These findings confirm the involvement of inflammation in the early pathogenesis of MCI.

**P2-130**

**METABOLIC SYNDROME AND COGNITIVE FUNCTION IN AN ELDERLY COHORT WITH MEMORY COMPLAINTS: A TRANSVERSAL STUDY**

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**Background:** Few studies have looked at the relationship between the components of Metabolic syndrome (Mets), a cluster of five cardiovascular risk factors including hypertension, abdominal obesity, high triglycerides, low High Density Lipoprotein (HDL)-Cholesterol, and elevated fasting glycaemia, as a whole, and cognition and these studies have provided controversial findings. **Methods:** Patients eligible for the study were over 70 years old, and benefited from a cognitive assessment with Mini Mental State Examination (MMSE/30) and Cognitive Efficiency Profile (CEP/100), a comprehensive, validated battery of neuropsychological tests. All patients had Blood Pressure (BP) and waist circumference measurements. A complete biological analysis and brain imagery were also performed. Mets was defined using the Third Adults Treatment Panel of the National Cholesterol Education Program criteria. Alzheimer Disease (AD), vascular dementia (Vad) and Mild Cognitive Impairment (MCI) diagnosis were performed according to the international criteria. **Results:** We included 412 elderly subjects, 67% of female, mean age: 76 ± 6 years. Mets prevalence was 14%. After adjustment for age, sex, educational level, albuminemia, MMSE and CEP score were significantly lower among people with Mets compared to those without (MMSE:23.8 ± 30 and 25.26 ± 30, p < 0.01 respectively and CEP score: 60 ± 100 and 49.9 ± 100, p < 0.001 respectively). Twenty seven % of patients were diagnosed with AD, 6.3% Vad, 43.7% MCI and 23% had normal cognition. Mets prevalence was 31% among patients with Vad, 17% in AD patients, 12% in MCI patients and 9% among cognitively normal subjects, p < 0.005. **Conclusions:** In this elderly cohort, Mets was associated with poorer cognitive function. The higher Mets prevalence of Mets was disclosed among Vad patients, then AD, then MCI patients. An association between Mets and AD was not found in recent prospective studies. Main strengths of our study are its homogenous elderly population with memory complaints, comprehensively assessed and adjustment for nutritional state. Its main limitation is its transversal design. A prospective follow-up of this cohort will be performed to assess the relationship between Mets and cognitive decline.

**P2-131**

**APOE-e4 ALLELE AND NEUropsychiatric SYMPTOMS IN MCI: FINDINGS FROM THE DESCRIPA STUDY**

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**Background:** Neuropsychiatric symptoms are common in subjects with MCI and may herald AD. The apolipoprotein E e4 allele (APOE-e4 allele) is the strongest known genetic risk factor for AD. The role of the APOE-e4 allele in the psychopathology associated with MCI remains in

**Predictive value of APOE e4 allele for psychopathology in MCI**

| NPI items | APOE-e4 | allele |
|-----------|---------|-------|
| Delusions | OR(95%CI) | Cut-off=0/1 |
| Hallucinations | 1.56(0.73-3.32) | 1.38(0.84-2.025) |
| Agitation | 1.37(0.85-2.20) | 1.37(0.85-2.20) |
| Depression | 0.82(0.52-1.29) | 0.72*(0.56-0.92) |
| Anxiety | 1.33(0.82-2.14) | 1.52(0.85-2.70) |
| Euphoria | 0.70(0.15-3.22) | – |
| Apathy | 1.40(0.85-2.31) | 0.88(0.54-1.42) |
| Disturbion | 0.70(0.41-1.12) | – |
| Irritability | 1.13(0.77-1.66) | 1.13(0.77-1.66) |
| Aberrant motor behavior | 2.97(1.08-8.18) | 2.97(1.08-8.18) |
| Night time behavior | 0.81(0.37-1.74) | 0.81(0.37-1.74) |
| Appetite | 1.74(0.95-3.18) | 1.74(0.95-3.18) |

APOE-e4=Apolipoprotein E e4 allele, AD=Alzheimer’s disease, NPI=Neuropsychiatric inventory

