Semantic Fluency Predicts Six-Year Progression to Mild Cognitive Impairment in Middle-Aged Men

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Abstract

Objective: Test the hypothesis that individual differences in episodic memory and verbal fluency in cognitively normal middle-aged adults will predict progression to amnestic MCI after 6 years.

Method: 1484 male twins, 842 of whom were cognitively normal at wave 1 completed measures of episodic memory and verbal fluency at up to 2 time-points (mean age 56 and 62 years).

Results: In the subgroup of 842, poor episodic memory predicted progression to both amnestic MCI (OR=4.42, 95% CI [2.44, 10.60]) and non-amnestic MCI (OR=1.92, 95% CI [1.32, 3.44]). Poor semantic verbal fluency also independently predicted progression to amnestic MCI (OR=1.86, 95% CI [1.12, 3.52]). In the full sample, a semantic-specific fluency latent variable at wave 1 (which controls for phonemic fluency) predicted change in episodic memory at wave 2 (β=.13), but not vice-versa (β=.04). Associations between episodic memory and verbal fluency factors were primarily explained by genetic, rather than environmental, correlations.

Conclusions: These findings emphasize the utility of memory and fluency measures in early identification of AD risk. Among individuals who were cognitively normal at wave 1, episodic memory moderately-to-strongly predicted progression to MCI at average age 62, emphasizing the fact that there is still meaningful variability even among cognitively normal individuals. Episodic memory, which is typically a primary focus for AD risk, declined earlier and more quickly than fluency. However, semantic fluency at average age 56 predicted of 6-year change in memory as well as progression to amnestic MCI even after accounting for baseline memory performance.

Introduction

The A/T/(N) framework emphasizes biomarkers in an effort to move toward a biological rather than a clinical diagnosis of Alzheimer's disease[1]. Although the framework is agnostic regarding the sequence of biomarker progression, it is generally assumed that biomarker positivity is present before cognitive manifestations. Amyloid beta and tau biomarkers indicate increased risk because they reflect underlying disease pathophysiology, but it does not necessarily follow that–with currently available detection techniques–these biomarkers will be the earliest predictors of progression to AD. In non-demented older adults, including those who already have mild cognitive impairment (MCI), cognitive markers–particularly episodic memory–do as well or better than AD biomarkers[2-5]. Semantic fluency impairment has also been associated with MCI and progression to AD in older adults[6-13]. However, because the AD process begins decades before dementia onset, there is a pressing need for earlier identification of risk[14,15].

With that in mind, there is still substantial variability in cognitive function even in cognitively normal, middle-aged adults. To address the need for early identification, we examined the ability of cognitive measures to predict progression to MCI in a 6-year follow-up of cognitively normal, community-dwelling men in their 50s who were rigorously defined as cognitively normal at baseline: 1) Does episodic memory predict progression to MCI? 2) Does semantic fluency predict progression to MCI after accounting for episodic memory? 3) Does episodic memory predict change in semantic fluency or vice versa? 4) To what extent do shared genetic influences underlie associations between semantic fluency and episodic memory?

Method

Subjects

Analyses were based on 1484 individual male twins who participated in at least one wave of the longitudinal VETSA project. All participants were recruited randomly from the Vietnam Era Twin Registry from a previous study[16]. All served in the United States military at some time between 1965 and 1975; nearly 80% did not serve in combat or in Vietnam[17,18]. Participants are generally representative of American men in their age group with respect to health and lifestyle characteristics[19]. See Table 1 for demographic characteristics of the sample, which includes the descriptive statistics for all covariates in the primary analyses involving MCI.

At wave 1 (N=1285), participants included 359 full monozygotic (MZ) twin pairs, 271 full dizygotic (DZ) twin pairs, and 25 unpaired twins. At wave 2 (N=1193), participants included 328 full MZ twin pairs, 231 full DZ twin pairs, and 74 unpaired twins. Most individuals participated at both sessions (N=1014). For analyses involving prediction of progression to MCI, we examined 842 individuals who were cognitively normal at wave 1, returned at wave 2, and had data for all relevant covariates. All participants completed informed consent, and all procedures were approved the Institutional Review Board of participating institutions.

Measures

Development of latent variable measures of episodic memory, verbal fluency, and other cognitive variables, and MCI diagnoses has been reported in our earlier work[20-23]. These measures are summarized briefly here.

