2006

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Genetic Influences on Free and Cued Recall in Long-Term Memory Tasks

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Long-term memory (LTM) problems are associated with many psychiatric and neurological illnesses and are commonly measured using free and cued recall tasks. Although LTM has been linked with biologic mechanisms, the etiology of distinct LTM tasks is unknown. We studied LTM in 95 healthy female twin pairs identified through birth records in the state of Missouri. Performance on tasks of free recall of unrelated words, free and cued recall of categorized words, and the vocabulary section of the Wechsler Adult Intelligence Scale (WAIS-R) were examined using structural equation modeling. Additive genetic and unique environmental factors influenced LTM and intelligence. Free recall of unrelated and categorized words, and cued recall of categorized words, were moderately heritable (55%, 38%, and 37%). WAIS-R vocabulary score was highly heritable (77%). Controlling for verbal intelligence in multivariate analyses of recall, two components of genetic influence on LTM were found; one for all three recall scores and one for free and cued categorized word recall. Recall of unrelated and categorized words is influenced by different genetic and environmental factors indicating heterogeneity in LTM. Verbal intelligence is etiologically different from LTM indicating that these two abilities utilize different brain functions.

Disturbances of memory are a feature or consequence of a variety of psychiatric and neurological illnesses such as Alzheimer's disease, schizophrenia, alcohol abuse, depression, and head injury. Despite a great deal of research addressing the role or impact of memory loss on psychopathology, many basic issues in the establishment and maintenance of memories are unresolved.

Long-term, or episodic, memory allows for the recording and storage of experiences over time (Roediger et al., 2002). Long-term memory (LTM) is thought to arise from increased and lasting changes in synapse plasticity, with several studies in animals implicating the role of the cAMP chain (Goda, 1995). Additionally, the proteins brain-derived neurotrophic factor (BDNF) and tissue plasminogen activator (tPA) have been associated with long-term potentiation in hippocampal cells, a mechanism thought to mimic the process of LTM (Pang et al., 2004). Taken together, these biologic and cellular level processes suggest a potential role of genetics in protein regulation and thus, LTM performance. Supporting this concept, examination of LTM in subjects at high genetic risk for Alzheimer's disease indicated that carriers of the APOE e4 allele are at increased risk for LTM decline before being classified as cognitively impaired (Caselli et al., 2004).

Twin studies have consistently demonstrated a low to moderate influence of genetic effects on general measures of memory. Heritability estimates range from 17% to 68%, with the task(s) used to assess memory performance and subject characteristics (age, source population) hypothesized to effect reported values (Alarcon et al., 1998; Finkel et al., 1995a, 1995b; Reynolds et al., 2005; Thapar et al., 1994). Few studies have specifically examined the influence of genetics on LTM. In a sample of older adult twins, the California Verbal Learning Test (CVLT) was subjected to principal components analysis. Free and cued word recall scores were associated with a combined verbal learning and LTM factor and found to be moderately heritable (56%; Swan et al., 1999). However, results from a study of twins discordant for schizophrenia suggests that LTM may also be influenced by nongenetic factors (Cannon et al., 2000).

Separate free and cued recall tasks are often used to measure LTM ability and additional work indicates that these tasks may measure different abilities. Results from dual task studies suggest that overall recall on free recall tasks is generally not affected by a simultaneous task while performance on cued recall tasks decreases substantially (Craik et al., 1996; Fernandes & Moscovitch, 2000; Naveh-Benjamin et al., 1998; Naveh-Benjamin & Guez, 2000). Only one study, which used a different concurrent, random

Received 8 February, 2006; accepted 14 July, 2006.

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stimulus task, indicated a similar decline in free recall performance (Rohrer & Pashler, 2003). Despite these decreases in cued recall performance, free recall is thought to be the more difficult and resource demanding of the two (Craik & McDowd, 1987). As demonstrated by Park et al. (1996), a greater amount of variance in free recall score was explained by speed and working memory than for the cued recall score. Conceptual repetition (following the target or cue with an associated idea) has been found to affect free recall with no effect on cued recall (McDermott & Roediger, 1996).

