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Anomia as a Marker of Distinct Semantic Memory Impairments in Alzheimer's Disease and Semantic Dementia

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Objective: Many neurologically constrained models of semantic memory have been informed by two primary temporal lobe pathologies: Alzheimer's disease (AD) and Semantic Dementia (SD). However, controversy persists regarding the nature of the semantic impairment associated with these patient populations. Some argue that AD presents as a disconnection syndrome in which linguistic impairment reflects difficulties in lexical or perceptual means of semantic access. In contrast, there is a wider consensus that SD reflects loss of core knowledge that underlies word and object meaning. Object naming provides a window into the integrity of semantic knowledge in these two populations. ***Method:*** We examined naming accuracy, errors and the correlation of naming ability with neuropsychological measures (semantic ability, executive functioning, and working memory) in a large sample of patients with AD ($n = 36$) and SD ($n = 21$). ***Results:*** Naming ability and naming errors differed between groups, as did neuropsychological predictors of naming ability. Despite a similar extent of baseline cognitive impairment, SD patients were more anomic than AD patients. ***Conclusions:*** These results add to a growing body of literature supporting a dual impairment to semantic content and active semantic processing in AD, and confirm the fundamental deficit in semantic content in SD. We interpret these findings as supporting of a model of semantic memory premised upon dynamic interactivity between the process and content of conceptual knowledge.

Keywords: Alzheimer's disease, semantic dementia, semantic memory, naming, error analysis

Object naming relies on the integrity of a series of discrete yet highly interactive cognitive processes working in concert to access meaning, assign a corresponding symbolic word form, and encode an appropriate lexical-phonological output sequence. Naming impairment (i.e., anomia) can potentially result from a number of etiologies affecting input, output, or core conceptual knowledge that supports word meaning. From the standpoint of cognitive neuropsychology, two primary temporal lobe pathologies have proven particularly informative toward elucidating the structure of semantic memory: Alzheimer's disease (AD) and Semantic Dementia (SD). Although the nature of the semantic impairment in AD and SD remains controversial, there exist few direct comparative investigations (for exceptions see Grossman et al., 2004; Rogers & Friedman, 2008). Our aim in the current work was to address this shortfall by directly contrasting naming accuracy and errors in AD and SD.

Process and Content in Semantic Memory

We have proposed a model of semantic memory that incorporates many of the merits of amodal and modality-specific theoretical approaches (Koenig & Grossman, 2007; Koenig, Smith, & Grossman, 2010; Peelle, Troiani, & Grossman, 2009; Reilly & Peelle, 2008; Reilly, Rodriguez, Peelle, & Grossman, in press). This framework is premised upon the idea that semantic memory is a dynamic system that relies on communication between heteromodal and modality-specific brain regions. Crucial to the proposed model is our hypothesis that object concepts are maintained in the brain in an abstract propositional format that we have previously described in terms of sparse representation (Reilly & Peelle, 2008; Reilly et al., in press). One might envision the concept of sparse representation in semantic memory as analogous to the filtering process that occurs during episodic memory consolidation (Moscovitch, Nadel, Winocur, Gilboa, & Rosenbaum, 2006; O'Kane, Kensinger, & Corkin, 2004; Squire, 2006; Squire & Bayley, 2007). That is, a durable representation in long-term memory is stripped of many nondiagnostic episodic features as a means of parsimony. Depending on task demands, however, it may be necessary to enrich sparse object representations by indexing cortical regions specialized for sensorimotor processing (Barsalou, 1999, 2008; Gallese & Lakoff, 2005). For example, consider two

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questions that require accessing qualitatively different types of semantic knowledge about skunks and groundhogs: (1) which is larger? and (2) which smells worse? A fully distributed theory of semantic memory holds that both tasks engage a comparable network of auto-associated features about skunks and groundhogs (Binder, Desai, Graves, & Conant, 2009; Gage & Hickok, 2005; Martin, 2007; Martin, Haxby, Lalonde, Wiggs, & Ungerleider, 1995; Wernicke, 1874). Our approach to semantic representation is somewhat different in that we view task demands as moderating patterns of activation (e.g., a relative size judgment engages regions dedicated to visual imagery, whereas a noxious odor judgment engages olfactory imagery). Importantly, in order to successfully negotiate both tasks one must first access core lexical knowledge about skunks and groundhogs. In this way, we hypothesize that abstract object representations are dissociable but also inextricably linked to their corresponding sensory features (for related accounts see Damasio, 1989; Damasio & Damasio, 1994; Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996; Patterson, Nestor, & Rogers, 2007). Moreover, the perceptual enactment processes that are necessary for enriching sparse representations demand executive resources, including attention and inhibitory control, components subsumed under the rubric of "process."

The Role of Process in Semantic Memory

Perhaps the most common association of semantic process relates to response selection and controlled retrieval among competing alternatives (Poldrack et al., 1999; Thompson-Schill, Aguirre, D'Esposito, & Farah, 1999; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997; Wagner, Pare-Blagoev, Clark, & Poldrack, 2001; Wagner, Poldrack et al., 1998). This association is well-justified in that lexical and conceptual retrieval are indeed critical aspects of semantic memory. However, the process component of semantic memory is not simply synonymous with retrieval, nor is it limited to conceptual response selection. A number of other active processes are critical for the representation of object knowledge, including object categorization, assimilation of incoming information with prior knowledge, perceptual imagery and enactment, differentiation of unique entities, short-term storage (i.e., working memory), and ad hoc allocation of other general cognitive or perceptual resources as dictated by task demands (e.g., attentional vigilance, filtering or suppressing noise) (Crosson et al., 1999; Farah & Feinberg, 2000; Grossman, Robinson, Bernhardt, & Koenig, 2001; Koenig et al., 2010; Martin, Gagnon, Schwartz, Dell, & Saffran, 1996; Martin & Gupta, 2004). Jefferies and colleagues (2008) have referred to this constellation of supporting process components collectively as semantic control.

