

Table 2
Association between presence of cortical CMIs and cognitive impairment/dementia

Presence of cortical CMIs	CIND mild (n=275) OR (95% CI)	CIND moderate (n=288) OR (95%CI)	Dementia (n=42) OR (95% CI)
Model I	1.26 (0.46-3.40)	3.12 (1.18-8.23)	16.92 (3.37-85.05)
Model II	1.23 (0.44-3.44)	3.48 (1.28-9.51)	15.65 (3.01-81.36)
Model III			
Intracranial stenosis	1.25 (0.45-3.49)	3.41 (1.23-9.44)	13.63 (2.52-73.69)
Cerebral microbleeds	1.24 (0.44-3.44)	3.54 (1.29-9.76)	13.21 (2.42-72.09)
WMH volume	1.19 (0.43-3.35)	2.94 (1.00-7.97)	16.38 (2.76-97.21)
Lacunar infarcts	1.04 (0.37-2.93)	2.43 (0.87-6.79)	4.83 (0.68-33.82)
Cortical infarcts	1.17 (0.41-3.31)	2.85 (1.00-8.02)	9.65 (1.59-58.48)

Abbreviations: CMI, cerebral microinfarct; CIND, cognitive impairment no dementia; OR, odds ratios; CI, confidence interval; WMH, white matter hyperintensity

Model I included age, gender, and education

Model II included age, gender, education, ethnicity, hypertension, diabetes

Model I + II + each individual MRI marker added separately

Table 3
Association between presence of cortical CMIs and cognition

Presence of cortical CMIs	MMSE Mean difference (95% CI)	MOCA Mean difference (95% CI)	Composite Z scores Mean difference (95% CI)
Model I	-1.85 (-2.84; -0.86) p = < 0.001	-2.55 (-3.79; -1.29) p = < 0.001	-0.42 (-0.62; -0.21) p = < 0.001
Model II	-1.83 (-2.81; -0.84) p = < 0.001	-2.47 (-3.69; -1.24) p = < 0.001	-0.39 (-0.59; -0.19) p = < 0.001
Model III			
Intracranial stenosis	-1.93 (-2.90; -0.96) p = < 0.001	-2.34 (-3.57; -1.12) p = < 0.001	-0.38 (-0.58; -0.18) p = < 0.001
Cerebral microbleeds	-1.95 (-2.92; -0.99) p = < 0.001	-2.51 (-3.74; -1.28) p = < 0.001	-0.41 (-0.61; -0.21) p = < 0.001
WMH volume	-1.49 (-2.48; -0.52) p = 0.003	-2.04 (-3.26; -0.81) p = 0.001	-0.31 (-0.50; -0.11) p = 0.002
Lacunar infarcts	-1.53 (-2.53; -0.53) p = 0.003	-2.01 (-3.25; -0.76) p = 0.002	-0.29 (-0.49; -0.09) p = 0.004
Cortical infarcts	-1.11 (-2.13; -0.09) p = 0.032	-1.95 (-3.23; -0.67) p = 0.003	-0.31 (-0.52; -0.10) p = 0.003

Abbreviations: CMI, cerebral microinfarct; MMSE, Mini Mental Status Examination; MOCA, Montreal Cognitive Assessment; CI, confidence interval; WMH, white matter hyperintensity

Model I included age, gender, and education

Model II included age, gender, education, ethnicity, hypertension and diabetes

Model I + II + each individual MRI marker added separately

disease. Presence of cortical CMIs was associated with CIND-moderate [Odds Ratios (OR): 3.12; 95% confidence interval (CI):1.18-8.23], dementia [OR: 16.92; 95% CI: 3.37-85.05] and poorer cognitive function [mean difference in composite

Z-score: -0.42; 95% CI: -0.62; -0.21]. Additional adjustments for cardiovascular risk factors and other MRI markers did not alter these associations. **Conclusions:** Cortical CMIs are a novel MRI marker of cerebrovascular disease and are independently associated with cognitive impairment and dementia. These findings provide new insights into the burden of cerebrovascular disease in cognitive impairment. Future research is needed to establish the additional etiologic and prognostic significance of cortical CMIs.

P1-279

FACE-NAME RECOGNITION MEMORY IN PRECLINICAL AUTOSOMAL-DOMINANT ALZHEIMER'S DISEASE: AN fMRI STUDY

Daniel J. Norton¹, Ana Baena², Brendan Pulsifier³, Francisco Lopera⁴, Yakeel T. Quiroz¹, ¹Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA; ²Grupo de Neurociencias, Universidad de Antioquia, Medellin, Colombia; ³Massachusetts General Hospital, Charlestown, MA, USA; ⁴University of Antioquia, Medellin, Colombia. Contact e-mail: djnorton@partners.org

Background: Carriers of autosomal-dominant Alzheimer's disease (ADAD) mutations provide a unique model for studying the earliest physiological changes associated with Alzheimer's disease (AD). In AD, deficits in episodic memory have been linked to failure of a large network of brain areas, including medial temporal lobe and parietal regions. This study used functional MRI (fMRI) to examine recognition memory function in a group of healthy, young, cognitively-unimpaired individuals, who carry the E280A presenilin-1 (PSEN1) genetic mutation, which causes ADAD with almost complete penetrance. These individuals will go on to develop the first symptoms of the disease around the age of 45 years. **Methods:** Subjects were 8 PSEN1 mutation carriers (6 female, mean age 35.2, SD=4.9) and 8 non-carrier family members (6 female, mean age = 36.4, SD = 6.5) from a Colombian kindred. All participants learned a set of 86 face-name pairs, and then performed a forced-choice recognition memory test while in a 1.5T fMRI scanner. During recognition, faces were presented with two names underneath—one name that had been paired with the face in the learning phase, and the other a distracter—and were asked to identify the correct one. fMRI task-related analyses were restricted to the contrast of correct vs. incorrect responses. **Results:** Behaviorally, there were no significant differences in accuracy or reaction time between the groups (p<0.05). Both carriers and non-carriers showed activation in fronto-parietal regions during recognition of face-name pairs. Asymptomatic carriers exhibited less activation in bilateral parietal regions (e.g. precuneus, p=0.002 uncorrected) during recognition, compared to age-matched controls. **Conclusions:** Our preliminary results demonstrate that functional changes within the memory system occur years before clinical onset in ADAD. These preclinical changes suggest that fMRI patterns during episodic memory retrieval may be useful for early detection of Alzheimer's disease. Further analysis and a larger sample size are needed to follow up on this preliminary result.

P1-280

RELATIVE RISK RATIO FOR MRI PATCH-BASED APPEARANCE METRIC FOR FUTURE DECLINE IN COGNITIVELY HEALTHY ADNI PARTICIPANTS

Olivier Potvin¹, Azar Zandifar², Vladimir S. Fonov², Louis Collins^{2,3}, **Simon Duchesne**^{1,3,4}, ¹Institut Universitaire en Sante Mentale de Quebec, Quebec, QC, Canada; ²Montreal Neurological Institute, McGill University, Montreal, QC, Canada; ³True Positive Medical Devices Inc., Quebec, QC,