To our knowledge, no study has examined etiologic influences on free and cued recall tasks separately. Here we assess genetic and environmental influences on free and cued recall in LTM using data from a population-based twin sample.

Materials and Methods

Subjects

Female–female twin pairs who met study criteria were randomly selected from the Missouri Adolescent Female Twin Study (MOAFTS). MOAFTS is a large prospective twin study designed to examine factors contributing to risk for alcoholism in young women and adolescents (Heath et al., 1994). Participants were identified through a computerized database recording all live-birth twin pairs born in Missouri between 1968 and 1997. A semistructured diagnostic interview was given over the telephone to all participants (Bucholz et al., 1994). Pairs were considered for inclusion in the present study if they did not live together, lived in the United States, had no history of seizure disorder, psychosis, autism or mental retardation, were not in special education classes, had no known memory problems, were not currently participating in another study, and were not currently taking medication.

A total number of 161 female-female twin pairs were contacted for enrollment in this study. Of the 161 contacted 52 pairs were ineligible for this study. Of these 52 pairs, 40 were participating in another study, eight were living together at time of interview, two were living overseas, and two could not be contacted. One hundred and nine pairs were further screened for enrollment in the study. Five pairs were excluded based on current medication use. Of the remaining 104 pairs, zygosity could not be reliably determined for seven pairs; therefore, memory testing was completed on 97 twin pairs. On the day of testing, all participants were asked about alcohol and illicit drug use that day; all denied current drug use or intoxication. Two twin pairs were subsequently excluded for cheating (one was heard writing down word lists and another was heard to be getting vocabulary word definitions from someone else in the room). This analysis utilizes data from 95 twin pairs (88 monozygotic [MZ] and 102 dizygotic [DZ] individuals) aged 18 to 24 years (mean 21.37 ± 1.44 years).

Measures of Long-Term Memory

Subjects enrolled in the study were contacted by telephone and participated in a three part series of tasks; the first two of which assessed LTM abilities, and the third assessed verbal intelligence. Instructions for each task were administered immediately prior to the task. For the free recall and cued recall tests, subjects were instructed to attempt to recall as many words as possible (in any order they wished) but not to guess; that is, they were asked to be certain that all words they recalled had indeed been read to them. Word lists were pretested on undergraduate students prior to beginning the study to ensure the absence of floor or ceiling effects in recall. These lists were then recorded as computer sound files equalized for volume in a Midwestern female voice and used throughout the study.

In the first task, subjects heard a recording of four sets of 15 unrelated words at the rate of one word every 2 seconds. Words within the lists were common words (mean frequency 114.5 words per million, mean length of 5.15 letters, ranging from one to three syllables; Kucera & Francis, 1967). After each set of 15 words, subjects recalled as many words from the list as possible in 1 minute. We will refer to this task as the free recall of unrelated words.

The second task consisted of two 30 word lists, each comprised of five words from six semantic categories taken from the Battig and Montague norms (Battig & Montague, 1969). Words were 5.7 letters in length on average, had a mean frequency of 32 words per million, and a mean of 1.8 syllables in length. Again, words were presented at the rate of one word every 2 seconds. Following presentation of the first 30-word list, subjects were first asked to recall as many of the words as possible in 2 minutes (free recall of categorized words). Subsequently, a single category name was provided to cue recall of grouped words and subjects were asked to recall as many words in that category as possible in 15 seconds (cued recall of categorized words). This process was repeated for each of the six categories, and then the entire process was repeated for the second 30-word list.

Finally for the third task, subjects were given a 5-minute vocabulary test using 19 words from the verbal performance section of the Wechsler Adult Intelligence Scale (WAIS-R). The subject was given increasingly difficult words until three words in a row were missed. The number of correct words was then transformed into the standardized WAIS vocabulary score for subject age. Separate vocabulary words were used for each twin.

