overall neuropsychological performance. Two measures of IIV, intraindividual standard deviation (ISD) and maximum discrepancy (MD), were calculated across all 13 cognitive tests for each participant. Intraindividual standard deviation was calculated by taking the standard deviation of the mean performance across each task for each participant. Maximum discrepancy was calculated by subtracted the lowest standard score from the highest standard score per participant across all 13 cognitive tests.
Results: Controlling for the impact of premorbid functioning, depressive symptoms, and gender, an analysis of covariance (ANCOVA) found significantly less ISD in athletes (M = 11.28, SD = 2.76) compared to non-athletes (M = 12.56, SD = 3.61) across all 13 neuropsychological tasks ($\eta 2 = 0.04$, $p = .004$). Similarly, significantly lower MD scores were found in athletes (M = 40.25, SD = 11.14) compared to non-athletes (M = 44.69, SD = 14.07) across all 13 neuropsychological tasks ($\eta 2 = 0.03$, $p = .008$). Post-hoc analyses revealed no significant differences when athletes were divided into contact and non-contact athletes.
Conclusions: Similar to prior findings that aerobic exercise may enhance cognitive performance, both contact and non-contact college athletes exhibited less neurocognitive dispersion (as measured by ISD and MD) compared to non-athlete college students. However, no significant differences were found between non-contact athletes and contact athletes (soccer players) who were exposed to repetitive subconcussive heading events. These findings suggest that athletic performance in college-aged athletes may lead to more consistent and therefore overall better neuropsychological performance despite exposure to repetitive subconcussive head impacts.

Categories: Other
Keyword 1: sports-related neuropsychology
Keyword 2: neuropsychological assessment
Correspondence: Eric McConathey, Fordham University, emcconathey@fordham.edu

92 To know or not to know: A case of CADASIL highlighting the ethical dilemmas of genetic testing among families carrying a highly heritable neurological condition

Alanna Coady, Jamie Piercy, Harry Miller
The University of British Columbia, Kelowna, BC, Canada

Objective: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a hereditary form of cerebral small vessel disease leading to early cerebrovascular changes. These changes result from mutations in the NOTCH3 gene that cause progressive accumulations of granular osmiophilic material (GOM) deposits, thickening arterial walls and reducing or restricting blood flow in the brain. The clinical presentation of CADASIL is characterized by migraines with aura, early and recurrent strokes, progressive cognitive impairment, and psychiatric disturbances. CADASIL is rare but frequently underrecognized or misdiagnosed. A genetic condition with a 50% risk of inheritance from an affected parent, the gold standard for diagnosis is genetic testing to determine the presence of mutations in the NOTCH3 gene. This presentation aims to familiarize neuropsychologists with the condition of CADASIL through a unique case study highlighting important psychological, social, and ethical considerations raised by genetic testing.
Participants and Methods: This case study presents a 67-year-old, right-handed, married female diagnosed with CADASIL who was referred for neuropsychological evaluation of cognitive function and low mood concerns following multiple ischemic events.
Results: Results revealed severe cognitive deficits in domains of attention, learning, and memory. Her superior verbal abilities and executive function remained largely intact. Assessment of mood revealed elevations in symptoms of depression and anxiety. The patient was aware of CADASIL in her father, paternal aunt, and younger brother, but elected to forego any genetic testing to confirm whether she had the condition until she experienced a stroke at age 61. She has two adult children who have also elected to forego testing and currently remain asymptomatic. Cognitive profile, mood disturbances, and patient perspectives on refraining from pre-symptomatic genetic testing for CADASIL diagnosis will be discussed.
Conclusions: Aspects of this case are consistent with a small body of literature evidencing distinct psychological, emotional, and social challenges among families carrying genetic risk of CADASIL. While providing an

https://doi.org/10.1017/S1355617723002965 Published online by Cambridge University Press

example of an often underrecognized neurological disorder with which neuropsychologists should be familiar, this case uniquely raises ethical questions relevant to care providers and current treatment guidelines regarding genetic testing among families carrying highly heritable neurological conditions. In particular, personal ethical challenges around deciding to pursue or forego pre-symptomatic testing, and implications for family planning, highlight the importance of genetic counseling for affected families.