All cognitive measures at the second wave of testing were adjusted to account for the fact that many of the subjects had encountered the tasks before[22]. Practice effects were computed according to the method of Rönnlund et al.[24,25] and utilized data from individuals who completed both waves of assessment (N = 1014), individuals who did not return at the second wave (N = 271), and attrition-replacement subjects randomly selected from the same twin registry who

completed the test battery for the first time at the second wave (N = 179) and were the same age as the wave 2 subjects (56 to 66). For each task, the practice effect calculation estimates a difference score (returnees minus attrition-replacements), and an attrition effect (returnees minus all wave 1 subjects). The practice effect is the difference score minus the attrition effect, and was subtracted from scores for all returnees. Although practice effects were small and often nonsignificant, it is important to correct for small and nonsignificant practice effects as ignoring these small differences result in the underdiagnoses of MCI in this sample[22].

Episodic memory. Episodic memory was measured with the Logical Memory (LM) and Visual Reproductions (VR) subtests of the Wechsler Memory Scale-III[26], and the California Verbal Learning Test-II (CVLT)[27]. The dependent measures were the LM and VR delayed recall scores, and the CVLT long delay free recall score. These measures were combined to create a latent factor.

Verbal fluency. Subjects performed 6 fluency subtests from the Delis-Kaplan Executive Function System (D-KEFS)[28]: phonemic (*F, A,* and *S*), followed by semantic subtests (*Animals, Boys' Names,* and *Fruits/Furniture*). Dependent measures were the number of words generated in 60 seconds. We utilized a latent variable model of verbal fluency that highlights unique variance in semantic fluency[29]. This model decomposes the variance in the 6 fluency subtests into two latent factors: a general fluency factor captures variance across phonemic and semantic subtests, and a semantic-specific factor captures the remaining variance shared among semantic subtests not captured by the general factor. To be comparable with the other measures, we used the number of words on Fruits/furniture switching condition, ignoring switching ability. This was validated in the previous work[29], which also showed that removal of this subtest has little impact on the factor structure of verbal fluency. Both factors were explained mostly by genetic

influences at mean ages 56 and 62 and demonstrated strong phenotypic and genetic correlations over this six-year interval[29].

Mild cognitive impairment. MCI was diagnosed using the Jak-Bondi actuarial/neuropsychological approach[30-32]. Impairment in a cognitive domain was defined as having at least two tests >1.5 SDs below the age- and education-adjusted normative means after accounting for "premorbid" cognitive ability by adjusting neuropsychological scores for performance on a test of general cognitive ability that was taken at a mean age of 20 years. The adjustment for age 20 cognitive ability ensures that the MCI diagnosis is capturing a decline in function rather than long-standing low ability. The validity of the VETSA MCI diagnoses is supported by previous studies[30-32] and in the present sample by evidence of reduced hippocampal volume in those diagnosed with amnestic MCI[33]. Although biomarker assays are not yet completed, higher AD polygenic risk scores were associated with significantly increased odds of MCI[21], indicating that our diagnosis is genetically-related to AD.

Because we were interested in transition to MCI, analyses involving MCI at wave 2 only included individuals who were cognitively normal at wave 1 and had data for all covariates. Of the 842 returnees meeting this criterion, 42 (5.0%) progressed to amnestic MCI, and 38 (4.5%) progressed to non-amnestic MCI. An additional 6 individuals who had language impairment (3 of which also had amnestic MCI), were assigned missing values for these analyses to prevent biasing estimates of the association between verbal fluency and MCI; 10 individuals were excluded for missing one or more covariates. However, odds ratios were nearly identical if these individuals were all included (in a model without any covariates).

Other cognitive measures (wave 1 only). To examine how other cognitive abilities account for the overlap between episodic memory and semantic fluency, a latent factor for

vocabulary was created using the vocabulary subtest of the Wechsler Abbreviated Scale of Intelligence (WASI)[34] and the multiple-choice vocabulary subtest of the Armed Forced Qualification Test[35,36]. Two executive function latent factors were included based on our previous work[37]. The first factor (Common Executive Function) captures common variance across 6 tasks assessing inhibition, shifting, and working memory[23]. The second latent factor (Working Memory-Specific) captures covariance among working memory tasks not already captured by the common factor.