Statistical Analysis

The total number of correctly recalled words was calculated for each task in order to minimize the total

amount of variance for each task. Free and cued recall scores were summed separately in the second task. Four summary scores — free recall of unrelated words, free recall of categorized words, cued recall of categorized words, and the standardized WAIS vocabulary score — were examined. Biometric models were used to examine genetic and environmental influence on the four scores described above. The per cent of total variance contributed by additive genetic (A), shared environmental (C), and unique environmental (E) factors was calculated using structural equation modeling. We assessed model goodness-of-fit using the likelihood function, which can be compared using a chi-square change statistic each time a parameter is added to or dropped from the base ACE model. Significant improvement from the base model can then be assessed by comparing the calculated chi-square statistic to the critical value for the appropriate number of degrees of freedom difference between the two models. We also compared Akaike information criteria (AIC) between models to determine the most parsimonious model which fit the data. The AIC is calculated from the –2 log-likelihood values plus 2 times the number of parameters in the model (Akaike, 1974). The model with the lowest AIC value can thus be determined to be the most parsimonious model. Age was added to the model as a covariate for all four scores. WAIS vocabulary score was included as a covariate in the model for each of the three word-recall scores.

Bivariate and multivariate analyses assessed genetic and environmental influences using a Cholesky decomposition model for the three word-recall summary scores and WAIS verbal performance score. Significant change in model fit from the base ACE Cholesky decomposition model was determined as described above. All analyses were carried out using Mx, a structural equation modeling program developed for the analysis of twin data (Neale et al., 2002).

Results

Description of Sample, Summary of Performance on Recall Tasks

This sample contains data from 95 female twin pairs (88 MZ and 102 DZ individuals) aged 18 to 24 years (mean 21.37 ± 1.44 years) attained from the MOAFTS. Subjects had a mean normalized WAIS score of 8.10 (± 2.27). The average number of words correct for each list of the two LTM tasks is reported in Table 1. Similar trial means with few significant differences between trials were found, supporting aggregation of correct words across trials.

Twin correlations for WAIS score and each memory task are presented in Table 2. For all measures, the correlation between MZ twins is greater than that for DZ twins. However, the DZ correlation is greater than half the MZ correlation for WAIS score and free recall of unrelated words suggesting that both genes and the shared family environment both explain familial resemblance. In contrast, these correlations, though moderate, suggest that resemblance in the recall of free and cued unrelated words may be influenced largely by genetic effects.

Table 1

Descriptive Characteristics of Sample and Long-Term Memory Scales

	Mean ± *SD*	Significance Test vs. List 1 for Each Section
Age	21.4 ± 1.44	
WAIS Verbal Score	8.2 ± 2.24	
Uncategorized Words		
Free Recall Summary Score	32.9 ± 9.13	
List 1	8.3 ± 2.41	
List 2	8.3 ± 2.65	$p = .67$
List 3	7.9 ± 2.91	$p = .0039$
List 4	8.2 ± 3.00	$p = .20$
Categorized Words		
Free Recall Summary Score	60.8 ± 16.6	
List 1	19.9 ± 6.27	
List 2	21.0 ± 5.91	$p = .0013$
List 3	19.5 ± 6.3	$p = .39$
Cued Recall Summary Score	64.8 ± 12.7	
List 1	21.0 ± 5.91	
List 2	22.3 ± 4.62	$p < .0000$
List 3	21.2 ± 4.81	$p = .46$

Note: * All significance tests conducted at the alpha = .05 level

Table 2

Phenotypic Correlations by Zygosity

	MZ	DZ
WAIS Score	.66	.54
Uncategorized Free Recall	.56	.34
Categorized Free Recall	.46	–.04
Categorized Cued Recall	.41	.10

Note: MZ = monozygotic twin, DZ= dizygotic twin

Genetic Analysis of Long-Term Memory

Univariate Models

As expected, model fitting demonstrated a substantial genetic influence on the WAIS vocabulary score. Dropping A from the model resulted in a significant deterioration of fit while dropping C did not significantly alter model fit. Thus, an AE model was chosen as the best fitting model (Table 3). Adding age or sibling interaction as a covariate to the model did not significantly improve model fit. Additive genetic factors contributed 78% and the unique environment contributed 22% of the variance in vocabulary score.

