the Risk of Bias. **Results:** Main outcomes (differences between carriers and non-carriers on each imaging modality) will be included in separate tables for each genetic group. Single case studies will be reported in the text. Measured cognitive deficits, expected age of onset of participants and differences between asymptomatic and symptomatic participants will be included if available. **Conclusions:** This systematic review will highlight to clinicians and researchers what are the earliest changes in the brains of pre-symptomatic carriers of FTD. It will also underline the strengths/weaknesses of current studies in the field and propose new avenues to future researchers.

P2-363 SERUM NEUROFILAMENT LIGHT PROTEIN AS A POTENTIAL BIOMARKER OF NEURODEGENERATION IN FORMER PROFESSIONAL ATHLETES WITH MULTIPLE CONCUSSIONS

Foad Taghdiri[1], Namita Multani[2], Miracle Ozzoude[3], Apameh Tarazi[4], Seyed Ali Naeimi[4], Mozhgan Khodadadi[4], Richard Wennberg[4], David Mikulis[4], Robin Green[4], Brenda Colella[4], Karen Davis[4], Kaj Blennow[5,6,7,8], Charles Tator[4], **Carmela Tartaglia**[2,9,10], *[1]Tanz Centre for Research in Neurodegenerative Diseases, Toronto, ON, Canada; [2]Division of Neurology, Krembil Neuroscience Centre, Toronto Western Hospital, University Health Network Memory Clinic, Toronto, ON, Canada; [3]Sunnybrook Research Institute, University of Toronto, Toronto, ON, Canada; [4]University Health Network, Toronto, ON, Canada; [5]The Sahlgrenska Academy at the University of Gothenburg, Mölndal, Sweden; [6]Institute of Neuroscience and Physiology, Department of Psychiatry and Neurochemistry, The Sahlgrenska Academy at University of Gothenburg, Mölndal, Sweden; [7]Clinical Neurochemistry Laboratory, Sahlgrenska University Hospital, Mölndal, Sweden; [8]Institute of Neuroscience and Physiology, the Sahlgrenska Academy at the University of Gothenburg, Gothenburg, Sweden; [9]Tanz Centre for Research in Neurodegenerative Diseases, University of Toronto, Toronto, ON, Canada; [10]University of Toronto, Toronto, ON, Canada. Contact e-mail: carmela.tartaglia@uhn.ca*

Background: The absence of *a*diagnostic tool for *in vivo* chronic traumatic encephalopathy (CTE) has made its diagnosis and treatment impossible.Increased concentrations of serum neurofilament light protein (NF-L) has been reported in individuals with neurodegenerative disease such as those with Alzheimer's disease and frontotemporal lobar degeneration. The aim of the study was to evaluate NFL as a biomarker of neurodegeneration and progression in former professional athletes with multiple concussions and at risk of CTE. **Methods:** Concentrations of serum NF-L of fifty-two former professional athleteswith a history of multiple concussions (ExPro]) [2 females, 50 males; age (mean±SD): 53.8±12.8 years of age]and twenty-three healthy controls (HC) with no history of concussions [all males; age: 49.1±10.8 years of age] were measured using single molecule array (SIMOA) technique. We determined, hippocampal, corpus callosum and total ventricular volumes. We performed seed-based probabilistic tractography of the uncinate, cingulum, and superior longitudinal and corpus callosum (CC) fasciculi to determine their integrity. We used CONN analysis to determine functional connectivity between the components of the resting state default mode network. We assessed the relationship between serum NF-L to the above-mentioned metrics. Eighteen ExPro underwent follow-up imaging after 2 years. All statistical analyses were controlled for age and intracranial volume. **Results:** Serum concentrations of NF-L were not significantly different between the ExPro and HC (11.63±7.4 vs. 10.61±5.3 pg/ml, respectively). However, in the ExPro, the levels of NF-L were significantly correlated with the mean diffusivity (MD) of CC (r=0.382, p=0.004), total volume of CC (r= - 0.401, p=0.004), total ventricular volume (r= 0.393, p=0.001), volumes of both right and left hippocampi (r= -0.342, p=0.003; r= -0.277, p=0.019; respectively), and the functional connectivity between the right and left hippocampi (beta=-0.03, p=0.005). Moreover, the levels of NF-L in the ExPro at the first visit were significantly correlated with the amount of increase in CC MD over the period of 2 years follow-up (r=0.475, p=0.04). **Conclusions:** Serum NF-L levels may reflect cerebral changes in athletes with multiple concussions and it may predict decrease in white matter integrity over time. Serum NFL might be a potential biomarker of neurodegeneration in athletes with a history of multiple concussions.

