examination and white matter hyperintensities and/or small basal ganglia infarcts on neuroimaging were observed in less than half of the MMICI cases but nevertheless distinguished MMICI from cases attributed to non-vascular lesions more reliably than other features.

SUNDAY, JULY 11, 2010
ORAL
O1-06
EPIDEMIOLOGY: DIET AND INTERVENTION

O1-06-01   **DIETARY INTAKE OF FLAVONOIDS AND COGNITIVE DECLINE IN THE NURSES' HEALTH STUDY**

Elizabeth E. Devore[1], Jae Hee Kang[1], Meir J. Stampfer[1,2], Francine Grodstein[1,2], *[1]Channing Laboratory, Brigham & Women's Hospital, and Harvard Medical School, Boston, MA, USA; [2]Department of Epidemiology, Harvard School of Public Health, Boston, MA, USA. Contact e-mail: edevore@hsph.harvard.edu*

Background: Flavonoids, with their antioxidant properties and ubiquitous presence in the diet, may have beneficial effects on cognition in older adults, but few epidemiologic studies have explored this association; thus, we evaluated dietary intake of flavonoids in relation to cognitive decline in a large, prospective cohort of older women. **Methods:** Beginning in 1995-2000, we measured cognitive function in 16,010 Nurses' Health Study participants, aged 70+ years, with dietary data; follow-up assessments were conducted twice, at two-year intervals. Dietary information was collected regularly beginning in 1980, using a semi-quantitative food frequency questionnaire (FFQ), and we cumulatively averaged flavonoid intake from 1980 until the initial cognitive interview. Multivariate-adjusted, random effects linear regression was used to estimate mean differences in slope of cognitive decline across quintiles of flavonoid intake. **Results:** Higher intake of total flavonoids was associated with a slower rate of cognitive decline in models adjusted for age and education (e.g. for the global score, combining all six of our cognitive tests, p-trend = 0.03). For example, the mean difference in slope of decline was 0.03 standard units per three years of follow up (95% CI, 0.01-0.06), comparing the top versus bottom quintiles. We found similar results after adjustment for antidepressant use, alcohol intake, smoking, physical activity, vitamin supplements, and history of cardiovascular risk factors and diabetes (e.g. p-trend = 0.04 for the global score; mean difference in slope of decline = 0.03 standard units per three years; 95% CI, 0.00-0.06, comparing extreme quintiles); the magnitude of this effect is equivalent to the difference that we observe in women 1-2 years apart. When we examined six subclasses of flavonoids separately, greater intakes of flavonols were significantly associated with slower rates of decline (e.g. p-trends = 0.05 for the global score), although similar patterns of association were observed for all subclasses. These associations were not modified by smoking status (p-interaction = 0.3). **Conclusions:** We found that higher intake of flavonoid-rich foods is associated with modestly slower rates of cognitive decline in this large cohort of older women.

O1-06-02   **DIET, WHITE MATTER HYPERINTENSITIES AND COGNITIVE FUNCTION**

Nikolaos Scarmeas[1,2], Yian Gu[1], Adam M. Brickman[1,2], Jeri W. Nieves[3,4], Yaakov Stern[1,2], Jose A. Luchsinger[1,5], Charles DeCarli[6], Richard Mayeux[1,2], *[1]Taub Institute for Research in Alzheimer's Disease and the Aging Brain, Columbia University Medical Center, New York, NY, USA; [2]Department of Neurology, Columbia University Medical Center, New York, NY, USA; [3]Clinical Research Center, Helen Hayes Hospital, West Haverstraw, NY, USA; [4]Department of Epidemiology, Columbia University Medical Center, New York, NY, USA; [5]Department of Medicine, Columbia University Medical Center, New York, NY, USA; [6]Department of Neurology and Imaging of Dementia and Aging Laboratory, Center for Neuroscience, University of California, Sacramento, Sacramento, CA, USA. Contact e-mail: ns257@columbia.edu*

