website.

Neuropsychologists should begin clear and cogent discussions about potential issues that may arise in the future regarding webinars including 1) how to design webinars using newer methods, 2) determining when is it ethical or unethical for an audience member to use webinar content for academic work, 3) whether speakers can impose copyright issues for all or some of their PowerPoint slides, 4) whether there should be a directory of webinars for easy browsing, and finally 5) if there should be a charge for viewing webinars over a free platform.

Summary

The present preliminary study shows that, from a pedagogical perspective, webinars are an effective delivery method for teaching special topics in neuropsychology. Despite multiple advantages, several methodological and ethical issues remain unanswered and should be addressed in the future. Neuropsychologists should engage in careful examination of this delivery method to benefit from the novel trend of webinar-based teaching.



Preeti Sunderaraman obtained her PhD from the Department of Psychology at Drexel University, Philadelphia, and is currently working as a postdoctoral fellow at Cognitive Neuroscience Division of the Taub Institute for Research on Alzheimer's Disease and the Aging Brain, New York City. She served as the student representative of APA Division 40's Ethnic Minority Affairs (EMA) Committee for three years during which she organized two webinars, and was involved in developing and enhancing EMA's mentorship program. She is currently the INS-SLC's International Student Liaison Student Representative wherein she is actively fostering a collaborative network of students and professionals from various countries with the goal of developing and sharing helpful resources. During her postdoctoral fellow, Preeti will study financial decision making in older adults, for which she was recently awarded the National Academy of Neuropsychology Clinical Research Grant 2016 Award.



Christina Eguizabal Love completed a neuropsychology internship at the Children's Hospital Colorado. She will be receiving her Psy.D. degree in Clinical Psychology from the Florida Institute of Technology in fall 2016 and will begin a post-doctoral fellowship in pediatric neuropsychology at the Kennedy Krieger Institute. She currently serves on the Mentoring and Education Subcommittee of the Hispanic Neuropsychological Society and is dedicated to providing bilingual and multi-cultural services to Spanish-speakers. Her research interests include the neuropsychological functioning of children with developmental and chronic medical conditions, especially in regards to the executive functioning and quality of life of pre-surgical candidates with intractable epilepsy.



Michelle R. Madore is a clinical neuropsychologist at the VA Palo Alto Health Care System (VAPA). She currently serves as the chair for APA's Society for Clinical Neuropsychology's Ethnic Minority Affairs sub-committee and the Financial Chair of the AAPA's Division on Filipino Americans. She received her Ph.D. in clinical psychology from the University of Cincinnati, where she received specialized training in neuropsychology. Dr. Madore completed her pre-doctoral internship at the VAPA. She has completed postdoctoral training focused on clinical neuropsychology and neurorehabilitation research at VA Martinez, San Francisco VA Medical Center, and VAPA.

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FDA Seeks Input on Neurodiagnostic Cognitive Assessment and External Neurostimulation Devices

Robin C. Hilsabeck, Ph.D.
INC Research

Tresa Roebuck-Spencer, Ph.D.
Private Practice

The Federal Drug Administration (FDA) hosted a public workshop on November 19-20, 2015 to seek input on cognitive assessment medical devices (e.g., computerized cognitive batteries) and non-invasive brain stimulation medical devices (e.g., transcranial magnetic stimulation). The overarching goal of the workshop was to further develop risk-based strategies within a regulatory framework that will facilitate advances in the technology while maintaining appropriate consumer protections. The first day of the workshop focused on neurodiagnostic devices to assess cognitive functioning and built upon what was learned from their prior public workshop on seizure detection, cognitive function, and TBI/concussion devices in June 2011, which was co-sponsored by NAN, the American Academy of Neurology, and the American Epilepsy Society and led to a joint position paper by NAN and the American Academy of Clinical Neuropsychology¹. The second day focused on external neurostimulation devices intended to improve normal cognitive functioning in healthy individuals.

Format of FDA Workshop

The format for both days was similar and included an introduction to the day's topic, an overview of the FDA's perspective on the topic, state of the science presentations by leaders in the field, panel discussions of risks and benefits by various stakeholders, and breakout sessions to seek public input into benefit and risk considerations, current scientific and clinical evidence, ethical considerations, and clinical trial design considerations. In addition, representatives from several government agencies presented overviews of their agencies and how their agency was relevant to the discussion of regulating these devices for their intended uses. For example, an attorney from the Federal Trade Commission (FTC), Michelle Rusk, explained that their broad mandate is to stop deceptive and unfair practices in commerce (e.g., companies such as Lumosity making false and unsubstantiated marketing claims). She encouraged the public to help the FTC regulate these devices by contacting them when there is concern about the validity of claims made in advertising (concerns about labeling claims should be directed to the FDA). Although all discussions were relevant to clinical neuropsychologists, highlighted in this summary are specific aspects of the workshops that might be of most interest to NAN members.