The best model for the uncategorized word free recall was an AE model without sibling interaction or covariates (age and WAIS vocabulary score). Dropping C from the base model did not significantly affect

Table 3

Univariate Models for WAIS and Long-Term Memory Test Scores

	Model	A^2	C^2	E^2	–2LL	*df*	AIC
WAIS Score	ACE	.65	.13	.23	801.97	186	807.97
	AE	.78	—	.22	802.10	187	806.10
	CE	—	.55	.45	810.08	187	814.08
Uncategorized Free Recall	ACE	.41	.13	.46	1356.40	186	1362.40
	AE	.55	—	.45	1356.63	187	1360.63
	CE	—	.43	.56	1358.18	187	1362.18
Categorized Free Recall*	ACE	.38	$.93x10^{-5}$	.62	1592.80	185	1598.80
	AE	.38	—	.62	1592.80	186	1596.80
	CE	—	.23	.77	1596.34	186	1600.34
Categorized Cued Recall*	ACE	.37	$0.66x10^{-5}$	.63	1485.32	185	1491.32
	AE	.37	—	.63	1485.24	186	1489.24
	CE	—	.25	.75	1487.69	186	1491.69

Note: *WAIS Score is included as a covariate for categorized free and cued recall models. Bolded model indicates best fit. All comparisons were made to the saturated (ACE) model. A = additive genetic, C = shared environment, E = unique environment, AIC = Akaike information criteria, *df* = degrees of freedom.

model fit while dropping A slightly decreased the AIC. As demonstrated in Table 3, additive genetic factors accounted for 55% and unique environmental factors accounted for 45% of the variance in total number of correctly recalled random words.

Model fitting results showed the AE model with WAIS vocabulary score included as a covariate to be the best fitting model for both free and cued recall of categorized word lists. Dropping C from the model did not significantly alter model fit from the base ACE model as the C term was very small. Dropping A from the model did not significantly change model fit, although the AIC indicated an AE model as the most parsimonious in each case. Additive genetic factors accounted for 38% of the variance in free recall and 37% of the variance in cued recall of categorized words (Table 3).

Bivariate Models

As the WAIS score was found to be an important covariate for free and cued recall of categorized words, bivariate analyses were conducted using Cholesky decomposition models. For all analyses, an ACE Cholesky decomposition was used as the base model. Additive genetic and unique environmental factors were found for the WAIS score and both free recall of uncategorized words and free and cued recall of categorized words. As the shared environment was not found to contribute significantly to the variance in the WAIS score or the recall score in the univariate analyses above, the C terms were dropped from each bivariate model resulting in a more parsimonious model (Table 4).

Reducing the models further suggested different underlying components accounting for the variation in the WAIS score and word recall. A model including specific genetic and specific unique environmental terms best fit the data when examining the WAIS score and uncategorized free recall (Table 4). These distinct genetic factors accounted for 77% and 55% of the variance in WAIS score and free recall of uncategorized words, respectively (Table 5).

In contrast, a model including specific genetic and overlapping unique environmental factors best explained the variance in the WAIS score and the free and cued recall of categorized words (Table 4). Specific genetic factors accounted for 74%, 31% and 35% of the variance in WAIS score, cued categorized word recall, and free categorized word recall, respectively (Table 5). A unique environmental factor contributed to the covariation of these tasks (27% WAIS, 7% cued recall, 3% free recall) while a second unique environmental factor specific to cued categorized word recall (62%) or free categorized word recall (62%) explained more variance in these tasks.