Categories: Stroke/Cerebrovascular Injury & Disease (Adult)
Keyword 1: genetic disorders
Keyword 2: cerebrovascular disease
Keyword 3: neuropsychological assessment
Correspondence: Alanna Coady, The University of British Columbia, alanna.coady@ubc.ca

93 Impact of Cardiovascular Risk on Cognitive and Brain Aging in Autosomal Dominant Frontotemporal Dementia

Anna M VandeBunte[1], Emily W Paolillo[1], Hyunwoo Lee[2], Ging-Yuek Robin Hsiung[2], Adam Staffaroni[1], Shannon Y Lee[1], Carmela Tartaglia[3], Hilary Heur[1], Joel H Kramer[1], Brad Boeve[4], Adam Boxer[1], Howie Rosen[1], Kaitlin B Casaletto[1]
[1]University of California, San Francisco, San Francisco, CA, USA. [2]University of British Colombia, Vancouver, BC, Canada. [3]University of Toronto, Toronto, ON, Canada. [4]Mayo Clinic, Rochester, MN, USA

Objective: Poor cardiovascular health occurs with age and is associated with increased dementia risk, yet its impact on frontotemporal lobar degeneration (FTLD) and autosomal dominant neurodegenerative disease has not been well established. Examining cardiovascular risk in a population with high genetic vulnerability provides an opportunity to assess the impact of lifestyle factors on brain health outcomes. In the current study, we examined whether systemic vascular burden associates with accelerated cognitive and brain aging outcomes in genetic FTLD.

Participants and Methods: 166 adults with autosomal dominant FTLD (C9orf72 *n*= 97; GRN *n*= 34; MAPT *n*= 35; 54% female; M^{age} = 47.9; $M^{education}$ = 15.6 years) enrolled in the Advancing Research and Treatment for Frontotemporal Lobar Degeneration (ARTFL) and Longitudinal Evaluation of Familial Frontotemporal Dementia Longitudinal FTD study (ALLFTD) were included. Participants completed neuroimaging and were screened for cardiovascular risk and functional impairment during a comprehensive neurobehavioral and medical interview. A vascular burden score (VBS) was created by summing vascular risk factors (VRS) [diabetes, hypertension, hyperlipidemia, and sleep apnea] and vascular diseases (VDS) [cerebrovascular disease (e.g., TIA, CVA), cardiac arrhythmia (e.g., atrial fibrillation, pacemaker, defibrillator), coronary artery disease (e.g., myocardial infarction, cardiac bypass, stent), and congestive heart failure] following a previously developed composite (range 0 to 8). We examined the interaction between each vascular health metric (VBS, VDS, VRS) and age (vascular health*age) on clinical severity (CDR plus NACC FTLD-SB), and white matter hyperintensity (WMH) volume outcomes, adjusting for age and sex. Vascular risk, disease, and overall burden scores were examined in separate models.
Results: There was a statistically significant interaction between total VBS and age on both clinical severity (β=0.20, p=0.044) and WMH burden (β=0.20, p=0.032). Mutation carriers with higher vascular burden evidenced worse clinical and WMH outcomes for their age. When breaking down the vascular burden score into (separate) vascular risk (VRS) and vascular disease (VDS) scores, the interaction between age and VRS remained significant only for WMH (β=0.26, p=0.009), but not clinical severity (β=0.04, p=0.685). On the other hand, the interaction between VDS and age remained significant only for clinical severity (β=0.20, p=0.041) but not WMH (β=0.17, p=0.066).
Conclusions: Our results demonstrate that systemic vascular burden is associated with an "accelerated aging" pattern on clinical and white matter outcomes in autosomal dominant FTLD. Specifically, mutation carriers with greater vascular burden show poorer neurobehavioral outcomes for their chronological age. When separating vascular risk from disease, risk was associated with higher age-related WMH burden, whereas disease was associated with poorer age-related clinical severity of mutation

https://doi.org/10.1017/S1355617723002965 Published online by Cambridge University Press