processing occur earlier in the AD trajectory than previously hypothesized, that is, at the preclinical stage. Measures of semantic knowledge may be useful cognitive outcome measures in secondary prevention trials.





O2-02-06 SLOW GAIT SPEED AND LOW GRIP STRENGTH ARE RELATED TO WORSE ATTENTION AND MENTAL SPEED IN PATIENTS WITH SUBJECTIVE COGNITIVE DECLINE AND MILD COGNITIVE IMPAIRMENT

Astrid M. Hooghiemstra¹, Inez H.G.B. Ramakers², Nicole Sistermans¹, Yolande A.L. Pijnenburg¹, Pauline Aalten², Renske E.G. Hamel², René J.F. Melis³, Frans R.J. Verhey², Marcel G.M. Olde Rikkert³, Philip Scheltens¹, Wiesje M. van der Flier¹, ¹VU University Medical Center, Amsterdam, Netherlands; ²Alzheimer Center Limburg, School for Mental Health and Neuroscience, Maastricht University, Maastricht, Netherlands; ³Radboud University Medical Center, Nijmegen, Netherlands. Contact e-mail: a.hooghiemstra@vumc.nl

Background: The association between gait speed and cognitive functioning has been reported in mild cognitive impairment (MCI). Studies on physical vitality in patients with subjective cognitive decline (SCD) are scarce. We studied whether baseline gait speed and grip strength are associated with cognitive performances and decline over time in patients with SCD and MCI. Methods: We included 315 non-demented patients (142 SCD; 173 MCI) from the Clinical Course of Cognition and Comorbidity in Mild Cognitive Impairment (4C-MCI) study. Gait speed (seconds over 15 feet) was assessed at fast pace and grip strength (kilograms) was assessed using a hydraulic hand dynamometer (Jamar®). We assessed four cognitive domains: memory (Rey Auditory Verbal Learning Test: immediate and delayed recall and recognition, Visual Association Test), attention-mental speed (Digit span forward, Letter Digit Substitution Test, Trail Making Test (TMT) A, Stroop I), executive functioning (Digit span backward, TMTB/A, Stroop III-II, Category fluency), and global cognitive functioning (Mini-Mental State Examination (MMSE)). Linear Mixed Models, corrected for age, sex, and education, were used to estimate associations between baseline gait speed and grip strength (independent variables in separate models) and cognitive functioning and decline in the abovementioned domains. Results: Patients were 70 ± 9 years old, 111(35%) were female, baseline MMSE was 27 \pm 3 points, 29% of SCD patients converted to MCI/dementia and 38% of MCI patients converted to dementia in a mean follow-up time of 2 \pm 1 years. At baseline, both slower gait speed (β =-.17(.05), p < .001) and lower grip strength ($\beta = .11(.05)$, p = .03) were related to more impaired attention-mental speed. There were no associations between baseline gait speed/grip strength and the other cognitive domains, nor with longitudinal cognitive decline. After stratification for group (SCD/MCI) and age (≤65/ >65 years), associations of attention-mental speed with gait speed were stronger in younger patients and patients with MCI. In addition, attention-mental speed associations regarding grip strength were stronger in older patients. **Conclusions:** These findings indicate cross-sectional relationships between both more impaired baseline gait speed and grip strength and worse attention-mental speed. Gait speed and grip strength do not seem to be indicative of imminent cognitive decline within two years.

MONDAY, JULY 20, 2015 ORAL SESSIONS O2-03 EPIDEMIOLOGY: DIET AND COGNITION

02-03-01 VITAMIN D AND DECLINE IN GLOBAL COGNITION AND MEMORY IN THE CARDIOVASCULAR HEALTH STUDY

Elzbieta Kuzma¹, Thomas J. Littlejohns^{1,2}, Maya Soni¹, Paulo H.M. Chaves³, Bryan R. Kestenbaum⁴, Lewis H. Kuller⁵, Oscar L. Lopez⁵, James T. Becker⁵, Kenneth M. Langa⁶, William E. Henley¹, Iain A. Lang¹, David J. Llewellyn¹, ¹University of Exeter, Exeter, United Kingdom; ²University of Oxford, Oxford, United Kingdom; ³Florida International University, Miami, FL, USA; ⁴University of Washington, Seattle, WA, USA; ⁵University of Pittsburgh, Pittsburgh, PA, USA; ⁶University of Michigan, Ann Arbor, MI, USA. Contact e-mail: e. kuzma@exeter.ac.uk