Studies from cognitive and clinical neuroscience have implicated a range of prefrontal and posterior parietal cortical regions in controlled semantic processing (Poldrack et al., 1999; Thompson-Schill, 2003; Thompson-Schill et al., 1999; Wagner, Desmond, Glover, & Gabrieli, 1998). Moreover, an elegant body of recent work has advanced our understanding of structure of semantic memory as a multiple component system through contrastive performance of patients with putative semantic control deficits (e.g., transcortical sensory aphasia) relative to other patient populations believed to show deficient content (e.g., semantic dementia) (Corbett, Jefferies, Ehsan, & Lambon Ralph, 2009; Jefferies & Lambon Ralph, 2006; Jefferies, Patterson, & Lambon Ralph, 2007; Lambon Ralph, Cipolotti, Manes, & Patterson, 2010; Lambon Ralph & Howard, 2000; Lambon Ralph, McClelland, Patterson, Galton, & Hodges, 2001; Patterson, 2007; Patterson et al., 2007). This comparative case study approach has particular relevance to the unique nature of the semantic deficits underlying Alzheimer's disease and Semantic Dementia. We further describe the content-process framework in relation these conditions to follow.

Semantic Dementia as a Lesion Model for Loss of Content

Semantic dementia is a variant of frontotemporal dementia (FTD) associated with relatively circumscribed atrophy that affects anterolateral and ventral temporal cortex, including the inferior temporal gyrus (BA 20), anterior fusiform gyrus (BA 37), and temporal pole (BA 38), with relative preservation of other brain regions (Galton et al., 2001; Mummery et al., 2000; Snowden, Goulding, & Neary, 1989; Snowden, Neary, & Mann, 2002). Structural neuroimaging often shows asymmetric atrophy worse in the dominant hemisphere; however, perfusion studies have demonstrated hypometabolism in contralateral homologues, supporting a bilateral locus of neuroanatomical damage (Nestor, Fryer, & Hodges, 2006). The extent to which the temporal lobe pathology associated with SD extends mesially to structures such as the hippocampi, parahippocampal gyri, and the perirhinal cortex remains controversial (Bussey & Saksida, 2002; Moss, Rodd, Stamatakis, Bright, & Tyler, 2005).

Since Warrington's (1975) seminal case study, SD has presented a unique challenge and compelling lesion model for studying both the structure of semantic memory and the interaction of semantic deficits with intact cognitive domains (e.g., reading, repetition, visuospatial functioning). Three decades worth of compelling work from Patterson, Hodges, Lambon Ralph, Rogers, and their many colleagues (hereafter referred to as the Cambridge-Manchester groups) has established SD as a disorder characterized by a progressive loss or "dimming" of amodal conceptual knowledge (Hodges, Bozeat, Lambon Ralph, Patterson, & Spatt, 2000; Hodges & Patterson, 2007; Lambon Ralph et al., 2001; Lambon Ralph, Patterson, Garrard, & Hodges, 2003; Lambon Ralph, Sage, Jones, & Mayberry, 2010; Patterson, Graham, & Hodges, 1994; Patterson & Lambon Ralph, 1999). In particular, the Cambridge-Manchester groups have argued that the anterior temporal lobes act as a hub for processing similarity relations and indexing semantic knowledge (Lambon Ralph, Pobric, & Jefferies, 2008; Patterson et al., 2007; Rogers et al., 2006; Rogers et al., 2004; Rogers & McClelland, 2004). As such, the progressive and inexorable loss of these regions in SD should, in theory, produce profound multimodal semantic impairments that impact all representational formats (e.g., behavioral performance on pictures = words = definitions = object function = environmental sounds = writing = listening = reading) (but for discussions of modality advantages in SD associated with abstract word processing see Bonner et al., 2009; Breedin, Saffran, & Coslett, 1994; Reilly, Cross, Troiani, & Grossman, 2007).

With respect to naming tasks, patients with SD sometimes show benefits of phonological cueing early during the course of the disease, but such facilitative effects soon appear to wane (Jefferies,

Patterson, & Lambon Ralph, 2006).[1] Also during the early stages of SD, patients tend to make many semantic errors, including superordinate naming errors (e.g., dog → "animal") demonstrating preservation of top-down hierarchically organized semantic knowledge (Hodges, 2003; Hodges, Graham, & Patterson, 1995; Lambon Ralph, Graham, Ellis, & Hodges, 1998; Lambon Ralph et al., 2001). Broad domain level knowledge is, however, insufficient to make the unique distinction needed for object naming. As such, patients with SD tend to show strong typicality effects among their many coordinate naming errors (Lambon Ralph et al., 1998; Lambon Ralph et al., 2001). These coordinate errors are often characterized by the substitution of a prototype in place of a target (e.g., "horse" as a prototypical four-legged mammal used to name both "hippo" and "zebra"). In the latest stages of decline, when semantic knowledge has deteriorated to the point that no approximation can be attempted, the majority of naming errors in SD are omissions with the greatest proportion of omission errors occurring for low typicality items (Woollams, Cooper-Pye, Hodges, & Patterson, 2008).

We view the impairment of semantic memory in SD as twofold. The dominant impairment arises from damage to anterolateral temporal regions (e.g., MTG) responsible for heteromodal semantic representations. A secondary impairment arises from damage to modality-specific regions of visual association cortex, including the anterior fusiform gyrus and inferior temporal gyrus (Binney, Embleton, Jefferies, Parker, & Lambon Ralph, 2010). As such, we predict that damage to the visual object recognition pathway will produce deficits in visual perceptual enactment and more generally compromise visual semantic features of objects. However, SD patients will show some degree of spared knowledge for words and concepts unmitigated by visual salience (e.g., abstract words) (for interpretations of reverse concreteness effects in SD see Bonner et al., 2009; Breedin et al., 1994; Papagno, Capasso, & Miceli, 2009; Reilly et al., 2007; Yi, Moore, & Grossman, 2007).

Alzheimer's Disease as a Lesion Model for Loss of Both Process and Content

AD is classically associated with severe impairments in episodic memory that have been linked primarily to damage to medial temporal lobe (MTL) structures (including entorhinal cortex, hippocampal formation) and agnosia resulting from damage to temporoparietal association cortices (Braak & Braak, 1997; Lewis, Campbell, Terry, & Morrison, 1987; Nestor et al., 2006; Thompson et al., 2003). As AD progresses, patients also commonly experience a constellation of symptoms associated with frontoparietal dysfunction, including deficits in inhibitory control, working memory, sustained attention and visuospatial functioning (Grossman et al., 1996; Grossman & Rhee, 2001; McKhann et al., 1984; Thompson et al., 2003).

