Table 2
Correlation between CERAD-K subtest performance and scores from visual rating scales of brain MRI

	GCA			MTA			WMH		
	0-1 N	2-3 N	P value	0-1 N	2-4 N	P value	0-1 N	2-3 N	P value
VFT									
Normal	11	10	0.031*	6	15	0.151	12	9	0.070
Mildly impaired	5	11		2	14		7	9	
Moderately impaired	4	21		2	23		6	19	
mBNT									
Normal	14	16	0.063	8	22	0.058	15	15	0.323
Mildly impaired	3	13		2	14		5	11	
Moderately impaired	3	13		0	16		5	11	
MMSE- KC									
Normal	1	2	0.056	0	3	0.002*	3	0	0.014*
Mildly impaired	6	3		5	4		6	3	
Moderately impaired	13	37		5	45		16	34	
WLMT									
Normal	9	11	0.088	6	14	0.103	10	10	0.451
Mildly impaired	6	8		2	14		6	8	
Moderately impaired	5	23		2	26		9	19	
CPT									
Normal	15	25	0.111	9	31	0.167	19	21	0.295
Mildly impaired	5	9		1	13		4	10	
Moderately impaired	0	8		0	8		2	6	
WLRT									
Normal	7	2	0.005*	4	5	0.042*	6	3	0.215
Mildly impaired	2	10		1	11		4	8	
Moderately impaired	11	30		5	36		15	26	
WLRcT									
Normal	11	9	0.021*	7	13	0.014*	10	10	0.301
Mildly impaired	4	9		0	13		3	10	
Moderately impaired	5	24		3	26		12	17	
CRT									
Normal	6	4	0.116	4	6	0.051	6	4	0.333
Mildly impaired	2	7		2	7		4	5	
Moderately impaired	12	31		4	39		15	28	

*: $p < 0.05$. VFT: Verbal Fluency Test, mBNT: Modified Boston Naming Test, MMSE-KC: Mini-Mental State Examination in the Korean Version of CERAD Assessment Packet, WLMT: Word List Memory Test. CPT: Construction Praxis Test, WLRT: Word List Recall Test, WLRcT: Word List Recognition Test, CRT: Construction Praxis Recall Test, GCA: Global Cortical Atrophy, MTA: Medial Temporal Atrophy, WMH: White Matter Hyperintensities

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RELATIONSHIPS BETWEEN BASELINE BIOMARKERS AND SUBSEQUENT COGNITIVE DECLINE IN COGNITIVELY UNIMPAIRED PSEN1 E280A MUTATION CARRIERS FROM THE COLOMBIAN KINDRED WITH AUTOSOMAL DOMINANT ALZHEIMER'S DISEASE

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Background: While brain imaging and cerebrospinal fluid (CSF) biomarkers have been used in the early detection and tracking of Alzheimer's disease (AD), their ability to predict subsequent clinical decline remains to be defined. In this study, we compared the ability of baseline PET amyloid-β (Aβ) and CSF measurements to predict subsequent cognitive decline in unimpaired Presenilin-1 (PSEN1) E280A mutation carriers from the Colombian autosomal dominant AD (ADAD) kindred, up to almost 25 years before the kindred's estimated median age of 44 at the onset of mild cognitive impairment. **Methods:** Thirty-seven cognitively unimpaired mutation carriers and non-carriers, aged 20-44 years, were recruited from the Alzheimer's Prevention Initiative (API) Colombia Registry. Baseline cerebral-to-cerebellar florbetapir PET standard-uptake-value ratios (SUVRs) and CSF Aβ1-42, total-tau and phospho-tau181 levels were related to 2-3 year subsequent decline on the API preclinical ADAD composite cognitive test score, previously found to be associated with preclinical progression. The mixed random effect model was used to estimate the relationship between baseline measures and subsequent cognitive decline in the mutation carriers. **Results:** In an independent replication, 2-3 year decline on the composite cognitive test score distinguished between carriers and non-carriers ($p=0.03$). In the carrier group, baseline florbetapir SUVRs and CSF p-tau/Aβ1-42 ratios were associated with subsequent decline on the composite cognitive test score ($p=0.008$, 0.04, respectively). CSF Aβ1-42, total tau, and p-tau levels alone were not ($p= 0.19$, 0.43 and 0.88, respectively), even after adjusting for age. Florbetapir SUVRs were slightly but not significantly better than CSF p-tau/Aβ1-42 ratios ($p=0.09$), and better than CSF Aβ1-42, total tau, and p-tau levels ($p= 0.04$, 0.04, and 0.06, respectively) in predicting subsequent cognitive decline. **Conclusions:** Aβ PET and, to a lesser extent, CSF p-tau/Aβ1-42 measurements may provide prognostic indicators of AD-related cognitive decline in ADAD mutation carriers. Indeed, Aβ PET may be a better prognostic indicator than CSF Aβ and tau levels in the group of mutation carriers assessed up to 25 years before their estimated age at clinical onset. Research is needed to further clarify the prognostic value of these biomarkers in cognitively unimpaired persons at risk for autosomal dominant and late-onset AD.

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STUDY OF SPONTANEOUS SEIZURE IN A MOUSE MODEL OF ALZHEIMER'S DISEASE

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Background: Seizures and epileptiform activity are prevalent clinical features, which presumably underlie some of cognitive decline, in patients with early stages of Alzheimer's disease (AD). In many mouse models of AD, spontaneous seizures and frequent epileptiform spike-wave discharges (SWD) have been described. Here, an APP/PS1 mouse model (Radde et al, 2006) was used to investigate differences in the time course of the occurrence of spontaneous and pharmacologically-induced seizures and SWD between ages of