A bivariate model was then constructed to examine overlapping influences on the free and cued recall of categorized words. An ACE Cholesky decomposition model was first fit to the data. Shared environmental factors were then dropped from the model resulting in a more parsimonious model. Further reduction of the model indicated that a single additive genetic factor accounted for 37% of the variance in both free and cued recall. The first unique environmental influence accounted for 62% of the variance in free recall and 44% of the variance in cued recall while the second unique environmental factor contributed 19% of the variance to cued recall score (Table 5).

In order to examine overlapping influences on the three memory tasks, a multivariate Cholesky decomposition model evaluated the number of correctly recalled words from task 1 (free recall of uncategorized words) and task 2 (free and cued recall of categorized words). A model including additive

Table 4

Bivariate and Multivariate Models for WAIS and Long-Term Memory Test Scores

	Working Model	Comparison Model	–2LL	*df*	Number Parameters	Chi-square change	*df* change	AIC
WAIS and Uncategorized Free Recall	ACE	—	2155.79	369	11	—	—	2177.79
	AE	ACE	2157.30	372	8	1.51	3	2173.30
	CE	ACE	2162.51	372	8	6.72	3	2178.51
	Specific A, E	AE	2157.53	373	7	0.23	1	2171.53
	A, Specific E	AE	2157.79	373	7	0.49	1	2171.79
	Specific A, Specific E	AE	2158.73	374	6	1.43	1	2170.73
WAIS and Categorized Free Recall	ACE	—	2394.20	369	11	—	—	2416.20
	AE	ACE	2394.87	372	8	0.67	3	2410.87
	CE	ACE	2405.20	372	8	11.0	3	2421.20
	Specific A, E	AE	2395.54	373	7	0.67	1	2409.54
	A, Specific E	AE	2396.19	373	7	1.32	1	2410.19
	Specific A, Specific E	A, Specific E	2398.98	374	6	2.79	1	2410.98
WAIS and Categorized Cued Recall	ACE	—	2289.87	369	11	—	—	2311.87
	AE	ACE	2287.39	372	8	2.48	3	2303.39
	CE	ACE	2297.19	372	8	7.37	3	2313.19
	Specific A, E	AE	2288.21	373	7	0.82	1	2302.21
	A, Specific E	AE	2292.52	373	7	5.13*	1	2306.52
Categorized Free and Cued Recall	ACE	—	2789.99	369	11	—	—	2811.99
	AE	ACE	2791.23	372	8	1.24	3	2807.23
	CE	ACE	2793.63	372	8	3.64	3	2809.63
	Shared A, E	AE	2791.41	373	7	0.18	1	2805.41
Uncategorized Free Recall,	ACE	—	4029.43	549	21	—	—	4071.43
Categorized Free and Cued Recall	**AE**	ACE	4029.32	555	15	0.11	6	4059.32
	2A,E	AE	4031.61	556	14	2.29	1	4059.61
Categorized Free Recall,	ACE	—	3573.74	549	21	—	—	3615.74
Categorized Cued Recall, WAIS	AE	ACE	3575.32	555	15	1.58	6	3605.32
	A, 1 shared E, 2 specific E	AE	3576.21	557	13	0.89	2	3602.21
	2A, 1 shared E, 2 specific E	A, 1 shared E, 2 specific E	3582.84	599	11	6.624*	2	3604.84

Note: Bolded model indicates best fit. A = additive genetic, C = shared environment, E = unique environment, *df* = degrees of freedom, AIC = Akaike information criteria.

*statistically significant at $p = .05$.

genetic and unique environmental factors best explained variance in these three tasks (Table 4). The first additive genetic factor contributed 56% of the variance in the free recall of uncategorized words, 28% of the variance in the free recall of categorized words and 29% of the variance in the cued recall of categorized words (Table 5).