Perhaps due in part to intrasubject variability, the neuropathology of AD represents a controversial lesion model for semantic memory. Some have argued that AD represents a disconnection syndrome characterized by impaired lexical and/or visual access to intact semantic representations (Bayles & Kim, 2003; Bayles & Tomoeda, 1983; Ober & Shenaut, 1999). AD patients, for example, have been observed to show "access-like" features such as preserved semantic priming (Nakamura, Nakanishi, Hamanaka, Nakaaki, & Yoshida, 2000; Rogers & Friedman, 2008) and facilitative effects of cueing when experiencing word finding or serial recall difficulties (Balthazar, Cendes, & Damasceno, 2008; Kuslansky, Buschke, Katz, Sliwinski, & Lipton, 2002). These symptoms of a disconnection syndrome are also evident in neuroanatomical staging of AD, wherein patients experience damage to the perforant pathway, connecting the hippocampus to the entorhinal cortex, prior to frank damage of the respective structures themselves (see also Braak staging of the early trans-entorhinal stage of AD) (Braak & Braak, 1997; Hyman, Van Hoesen, Kromer, & Damasio, 1986).

Although there is some evidence to support process-based accounts of semantic deficits in AD, a stronger consensus holds that AD results in the degradation of core semantic representations (Chertkow & Bub, 1990; Devlin, Gonnerman, Andersen, & Seidenberg, 1998; Gonnerman, Andersen, Devlin, Kempler, & Seidenberg, 1997; Hornberger, Bell, Graham, & Rogers, 2009). Patterns of naming and error distributions have been particularly informative toward the evolution of this view. Early work demonstrated a strong correlation between "naming and knowing" in AD as revealed by the superior quality of concept definitions for items that patients were able to successfully name relative to anomic items (Hodges & Patterson, 1995; Lambon Ralph, Patterson, & Hodges, 1997). More recent experimental investigations have delineated the loss of feature knowledge in AD through techniques such as multidimensional scaling as a means for discerning the integrity of semantic category distinctions (e.g., land/water animal, bird/mammal) (Hornberger et al., 2009).

Based on a range of neurobehavioral characteristics, there is compelling support for the hypothesis that AD results in a dual impairment of process and content in semantic memory (Rogers & Friedman, 2008). Within the context of our proposed model, damage to the lateral temporal cortex in AD should impact multimodal conceptual knowledge (i.e., content), whereas the distribution of frontoparietal damage in AD should correspondingly impair a range of process components, including controlled retrieval, semantic categorization, and indexing of modality-specific cortical regions.

Anomia as Marker of Unique Semantic Impairments in AD and SD

As a cognitive task, naming provides a window into the nature of semantic impairments in AD and SD. Curiously, however, few studies have directly contrasted naming ability in a well characterized sample of AD and SD patients. We do so here with attention to naming errors and correlations with cognitive factors such as semantic categorization ability, working memory, and executive functioning. By examining the correlations between neuropsychological variables linked strongly to either the process component of semantic memory (e.g., executive functioning underlying controlled semantic retrieval) or the content component (e.g., verbal vs. nonverbal semantic association ability), we can derive empirical support for our hypotheses regarding the selective compromise of these abilities in AD and SD. The advantage of this

[1] A different yet highly influential perspective on function of the anterior temporal lobes *has been* proposed by Damasio, Tranel, and colleagues (Damasio, Tranel, Grabowski, Adolphs, & Damasio, 2004). Specifically, that the temporal poles play a critical role in lexical retrieval.

approach relative to previous studies of naming in AD and SD is that given large statistical power and the same stimulus set balanced across a range of semantic categories, we can empirically validate predictors of naming accuracy and examine the discriminant power of specific naming errors.

Method

Participants

Participants included 36 patients with AD diagnosed via a consensus review mechanism in accord with NINDS-ARDCA criteria (McKhann et al., 2001), and 21 patients with SD diagnosed through a consensus review in accord with a modification of published criteria (Neary et al., 1998). Exclusionary criteria were as follows: (1) non-native English speaker; (2) currently taking sedating or psychotropic medications; and (3) comorbid neurological conditions (e.g., stroke, tumor). Neuropsychological and demographic data obtained contemporary to the naming data reported to follow appear in Table 1.

Notably, the AD and SD groups showed comparable verbal and nonverbal semantic impairment as assessed by their scores on the Pyramids and Palm Trees Test (Howard & Patterson, 1992) [word version: $t(41) = 1.16$, $p = .25$; picture version $t(41) = .78$, $p = .44$]. Patients also showed comparable baseline global cognitive impairment as assessed by scores on the Mini Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) [$t(54) = .70$, $p = .49$]). Raw scores within these ranges achieved by the AD and SD patients on the *PPT* and the *MMSE* have previously been linked with mild-moderate semantic impairment and mild global cognitive decline respectively (Balthazar et al., 2008; Lambon Ralph, Patterson, Graham, Dawson, & Hodges, 2003). AD and SD patients did not significantly differ in terms of education [$p > .05$] but did differ by age, with the average AD patient being almost six years older than their SD counterpart [$t(55) = 2.46$, $p = .02$].[2]

We contrasted patient performance with that of 11 healthy community-dwelling adults from the Philadelphia region. The patient and control groups were matched on age, education, and sex. Exclusionary criteria for the controls were as follows: (1) non-native English speaker; (2) currently taking sedating or psychotropic medications; (3) comorbid neurological conditions (e.g., stroke, tumor); and (4) MMSE < 27.

All participants and their primary caregivers provided informed consent in accord with the protocol approved by the University of Pennsylvania institutional review board.

Materials

We administered a battery of neuropsychological tests with the goal of correlating specific semantic, executive, amnestic, and linguistic functions with naming ability. We administered the MMSE as a means for assessing global cognitive ability among patients and for screening the control participants. The Pyramids and Palm Trees test provided indices of verbal and nonverbal semantic functioning. For executive functioning we administered Stroop, letter fluency, and Symbol Trails tests. As an assessment of working memory, we obtained forward and backward digit span. For additional detail regarding these particular tasks, see Table 3.

