behavior (SB) by the percentage of daily wear time spent in LPA.
Results: On average, participants (mean age = 43.22 years old and BMI = 45.83 kg/m^2) wore the accelerometer for 909±176 minutes/day and spent 642±174 minutes/day in ST, 254±79 minutes/day in LPA, and 14±13 minutes/day in MVPA. Mean daily ST-to-LPA time ratio was 2.81 ± 1.3 (0.73-7.11). Overall, bivariate Pearson correlations found no significant relationships between LPA and cognitive performance on any of the NIH Toolbox subtests (*r* values = -.002 to -.158, all *p* values >.05). Additionally, bivariate Pearson correlations also found no significant relationships between daily ST-to-LPA time ratio and cognitive performance on any of the subtests (*r* values = .003 to .108, all *p* values >.05). However, higher ST-to-LPA was associated with lower scores on the Dimensional Change Card Sort Test in women (*r* = -.26, *p* = .01).
Conclusions: Results showed that participants' mean daily time spent in ST was 2.5 times higher than that spent in LPA and a higher ratio of ST-to-LPA was associated with poorer set-shifting in women with Class III obesity. Future studies should look to clarify underlying mechanisms, particularly studies examining possible sex differences in the cognitive benefits of PA. Similarly, intervention studies are also needed to determine if increasing LPA levels for individuals with Class III obesity would lead to improved cognitive performance by means of reducing ST.

Categories: Other
Keyword 1: cognitive functioning
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91 Investigation of Cognitive Differences in Pre-Symptomatic Known PRNP Mutation Carriers vs. Non-Carriers

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Objective: Prion disease is a rare, invariably fatal neurodegenerative disease characterized by rapid neuronal degeneration; Mutations to PRNP gene cause genetic prion disease (GPD). In animal models, microglial activation, astrocytosis, and release of neurofilament precede the onset of frank symptoms (Sorce & Nuvolone 2020, Minikel 2020). In humans at risk for GPD, prodromal pathology appears to occur in only a brief window prior to symptom onset (Vallabh et al. 2020, Thompson et al. 2021), but some data suggest that known PRNP mutation carriers may exhibit cognitive abnormalities prior to meeting clinical diagnostic criteria (Mole et al., 2021). We aim to examine pre-symptomatic differences in cognitive processing speed (CPS) and executive function (EF) in PRNP mutation carriers and controls.
Participants and Methods: Our sample includes two groups from an ongoing observational study on GPD (Vallabh et al., 2020): known PRNP mutation carriers (N = 32, Age M = 45.77, SD = 14.75) and control group of non-carriers with a family history of GPD and healthy controls with no known history (N = 11, Age M = 42.01, SD = 12.43). All participants completed a full cognitive battery at baseline and on an annual basis. We compared first visit cognitive testing measuring CPS and EF using: National Institute of Health (NIH) Toolbox [Pattern Comparison (NIH-PC), Flanker, Dimensional Card Sorting Task (NIH-DCCS)], Trail Making Test (TMT) A and B, and Delis-Kaplan Executive Function System (D-KEFS) Color-Word Interference Test (CWIT).
Results: Independent t-tests and Mann-Whitney U tests compared cognitive test performance between groups. Across all cognitive test measures assessed, none exhibited significant differences between groups after Bonferroni correction for N=10 tests (corrected P > 0.05). Mean scores for mutation carriers were non-significantly lower than controls on TMT-B (Z-score Mdn = .29, SD = 1.33 vs. Z-score Mdn = .96, SD = .97), NIH-PC (Age-corrected Standard Score [ACSS] M = 100.13, SD = 20.76 vs. ACSS score M = 114.82, SD = 14.61) and NIH-Flanker (ACSS score M = 83.58, SD = 9.72 vs. ACSS score M = 90.64, SD = 10.94), and NIH-DCCS (ACSS M = 101.29, SD = 16.37 vs. ACSS score M = 112.00, SD = 16.28) but not for TMT-A or all four conditions of CWIT.
Conclusions: We did not detect any significant cognitive deficits in known PRNP mutation carriers. This is consistent with the lack of prodromal pathological biomarker changes or

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cognitive changes as reported in Vallabh et al 2020, and with the finding of Mole et al. 2021 that most tests reveal impairment only at a stage where carriers report subjective symptoms. Our results suggest an opportunity for primary prevention to preserve full cognitive health in at-risk individuals. However, small sample size and limited test sensitivity may leave us underpowered to detect subtle deficits. Future research is warranted to further investigate the neuropsychological profile of pre-symptomatic GPD.

Categories: Genetics/Genetic Disorders
Keyword 1: cognitive functioning
Keyword 2: neuropsychological assessment
Keyword 3: genetic disorders
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Program Welcome by Co-Chairs: Julie Bobholz and Sakina Butt

4:15 - 4:30pm Wednesday, 1st February, 2023
Pacific Ballroom A

Plenary A: Presidential Address: Anesthesia: A Wake-Up Call. Part 2. Developmental Risk or Resilience?

Presenter: Ida Sue Baron

4:30 - 5:25pm
Wednesday, 1st February, 2023
Pacific Ballroom A

Abstract & Learning Objectives:
This presentation is a clarion call to neuropsychologists to contribute their specialized knowledge to help answer a critical question: Is there a Fetal Anesthesia Syndrome that results in subtle and persistent adverse effects over an individual's lifespan? Neuropsychologists are uniquely positioned to make substantial contributions to conceptualization, methodology, and interpretation in studies of human exposure to general anesthesia (GA). Part 1, presented at the 2022 INS Barcelona meeting, reviewed preclinical data that documented effects on the central nervous system and long-term behavioral adversities of GA exposure during an animal's critical growth spurt developmental period. Studies of human adult exposure were also summarized, and attention directed to the absence of prospective studies from childhood to adulthood. Part 2 extends the conversation to GA exposure during the highly vulnerable in utero and early childhood developmental periods. Human retrospective study results began to be published in the early 2000s, and prospective studies only within the last decade. Reports of associations between GA and attentional problems, learning disorder, neuropsychological deficit, and neuropsychiatric disorder are emerging. Yet, due to methodological weaknesses and multiple confounders, clear evidence of causality remains lacking in this nascent literature. A 'developmentalistic' way forward for neuropsychologists will be suggested, one using neuropsychological expertise along with the application of innovative technologies that is informed by the extensive preclinical data showing cellular, synaptic, and neural circuitry disruption during critical growth periods and short- and long-term neuropsychological effects.
Upon conclusion of this course, learners will be able to:
1. Describe types of central nervous system disruption that result in animals following exposure to general anesthesia
2. Identify neuropsychological domains at high potential risk following exposure to general anesthesia during the human critical growth spurt period
3. Explain what is meant by 'vertical transfer'

INS Awards Ceremony

5:30 - 6:30pm
Wednesday, 1st February, 2023
Pacific Ballroom A

Reception

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