Approximately 6% of the variance in the free and cued recall of categorized words was explained by a second additive genetic factor, while the third accounted for 1% of the variance in the cued recall of categorized words alone. Examination of unique environmental factors indicated that a factor influencing all three scores accounted predominantly for variance in uncategorized free recall score (45%), while a second unique environmental factor contributed similar amounts of variance to categorized free and cued recall scores (48% and 31% respectively). A third unique environmental factor also accounted for 18% of the variance in the categorized cued recall score.

Since we found that the WAIS score was an important covariate of free and cued categorized word recall in the univariate analyses, we also examined the contribution of genetic and environmental factors on the free and cued recall of categorized words (part 2) and the WAIS score. The first additive genetic factor contributed 40% of the variance in the free and cued recall of categorized words and only 6% of the variance in the WAIS score. A second additive genetic factor primarily explained variance in the WAIS score (55%) and a small portion of that in cued recall of categorized words (2%). The third additive genetic factor was specific to the WAIS score

Table 5

Path Values for Bivariate and Multivariate Models

Model		A1	A2	A3	C1	C2	C3	E1	E2	E3
ACE	WAIS	.76	—	—	.43	—	—	.49	—	—
	Uncategorized Free Recall	.07	.54	—	.01	.48	—	.10	.68	
AE	WAIS	.88	—	—	—	—	—	.48	—	—
	Uncategorized Free Recall	.06	.74	—	—	—	—	.07	.67	—
CE	WAIS	—	—	—	.75	—	—	.66	—	—
	Uncategorized Free Recall	—	—	—	.01	.67	—	.17	.72	—
Specific A, E	WAIS	.88	—	—	—	—	—	.48	—	—
	Uncategorized Free Recall	—	.74	—	—	—	—	—	.10	.67
A, Specific E	WAIS	.88	—	—	—	—	—	.47	—	—
	Uncategorized Free Recall	.09	.74	—	—	—	—	—	.66	—
Specific A, Specific E	WAIS	.88	—	—	—	—	—	.47	—	—
	Uncategorized Free Recall	—	.74	—	—	—	—	—	.67	—
ACE	WAIS	.78	—	—	.40	—	—	.48	—	—
	Categorized Free Recall	.14	.60	—	.00	.00	—.10	.78	—	
AE	WAIS	.88	—	—	—	—	—	.47	—	—
	Categorized Free Recall	.12	.61	—	—	—	—	.09	.78	—
CE	WAIS	—	—	—	.74	—	—	.67	—	—
	Categorized Free Recall	—	—	—	.02	.47	—	.18	.86	
Specific A, E	WAIS	.88	—	—	—	—	—	.48	—	—
	Categorized Free Recall	—	.59	—	—	—	—	.10	.67	
A, Specific E	WAIS	.88	—	—	—	—	—	.47	—	—
	Categorized Free Recall	.09	.74	—	—	—	—	—	.66	—
Specific A, Specific E	WAIS	.88	—	—	—	—	—	.47	—	—
	Categorized Free Recall	—	.74	—	—	—	—	.00	.67	—
ACE	WAIS	.88	—	—	.22	—	—	.47	—	—
	Categorized Cued Recall	.21	.00	—	.49	.00	—	.12	.84	—
AE	WAIS	.88	—	—	—	—	—	.47	—	—
	Categorized Cued Recall	.24	.59	—	—	—	—	.10	.77	—
CE	WAIS	—	—	—	.74	—	—	.67	—	—
	Categorized Cued Recall	—	—	—	.12	.48	—	.22	.84	—
Specific A, E	WAIS	.86	—	—	—	—	—	.52	—	—
	Categorized Cued Recall	—	.56	—	—	—	—	.26	.79	—
A, Specific E	WAIS	.89	—	—	—	—	—	.46	—	—
	Categorized Cued Recall	.28	.58	—	—	—	—	—	.76	—
ACE	Categorized Free Recall	.61	—	—	.06	—	—	.79	—	—
	Cued Recall	.58	.00	—	.22	.00	—	.67	.41	—
AE	Categorized Free Recall	.61	—	—	—	—	—	.79	—	—
	Cued Recall	.61	.11	—	—	—	—	.67	.42	—
CE	Categorized Free Recall	—	—	—	.47	—	—.88	—	—	
	Cued Recall	—	—	—	.47	.15	—	.76	.41	—
Shared A, E	Categorized Free Recall	.61	—	—	—	—	.79	.00	—	
	Cued Recall	.61	—	—	—	—	.66	.44	—	
ACE	Uncategorized Free Recall	.65	—	—	.39	—	—	.67	—	—
	Categorized Free Recall	.52	.23	—	.17	.02	—	.42	.68	—
	Cued Recall	.56	.17	.00	.09	.14	00	.39	.56	.40
AE	Uncategorized Free Recall	.74	—	—	—	—	—	.67	—	—
	Categorized Free Recall	.53	.25	—	—	—	—	.42	.69	—
	Cued Recall	.54	.23	.11	—	—	—	.40	.56	.42
2A, E	Uncategorized Free Recall	.74	—	—	—	—	—	.67	—	—
	Categorized Free Recall	.53	.25	—	—	—	—	.43	.69	—
	Cued Recall	.54	.23	—	—	—	—	.39	.56	.44