With respect to the experimental materials, there is an extensive literature on category-specific naming effects in AD, with the most common impairment existing for natural kinds relative to manufactured artifacts (Gonnerman et al., 1997; Humphreys & Riddoch, 2003). In the analyses to follow, we attempted to avoid a specific category bias by sampling across a wide range of natural kinds and manufactured artifacts. To accomplish this, stimuli included a selection of 60 black-and-white line drawings from the Snodgrass and Vanderwart picture series (Snodgrass & Vanderwart, 1980), spanning nine basic level categories within natural kinds ($n = 22$ items) and artifacts ($n = 38$ items). The three natural kind basic level categories included: Fruits/Vegetables ($n = 9$), Mammals ($n = 7$), and Nonmammals ($n = 6$). Six artifacts subcategories included: Clothing/Accessories ($n = 5$), Household Items ($n = 8$), Kitchen Items ($n = 5$), Tools ($n = 7$), Toys ($n = 7$), and Vehicles ($n = 6$).

We matched the basic level subcategories ($p > .05$ all) for familiarity [mean familiarity = 525 on a 100–700 scale] and word frequency [mean frequency = 27.69 per-million words] using the MRC Psycholinguistic Database norms (Coltheart, 1981)]. Stimuli were also matched for rated visual complexity [mean rating = 3.07] across the semantic categories using the Snodgrass and Vanderwart (1980) norms. In a post hoc fashion, we also evaluated age-of-acquisition (AOA) across the semantic subcategories using a combination of published AOA norms from the Bristol database (Stadthagen-Gonzalez & Davis, 2006) and the MRC Psycholinguistic database (1981). For missing AoA values, we obtained in-house ratings of AOA ($n = 22$) from naïve raters by replicating the Bristol psycholinguistic database (Stadthagen-Gonzalez & Davis, 2006) rating instructions and scale. The average rated AOA across all items was 273 (on a 100–700 scale), and the nine semantic subcategories did not differ by AOA [one-way ANOVA, $F(8, 59) = 1.10$, $p = .38$].

Naming Accuracy

Procedure

We presented the 60 line drawings, each centered on an individual piece of 8" × 10" white paper. Stimuli appeared in a fixed pseudorandom order, prerandomized such that no single semantic category examplar appeared more than twice in succession. Our aim in pseudorandomizing the stimuli was to avoid the confounding possibility of a category blocking effect (i.e., many tools or animals appearing in succession). Participants were asked to name each item, and responses were recorded and scored offline. Participants received no feedback on accuracy of production. We treated failure to name an item within 60 sec as an omission/nonresponse. On trials in which participants made multiple naming attempts, the final response was accepted. For the accuracy analyses, we used a categorical scoring classification system (correct or incorrect) as evaluated by three independent judges. These judges were graduate research assistants blind to study aims and not represented among the authorship.

[2] This age discrepancy is not unexpected as a function of the distinct pathologies that underlie AD and FTD. The prevalence of frontotemporal dementia typically follows a roughly Gaussian distribution with a mean age of onset occurring early during the sixth decade of life, whereas the onset of Alzheimer's disease is often a full decade later with an increasing linear risk during advancing age (Grossman et al., 2007; Hodges, Davies, Xuereb, Kril, & Halliday, 2003; Hodges et al., 2004; Neary, Snowden, & Mann, 2005).

Table 1
Neuropsychological and Demographic Data

Group	Stat	Age	EDU	MMSE	BNT	Pyramids and palm trees		Digit span		FAS	STROOP
						Pics	Words	For	Back		
AD	Mean	72.3	13.8	22.5	9.7	44.3	42.6	4.3	3.18	6.1	.06
	*z	.35	−.20	−6.1	−3.9	−4.0	−7.4	n/a	n/a	−1.8	n/a
	SD	7.8	3.1	4.1	4.2	5.7	9.1	1.6	1.6	4.6	.92
SD	Mean	66.6	16.3	21.5	8.0	42.9	44.1	3.3	5.67	5.7	.19
	*z	−.37	.11	−7.0	−5.3	−4.8	−6.1	n/a	n/a	−1.9	n/a
	SD	8.4	2.9	5.8	4.2	5.2	5.0	1.4	1.57	3.2	.21
Ctrl	Mean	69.5	15.4	29.2	14.25	50.4	50.6	n/a	n/a	13.1	n/a
	SD	7.8	8.0	1.1	1.2	1.6	1.6	n/a	n/a	3.9	n/a

Note. Age in years; EDU = Education in Years; MMSE = Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975); BNT = Boston Naming Test 15-item Short Form (Control $M = 14.25$, $SD = 1.19$) (Kaplan, Goodglass, & Weintraub, 1983); P&p = Pyramids and Palm Trees Test (Howard & Patterson, 1992); STROOP score reflects the total number of correct responses minus incorrect responses divided by the total time to complete the task. * Z-score represents performance relative to the mean and standard deviation of an age-matched control group from the Philadelphia community ($N = 24$, mean age = 69.46).

Judges were instructed to score phonemic distortions or other phonological approximations of the target word as incorrect (see error analysis). Judges were also instructed to score near synonyms or derivations of the target word as correct. For example, for the target picture of a football helmet, we accepted either "helmet" or "football helmet" but not errors such as "football" or "thing that is used to protect your head." Likewise, for the target picture of a "spool of thread," we accepted spool, thread, or spool of thread. Examples of incorrect responses are explicitly described in the naming error analyses to follow.

Item level detail regarding order of presentation and accuracy are available for download at: http://phhp.ufl.edu/~jjreilly/data/.

Results

Naming accuracies across the nine semantic categories are listed in Table 2. Correlation matrices assessing the relationship between neuropsychological measures of executive functioning, immediate recall, semantic association ability, and naming performance are shown in Tables 3 and 4.

