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random effects model, the estimate was 0.368 (SE = 0.0198) points per year. **Conclusions:** The naïve estimate of cognitive decline that ignored potentially informative missing data was underestimated by approximately 10%. Joint modeling with auxiliary telephone data has the potential to correct some of the bias associated with informative missingness. This or similar strategies could improve estimates of age and dementia related cognitive decline.

P1-500

EXPLORATION OF THE RAVEN APM - NART DISCREPANCY AS A MEASURE OF INTELLECTUAL DECLINE

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Background: Detecting decline in intellectual functioning following brain injury is an important objective of neuropsychological assessment. As a person's previous level of functioning is generally not available, the premorbid level must be estimated. This study explored the validity of the discrepancy between performance of the Raven Advance Progressive Matrices (APM) score (current intellectual level) and the National Adult Reading Test (NART; estimated premorbid level) as a measure of intellectual decline. Methods: A multiple regression equation was created to predict the Raven Advanced Progressive Matrices (APM) score from the National Adult Reading Test (NART) score and demographic variables in a large sample of healthy older persons (n = 270, age 68±8). Predicted scores were subtracted from the obtained scores. The resulting discrepancy score was converted into a percentile distribution. The validity of this procedure was examined by application of the regression equation to two patient samples (110 patients with cerebral stroke, age 60±14; 387 patients with diabetes mellitus, aged 65±7). The proportion of patients with a discrepancy score below the 16th and 5th percentile was compared between the groups with chi² analysis. Results: Within the healthy control group the following regression equations were created: Estimated Raven APM score = 7.21 - $(0.07 \times \text{age}) + (0.11 \times \text{years of education}) + (0.05 \times \text{NART})$ for males; Estimated Raven APM score = $7.24 - (0.11 \times age) + (0.16 \times years of educa$ tion) + $(0.06 \times NART)$ for females. When applied to the patient groups the results showed a significantly higher rate of decline at the 16% ("below average") and 5% ("impaired") significance cut-off levels for stroke patients compared with patients with diabetes (stroke 35% and 15%; diabetes 16% and 5%, < 0.05). Conclusions: The present study developed a multiple regression equation that estimated Raven APM scores from the NART score and demographic variables in a large sample of 270 healthy older persons. When compared with the current Raven APM scores in this group the procedure resulted in discrepancy scores that aid clinicians in detecting global intellectual impairment in older persons. The Raven APM - NART discrepancy may be a useful measure of intellectual decline in older persons.

P1-501

TYPE 2 DIABETES, BUT NOT HYPERCHOLESTEROLEMIA, IS RELATED TO DECREMENTS IN COGNITIVE FUNCTIONING AMONG PATIENTS WITH MCI

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Background: Research suggests an elevated risk of cognitive impairment, particularly memory, attention and executive functioning deficits in older adults

