

FTD phenotypes, and to understand the rates and effects of more extensive repetitive head trauma (symptomatic and asymptomatic) in patients with FTD.

**Categories:** Neurodegenerative Disorders

**Keyword 1:** traumatic brain injury

**Keyword 2:** dementia - other cortical

**Correspondence:** Jessica Bove, University of Florida, Department of Clinical and Health Psychology, [bovej@ufl.edu](mailto:bovej@ufl.edu)

## 58 Highly Educated Professionals with Dementia: More than just Physicians

John F Linck<sup>1,2</sup>, Julia E Maietta<sup>2</sup>, Christopher T Copeland<sup>2</sup>

<sup>1</sup>Spectrum Health, Grand Rapids, MI, USA. <sup>2</sup>OU Health, Oklahoma City, OK, USA

**Objective:** Findings from cognitive screenings have resulted in lower-than-expected scores amongst late-career physicians (Moutier et al., 2013). Similar to healthy aging samples and those with mild cognitive impairment, inconsistencies in self-report and objective neuropsychological functioning have been noted in physicians (Nasreddine et al., 2005). Little research has focused on neuropsychological functioning of other highly educated groups, including PhD and JD degrees. We addressed a lack of normative cognitive performance data for populations with advanced degrees by exploring cognitive test scores in a mixed clinical sample of adults.

**Participants and Methods:** Archival data are from 208 neuropsychology clinic outpatients with 20 years of education ( $M_{age}=67.7$ ,  $SD_{age}=12.3$ ; 25% female; 95% White). Academic degrees were PhD (35.6%), JD (28.4%), MD/DO (21.6%), and 6% other. Referrals sources were physicians (93.8%), licensing boards/employers (3.8%), self-referrals (1.4%), and attorneys (1.0%). Employment status was 55.3% employed and 44.7% not employed. Final DSM-5 neurocognitive diagnosis (NCD) status was: no NCD (45.2%), mild NCD (35.6%), and major NCD (19.2%). Etiologies were: possible Alzheimer's disease (41.2%), unspecified (13.2%), and possible vascular (12.3%). Chi-square tests denoted diagnostic status differences between degree type and employment status. ANOVAs denoted

differences in global cognitive and intellectual functioning (on the Repeatable Battery for Neuropsychological Status [RBANS] Total Index, Weschler Adult Intelligence Scale-IV (WAIS-IV), Weschler Abbreviated Scale of Intelligence-II [WASI-II] FSIQ-4 and FSIQ-2) between degree types. Cumulative frequency rates for low scores in the entire sample on normally distributed tests of general intellectual and cognitive functioning were computed for -1.0, -1.5, -2.0, and -2.5 standard deviations (SDs) at or below the population mean.

**Results:** NCD diagnosis did not differ by degree ( $\chi^2[14]=8.73$ ,  $p=.848$ ) but did differ by employment status ( $\chi^2[2]=40.98$ ,  $p<.001$ ,  $\phi=0.44$ ). Employment rate was highest for the no NCD group (66.0%), followed by mild NCD (37.8%), and major NCD (7.5%). For cases below retirement age (<65 years), employment status did not significantly differ between NCD diagnostic groups ( $\chi^2[2]=5.97$ ,  $p=.050$ ). Low scores on an FSIQ measure were: -1 SD (7.0%), -1.5 SD (2.6%), -2.0 SD (0.9%), and -2.5 SD (0.0%) compared to general cognitive test scores which demonstrated 42.5% at -1 SD, 30.5% at -1.5 SD, 19.0% at -2.0 SD, and 9.2% at -2.5 SD below the population mean.

**Conclusions:** The high-education literature is limited to medical degree samples. This sample included multiple degree types. Unsurprisingly, employment rates were higher for healthy versus impaired samples; however, employment rates were similar across these groups for people below retirement age. Our findings suggest that cognitively impaired people with 20 years of education often perform at or near the general population average on tests of general intellectual functioning but below the general population average on tests of general cognitive functioning. Future work should include base rates of low scores on a broader array of cognitive tests across diagnostic groups.

