control groups during the first year, and the proportion of energy from fat increased (paired t-test for groups separately, p<0.01). Intakes of vitamin E, vitamin D, and dietary fiber increased in the intervention group (p<0.05), and decreased in the control group (table 1). Furthermore, quality of fat improved in the intervention group: intake of saturated fatty acids decreased while the intake of unsaturated fatty acids increased. Participants were categorized into 3 groups based on their level of cognitive function, and dietary changes were compared across the tertiles. In the control group, those with highest cognitive scores had less decrease in vitamin D (p for trend across tertiles 0.032) and in vitamin E (p= 0.012) during the 1 st year. In the intervention group the positive dietary changes were observed irrespective of cognitive function. The FINGER dietary intervention shows that beneficial dietary changes can be achieved in a group of older, high-risk individuals. The lack of association between baseline cognition and dietary changes in the intervention group suggests that participants with different levels of baseline cognitive function benefit from a dietary intervention. The future results of the FINGER study will show, if these positive changes in the diet will also prevent cognitive decline and dementia.

Table 1
Nutrient intakes at baseline and the change during the first year of intervention in the FINGER study. Data are presented as mean (sd) and stars indicate statistically significant difference between the groups (***p<0.001)

	Intake at baseline		Change during the 1st year of intervention	
	Intervention group	Control group	Intervention group	Control group
Energy intake (MJ)	7.6 (2.3)	7.6 (2.2)	-0.2 (1.8)	-0.2 (1.8)
Fat (E%)	33.7 (6.5)	33.6 (6.6)	1.0 (6.8)	1.0 (6.9)
Saturated fat (E%)	12.4 (3.5)	12.3 (3.6)	-0.6 (3.4)	0.6 (3.4) ***
Unsaturated fat (mono- and poly-combined, E%)	16.6 (3.9)	16.7 (4.1)	1.4 (4.7)	0.4 (4.5) ***
Protein (E%)	17.4 (3.2)	17.3 (3.4)	0.1 (3.7)	-0.1 (3.6)
Carbohydrate (E%)	46.0 (7.3)	46.2 (7.2)	-0.5 (6.8)	-0.7 (6.8)
Fiber (g/MJ)	2.87 (0.90)	2.91 (0.91)	0.14 (0.93)	-0.13 (0.84) ***
Vitamin E (mg/MJ)	1.45 (0.92)	1.60 (2.70)	0.10 (0.96)	-0.14 (1.42) ***
Vitamin D (µg/MJ)	1.85 (1.52)	1.88 (1.62)	0.34 (1.89)	-0.09 (1.68) ***
Folate (µg/MJ)	36.0 (18.4)	36.4 (19.4)	1.6 (20.8)	-0.5 (25.9)

MONDAY, JULY 14, 2014
ORAL SESSIONS
O2-03
NEUROIMAGING: IMAGING IN MILD COGNITIVE IMPAIRMENT AND SUBJECTIVE MEMORY COMPLAINT

O2-03-01 INCREASED AMYLOID DEPOSITION IN OLDER ADULTS AT RISK FOR PROGRESSION TO ALZHEIMER'S DISEASE DUE TO GENETIC BACKGROUND AND/OR THE PRESENCE OF SIGNIFICANT MEMORY CONCERNS

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Background: Older adults with significant memory concerns (SMC) and/or genetic risk for AD are key groups of interest due to risk of progression. Our goal was to evaluate amyloid deposition, glucose metabolism, and medial temporal lobe (MTL) atrophy in SMC participants from ADNI. **Methods:** 569 participants were selected from the ADNI cohort, including 177 healthy controls (HC), 93 participants with SMC, and 299 patients with early mild cognitive impairment (EMCI). The HC participants were further divided into those with genetic risk (APOE ε4 positive and/or family history of AD (HC-risk)) and those without genetic risk (HC). The SMC participants were also further divided by the presence or absence of informant complaints about the participant's memory. SMC participants were considered to have self-only concerns (SMC) if the informant did not endorse >1.5 SD above the HC mean on the ECog Memory domain (~62% of items) and to have self-plus-informant concerns if the informant endorsed > 62% (SMC-plus). Florbetapir and structural MRI scans were downloaded from the ADNI site for the baseline visit and processed as previously described [1]. FDG PET scans were also downloaded from this timepoint and processed using standard techniques. Average Florbetapir SUVR, FDG SUVR, and structural grey matter density were extracted from the global cortex (PET) and bilateral hippocampus (MRI). Florbetapir PET scans were also compared between groups on a voxel-wise level in SPM8. **Results:** A significant difference in amyloid deposition was observed between groups on both voxel-wise and ROI analyses. HC-risk, SMC, and SMC-plus demonstrated more global and regional amyloid deposition than HC without risk, including in the global cortex (p=0.004) and precuneus (p<0.001). Differences in glucose metabolism and MTL atrophy were more variable with some regions showing a trend towards hypometabolism and MTL atrophy in HC-risk and SMC groups. **Conclusions:** Participants with SMC and HC participants at risk for progression to AD due to genetic background show increased amyloid deposition relative to HC without risk. This suggests that older adults with SMC, especially those with informant corroboration, are at increased risk for future cognitive decline and therefore may be a good target population for enrichment of clinical trials.[1] Risacher et al. (2013) Frontiers in Aging Neuroscience.

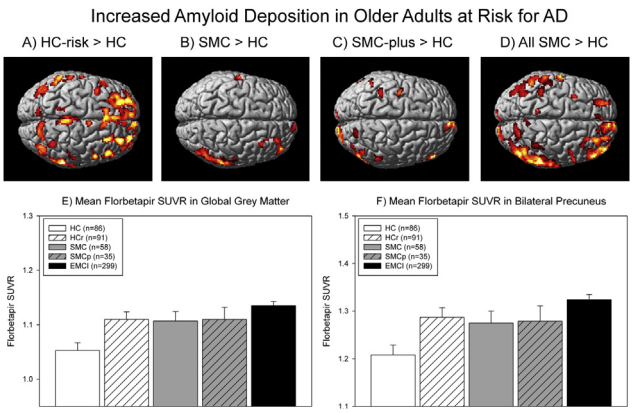

Figure 1. Increased Amyloid Deposition in Older Adults at Risk for AD

O2-03-02 REGIONAL BRAIN METABOLISM AND CORTICAL THICKNESS IN F18-FLUTEMETAMOL AMYLOID-POSITIVE VERSUS -NEGATIVE MILD COGNITIVE IMPAIRMENT PATIENTS

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Background: New criteria for Alzheimer's disease (AD) define prodromal AD as patients suffering from mild cognitive impairment (MCI) and presenting both an amyloid and a neurodegenerative marker. However, recent studies proved that some MCI do not carry brain amyloid. These patients have been named 'neurodegenerative only' MCI (NO-MCI). We aimed at comparing the regional pattern of cortical thinning and hypometabolism