Data Analysis

Phenotypic analyses involving MCI were conducted with mixed effects logistic regression using the lme4 package in R version 3.3.1[38]. Pair ID was included as a random effect to account for the clustering of data within families. In these analyses, we also controlled for wave 1 age, diabetes, hypertension, depression symptoms (based on Center for Epidemiologic Studies–Depression scale)[39], the time interval between wave 1 and wave 2, *ApoE* status (ε4+ alleles vs. ε4-), and years of education. The lme4 package uses list-wise deletion with missing observations, and reports profile-based 95% confidence intervals (95% CIs).

Additional phenotypic regression and cross-lagged analyses in the full sample were conducted using Mplus Version 7[40]. Genetic analyses were conducted using the structural equation modeling package OpenMx in R[41]. Both programs account for missing observations using a full-information maximum likelihood approach. Model fit for these structural equation models was determined using χ^2 tests, the Root Mean Square Error of Approximation (RMSEA), and the Comparative Fit Index (CFI). Good fitting models had χ^2 values less than 2 times the degrees of freedom, RMSEA values<.06, and CFI values>.95[42]. Additionally, good fitting genetic models did not fit significantly worse than a full Cholesky decomposition of all

measures, a common baseline model in twin studies. Significance of individual parameters was established with χ^2 difference tests and standard-error based (Mplus) or likelihood-based (OpenMx) 95% CI.

Genetically-informed models were based on the standard assumptions in twin designs, which decompose variance in phenotype (and covariance among phenotypes) into three sets of influences: additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E). Additive genetic influences are correlated at 1.0 in MZ twin pairs and 0.5 in DZ twin pairs because MZ twins share all their alleles identical-by-descent and DZ twins share, on average, 50% of their alleles identical-by-descent. Shared environmental influences (C), which are environmental influences that makes twins in a pair more similar, are correlated at 1.0 in both MZ and DZ twins. Nonshared environmental influences (E), which are environmental influences that make twins in a pair dissimilar, are correlated at 0.0 in both MZ and DZ twin pairs, by definition. We also assume equal means and variances within pairs and across zygosity. These assumptions for univariate analyses apply to multivariate cases and to situations where phenotypic correlations between constructs are decomposed into their genetic ($r_{genetic}$) and nonshared environmental components ($r_{environmental}$). Shared environmental influences on verbal fluency factors, and their correlations with episodic memory, were not estimated based on our previous work showing no evidence for shared environment on verbal fluency[29].

Data Availability

VETSA data are publicly available. Information regarding data access can be found at our website: https://medschool.ucsd.edu/som/psychiatry/research/VETSA/Pages/default.aspx

Results

Descriptive Statistics

Demographic and clinical characteristics are displayed in Table 1. There were no significant differences between cognitively normal and MCI groups in any of these characteristics, except that individuals who progressed to amnestic MCI were older than those who remained cognitively normal at both wave 1 (p=.008) and wave 2 (p=.012). Fluency and memory scores are displayed in Table 2.

Predicting Progression to MCI

Table 3 displays the longitudinal logistic regression analyses. Progression to amnestic MCI (Table 3A) or non-amnestic MCI (Table 3B) at wave 2 is predicted by general fluency, semantic-specific and episodic memory factor scores at wave 1 (exported from Mplus), as well as the covariates. Higher odds ratios indicate increased odds of progression to MCI (estimated for -1 *SD* decrease on the original factor score scale).

Results indicated that episodic memory at wave 1 strongly predicted progression to amnestic MCI at wave 2, even when controlling for both verbal fluency factors, OR=4.42, p<.001, 95% CI [2.44, 10.60]. Poor semantic-specific fluency was also associated with nearly double the odds of being diagnosed with amnestic MCI at wave 2 even when controlling for episodic memory, OR=1.86, p=.025, 95% CI [1.12, 3.52]. Poor general fluency was not associated with increased odds of amnestic MCI, OR=1.29, p=.332, 95% CI [0.79, 2.35]. Interestingly, poor episodic memory was also associated with progression to non-amnestic MCI, OR=1.92, p=.009, 95% CI [1.21, 3.34]. Neither fluency factor predicted progression to non-amnestic MCI, both ps>.274.