with type 2 diabetes (T2DM) even after controlling for other comorbid conditions. Further, research has also shown that high cholesterol levels have been associated with difficulties in cognitive functioning and risk of Alzheimer's disease. Mild Cognitive Impairment (MCI) is characterized as the transitional stage between normal cognitive aging and dementia. However, little is known about the role of medical disorders such as T2DM and hypercholesterolemia and the impact they may have on cognition in patients with MCI. Further, because the diagnostic classification of MCI and MCI subtyping has been documented to be variable over time, it is possible that other factors, including these medical comorbidities, may impact specific neuropsychological domains, and as a result, diagnostic classification. This study examines the association of T2DM and hypercholesterolemia on cognitive functioning in MCI patients enrolled in the Uniform Data Set (UDS) from the National Alzheimer's Coordinating Center (NACC), including patients whose baseline evaluations resulted in a diagnosis of amnestic MCI (aMCI), alone. Methods: A total of 3648 participants diagnosed with MCI based on established UDS criteria underwent baseline medical, neurological and neuropsychological evaluations (mean age = 75.09; 52.3% female; mean education level = 15.28 years) at the time of their enrollment in their respective NACC- Alzheimer's disease research centers. Patients' demographics and medical and health histories were recorded, including the presence or absence of T2DM and hypercholesterolemia. The following neuropsychological test measures were administered to patients as part of the national UDS battery: MMSE, Logical Memory (initial learning and delayed recall), Digits forwards and backwards, Trail Making Test A and B, Digit Symbol, Boston Naming Test, Geriatric Depression Scale (GDS). Statistical analyses using analysis of covariance were computed using SAS version 9.1.3 adjusting for age, education level, and GDS results. Results: Overall, 11.0% of participants reported the presence of both hypercholesterolemia and T2DM diagnoses, whereas only 3.9% reported the presence of a diagnosis of T2DM without comorbid diagnosis of hypercholesterolemia. In addition, 42.1% of the sample reported the presence of a diagnosis of hypercholesterolemia, without comorbid diagnosis of T2DM. For patients who reported the presence of a diagnosis of hypercholesterolemia after adjusting for age, education, and self-reported symptoms of depression, no significant effect was observed on any cognitive measure. For those patients with the presence of a diagnosis of T2DM only, results showed significant main effects for the MMSE (F = 25.96, p < .0001), Digits Forward (F = 62.67, p < .0001), Digits Backwards (F = 51.91, p < .0001), Trails A (F = 55.40, p < .0001), Trails B (F = 43.66, p < .0001), WAIS/Digit Symbol (F = 91.63, p < .0001), and Boston Naming (F = 37.23, p < .0001). However, there were no significant effects found for Logical Memory. In all significant cases, endorsement of the presence of a diagnosis of T2DM was associated with lower scores than those participants who did not endorse the presence of a diagnosis of T2DM. There were no significant interactions between T2DM, hypercholesterolemia and neuropsychological test performances suggesting that the presence of both diagnoses did not lead to greater burden than T2DM, alone. When the analyses were repeated with the amnestic MCI patients with memory impairments only, results showed a significant main effect for the presence of hypercholesterolemia and performance on Trails A and B, only. The presence of T2DM was associated with decrements on all cognitive measures but Boston Naming and Logical Memory. Conclusions: These findings suggest that medical comorbidities may contribute to differential patterns of cognitive difficulties in older adults diagnosed with MCI, even when considering MCI patients who report only memory impairment. Endorsement of high cholesterol levels, alone, was not associated with lower test performance, but endorsement of T2DM was associated with lower test scores on measures of attention/working memory (Digit Span forwards and backwards, Trails A), executive functioning (Trails B and Digit Symbol), as well as on measures of general mental status (MMSE) and confrontational naming (Boston Naming Test). Difficulties on measures of attention and executive functioning have been reported in older patients with T2DM, and our results support this finding in patients with MCI. Lower scores on measures of mental status and language ability among individuals with T2DM may represent particular vulnerabilities for MCI patients with T2DM who already exhibit compromised memory functioning. Further, the cognitive

difficulties associated with T2DM reported in this study may contribute to some of the variability in diagnoses of patients with MCI over time, or to possible over-classification of subtypes of MCI with executive functioning or language. Longitudinal follow-up of these individuals will be important in more clearly understanding the impact of this medical comorbidity in predicting cognitive decline over time and better clarifying the relationship between this disorder and possible underlying pathology associated with MCI.

P1-502

UTILITY OF BARRAT SCALE TO MEASURE IMPULSIVITY IN A SERIES OF CASES WITH BEHAVIORAL VARIANT-FRONTOTEMPORAL LOBAR DEGENERATION (BV-FTD)

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Background: To describe the utility of Barratt scale as a measure of impulsivity and the divergence of perceptions between patients and relatives, in cases diagnosed with by-FTD. Methods: The scale was applied to 9 patients (5 men / 4 women, mean age 67.16 years old and average years of schooling, 8.5), diagnosed with bv-FTD (Lund-Manchester criteria) and close family, filling separately, double-blind. Assessment was made more extensive with neurobehavioral and neuropsychological tests (MMSE, Blessed personality subscale and digit span, TMT A, Stroop). Barratt Impulsiveness Scale was chosen for brevity, easy application, and original division into three impulsivity subscales (cognitive, motor and impulsivity unplanned). We compared raw scores obtained by patients and families, with median scores in Hispanic population validation. Scores were compared with each patientfamily. Considering the discrepancy between them as an index of lack of insight or anosognosia on its own momentum. **Results:** MSSE average 26.6 ± 3.2. Direct Media span 4.33 \pm 0.8, and 3.16 \pm 0.75 reverse. Media personality Blessed 3 ± 1.6. Media Barratt scale of 71.6 patient, family member 75.4. Of all cases, major discrepancies were observed in 5 patients, showing difference of 7.2 points between scores of patient and family, being of 0.5 among patients who did not differ. Conclusions: Impulsivity is a common neuropsychiatric symptom and underdiagnosed in bv-FTD. The Barratt Impulsiveness Scale is a useful tool in order to characterize this symptom and implement an appropriate treatment.