Table 5 (CONTINUED)

Path Values for Bivariate and Multivariate Models

Model		A1	A2	A3	C1	C2	C3	E1	E2	E3
ACE	Categorized Free Recall	.62	—	—	.00	—	—	.78	—	—
	Cued Recall	.61	.04	—	.14	.07	—	.66	.41	—
	WAIS	.18	.77	.02	.34	.17	.02	.06	.05	.47
AE	Categorized Free Recall	.62	—	—	—	—	—	.78	—	—
	Cued Recall	.62	.13	—	—	—	—	.65	.41	—
	WAIS	.18	.72	.47	—	—	—	.05	.03	.47
A, 1 shared E, 2 specific E	Categorized Free Recall	.63	—	—	—	—	—	.78	—	—
	Cued Recall	.63	.13	—	—	—	—	.63	.41	—
	WAIS	.24	.74	.42	—	—	—	—	—	.46
2A, 1 shared E, 2 specific E	Categorized Free Recall	.12	—	—	—	—	—	.99	—	—
	Cued Recall	.09	.62	—	—	—	—	.72	.31	—
	WAIS	.69	.59	—	—	—	—	—	—	.41

Note: Bolded model indicates best fit. A = additive genetic, C = shared environment, E = unique environment.

(17%; Table 5). Unique environmental factors were found to influence both free recall and cued recall of categorized words. Variance in the WAIS vocabulary score was explained by a separate, specific unique environmental factor (22%).

Discussion

Results from univariate structural equation models indicate that the WAIS verbal score is highly influenced by genetic factors. However, the free recall of uncategorized words and both the free and cued recall of categorized words were only moderately heritable. The WAIS verbal score, used here as an indicator of IQ, was found to be a covariate for both the free and cued recall of grouped words, but not the uncategorized word lists in part 1.

Bivariate model results support these findings and further demonstrate that while free and cued recall are largely influenced by the same factors, there appear to be separate influences that also independently influence cued recall. Specific additive genetic and unique environmental factors influenced the WAIS score and free recall for both LTM tasks while the WAIS score and cued categorized word recall were seen to have the same unique environmental influence. This shared factor demonstrates a marked difference between free and cued recall processes and related constructs, such as intelligence, which may influence performance on tests of memory. This distinction is present regardless of the type of words used in the free recall task.