Longitudinal Associations Between Episodic Memory and Verbal Fluency

We next fitted a phenotypic cross-lagged model using fluency and memory data from both waves in the full sample. As seen in Figure 1, memory at wave 2 was significantly predicted

by semantic-specific fluency at wave 1, β=.13, p=.035, 95% CI [.01, .25], even after accounting for memory at wave 1, β=.85, p<001, 95% CI [.74, .98]. In contrast, memory at wave 1 did not predict semantic-specific fluency at wave 2, β=.04, p=.582, 95% CI [-.09, .16]. The general fluency factor also did not predict later memory (or vice versa).

Biometric Models of Cognitive Abilities

In a biometric twin models of wave 1 and 2 data (Figure 3), the phenotypic correlations between memory and semantic-specific fluency were explained primarily by genetic influences, $r_{genetic}$=.65, 95% CI [.46, .88] at wave 1, $r_{genetic}$=.73, 95% CI [.58, .94] and wave 2, explaining 87% (wave 1) and 86% (wave 2) of the total phenotypic correlations. There was also a significant nonshared environmental correlation between episodic memory and semantic-specific fluency at wave 2 only, $r_{environmental}$=.28, 95% CI [.01, .55]. This environmental correlation was nonsignificant at wave 1, but similar in magnitude, $r_{environmental}$=.25, 95% CI [-.08, .60]. Genetic correlations explained 92% (wave 1, $r_{genetic}$=.50, 95% CI [.36, .70]) and 94% (wave 2, $r_{genetic}$=.36, 95% CI [.25, .50]) of the correlation between memory and general fluency.

Using only the wave 1 data, we fit a regression model (Figure 2) in which the 2 fluency factors were regressed on factors for memory, vocabulary, and the 2 executive function factors. Although semantic-specific fluency was positively correlated with episodic memory (r=.46, p<.001), vocabulary (r=.19, p<.001), and common executive function (r=.20, p=.003), only the association with memory remained significant in the regression model, β=.60, p<.001. Conversely, the general fluency factor was correlated with all other cognitive abilities (rs=.32 to .50) including memory, r=.38, p<.001, but this association with memory was nonsignificant in the regression model, β=.04, p=.538. These findings suggest that the genetic correlations between memory and general fluency were likely explained by their overlap with other cognitive

abilities, but the association episodic memory and semantic-specific fluency reflects unique genetic associations between these constructs.

Discussion

These findings highlight the importance of episodic memory and semantic fluency as risk factors for cognitive decline and MCI. Among middle-aged men (ages 51-60) who were cognitively normal at baseline, both measures predicted amnestic MCI 6 years later. Importantly, our previous results showing that higher AD polygenic risk scores were associated with significantly increased odds of having MCI in this sample provide support for this diagnosis being AD-related MCI[21]. The current study demonstrated that baseline semantic-specific fluency predicted progression to amnestic MCI even after controlling for baseline memory. In other previous work, we found that there was no change in the semantic-specific factor (*d*=-.01) over this 6-year interval[29], yet in the current study it did independently predict episodic memory 6 years later. Thus, semantic-specific fluency is associated with episodic memory, but it does not necessarily change at the same time or rate as episodic memory. Nevertheless, it does add to the prediction of memory and progression to amnestic MCI beyond the predictive value of baseline memory itself.

With respect to amnestic MCI, the focus is naturally on episodic memory, but our results indicate that relatively poor semantic-specific fluency performance among cognitively normal adults may precede decline in episodic memory. The associations between semantic-specific fluency and episodic memory were driven by genetic influences at both waves, suggesting that these risk factors are not new to middle age, but may reflect genetic risk factors that impact cognitive performance throughout the lifespan. The associations also had significant non-shared environmental influences at wave 2, suggesting unique environmental risk factors that may

present themselves later in life. Although the prediction was not as strong, episodic memory also predicted non-amnestic MCI, suggesting it can also be a predictor of cognitive decline in other domains (except fluency).