P1-503

SUPPRESSION OF INTRINSIC APOPTOSIS BY SYNAPTIC RELEASED ZINC

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Background: Increased physiological electrical activity has been reported to promote cell survival in many neuronal types, but the exact molecular events that underlie the anti-apoptotic effect of synaptic activity are not completely understood. We have found that zinc release in synaptic transmission induced tau hyperphosphorylation, since tau phosphorylation suppress apoptosis (as evidenced by the higher viability and lower level of activated caspase-3 in phosphorylated tau positive neurons), in this study, the effects of synaptic released zinc on apoptosis is investigated. Methods: Competent rat brain hippocampus slices and cultured primary neurons were incubated with glutamate or bicuculline/4-aminopyridine (bic/4-AP) to induce bursts of action potential firing, with or without the pre-treatment of Ca-EDTA, a membrane-impermeable zinc chelator. Then the brain slices or neurons were harvested, cell viability was measured by using CCK-8 assay, the expression and activity of apoptotic markers p53 and caspase-3 were detected. Results: Glutamate treatment for 20 minutes have no effect on p53 expression and caspase-3 activity, no cell apoptosis was observed; Bic/4-AP treatment for 3h increased cell viability as previously reported. While preincubation with Ca-EDTA in both glutamate and bic/4-AP treated brain slices and primary neurons up-regulated p53 expression and caspase-3 activity, with decreased cell viability, indicating that synaptic released zinc inhibits apoptosis. **Conclusions:** Synaptic released zinc inhibits intrinsic apoptosis in neurotransmission, however, whether this anti-apoptotic effect is mediated by tau phosphorylation needs to be further explored.

P1-504

THE PREVALENCE OF UNDERLYING DEMENTING ILLNESS CONTRIBUTING TO THE PRESENTATION OF DELIRIUM IN AN ACUTE HOSPITAL SETTING IN THE ELDERLY

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Background: In Australia, a marked increase in the aged population is affecting many domains of the health care system including the Emergency Departments (ED) of public hospitals. Currently, older people are higher users of EDs compared to their proportion in the general population. In 2004-05, 35% of all episodes of admitted hospital care in the public setting involved individuals over the age of 65. Furthermore, the proportion of the total population aged over 65 is likely to rise to 22% by 2031 compared to 12% in 1997. Consequently, older patients will continue to make up an increasingly important group treated by EDs. Delirium is a frequent presentation at ED or complication of hospital admissions, particularly among the elderly. Recent literature has suggested that approximately 10-15% of older people are delirious at the time of admission. However, recent literature has indicated rates as high as 76% of cases, particularly the hypoactive (quiet) subtype is often overlooked. This lack of detection is estimated be even higher if the individual has a pre-existing dementia. Differentiating delirium and dementia can be less apparent in those elderly who develop "persistent cognitive impairment" following an episode of delirium. Given it has been proposed that delirium may be an indicator for early or subclinical dementing - an episode of delirium in the elderly may be masking an underlying undiagnosed dementia and vice versa. As a greater proportion of elderly individuals are likely to present to ED with altered mental state in the ensuring years, early diagnosis is likely to lead to appropriate treatment and management with better outcomes for the patient as well as family, and guide admission, as well as follow up and management. Methods: Study 1 Statistical information from Eastern Health Decision Report Records for the preceding two years will be accessed and reviewed to determine the prevalence of detected delirium in older populations at ED. This information will be compared to current estimates as reported in recent literature to determine whether delirium is under-detected at ED and if so, by what degree. Study 2 Patients over the age of 65 who presented to the ED with increased confusion and were subsequently admitted to hospital will be recruited for the study. Patients will be excluded if they have a confirmed diagnosis of Dementia, are unarousable to verbal stimuli for all delirium assessments, or a medical cause is established to account for the delirium. Patients will be assessed while in ED using the Confusion Assessment Method - Intensive Care Unit to establish the presence of delirium. Once admitted a delirium screen will be performed (i.e., full blood examination, MRI brain) and family members will be interviewed to determine premorbid level of functioning using the Informant Questionnaire of Cognitive Decline in the Elderly (IQCODE). During admission, daily monitoring of confusion will be conducted using the 'One Day Fluctuation Assessment Scale (ODFAS). This scale consists of seven items of confusional behaviour (falls, fluctuation, drowsiness, attention, disorganised thinking, altered level of consciousness, communication). Those with persistent confusion will be selected for further evaluation. Study 3 All patients admitted with increased confusion will be seen six weeks post discharge. The ODFAS will be re-administered and patients with no evidence of impaired cognitive functioning will be discharged with a recommendation for GP monitoring/follow up if necessary. Patients with evidence of ongoing confusion/impaired cognition will be referred for neuropsychological opinion to determine/discount the possibility