We used an ANOVA with a group (2: AD, SD) by semantic category (2: natural kinds, manufactured artifacts) design to assess naming accuracy. There was a significant main effect of group [$F(1, 55) = 4.15$, $p = .04$]. This was confirmed by post hoc testing demonstrating that the AD group named more accurately than the SD group [AD = 75.8%; SD = 61.8%; $t(55) = 1.99$, $p = .05$]. Participants also showed a significant group by basic level semantic category interaction in their naming performance [Greenhouse-Geisser Corrected $F(8, 440) = 3.91$, $p = .01$]. This heterogeneity is reflected in differential naming performance across the basic level category distinction. A planned contrast Bonferroni-corrected for nine multiple comparisons demonstrated that the only significant basic level category impairment was seen in SD patients relative to AD for naming household items [AD = 80.1%; SD = 52.9%; $t(55) = 3.54$, $p = .001$].

Within the AD group, patients were comparable in their accuracy of naming natural kinds (73% accuracy) versus manufactured artifacts (76% accuracy) [$t(35) = 1.39$, $p > .05$]. In contrast, SD

Table 2
Naming Accuracies by Semantic Category

	Natural kinds ($n = 22$)	Artifacts ($n = 38$)	Natural kinds			Manufactured artifacts					
			Foods ($n = 9$)	Mammals ($n = 7$)	Nonmammal ($n = 6$)	Clothes ($n = 5$)	House ($n = 8$)	Kitchen ($n = 5$)	Tools ($n = 7$)	Toys ($n = 7$)	Vehicles ($n = 6$)
AD											
Raw	16.13	29.03	6.25	5.42	4.47	4.16	6.47	3.83	4.42	5.03	5.11
% Correct	73	76	69	77	75	83	80	77	63	.72	.85
Z-score	−4.98	−4.93	−6.05	−2.66	−2.18	−2.32	−4.52	−3.37	−1.37	−4.20	−.73
SD											
Raw	14.90	22.05	5.90	5.10	3.90	3.38	4.24	2.76	3.48	4.00	4.19
% Correct	68	58	65	73	.65	.68	.53	.55	.50	.57	.70
Z-score	−6.23	−9.87	−6.87	−3.32	−3.28	−4.80	−11.60	−6.76	−2.19	−6.64	−2.48
Control											
Raw score	21.1	36	8.8	6.7	5.6	4.9	7.9	4.9	6.0	6.8	5.5
% Correct	.96	.95	.98	.96	.93	.98	.99	.98	.86	.97	.92

Table 3
Correlation Matrix of Naming, Demographic, and Neuropsychological Measures for Alzheimer's Disease

Measure	Stat	Naming accuracy	PP-words	PP-pics	STROOP	FAS	BNT	Dig-F	Dig-B	EDU	Age	MMSE
Naming accuracy	*r*	1										
	n	36										
PP-words	*r*	.30	1									
	n	28	28									
PP-pics	*r*	.53**	.45*	1								
	n	27	26	27								
STROOP	*r*	.48**	.43*	.55**	1							
	n	30	23	24	30							
FAS	*r*	.43*	.19	.46*	.56**	1						
	n	35	28	27	30	35						
BNT	*r*	.75**	.25	.66**	.56**	.47**	1					
	n	35	28	27	30	35	35					
Dig-F	*r*	.43*	−.03	.37	.25	.45**	.44**	1				
	n	35	28	27	30	35	35	35				
Dig-B	*r*	.39*	.24	.60**	.70	.65**	.56**	.53**	1			
	n	35	28	27	30	35	35	35	35			
EDU	*r*	−.03	−.08	.10	.05	.05	−.08	−.01	−.03	1		
	n	35	28	27	30	35	35	35	35	35		
Age	*r*	−.12	−.01	−.19	.18	.01	−.17	−.00	.30	−.06	1	
	n	36	28	27	30	35	35	35	35	35	36	
MMSE	*r*	.46**	.21	.44*	.35	.72**	.39*	.30	.45**	.10	.02	1
	n	35	28	27	30	35	35	35	35	35	35	35

Note. AGE = age in years; EDU = Education in Years; MMSE = Mini Mental State Examination score of 30 possible (Folstein, Folstein, & McHugh, 1975); BNT = Boston Naming Test 15-item Short Form (Control $M = 14.25$, $SD = 1.19$) (Kaplan, et al., 1983); PP = Pyramids and Palm Trees Test (Howard & Patterson, 1992); STROOP = Color-Word Reading Stroop Test result; FAS = Average number of responses for the letters F,A,S in one minute; Dig-F = Forward digit span; Dig-B = Backward digit span.
* Pearson correlation significant at $p < .05$. ** Pearson correlation significant at $p < .001$. Naming Accuracy = Based on proportion correctly named across the 60 Snodgrass and Vanderwart (1980) items.

patients showed a relatively small but significant category naming advantage for naming natural kinds (67% accuracy) relative to manufactured artifacts (58% accuracy) [paired $t(20) = 2.94$, $p = .008$].

SD patients showed a near perfect correlation between their performance on verbal and nonverbal semantic categorization tasks (Pyramids and Palm Trees Test word vs. picture versions, Pearson $r = .97$, $p < .001$), consistent with a uniformity in semantic impairment irrespective of the modality of presentation (see Tables 3 and 4). Moreover, for SD patients, semantic association ability was the single strongest predictor of naming accuracy, as revealed by the strong bivariate correlations between naming accuracy and scores on a standardized measure of semantic association [Pyramids and Palm Trees Test word version; $r = .73$; picture version; $r = .57$]. In contrast, measures linked to working memory and executive functioning (digit span and Stroop) were not significant predictors of naming ability in SD.

AD patients showed a weaker correlation between performance on verbal and nonverbal semantic categorization tasks (Pyramids and Palm Trees Test word vs. picture versions, $r = .45$, $p < .001$). Moreover, AD patients tended to show an advantage for the picture relative to the word version of the test, suggesting at least some degree of a modality-specific advantage when processing pictures relative to words, consistent with accounts of privileged access to the semantic system for pictures (Caramazza & Shelton, 1998). In addition, semantic association abilities were only modestly predictive of naming accuracy for AD patients. A number of other factors were significant predictors of naming ability in AD, including common measures of global cognitive functioning [MMSE: $r = .46$] and working memory [digits forward recall $r = .43$; digits backward recall $r = .39$]. Neuropsychological evidence for process impairment in AD is perhaps more convincingly derived from correlations between naming ability and an explicit measures of executive functioning, including Stroop performance [$r = .48$] and letter fluency [FAS, $r = .46$].