Although biomarkers are necessary for biologically-based diagnosis, and are important for identifying individuals at greatest risk for cognitive decline and dementia, several studies have shown that neuropsychological tests are often better and earlier predictors of progression to AD than biomarkers[3-5,43-45]. One reason may be that current techniques for measuring biomarkers are not necessarily able to detect the earliest stages of disease progression. For example, beta-amyloid accumulates slowly for as much as 2 decades before symptom onset, but PET ligands have high affinity only or later stage neuritic beta-amyloid plaques[46,47]. Continuing the search for earlier biomarker detection is critically important. Meanwhile, the present results suggest that measures of memory and fluency are effective early indicators of progression to amnestic MCI given that participants were only in their 50s at the baseline assessment. They can also be completed in a short amount of time at little expense. Fluency and memory measures may be combined with biomarkers to further improve prediction of MCI and AD. Biomarker data will improve determination of specificity for AD-related deficits, and fluency and memory measures may also be useful as screening tools for identifying individuals who should be followed up with biomarker testing.

Strengths and Limitations

This sample comprised primarily white, non-Hispanic men, so these findings may not generalize across sex and race/ethnicity. It will also be important for future work to directly compare these odds ratios with those for biomarkers (preferably in the same model) and to examine whether neuropsychological tests more strongly predict progression to MCI in subsets

of individuals (e.g., who are already amyloid positive). Strengths of the study are that this is a national, rather than a local, community-based non-clinical sample of middle-aged men with health, education, and lifestyle characteristics that are representative of American men in their age range. The young age of the baseline assessment is also a strength with respect to early identification of risk.

Conclusions

This study demonstrated that episodic memory and semantic fluency independently predicted progression to amnestic MCI in cognitively normal, middle-aged men who were only in their 50s at baseline. These findings demonstrate the usefulness of examining neuropsychological variability among cognitively normal individuals for early identification of risk for AD, highlighting the fact that cognitively normal adults should not treated as a homogeneous group. It will also be important to further examine the cognitive processes that distinguish semantic from phonemic fluency and their underlying neural circuitry, to understand why semantic-specific fluency predicts change in episodic memory (but not vice-versa). This may lead to further insights regarding the mechanisms that drive cognitive decline and the progression to AD. In the meantime, the results suggest that it will be important to take advantage of the predictive ability of episodic memory and verbal fluency tests in studies designed to identify and individuals at greatest risk for MCI (and later dementia). Ultimately, the combination of these cognitive abilities and biomarkers may further improve prediction of MCI and AD.

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Table 1

Demographic Characteristics of the Full Sample and Covariates Included in Analyses Involving Mild Cognitive Impairment (MCI)

Demographic Variable	N	M	SD	Range	*p* (CN vs. aMCI)	*p* (CN vs. nMCI)
All Subjects						
Lifetime Education	1483	13.8	2.11	5, 30	0.229	0.168
Ethnicity (% white non-Hispanic)	1484	88.1	-	-	0.793	0.743
ApoE status (% e4 positive)	1444	29.4	-	-	0.499	0.093
Wave 1						
Age	1290	55.9	2.44	51.08, 60.67	0.136	0.008
Depression Symptoms	1283	8.4	8.22	0, 52	0.809	0.818
Diabetes (% yes)	1237	11.2	-	-	0.747	0.420
Hypertension (% yes)	1237	59.8	-	-	0.259	0.140
Wave 2						
Age	1207	61.7	2.45	56.00, 66.92	0.098	0.012
Age Interval (wave 2 - wave 1)	1013	5.7	0.69	4.25, 9.42	0.607	0.510

Note: The final two columns display the *p* value for comparisons between individuals who were cognitively normal at wave 1 and remained cognitively normal (N=762) and either progressed to amnestic MCI (aMCI; N=42) or nonamnestic MCI (nMCI; N=38) at wave 2. Lifetime education was the number of years of school completed. Depression symptoms were measured with the Center for Epidemiologic Studies – Depression Scale[39], with scores above 15 indicating risk for clinical depression.