Highlights from Day 1: Neurodiagnostic Devices

With regard to the neurodiagnostic devices topic, it is important for neuropsychologists to know that the FDA considers computerized cognitive measures, including tests that assess a single cognitive ability (e.g., reaction time) and those that measure multiple cognitive functions (e.g., a test battery), to be under the

purview of neurodiagnostic medical devices that may require FDA regulation. It was noted that many of these devices do not yet have FDA regulations so this is an emerging area. An example of one of the topics the FDA was seeking input about was how differences in intended use of a neurodiagnostic device would affect the risk-benefit profile (e.g., what kind of protections need to be in place if a device is intended to be used to identify a specific cognitive deficit versus to screen for cognitive impairment?). Recent FDA approvals of neurodiagnostic devices used for assessment of cognitive functioning were discussed, including Cognivue, which has been approved "for use as an adjunctive tool for evaluating perceptual and memory function in individuals aged 55-95 years old" as written in the FDA approval letter dated June 5, 2015. The FDA approved this device as a class II computerized cognitive assessment aid that is not intended to be used as a stand-alone or adjunctive diagnostic device. Deborah Wolf from the FDA cautioned in her presentation that when a device is approved as an aid in assessing, the company should not make any statements about the device detecting or diagnosing, which would be considered a false or misleading claim, going beyond the scope of intended use, and subject to investigation and potential penalties. The only other computerized test mentioned specifically was DANA, which has been cleared by the FDA as a "tool that provides an objective measurement of reaction time." Of note, other computerized cognitive batteries have also been cleared by the FDA, and there may be other computerized cognitive batteries undergoing this process currently.

Highlights from Day 2: External Neurostimulation Devices

With regard to the external neurostimulation devices topic, the discussion was limited to those devices that "apply external electromagnetic neurostimulation to the head, with the intent of improving, enhancing, or otherwise favorably altering normal cognitive function in healthy individuals" (p. 3 of the Discussion Paper on this topic provided by the FDA as workshop materials). The intent of these devices could be to affect the structure (anatomy) or function (physiology) of the brain but not to prevent or treat medical conditions. Of particular interest to the FDA in order to provide regulatory oversight on these devices are considerations of whether the use of the device presents a potential unreasonable risk of illness or injury and how devices may relate to other medical devices that deliver energy externally to the head. It was acknowledged that research on external neurostimulation devices intended to improve normal cognitive functioning in healthy individuals is limited but is a rapidly evolving area of interest to the FDA. In fact, the FDA has played a

key role in understanding non-invasive neurostimulation through their regulatory science research using computational modeling conducted in the Center for Devices and Radiological Health in the Office of Science and Engineering Laboratories. Dr. Leonardo Angelone, a biomedical engineer in the Division of Biomedical Physics, informed the group of the FDA's collaboration with several academic institutions to produce a model of the human head and neck, the "MIDA" model², which is freely available to the public to facilitate research in this area. During breakout sessions discussing the use of non-invasive neurostimulation devices, some ethical concerns that may be of interest to neuropsychologists were use or misuse of these devices by children and adolescents whose brains have not fully developed and by individuals with neuropsychiatric conditions in which use of these devices may be contraindicated. This concern was raised because these devices may be purchased for individual use and not by prescription or at the recommendation of a health care provider who will monitor use or apply the technology.

Closing Comments and Additional Information:

Neuropsychology was well represented by Dr. Alison Cernich who spoke on technical aspects in computerized cognitive assessment and Dr. Philip Harvey who spoke on technology assisted cognitive assessment. In addition, Drs. Tresa Roebuck Spencer and Peter Como served as breakout session facilitators. The full agenda, speakers, slide presentations, webcasts links, transcripts from the workshop, discussion papers distributed prior to the workshop, and contact information for FDA representatives for each topic can be found here: <http://www.fda.gov/MedicalDevices/NewsEvents/WorkshopsConferences/ucm458018.htm>.



Robin C. Hilsabeck, PhD, ABPP earned her Ph.D. in clinical psychology with a sub-specialization in neuropsychology from Louisiana State University, and she completed a 2-year postdoctoral fellowship in neuropsychology at University of California, San Diego. Dr. Hilsabeck is a board certified clinical neuropsychologist who works at a large, global contract research organization, INC Research, as Senior Clinical Scientist on the Clinical Surveillance Team in the CNS Business Unit. Prior to joining INC Research in 2012, Dr. Hilsabeck worked for 13 years in clinical, administrative, and academic research positions, most recent as Associate Professor of Psychiatry with tenure at University of Texas Health Science Center in San Antonio (UTHSCSA). She continues to carry an appointment as Associate Clinical Professor of Psychiatry at UTHSCSA where she has been on faculty since 2006. Dr. Hilsabeck currently serves as Associate Editor for *The Clinical Neuropsychologist* and is on the editorial board of *Archives of Clinical Neuropsychology* and *Neuropsychology Review*. She is Past President of the National Academy of Neuropsychology.

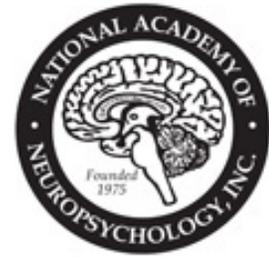


Dr. Roebuck Spencer earned her PhD in clinical psychology with a specialization in neuropsychology from the University of California, San Diego and San Diego State University Joint Doctoral Program. She completed a postdoctoral fellowship at Baylor College of Medicine and The Institute for Rehabilitation and Research (TIRR) in Houston, TX. She currently works in private practice in New Orleans at the Jefferson Neurobehavioral Group and remains active in research within her practice and through collaborations at the University of Oklahoma. She is a Fellow and current board member of NAN and previously chaired the NAN Professional Affairs and Information Committee (PAIC). Her research interests include traumatic brain injury, computerized neuropsychological testing, and performance validity testing.

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