Background: Recent meta-analyses suggest that low serum vitamin D levels are associated with cognitive impairment. However, the relationship with specific cognitive domains remains largely unknown in older adults: vitamin D deficiency has been linked with executive dysfunction though there are no prospective studies investigating this association with memory. Methods: We used data on older adults free from dementia and cardiovascular disease with valid baseline serum 25-hydroxyvitamin D (25(OH)D) measurements who participated in the Cardiovascular Health Study between 1992-1993 and 1999. Further we excluded those with no follow-up cognitive assessment and those with low scores that precluded substantial decline resulting in 1,564 and 1,250 participants in our fully adjusted analyses of global cognition (mean follow-up 5.4 years, standard deviation [SD] 1.3) and memory (mean followup 4.5 years, SD 1.1) respectively. Serum 25(OH)D levels were measured using high-performance liquid chromatography-tandem mass spectrometry. The Modified Mini-Mental State Examination and the Benton Visual Retention Test were used to assess global cognition and memory respectively. Global cognitive impairment was defined as a decline of at least five points from baseline and memory impairment as at least one standard deviation decrease greater than the mean change score. Poisson regression models with a robust error variance and linear mixed-effects models were adjusted for sociodemographics, body mass index, smoking, alcohol consumption, depressive symptoms and gait impairment. Results: The relative risk of incident global cognitive impairment in participants severely deficient in serum 25(OH)D (<25 nmol/ L) and deficient (≥25 to <50 nmol/L) was 1.73 (95% confidence interval [CI]: 1.22-2.45) and 1.22 (95% CI: 0.97 - 1.53) respectively compared to sufficient levels (≥50 nmol/L). Annual rates of decline did not differ significantly between groups. Those severely deficient and deficient declined in memory -0.17 (95% CI: -0.31 - -0.03) and -0.04 (95% CI: -0.10 - 0.01) points per year respectively compared to sufficient levels. The association

with incident memory impairment was not significant but in the same direction. **Conclusions:** Our results confirm that severe vitamin D deficiency is linked with global cognitive impairment and suggest a weaker association with memory decline. Neuroimaging studies are needed to investigate the potential neurodegenerative and cerebrovascular mechanisms.

02-03-02 VITAMIN D AND THE RISK OF DEVELOPING NEUROIMAGING ABNORMALITIES

Thomas J. Littlejohns^{1,2}, Katarina Kos², William E. Henley³, Cedric Annweiler⁴, Olivier Beauchet⁴, Paulo H.M. Chaves⁵, Iain A. Lang³, Lewis H. Kuller⁶, Kenneth M. Langa⁷, Oscar L. Lopez⁶, Bryan R. Kestenbaum⁸, **David J. Llewellyn**³, ¹University of Oxford, Oxford, United Kingdom; ²University of Exeter Medical School, Exeter, United Kingdom; ³University of Exeter, Exeter, United Kingdom; ⁴University of Angers, Angers, France; ⁵Florida International University, Miami, FL, USA; ⁶University of Pittsburgh, Pittsburgh, PA, USA; ⁷University of Michigan, Ann Arbor, MI, USA; ⁸University of Washington, Seattle, WA, USA. Contact e-mail: david.llewellyn@exeter.ac.uk

Background: Low vitamin D concentrations have been associated with an increased risk of cognitive decline, all-cause dementia and Alzheimer's disease. Neuroimaging findings could provide an insight into the cerebrovascular and/or neurodegenerative pathologies underlying these associations. We investigated whether low vitamin D concentrations are associated with the risk of developing neuroimaging abnormalities in a population-based prospective cohort of elderly adults. Methods: The population consisted of 1,658 participants aged ≥65 years from the US-based Cardiovascular Health Study who were free from prevalent cardiovascular disease, stroke and dementia. Serum 25-hydroxyvitamin D (25(OH)D) concentrations were determined by liquid chromatography-tandem mass spectrometry from blood samples collected at baseline from participants in 1992-93. The first MRI scan was conducted between 1991-1994 and the second MRI scan was conducted between 1997-1999. Change in white matter grade, ventricular grade and presence of infarcts between MRI scan one and two were used to define neuroimaging abnormalities. Logistic regression models were adjusted for age, education, season of vitamin D collection, sex, BMI, smoking, alcohol consumption, depressive symptoms and length of follow-up. Results: Participants were followed-up for a mean of 5.0 years (Standard Deviation = 0.6). There were no statistically significant associations between 25(OH)D concentrations and the development of any selected neuroimaging abnormalities. To expand, the multivariate adjusted odds ratios (95% CI) for worsening white matter grade in participants who were severely 25(OH)D deficient (<25 nmol/L) and deficient (≥25-50 nmol/L) were 0.76 (0.35-1.66) and 1.09 (0.76-1.55) compared to participants with sufficient concentrations (\geq 50 nmol/L). The multivariate adjusted odds ratios for ventricular grade in participants who were severely 25(OH)D deficient and deficient were 0.49 (0.20-1.19) and 1.12 (0.79-1.59) compared to participants with sufficient concentrations. The multivariate adjusted odds ratios for incident infarcts in participants who were severely 25(OH)D deficient and deficient were 1.95 (0.84-4.54) and 0.73 (0.47-1.95) compared to participants with sufficient concentrations. Conclusions: There were no significant associations observed between serum vitamin D concentrations and the risk of worsening white matter grade, worsening ventricular grade or incident infarcts. Further studies are necessary to elucidate the potential mechanisms underlying the previously observed relationships between vitamin D concentrations and dementia related disorders.

```
02-03-03
```