Background: White matter hyperintensities (WMH) have been associated with increased risk of stroke, cognitive decline, and dementia, but little is known about the relations between WMH and dietary habits. **Methods:** High-resolution structural magnetic resonance imaging (MRI) was collected on 603 non-demented community elderly participants of the Washington Heights-Inwood Columbia Aging Project imaging sub-study with available dietary assessments administered at the same visit. WMH volume was adjusted for total cranial volume and was log-transformed. The Petersen criteria were used to define mild cognitive impairment (MCI). Using Reduced-Rank Regression we examined whether dietary patterns (DP; linear combinations of 33 food groups) could explain the variation in WMH. We then examined potential associations of the WMH-related DP with cognitive performance (4 cognitive domain z-scores derived from a comprehensive neuropsychological battery) and with odds of MCI (n = 139). Models were adjusted for age, gender, ethnicity and education. Further adjustment for caloric intake, BMI, APOE-e4, smoking status, and comorbid diseases, did not change the associations materially. **Results:** We identified a DP that was positively correlated with WMH (r = 0.32, p < 0.0001; 11.4% of WMH variability explained). A higher DP-score (representing higher adherence to the DP) was characterized by higher intakes of beer, organ meat, snacks, and green leafy vegetables; and lower intakes of nuts, low fat dairy, coffee, low energy drinking, salad dressing, and whole grain. In adjusted models, individuals with higher DP-scores tended to have worse language (r = -0.11, p = 0.01), visuospatial (r = -0.14, p = 0.002), and marginally worse memory (r = -0.09, p = 0.06) z-score cognitive performances. The DP was not related to executive-speed z-score performance (r = -0.07, p = 0.12). Adjusted logistic regression analysis showed that, compared to the lowest DP-score tertile, the prevalence odds ratios (95%CI) of MCI for the middle and highest tertiles were 1.21(0.63-2.32) and 2.45(1.32-4.55), respectively (p for trend = 0.003). The DP was associated with both amnestic and non-amnestic MCI odds (p for trend < 0.05 for both). **Conclusions:** WMH may be related to dietary factors. The pattern of food consumption that is associated with more WMH is also associated with worse cognitive performance (in the domains of language and visualspatial function) and with increasing odds of MCI.

O1-06-03   **VITAMIN D AND COGNITIVE IMPAIRMENT IN NHANES III**

David J. Llewellyn[1], Iain A. Lang[1], Kenneth M. Langa[2], David Melzer[1], *[1]Peninsula Medical School, University of Exeter, Exeter, United Kingdom; [2]University of Michigan, Ann Arbor, MI, USA. Contact e-mail: david. llewellyn@pms.ac.uk*

Background: Recent European studies suggest vitamin D deficiency is associated with increased odds of cognitive impairment in later life, although findings from the US have been mixed. **Methods:** 3,325 adults aged 65 years or more completed cognitive assessments and provided blood samples in the Third National Health and Nutrition Examination Survey (NHANES III), a nationally representative cross-sectional study of the US non-institutionalized population. Cognitive impairment was assessed using measures of immediate and delayed verbal memory, orientation and attention (impairment was defined as the worst 10% of the distribution of combined scores). **Results:** Multivariate adjusted odds ratios (95% confidence interval) for cognitive impairment in participants who were 25(OH)D insufficient (≥50 <75 nmol/L), deficient (≥25 <50 nmol/L), and severely deficient (<25 nmol/L) in comparison with those sufficient (≥75 nmol/L) were 0.9 (0.6-1.3), 1.5 (1.0-2.2), and 4.2 (1.5-12.1) respectively (*P* for linear trend = 0.02). Log transformed levels of 25(OH)D were also significantly associated with cognitive impairment (*P* = 0.02). **Conclusions:** Vitamin D deficiency is associated with increased odds of cognitive impairment in the US elderly population. Further exploration of the possible causal relationship between vitamin D deficiency and cognitive impairment and dementia is warranted.

O1-06-04   **DIET, HIPPOCAMPAL VOLUME AND COGNITIVE FUNCTION**

Yian Gu[1], Adam M. Brickman[1,2], Jeri W. Nieves[3,4], Yaakov Stern[1,2], Jose A. Luchsinger[1,5], Charles DeCarli[6], Richard Mayeux[2,7], Nikolaos Scarmeas[1,2], *[1]Taub Institute for Research in Alzheimer's Disease*