P2-364 NEUROANATOMICAL AND NEUROPSYCHOLOGICAL CORRELATES OF RESTING STATE EEG DIAGNOSTIC FEATURES IN PATIENTS WITH ALZHEIMER'S DISEASE

William J. McGeown[1], Raymundo Cassani[2], Tiago H. Falk[2], Marco Cecchi[3], K. C. Fadem[3], *[1]University of Strathclyde, Glasgow, United Kingdom; [2]University of Quebec, Montreal, QC, Canada; [3]Cognision, Louisville, KY, USA. Contact e-mail: william.mcgeown@strath.ac.uk*

Background: In the search for accurate, low cost biomarkers for Alzheimer's disease (AD) and other dementias, quantitative electroencephalography (EEG) may offer a solution. In a recent multi-site study by Cognision, patients with AD were assessed using the Alzheimer Disease Neuroimaging Initiative protocol, plus EEG assessment. The primary objective of the current analysis, was to examine the relationships between a resting-state (rs)EEG feature set (that best discriminated AD patients from controls) and neuroanatomical measures. The second objective was to identify the rsEEG measures that reflected disease staging. **Methods:** Eighty-nine patients with mild AD (MMSE 21-26) were evaluated using a comprehensive neuropsychological assessment battery, 5 minute eyes-open rsEEG, and structural MRI. Correlations (Spearman's) were assessed between the 35 rsEEG features (that most accurately discriminated the AD patients), neuroanatomical measures (derived using Freesurfer), and neuropsychological test results. **Results:** Cortical Thickness (CT) measures within the left posterior cingulate and right precuneus were related to alpha features. Beta features were associated with regions including the right entorhinal cortex, middle temporal, supramarginal, lingual, and paracentral cortex, in addition to the anterior cingulate cortex (ACC) and precuneus, bilaterally. Gamma features correlated with regions that included the right ACC and fronto-parietal cortex. Delta features were linked to the left fronto-parietal and right entorhinal cortex. Theta features were associated with the left ACC and visual cortex. In relation to disease staging – Clinical Dementia Rating scores were correlated with gamma features at frontal electrode sites, and with power over frequency bands, delta to beta, at Fz. Alpha features were associated with hippocampal volume (bilaterally), whereas some delta features and a beta feature were linked to left hippocampal volume. **Conclusions:** These preliminary correlation analyses highlight multiple brain regions that appear to underpin the rsEEG abnormalities that occur due to AD. Given the rich

data offered by both rsEEG and by structural MRI, future studies could investigate the combined potential for these techniques to classify the dementias.

P2-365 ADDED DIAGNOSTIC VALUE OF 18F-FLUTEMETAMOL PET SCANNING IN YOUNG PATIENTS WITH DEMENTIA

Ron Handels[1,2], Marissa D. Zwan[3], Wiesje Pelkmans[4], Femke H. Bouwman[5], Geert Jan Biessels[6], Gerwin Roks[7], Niki S. M. Schoonenboom[8], Nicolaas A. Verwey[9], Bart N. M. Van Berckel[10], Wiesje M. van der Flier[11], Frans R. J. Verhey[12], Philip Scheltens[5], *[1]Maastricht University; Department of Psychiatry and Neuropsychology; Alzheimer Centre Limburg; School for Mental Health and Neurosciences, Maastricht, Netherlands; [2]Karolinska Institutet, Department for Neurobiology, Care Sciences and Society, Center for Alzheimer Research, Div of Neurogeriatrics, Solna, Sweden; [3]VU University Medical Center, Amsterdam, Netherlands; [4]Alzheimer Center and Department of Neurology, VU University Medical Center, Amsterdam, Netherlands; [5]Alzheimer Center Amsterdam, Department of Neurology, Amsterdam Neuroscience, Vrije Universiteit Amsterdam, Amsterdam UMC, Amsterdam, Netherlands; [6]Department of Neurology, Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, Netherlands; [7]Elisabeth-TweeSteden Ziekenhuis, Tilburg, Netherlands; [8]Department of Neurology, Spaarne Gasthuis Haarlem, Haarlem, the Netherlands; [9]Medisch Centrum Leeuwarden, Leeuwarden, Netherlands; [10]Amsterdam UMC, Amsterdam, Netherlands; [11]VU University Medical Center, Amsterdam Neuroscience, Amsterdam, Netherlands; [12]Maastricht University, Maastricht, Netherlands. Contact e-mail: ron.handels@maastrichtuniversity.nl*