3-03 HIGH GLYCEMIC DIET ASSOCIATED WITH BRAIN NEURODEGENERATION IN A HEALTHY MIDDLE-AGED COHORT

Martha Clare Morris¹, Christy C. Tangney², Yamin Wang¹, Rebecca L. Koscik^{3,4}, Corinne D. Engelman⁵, Mark A. Sager^{4,6}, Barbara B. Bendlin^{4,6,7}, Sterling C. Johnson^{4,6,7,8}, ¹*Rush University, Chicago, IL, USA*; ²*Rush University, Chciago, IL, USA*; ³*University of Wisconsin - Madison, Madison, WI, USA*; ⁴*Wisconsin Alzheimer's Institute, University of Wisconsin School of Medicine and Public Health, Madison, WI, USA*; ⁵*University of Wisconsin School of Medicine and Public Health, Madison, WI, USA*; ⁵*University of Wisconsin School of Medicine and Public Health, Madison, WI, USA*; ⁶*Alzheimer's Disease Research Center, University of Wisconsin School of Medicine and Public Health, Madison, WI, USA*; ⁷*Geriatric Research Education and Clinical Center, WI, S. Middleton Veterans Hospital, Madison, WI, USA*; ⁸*Waisman Laboratory for Brain Imaging and Behavior, University of Wisconsin-Madison, Madison, MI, USA. Contact e-mail: martha_c_morris@rush.edu*

Background: Insulin resistance during the preclinical stage of dementia is a risk factor for symptomatic MCI-AD. Diet composition is a major, established determinant of insulin resistance and diabetes, the most important components being a high glycemic diet and low quality carbohydrates, fats and fiber. Methods: We investigated the relations of dietary determinants of insulin resistance (glycemic index, fat composition, carbohydrates and fiber) to: 1) cognitive decline using mixed models, 2) total brain and hippocampal/total brain volumes at baseline using magnetic resonance imaging (MRI), and 3) MRI volumetric brain changes over 2 years using linear regression models among a cognitively healthy sample of 489 participants of the Wisconsin Registry for Alzheimer's Prevention study who completed a semi-quantitative food frequency questionnaire. All analyses were adjusted for age, sex, education, energy intake, and APOE- $\varepsilon 4$. Results: The analytic sample was on average 59.8 years of age at baseline (46 to 76 years). Low dietary consumption of simple carbohydrates was associated with slower cognitive decline. For example, rates of decline in verbal learning were slower for participants in the lowest quintile of sugar intake versus higher quintiles (β =0.0267, p=0.03) in adjusted mixed models. High glycemic index was associated with faster decline in working memory (β = -0.0195, p=0.04). High intakes (top 20%) of simple carbohydrates, trans fats, and glycemic load were associated with decreased hippocampal/total brain volumes in 188 participants at baseline (e.g. for glucose, $\beta = -0.427$ p=0.03; for trans, $\beta =$ -0.407 p=0.05; for glycemic load β = -0.3034 p=0.02). Greater atrophy in total brain volume over 2 years was associated with higher glycemic index score ($\beta = 0.002$ p=0.06). Conclusions: Dietary determinants of insulin resistance, including glycemic index, simple carbohydrates and trans fats, may contribute to neurodegenerative processes of the brain in late middle-age.

02-03-04 DIETARY NUTRIENT PATTERNS AND BRAIN STRUCTURE IN AN ELDERLY POPULATION

Yian Gu¹, Christian Habeck¹, Adam M. Brickman², Jose A. Luchsinger², Jennifer J. Manly³, Nicole Schupf¹, Yaakov Stern¹, ¹Columbia University, New York, NY, USA; ²Columbia University, New York, NY, USA; ³Columbia University Medical Center, New York, NY, USA. Contact e-mail: yg2121@ columbia.edu

Background: Mounting evidence have suggested that certain dietary nutrient intakes are associated with reduced risk for Alzheimer's disease and slower cognitive decline among the elderly. However, little is known about the association of dietary nutrient patterns with brain imaging markers such as brain atrophy, surrogate markers for clinical dementia. **Methods:** 674 dementia-free elderly