Table 2

Descriptive Statistics for Measures of Verbal Fluency and Episodic Memory in the Full Sample

Task	*N*	*M*	*SD*	Range	Skewness	Kurtosis
Wave 1 (age 56)						
Letter F	1277	12.28	4.09	1 - 29	0.29	-0.02
Letter A	1277	11.15	3.90	1 - 29	0.41	0.34
Letter S	1277	13.48	4.32	1 - 31	0.25	0.08
Animals	1275	19.20	4.43	6 - 39	0.26	0.26
Boys' Names	1276	19.09	4.48	6 - 40	0.34	0.58
Fruits / Furniture	1277	12.75	2.55	4 - 22	-0.01	0.30
Wave 2 (Age 62)						
Letter F	1189	11.68	4.04	0.82 - 28.00	0.32	0.14
Letter A	1189	10.39	3.87	1.44 - 26.00	0.31	-0.06
Letter S	1189	12.70	4.31	0.00 - 28.83	0.24	-0.08
Animals	1189	19.11	4.51	5.08 - 35.08	0.17	0.11
Boys' Names	1189	18.32	4.50	4.31 - 37.31	0.23	0.47
Fruits / Furniture	1188	12.30	2.58	2.71 - 21.71	0.02	0.38

Note: In all analyses involving the full sample, these dependent measures were standardized residual scores after removing the effect of age on each measure, but the unadjusted scores are presented here. However, the wave 2 scores reported here reflect the adjustments for practice effects for individuals who participated at both waves of assessment.

Table 3

Logistic Regression for Mild Cognitive Impairment (MCI) Predicted by Fluency and Memory Factor Scores

Dependent Variable	A) No MCI vs. Amnestic MCI		B) No MCI vs. Non-Amnestic MCI	
	OR	95% CI	OR	95% CI
Cognitive Factor Scores				
General Fluency	1.29	[0.79, 2.35]	1.29	[0.82, 2.12]
Semantic-Specific	**1.86**	**[1.12, 3.52]**	0.92	[0.59, 1.42]
Episodic Memory	**4.42**	**[2.44, 10.60]**	**1.92**	**[1.32, 3.34]**
Covariates				
Age (wave 1)	1.28	[0.76, 2.32]	**1.77**	**[1.13, 3.05]**
Age Interval (wave 2 - wave 1)	1.41	[0.87, 2.44]	0.99	[0.58, 1.57]
Depression (wave 1)	0.92	[0.56, 1.47]	1.01	[0.66, 1.52]
ApoE e4+	1.49	[0.54, 4.95]	0.47	[0.13, 1.25]
Diabetes (wave 1)	1.05	[0.21, 4.47]	1.55	[0.43, 4.94]
Hypertension (wave 1)	1.35	[0.52, 4.28]	1.69	[0.72, 4.48]
Years of Education	0.96	[0.57, 1.65]	0.82	[0.49, 1.29]

Note: Significant odds ratios (ORs) are displayed in bold ($p < .05$). Factor scores for verbal fluency and episodic memory were scored and standardized so the odds ratio indicate the increase in odds of converting to amnestic MCI (A) or non-amnestic MCI (B) at -1 *SD* for that variable. Measures of age, depression, and years of education were also standardized, but not reverse scored. Odds ratios for ApoE status, diabetes, and hypertension reflect increase in odds for having an ε4 allele, diabetes, or hypertension, respectively. CI = Confidence interval.

Figure Captions:

