

with 65% accuracy. Interestingly, coherence indices showed only modest correlation with MMSE scores ($r = .19$).

Conclusions: GC metrics of spontaneous speech differentiated between persons with AD and controls, but did not strongly correlate with MMSE performance. Such findings support the notion that many aspects of language are impacted in persons with AD. In addition to replication, future work should evaluate whether GC is also disrupted in persons with pre-clinical AD and its potential to assist with early detection.

Categories: Dementia (Alzheimer's Disease)

Keyword 1: speech

Keyword 2: dementia - Alzheimer's disease

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21 Assessment of Semantic Memory Decline in aMCI : Naming and Semantic Knowledge of Unique and Non-Unique Entities

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Objective: Semantic memory deficits have been reported in both Alzheimer's disease (AD) and amnesic mild cognitive impairment (aMCI). However, the nature of this decline is still a matter of debate. The aim of this study was to explore the patterns of semantic memory impairment in aMCI by examining performance on naming tasks, and on tests assessing both general and specific semantic knowledge.

Participants and Methods: Participants were divided in two groups matched for age and education, one comprising 33 aMCI individuals and the other 39 healthy controls. Three experimental tests assessing naming and semantic knowledge of unique items of famous persons (FACE) and places (PLACE), logos recognition (LOGO: brands and pictograms), and non-unique entities (Boston Naming Test: BNT) were administered, and the performance of the two groups was compared.

Results: Lower scores were observed on all naming tests (PLACE, FACE, LOGO and BNT) in the aMCI group compared to controls. On the PLACE test, the general knowledge mean score ($M=84.5$, $SD=12.9$) was significantly higher than the specific knowledge mean score ($M=54.2$, $SD=18.5$) in aMCI participants ($t(31)=11.9$, $p<.001$), but not in controls (general: $M=92.2$, $SD=11.1$; specific: $M=73.7$, $SD=15.8$), and there was a significant Group X Type of knowledge interaction ($F(1,1)=15.13$, $p<.001$, $\eta^2=.18$). On the FACE test, in addition to significant group and condition (naming, semantic questions) main effects, a significant interaction was found ($F(1,1)=7.19$, $p=.009$, $\eta^2=.09$). On the LOGO task, controls were significantly better on brand items ($M=94.4$, $SD=10.5$) than on pictograms ($M=83.3$, $SD=12.2$), while no significant difference was noted in aMCI (brands: $M=81.5$, $SD=22.6$; pictograms: $M=77.5$, $SD=14.1$). Lastly, on the BNT, aMCI participants benefited more from phonemic cues than controls ($F(1,1)=16.56$, $p<.001$, $\eta^2=.19$), suggesting a lexical access deficit, in addition to their semantic memory impairment.

Conclusions: This study adds to the growing evidence confirming the presence of semantic memory deficits in aMCI. Specific semantic knowledge seems to be more affected than general semantic knowledge, a finding reported in previous studies. Lexical access deficits, in addition to semantic decline, were also observed in the aMCI group. These results allow for a better understanding of the pattern of semantic memory deficits in the prodromal stage of AD and could potentially facilitate diagnosis of aMCI.

Categories: Dementia (Alzheimer's Disease)

Keyword 1: mild cognitive impairment

Keyword 2: dementia - Alzheimer's disease

Keyword 3: semantic processing

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22 Semantic Processing and its Relation to Brain Pathology in Individuals with Autosomal Dominant Alzheimer's Disease: Preliminary Findings from the Colombia-Boston Biomarker Study

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Objective: Semantic processing dysfunction has been shown to be an early indicator of cognitive decline in Alzheimer's disease (AD) and has been linked to early accumulation of AD-pathology. We examined semantic processing and its relation to AD pathology in non-demented individuals from a Colombian kindred with autosomal dominant AD due to the Presenilin1 E280A mutation (*PSEN1*).

Participants and Methods: A total of 13 cognitively unimpaired *PSEN1* mutation carriers (mean age: 36.92± 4.94), 7 carriers with mild cognitive impairment (MCI; mean age: 45±2.65), and 17 family non-carriers (mean age: 36±6.38) from the Colombia-Boston (COLBOS) longitudinal biomarker study were included. We used the Bateria IV Woodcock-Muñoz verbal analogies and text comprehension subtests to examine semantic processing, the Mini-Mental State Examination (MMSE) to assess global cognition and the CERAD word list delayed recall task to measure verbal memory. Participants also underwent PiB and florataucipir-PET to measure mean cortical amyloid and regional tau burden (entorhinal cortex and precuneus), respectively. Mann-Whitney U tests and Spearman's Rho correlations compared group differences in semantic processing, and its associations with age and pathological

markers. Post-hoc analyses excluded carriers with MCI and controlled for education.

Results: Carriers (including cognitively unimpaired and symptomatic individuals) performed significantly worse on the MMSE (carriers: 14.55, non-carriers: 24.24; $U=81.00$, $p=.006$), CERAD word list delayed recall (carriers: 13.63, non-carriers: 25.32; $U=48.00$, $p=.001$), and text comprehension (carriers: 16.36, non-carriers: 23.81; $U=107.00$, $p=.042$), than non-carriers, and showed a trend towards worse performance on verbal analogies (carriers: 17.16, non-carriers: 23.68; $U=124.50$, $p=.077$). There were no differences in text comprehension or verbal analogies performance between cognitively-unimpaired carriers and non-carriers. Across the whole sample, age was negatively associated with performance on verbal analogies ($r=-.341$, $p=.039$), but not text comprehension ($r=-.136$, $p=.428$). Among carriers only, better MMSE and CERAD delayed recall performance was associated with higher verbal analogies ($r=.561$, $p<.001$; $r=.662$, $p<.001$, respectively) and text comprehension scores ($r=.468$, $p=.004$; $r=.480$, $p=.003$, respectively). Greater amyloid burden was associated with worse verbal analogies performance ($r=-.432$, $p=.007$) and text comprehension ($r=-.430$, $p=.008$). Greater entorhinal cortex ($r=-.384$, $p=.016$) and precuneus tau burden ($r=-.318$, $p=.049$) was associated with worse performance on verbal analogies, but not text comprehension. These associations did not survive when excluding carriers with MCI or controlling for education.

Conclusions: Preliminary results show that non-demented mutation carriers had worse performance in semantic processing than non-carriers and performance was associated with markers of AD pathology. These findings suggest that changes in semantic processing may be early indicators of disease progression in individuals at increased risk for Alzheimer's disease dementia. Future studies with larger samples need to examine the role of education and the longitudinal trajectory of semantic processing dysfunction in AD.

Categories: Dementia (Alzheimer's Disease)

Keyword 1: dementia - Alzheimer's disease

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