We conducted significance testing on the differences between two specific correlations in AD and SD by converting the original Pearson correlations to z-scores and accounting for the pooled sample size (Fischer's Z-score conversion). Using this comparison method, AD patients showed a significantly stronger correlation between naming and executive functioning (as gauged by the Stroop test) than did SD patients [$z = 2.05$, $p = .02$]. In contrast, SD patients showed a stronger correlation between naming and semantic association ability for words (as gauged by Pyramids and Palm Trees Word version) [$z = -1.71$, $p = .04$], but groups did not differ by their correlations of naming with semantic association ability for pictures (as gauged by Pyramids and Palm Trees Picture version [$z = -.14$, $p > .05$].

Interim Discussion of Naming Accuracy Results

Despite comparable degrees of semantic and global cognitive ability (assessed by neuropsychological measures), AD and SD patients differed in their severity of naming impairment. That is, SD patients tended to be more anomic than AD patients, as well as showing different predictors of naming accuracy. For AD patients,

Table 4
Correlation Matrix of Naming, Demographic, and Neuropsychological Measures for Semantic Dementia

Measure	Stat	Naming accuracy	PP-words	PP-pics	STROOP	FAS	BNT	Dig-F	Dig-B	EDU	Age	MMSE
Naming accuracy	*r*	1										
	n	21										
PP-words	*r*	.73**	1									
	n	15	15									
PP-pics	*r*	.57*	.97**	1								
	n	16	15	16								
STROOP	*r*	−.30	.05	−.08	1							
	n	11	9	9	11							
FAS	*r*	.50*	.57*	.46	−.38	1						
	n	19	15	16	11	19						
BNT	*r*	.82**	.50	.44	−.53	.65**	1					
	n	20	15	16	11	19	20					
Dig-F	*r*	−.17	.22	.01	−.08	.56*	.12	1				
	n	19	14	15	11	18	19	19				
Dig-B	*r*	−.06	.40	.17	−.21	.57*	.23	.82**	1			
	n	19	14	15	11	18	19	19	19			
EDU	*r*	−.37	−.04	.04	−.24	−.35	−.25	−.06	.06	1		
	n	21	15	16	11	19	20	19	19	21		
Age	*r*	.21	−.20	−.27	−.02	.16	.37	.18	.15	−.16	1	
	n	21	15	16	11	19	20	19	19	21	21	
MMSE	*r*	.42	.79**	.71**	−.33	.52*	.36	.32	.44	.10	−.07	1
	n	21	15	16	11	19	20	19	19	21	21	21

Note. For description of variables see note for Table 3.
* Pearson correlation significant at $p < .05$. ** Pearson correlation significant at $p < .001$.

a combination of working memory, executive functioning, and semantic association abilities predicted naming ability. Moreover, as a group the AD patients showed relative homogeneity in their naming ability as a function of semantic category. In contrast, the single strongest predictor of naming for SD patients was semantic association ability. For further insight into processing differences between AD and SD, we turn to naming error analyses.

Error Analysis I: Major Naming Errors

We contrasted distributions of naming errors in AD and SD patients via two separate error analyses. The first of these error analyses targeted coarse differences between visual, phonemic, and semantic levels of processing. The second analysis focused exclusively on semantic errors. For both analyses, we first isolated impaired patients by establishing a threshold for naming impairment using a z-score criterion relative to controls ($z < -1.96$, corresponding to an alpha level of .05, two-tailed). Using this criterion, 69% of AD patients ($n = 25$) and 86% of SD patients ($n = 18$) were considered anomic. We then analyzed anomic participants' naming errors. Two independent judges evaluated each error, and in the event of a disagreement a third judge broke the tie.

Method

Coding procedures. Raters classified responses as one of the following subtypes:

1. Visual: Naming a selected part of the target item (e.g., banana → "stem") or substituting a visually similar item from a different semantic category (e.g., asparagus → "pencil").
2. Phonemic: Distortions or phonemic approximations that share at least one syllable in common with the target (e.g., umbrella → "umbellug").
3. Unrelated: Real word responses visually dissimilar and semantically unrelated to the target item (e.g., cat → "apple").
4. Omission: Nonresponses and Empty Responses (e.g., 'I know . . . It's That Thing.').
5. Semantic: Errors Conceptually Related to the Target Item (e.g., dog → "animal").
6. Uninterpretable: Incomprehensible Responses.

Data analysis procedures. We employed a mixed-model ANOVA to evaluate the distribution of major errors. The within-subjects factor was proportion of each major error type (5 levels); the between-subjects factor was diagnosis (AD or SD). We eliminated uninterpretable responses (5.1% of total responses) prior to this analysis.

Results: Major Naming Errors

Table 5 summarizes the distribution of major naming errors. The ANOVA showed a significant main effect of major error type [$F(4, 164) = 36.06$, $p < .001$]. The interaction between major error type and patient group approached but did not attain statistical significance [$F(4, 164) = 2.29$, $p = .06$]. For both patient groups, the most common naming error was semantic, followed by omission. We recorded no phonemic errors from the AD group but observed a small proportion of phonemic errors in SD.

Interim Discussion: Major Naming Errors

Major error distributions were roughly similar between AD and SD patients, and therefore not particularly discriminative. In order

Table 5
Major Naming Errors

	Major error type									
	Visual		Semantic		Phonemic		Unrelated		Omission	
Semantic category	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
AD										
*Total errors	2.96	1.46	11.28	8.56	0	0	.44	1.04	5.04	6.45
Natural kinds	.92	.76	4.72	4.05	0	0	.12	.44	2.00	3.56
Artifacts	2.04	1.10	6.56	5.49	0	0	.32	.69	3.04	4.39
SD										
Total errors	2.11	1.37	9.50	5.80	.33	.84	.94	2.92	9.11	10.06
Natural kinds	.44	.71	2.72	2.45	.06	.24	.17	.27	3.44	3.55
Artifacts	1.67	.97	4.22	3.80	.28	.67	.78	2.42	5.67	7.13

Note. These values reflect raw total of each error type (of 60 items); natural kinds errors only ($n = 22$ possible); and manufactured artifacts only ($n = 38$ possible).
* Cell totals reflect the mean number of errors incurred within a patient group for a specific category.

to further elucidate semantic processing differences between these patient populations, we turn to a finer grained analysis of semantic naming errors.