**Categories:** Neurodegenerative Disorders

**Keyword 1:** cognitive functioning

**Keyword 2:** memory disorders

**Correspondence:** John F Linck, Spectrum Health, [John.Linck@spectrumhealth.org](mailto:John.Linck@spectrumhealth.org)

## 59 Effects of Cognitive Impairment, Geriatric Depression, and Anxiety on the Texas Functional Living Scale (TFLS) in a Memory Disorder Clinic

Karina E. Guerra-Guzman, Dominique R. Ghirardi, Anthony LoGalbo  
Florida Institute of Technology, Melbourne, FL, USA

**Objective:** The Texas Functional Living Scale (TFLS) is a measure of adaptive functioning commonly utilized across the geriatric population. Current research suggests that those with Alzheimer's disease and other dementias perform poorly on the TFLS, compared to those with mild cognitive impairment (MCI) and normal cognition (Cullum et al., 2001). Additional research is needed to examine the influence anxiety and depressive symptoms have on activities of daily living (ADLS) in individuals being evaluated for memory disorders. This study will examine the effects of anxiety and depression on adaptive functioning across all patients, and within samples of those with dementia and MCI. It is hypothesized that higher reported anxiety and depressive symptoms will predict lower scores of ADLS.

**Participants and Methods:** Patients at a memory disorder clinic ( $N = 756$ ; 58.2% female) were screened for cognitive impairment using the Montreal Cognitive Assessment (MoCA). A brief neuropsychological evaluation (BNE) was then conducted in which the TFLS, Geriatric Depression Scale (GDS), and Geriatric Anxiety Inventory (GAI) were administered, among other measures.

**Results:** A stepwise hierarchical regression was conducted on the entire sample to examine the effects of anxiety and depressive symptoms on TFLS performance, controlling for cognitive impairment using the MoCA. Lower MoCA scores explained a significant amount of variance in TFLS performance ( $R^2 = 0.456$ ,  $F(1, 754) = 632.78$ ,  $p < .001$ ). MoCA scores ( $b = 1.27$ ,  $p < .001$ ), the GAI ( $b = 0.14$ ,  $p = .019$ ), and the GDS ( $b = 0.10$ ,  $p = 0.039$ ) were significant predictors of poor TFLS performance across the entire sample. Although the MoCA, GDS, and GAI were each significant predictors of the TFLS, the increased variance explained by the GDS and GAI individually was incremental ( $\Delta R^2 = 0.003$ ,  $F(1, 752) = 3.90$ ,  $p = .049$ ). Stepwise hierarchical regressions were also conducted on subsamples diagnosed with MCI ( $N = 171$ ) and dementia ( $N = 394$ ). For those with MCI, MoCA scores explained a significant amount of variance in TFLS performance ( $R^2 = 0.044$ ,  $F(1, 169) = 7.80$ ,  $p = .006$ ). Neither the

GAI nor GDS explained significant additional variance. Only MoCA scores ( $b = .30$ ,  $p = .006$ ) predicted TFLS performance. For those with dementia, MoCA scores explained significant variance in TFLS scores ( $R^2 = 0.338$ ,  $F(1, 392) = 200.47$ ,  $p < .001$ ). The GAI explained additional significant variance when added ( $\Delta R^2 = 0.009$ ,  $F(1, 391) = 5.26$ ,  $p = .022$ ). The GDS did not explain any additional variance. Both the MoCA ( $b = 1.29$ ,  $p < .001$ ) and the GAI ( $b = -0.15$ ,  $p = .002$ ) significantly predicted TFLS performance. **Conclusions:** While results suggest that anxiety and depressive symptoms alone do not explain a significant degree of variance within scores of adaptive functioning across the entire sample, elevated ratings of anxiety and depressive symptoms were significant predictors of lower scores of ADLS, suggesting some support for our hypothesis. Additionally, anxiety symptoms significantly explained increased variance in TFLS scores for those diagnosed with dementia, suggesting a potential relationship between anxiety levels and poor adaptive functioning for dementia patients.

**Categories:** Neurodegenerative Disorders

**Keyword 1:** memory disorders

**Keyword 2:** adaptive functioning

**Keyword 3:** depression

**Correspondence:** Karina Guerra-Guzman, Florida Institute of Technology, kguerraguzma2020@my.fit.edu

## 60 Impact of Reducing the Nuclear Mutant ATXN1 on Spinocerebellar Ataxia-Like Phenotype

Kathleen B Mather<sup>1</sup>, Lisa A Duvick<sup>2</sup>, Hillary P Handler<sup>2</sup>, Harry T Orr<sup>2</sup>

<sup>1</sup>Loyola University Chicago, Chicago, IL, USA.

<sup>2</sup>University of Minnesota, Minneapolis, MN, USA

**Objective:** Spinocerebellar ataxia type one (SCA1) is an autosomal dominant neurodegenerative disease caused by an expanded CAG repeat that encodes glutamine (polyQ) in the affected ATXN1 gene. SCA1 pathology is commonly characterized by the degeneration of the cerebellar Purkinje cells (PC) and brainstem. Symptoms include motor dysfunction, cognitive impairments, bulbar dysfunction, and premature death. *Atxn1*<sup>175Q/2Q</sup> knock-in mice were previously developed to