* Significant at p<0.05.
** Significant at p<0.001-cell with 0 data,not computed

**P2-132**

**BODY ADIPOSIty IN LATER LIFE AND THE INCIDENCE OF DEMENTIA: THE HEALTH IN MEN STUDY**

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**Background:** Adiposity in mid life has been postulated as a risk factor for dementia. It is unclear whether adiposity in late life is also a risk factor for dementia. The aim of this study was to determine if adiposity in later life increases the hazard of dementia. **Methods:** Cohort study of 12,047 men
aged 64-84 years (mean age 72.1 years) living in Perth, Australia. Adiposity exposures were baseline body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR). We used the Western Australian Data Linkage System to establish the presence of new cases of dementia between 1996 and 2009 according to the International Classification of Diseases, Crude and adjusted hazard ratio (HR, 95% confidence interval, 95%CI) of dementia for each adiposity marker was calculated using Cox regression models. Other measured factors included age, marital status, education, alcohol use, smoking, fat consumption, physical activity, and prevalent diabetes, dyslipidaemia and cardiovascular disease. Results: Compared with men with BMI < 25, participants with BMI between 25-30 had lower adjusted HR of dementia (HR = 0.83, 95% CI = 0.72-0.97). The HR of dementia for men with BMI = 30 was comparable to men with BMI < 25 (HR = 0.85, 95%CI = 0.70-1.05). Waist circumference showed no obvious association with dementia hazard. Men with WHR = 0.9 had lower adjusted HR of dementia than men with WHR < 0.9 (HR = 0.82, 95%CI = 0.68-0.98). We found a "J" shape association between measures of obesity and the hazard of dementia, with the nadir of risk being in the overweight range of BMI and about 1 for WHR. Conclusions: Higher adiposity is not associated with incident dementia in older men. Overweight men and those with WHR = 0.9 have lower hazard of dementia than men with normal weight and with WHR < 0.9.

P2-136 CAROTENOIDS AND COGNITIVE DECLINE IN OLDER WOMEN

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Background: Most clinical trials have shown no effect of antioxidant supplements on cognition in older adults. However, one large trial found that men assigned to very-long-term beta carotene supplementation demonstrated better cognitive function than those receiving placebo, although the trial only had a single measure of cognition at its close, and thus could not identify rates of cognitive decline. To explore this area further, we examined long-term intake of total and individual carotenoids in relation to cognitive decline over six years. Few other epidemiologic studies have specifically considered carotenoids. Methods: Beginning in 1995-2000, we measured cognitive function in 16,010 participants of the Nurses' Health Study, aged = 70 years; follow-up cognitive assessments were conducted three times, at two-year intervals. Dietary information was collected repeatedly beginning in 1980, with a semi-quantitative food frequency questionnaire approximately every four years; we averaged carotenoid intakes from 1980 through the initial cognitive interview to measure long-term intake. We used multivariable-adjusted, mixed linear regression to obtain mean differences in slopes of cognitive decline across quintiles of carotenoid intake (total carotenoids, alpha carotene, beta carotene, lutein/zeaxanthin, lycopene, and beta cryptoxanthin). Results: In models adjusted for age and education, higher lycopene intake was significantly associated with slower cognitive decline (e.g., p-trend = 0.02 for a global score, averaging together all six tests in our cognitive battery). For example, after adjusting for age and education, participants in the highest quintile of lycopene intake had a mean slope of decline in the global score that was 0.05 standard units less (95% CI: 0.00-0.10) compared to those in the lowest quintile. This mean difference is similar to difference we observe for women one to two years apart in age, suggesting that increasing intake of lycopene might delay cognitive aging by one to two years. The association remained after additional adjustment for multiple potential confounders. Alpha carotene, beta carotene, lutein/zeaxanthin, and beta cryptoxanthin were not associated with cognitive decline. Conclusions: Long-term consumption of lycopene-rich foods may be associated with modest delays in cognitive aging, although the relation with other carotenoids is less clear. This area merits further research.

P2-137 COGNITIVE DECLINE AND DEPRESSION IN 29 U.S. ALZHEIMER'S DISEASE CENTERS

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Background: Depression and cognitive impairment in the elderly frequently co-occur, but the effect of depression on cognitive deterioration is poorly understood. The relationship is complex and possibly multi-directional. Previous studies have provided conflicting evidence. To gain further insight into the relationship between depression, antidepressant use, and risk of progression from healthy aging to MCI and AD, we employed the National Alzheimer’s Coordinating Center (NACC) database to assess the risk of cognitive deterioration in patients with normal cognition or MCI. Methods: Individuals with either normal cognition (n = 4,009, 15% depressed) or MCI (n = 1,689, 31% depressed) were followed prospectively