Error Analysis II: Semantic Errors

We isolated semantic errors identified in the major naming error analysis described previously and conducted an additional coding of these error types.

Coding Procedure

Three independent judges coded semantic errors as belonging to one of the following subtypes:

1. Coordinate: Responses from the same superordinate semantic category and the same taxonomic level as the target (e.g., zebra → "horse").
2. Functional-Associative: Responses that state a function or action of the target item (e.g., piano → "you play music on it" or "you hit the keys").
3. Physical Attribute: Responses that describe a feature of the item that is not part of the line drawing (e.g., pumpkin → "orange").
4. Contextual: Responses that identify the context where the target item might be found or used (fish → "pond"; or rolling pin → "baker").
5. Subordinate: Responses that include a specific subordinate exemplar of the target (e.g., dog → "poodle") or a proper name (e.g., volcano → "Vesuvius").
6. Superordinate*:* Responses that state the general category to which the target belongs (e.g., dog → "animal").

Data Analysis Procedures

We employed a mixed-model ANOVA to evaluate the distribution of major errors. The within-subjects factor was proportion of semantic error type (6 levels); the between-subjects factor was diagnosis (AD or SD).

Results: Semantic Naming Errors

Table 6 summarizes the distribution of semantic errors. There was a significant main effect of semantic error type [$F(5, 205) = 29.05$, $p < .001$] and a main effect of diagnosis [$F(1, 41) = 79.13$, $p < .001$]. The interaction between semantic error type and diagnosis was also significant [$F(5, 205) = 5.68$, $p < .001$].

Within the AD group, the predominant error type was coordinate. Significant Bonferronni-corrected paired *t* tests were as follows: coordinate > superordinate [$t(24) = 4.84$, $p < .001$]; coordinate > subordinate [$t(24) = 6.27$, $p < .001$]; coordinate > contextual [$t(24) = 5.53$, $p < .001$]; coordinate > physical attribute [$t(24) = 6.17$, $p < .001$]; coordinate > functional-associative [$t(24) = 3.63$, $p < .001$]. The second most common naming error type in AD was functional associative. Paired *t* test results are as follows: functional-associative > subordinate [$t(24) = 3.72$, $p < .001$]; functional-associative > physical attribute [$t(24) = 3.71$, $p < .001$]. Finally, AD patients committed more superordinate errors than either subordinate errors [$t(24) = 3.78$, $p < .001$] or physical attribute errors [$t(24) = 3.69$, $p < .001$].

Within the SD group, the dominant error type was functional-associative [paired *t* test results: functional-associative > subordinate $t(17) = 4.10$, $p < .005$; functional-associative > physical attribute $t(17) = 3.91$, $p < .001$]. The second most common error for SD was coordinate [coordinate > subordinate $t(17) = 5.82$, $p < .001$, coordinate > contextual [$t(17) = 3.72$, $p = .002$, coordinate > physical attribute $t(17) = 4.62$, $p < .001$]. We observed no superordinate errors in the SD group.

The interaction between diagnosis (AD or SD) and semantic error type was primarily driven by the differing distributions of coordinate and functional associative errors and the lack of superordinate errors for the SD group. AD patients made more coordinate errors than SD patients [$t(41) = 2.16$, $p = .03$]. In contrast, SD patients showed a trend toward committing more functional-associative errors than did AD patients [$t(41) = 1.72$, $p = .09$].

Table 6
Semantic Naming Errors

	Semantic error type											
	Coordinate		Functional-associative		Physical attribute		Contextual		Subordinate		Superordinate	
Category	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
AD												
Total errors	5.88	4.62	2.60	3.40	.16	.47	.68	1.03	.04	.20	1.92	2.47
Natural kinds	2.60	2.81	.28	.54	.12	.44	.20	.50	0	0	1.52	2.14
Artifacts	3.28	2.73	2.32	3.11	.04	.20	.48	.77	.04	.20	.40	.58
SD												
Total errors	3.33	2.22	4.72	4.68	.44	.78	.83	1.34	.17	.38	0	0
Natural kinds	1.44	1.34	.72	1.07	.22	.43	.22	.55	.11	.32	0	0
Artifacts	1.89	1.28	4.00	4.06	.22	.55	.61	.98	.06	.24	0	0

Note. These values reflect raw total of each error type (of 60 items); natural kinds errors only ($n = 22$ possible); and manufactured artifacts only ($n = 38$ possible).
* Cell totals reflect the mean number of errors incurred within a patient group for a specific category.

Interim Discussion: Semantic Error Analyses

At a coarse level of processing (i.e., major naming errors), AD and SD patients appeared roughly similar. However, at a finer-grained level of semantic analysis, several differences emerged between AD and SD patients. In the context of an anomic response, SD patients tended most frequently to revert to functional-associative knowledge about objects (e.g., knife → "you cut with it"), whereas this error type was relatively rare in the AD group. AD patients instead produced many coordinate naming errors but relatively few superordinate errors.

The lack of superordinate errors in AD is discrepant with a number of previous naming studies and necessitates explanation (Coccia, Bartolini, Luzzi, Provinciali, & Lambon Ralph, 2004; Gainotti, Daniele, Nocentini, & Silveri, 1989). We attribute several potential causes to lack of this error type, both intrinsic (patient-based) and extrinsic (item-based). One such extrinsic factor involves semantic category structure of the particular stimuli: patients named more manufactured artifacts ($n = 38$) than natural kinds ($n = 22$). Artifacts lack the high degree hierarchical organization that is typical among animals and other natural kinds (Gonnerman et al., 1997). As such, broad, domain level responses such as "tool" may be less likely for target items such as "knife" and "pencil."

Another possible reason for the lack of superordinate errors is disease severity. Superordinate naming errors likely provide a linguistic marker of advanced semantic impairment (Lambon Ralph et al., 1998), and the majority of patients here were at a mild to moderate stage of disease severity, and therefore may not have reached the stage of impairment where they can only provide domain level information.