Background: A correct diagnosis of Alzheimer's Disease (AD) is considered important for access to correct treatment, advice, and care. We aim to estimate the diagnostic accuracy of 18F-Flutemetamol PET. **Methods:** In the Dutch Flutemetamol Study, 211 participants, suspected of dementia, aged $\leq$ 70, diagnostic confidence <90% and mentally competent (MMSE $\geq$ 18) received an 18F-Flutemetamol PET scan and were followed up for 2 years. At baseline and 1- and 2-year follow-up, participants received a standardized clinical dementia evaluation including neuropsychological testing and (mainly at baseline) MRI. 18F-Flutemetamol PET scans were made in clinical practice at baseline and the etiological diagnosis before (pre-PET diagnosis; standard clinical dementia evaluation without PET) and after (post-PET diagnosis; standard clinical dementia evaluation with PET) was recorded. Independently from this, a panel of 3 cognitive neurologists rated all 2-year clinical information summarized in a patient vignette (blinded for PET) and their majority (minimal 2 identical ratings) etiological diagnosis (reference diagnosis). The primary outcomes were the diagnostic accuracy of post-PET compared to the reference diagnosis, and the correctness of diagnostic changes from pre- to post-PET. **Results:** The panelists rated 95 cases. A reference diagnosis was set in 77 (81%), and in 17 (19%) cases no majority diagnosis was present. In 67 (87%) cases post-PET was in line with the reference, and in 65 (84%) the pre-PET diagnosis was in line with the reference (p=0.754). In 16 cases (21%) the diagnosis changed between pre-PET to post-PET. Of those 16, 6 changes were correct (i.e. post-PET in line with reference), 6 changes were incorrect (i.e. post-PET differed from reference), 4 changes were within a mixed reference etiology (e.g. pre-PET AD to post-PET FTD, consensus etiology was mixed AD/FTD). **Conclusions:** Preliminary results show a high accuracy of post PET. With regard to added diagnostic value, PET did not improve the diagnosis compared to standard clinical dementia evaluation. Further research on factors predictive for correct diagnostic change could identify a subpopulation in which PET better correlates to the course of clinical symptoms, and potentially improves clinical management and patient health.

P2-366 QUANTIFYING MITOCHONDRIAL AND SYNAPTIC FUNCTION IN ALZHEIMER'S DISEASE USING [^{18}F]BCPP-EF, [^{11}C]SA4503 AND [^{11}C]UCB-J PET IMAGING

Ashwin V. Venkataraman[1,2,3], Ayla Mansur[1,3,4], Yvonne Lewis[3,4], Ece Kocagoncu[5], Anne Lingford-Hughes[1], Mickael Huiban[3,4], Jan Passchier[3,4], James B. Rowe[3,5], Hideo Tsukada[3,6], David J. Brooks[3,7], Roger N. Gunn[3,4,8], Paul M. Matthews[1,2,3], Eugenii A. Rabiner[3,4,9], Mindmaps Consortium[3], *[1]Division of Brain Sciences, Imperial College London, London, United Kingdom; [2]UKDRI at Imperial College London, London, United Kingdom; [3]MINDMAPS Consortium, London, United Kingdom; [4]Invicro LLC, London, United Kingdom; [5]University of Cambridge, Cambridge, United Kingdom; [6]Hamamatsu Photonics, Hamamatsu, Japan; [7]Newcastle University, Newcastle upon Tyne, United Kingdom; [8]Imperial College London, London, United Kingdom; [9]Centre for Neuroimaging Sciences, IoPPN, King's College, London, United Kingdom. Contact e-mail: a.venkataraman@imperial.ac.uk*

Background: Mitochondrial deficits leading to synaptic dysfunction have been hypothesised in the pathophysiology of AD with Aβ/tau impairing mitochondrial function. To date a combined evaluation of human mitochondrial and synaptic function has not been performed directly *in vivo*. We describe the pilot results of MINDMAPS-AD aiming to evaluate mitochondrial and synaptic function in the brain of patients with MCI/AD. The novel radioligands [^{18}F]BCPP-EF, [^{11}C]SA4503 and [^{11}C]UCB-J are used to compare the regional density of mitochondrial complex I (MC1), the sigma 1 receptor (s1R) and synaptic vesicular protein 2A (SV2A) respectively. **Methods:** Six participants with AD related pathologies, EMCI (n=2), LMCI (n=2), and AD (n=2) and six cognitively normal (CN) subjects were enrolled. Participants fulfilled NIA-AA criteria and were Aβ +ve confirmed by [^{18}F]Florbetaben PET. Participants underwent PET scans with [^{18}F]BCPP-EF, [^{11}C]SA4503 and [^{11}C]UCB-J. Regions of interest (ROIs) were defined on subject MR images using an anatomical atlas. Target density was evaluated using the V_T, as well as V_T corrected for the plasma free fraction (f_P; V_T/fp), and the regional V_T ratio versus the V_T in the centrum semiovale, expected to have low levels of the targets (DVR). Comparison of regional target density and f_P between AD and CN was performed using a two-tailed, unpaired student's t-test. **Results:** The f_P values in the AD participants were higher for [^{18}F]BCPP-EF and [^{11}C]UCB-J (27%, $p < 0.02$; and 14%, $p < 0.08$ respectively) and hence V_T/f_P and DVR were chosen as the parameters of interest. V_T/f_P and DVR analyses provided consistent results, with lower mean density of MC1 (-10%) and SV2A (-16%) across the brain regions, and higher density of s1R (+16%) in participants with AD. Although statistical significance was reached in only some of the ROI, the overall pattern was consistent across ROI in this small pilot. **Conclusions:** Differences