However, the free and cued recall of categorized words were influenced by similar additive genetic and unique environmental factors, which indicates that the free and cued recall of categorized words tap into similar processes. The unique environmental factor accounting for variance in the free and cued recall score is likely the category name cue itself. The additional unique environmental factor related to cued recall may come from the variance shared with IQ. The free recall of uncategorized words, however, may measure a different aspect of LTM. Multivariate model results indicate that the factors accountable for much of the variance in the free recall of uncategorized words are less important in the recall of categorized words. Additionally, the free recall of categorized words is influenced by genetic and environmental variance in a similar manner as the cued recall of these same lists rather than the free recall of uncategorized lists. Again, the unique environmental component of categorized word cued recall may include a contribution from verbal IQ. This influence of verbal IQ may come from response to classroom education, cognitive ability, or differential social treatment, all of which could affect cue-related word recall performance.

These results partially agree with those indicating that free and cued recall are distinct processes (Tulving & Pearlstone, 1966; Tulving & Psotka, 1971). The larger estimated genetic effect on the free recall of uncategorized words suggests that there may be an etiologic difference in LTM. However, we also find that these recall processes are influenced by the same unique environmental factors. Additionally, our estimates of heritability for IQ (as measured by WAIS verbal score) and LTM components are consistent with those from previous twin studies (Alarcon et al., 1998; Finkel et al., 1995a, 1995b; Reynolds et al., 2005; Thapar et al., 1994). The present analyses extend this work by suggesting that etiologic differences in recall come from two sources. First, while shared additive genetic and unique environmental variance is present, these factors differentially affect the recall of categorized and uncategorized words. Thus, ability to recall different types of words may involve different types of brain function. Second, our results indicate shared variance between IQ and categorized word recall. In the bivariate models, the shared variance between IQ and free and cued

categorized word recall is attributed to a unique environmental factor indicating the potential for an outside event or stimulus to influence these traits jointly. However, in the multivariate model we find a small amount of additive genetic variance in the WAIS score explained by factors also related to categorized word recall. Thus, processes measured by these tasks may not be etiologically distinct. Application of these results to neuroimaging studies may provide valuable insight into the etiology of recall processes and enable better understanding of LTM function in individuals with disorders of memory.

This study is not without limitations. The analysis was conducted on a sample of young adult females and may not be generalizable to males or subjects of younger or older age. However, estimates of genetic effects here fall into the range reported earlier in older samples of twins of both genders (Swan et al., 1999). We may not have had sufficient power in our sample to differentiate between more complex models. However, this work is conducted on healthy individuals from the general population and may serve as an introduction to future work on the study of the genetics of specific memory tasks and processes. Additionally, the WAIS verbal score was used here as a proxy measure for a full IQ assessment. A more accurate determination of IQ may have provided different results. Finally, although the twins were screened for major neurological, psychiatric, and learning disorders we do not have specific measures of psychopathology or neuroimaging data which may have provided additional insight into the unshared and environmental causes of variation indicated in this study. Future work integrating neurologic, psychiatric, and cognitive assessments will aid in answering remaining questions regarding shared etiologic influences.

These results provide a valuable insight into the etiology of disorders of memory. If different genetic influences underlie different parts of LTM, then it is possible that different genes are also are implicated in different memory-based disorders. The genetic variation underlying different forms of memory likely differentially impacts psychiatric and neurological disorders characterized by memory problems. This information will help focus molecular genetic studies for known heritable conditions, such as Alzheimer's disease, where specific types of memory are affected. Additionally, this study underlines the need to use genetically specific memory tasks when studying familial disorders with memory problems. Memory tasks which are influenced by genetic factors may be more suited for diseases with a known genetic component, like schizophrenia. Likewise, the effect of head trauma on memory may be better assessed using a memory test with a strong environmental influence. In this manner, memory ability can be better assessed leading to an increased understanding of the etiology of debilitating illnesses associated with memory impairment.

Acknowledgments

This work is supported by 1F31MH074272 (HEV) and the Blanche F. Ittleson endowment fund. We thank Endel Tulving, PhD for assistance in study design, John Engel for data collection, and Andrew Heath, DPhil for access to twins from the Missouri Adolescent Female Twin Study.

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