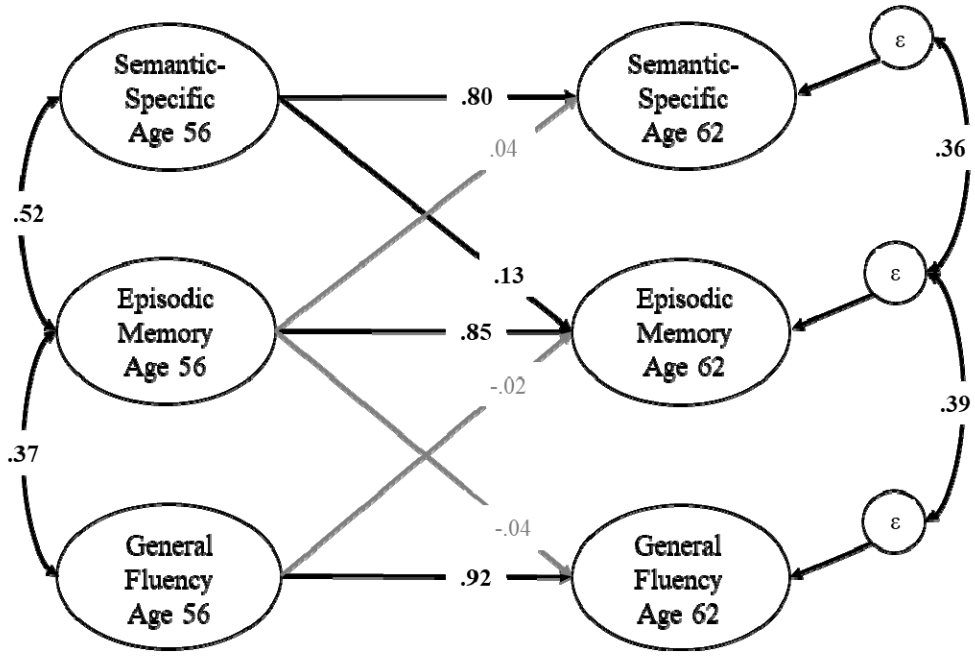

Figure 1: Phenotypic cross-lagged model of verbal fluency factors and episodic memory. Both fluency latent factors are correlated with episodic memory at wave 1 (age 56) and predict episodic memory at wave 2 (age 62). Episodic memory at wave 1 also predicts the fluency factors at wave 2. Each factor at wave 1 also predicts itself at wave 2, and there are residual correlations between the residual variances (ε) on the fluency factors at wave 2 and the episodic memory factor at wave 2. Not pictured are the factor loadings on individual tasks (which were equated across time and similar in magnitude to those displayed in Figure 3) or residual correlations between all observed variables across time (e.g., *Animals* at wave 1 correlated with *Animals* at wave 2). Significant paths and correlations are displayed in bold, with black text and lines ($p < .05$). The semantic-specific factor is the only factor to significantly predict another construct at the second wave ($\beta = .13$).

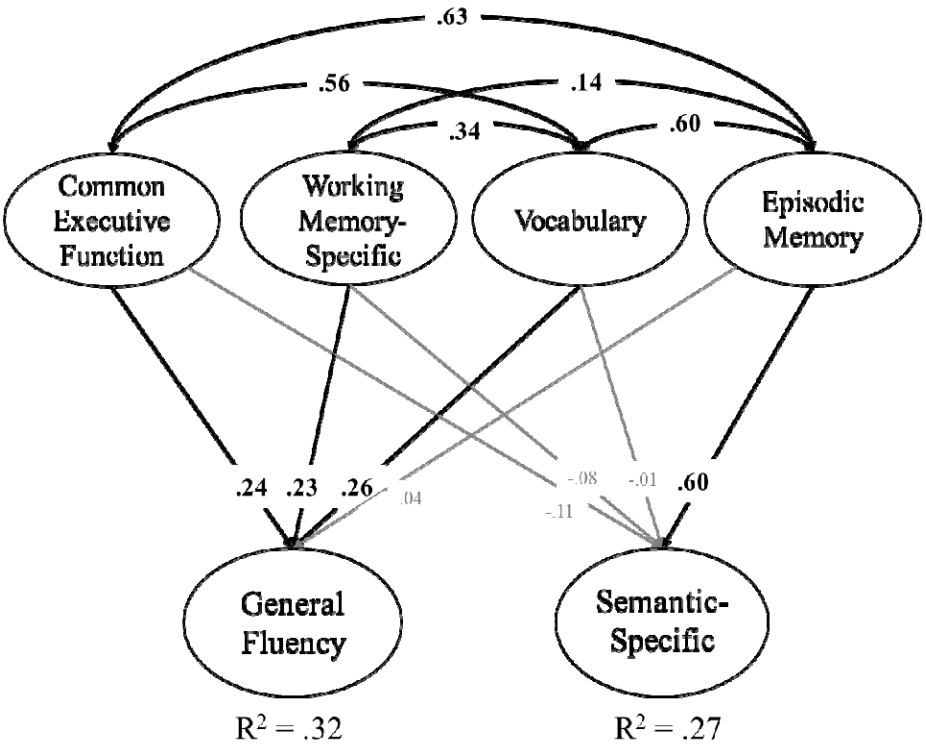

Figure 2: Structural equation model where both fluency factors are regressed on latent variables for executive function (Common Executive Function, Working Memory-Specific), vocabulary, and episodic memory. Not pictured are factor loadings on latent factors (which are similar to our previous work and those displayed in Figure 3). Significant paths and correlations are displayed in bold, with black text and lines ($p < .05$).

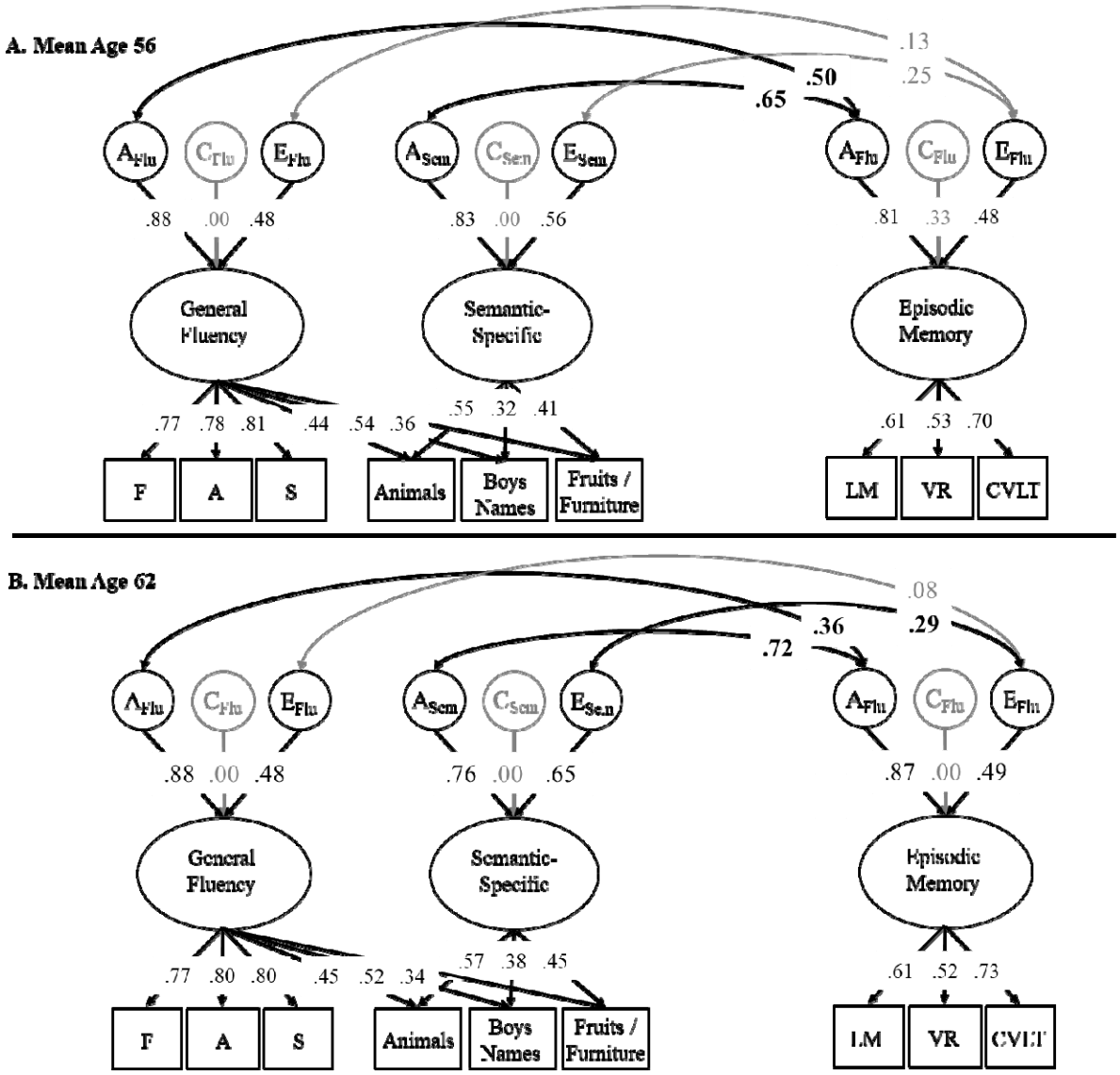

Figure 3: Full correlational models of the genetic (A), shared environmental (C), and nonshared environmental (E) influences on the general fluency, semantic-specific, and episodic memory latent variables at mean age 56 (top) and mean age 62 (bottom). Ellipses indicate latent variables and rectangles indicate measured variables. Significant factor loadings are displayed in bold, with black text and lines ($p < .05$). Variation explained by latent factors can be computed by squaring the factor loadings.