Relative to their AD counterparts, SD patients showed a trend toward producing more functional-associative naming errors. One possible explanation for the disparate rate of functional associative errors is that SD impacts inferior and ventral temporal structures that impact visual feature knowledge with relative sparing of frontoparietal structures that support functional information regarding artifact representation (e.g., "knife" → you cut with it) and associative information about natural kinds (e.g., "lion" → in the jungle). Buxbaum and colleagues (1997) reported just such a dissociation between semantic memory and object use in semantic dementia for patient DM, who showed preservation of object use in the context of more pervasive loss of semantic knowledge across alternative modalities (e.g., naming) (but for failures to replicate see Bozeat, Lambon Ralph, Patterson, & Hodges, 2002; Coccia et al., 2004).

Interestingly, both patient groups produced a high proportion of coordinate naming errors, an error that can potentially emerge from deficits in either process or content in semantic memory. With respect to content, it is feasible that "dimmed" semantic representations related to the loss of semantic feature knowledge affords access only to the most frequent or highly typical members of a semantic category. Thus, loss of content results in all medium-sized animals becoming "dogs" (a common coordinate error). An alternative account of coordinate naming errors, rooted more toward process, holds that the density of visual features (particularly among natural kinds) places increased demands on selection processes, resulting in more visual similarity and/or lexical selection errors. Given the potential for several distinct causes of a single naming error, a one-size-fits-all mechanistic account of a coordinate naming error seems implausible (see also Budd et al., 2010). Thus, it is not the presence or absence of a particular naming error that marks a difference between SD and AD, but rather the distinctiveness of their naming error distributions considered as a whole (see Figure 1).

General Discussion

We examined naming in AD and SD as a means of elucidating distinct components that support semantic memory. Our approach involved special attention to naming errors and correlations with cognitive factors such as semantic categorization ability, working memory, and executive functioning. We found that many of these factors differed between SD and AD patients, and that such differences support distinctiveness of the semantic impairments associated with both populations. More specifically, the strong correlations we observed between se-

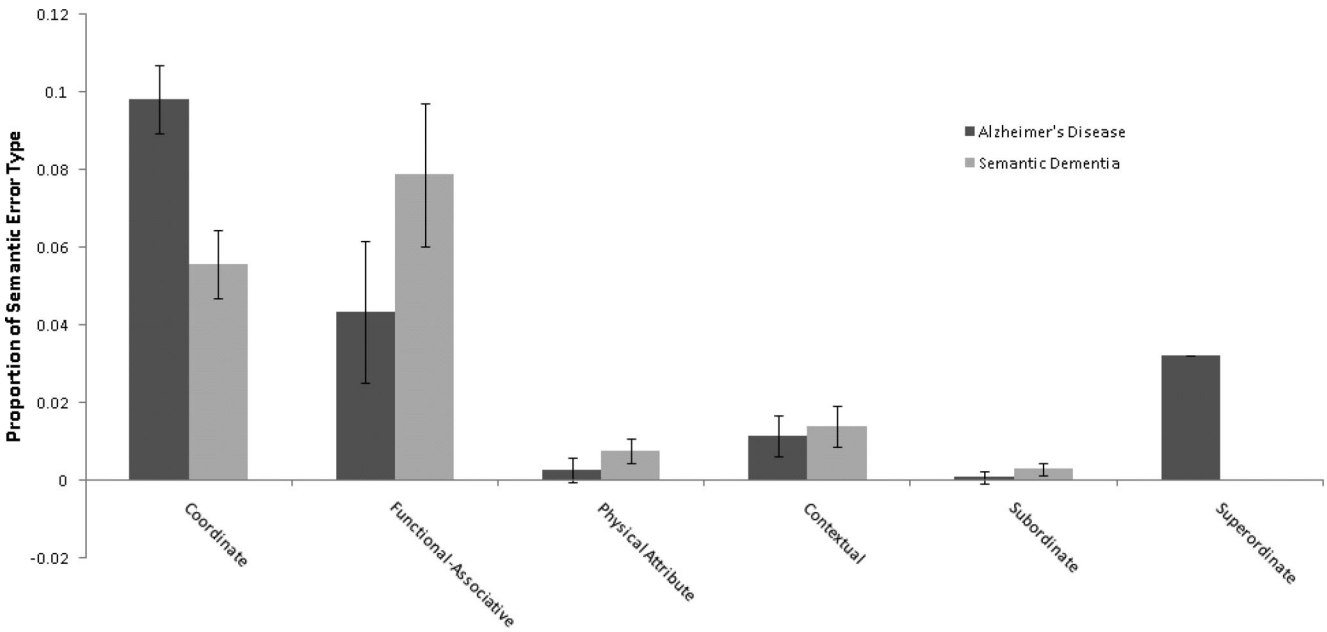

Figure 1. Semantic naming error distributions in Alzheimer's disease and Semantic Dementia.

mantic measures and naming in SD lend further support to the consensus that SD primarily reflects a disorder of degraded conceptual knowledge (Hodges, 2003; Lambon Ralph et al., 2008; Pobric, Jefferies, & Lambon Ralph, 2007; Warrington, 1975; Woollams et al., 2008). In contrast, the patterns of anomia and their neuropsychological predictors in the AD group are more consistent with a multifactorial impairment of both process and content in semantic memory.

We have proposed a model that may prove useful toward delineating the semantic impairments in SD and AD. We hypothesize that the long term consolidation of object concepts involves a process wherein objects are stripped of their original sensorimotor salience and stored in a perceptually sparse format. This concept of sparse representation, although novel as an application to semantic memory, is a well-accepted phenomenon in episodic memory research (Budson et al., 2007; Gallo et al., 2006; Rosenbaum, Gilboa, Levine, Winocur, & Moscovitch, 2009). That is, during encoding and consolidation of long-term memories, a substantial amount of peripheral detail is often forgotten while maintaining essential details in favor of a gist-like representation. Episodic gist-like analogs exist for object concepts and that the regions of the lateral temporal cortex (e.g., posterior superior temporal sulcus, middle temporal gyrus) are uniquely suited for such abstracted representations due to their central proximity and connectivity between regions of cortex that are highly specialized for specific input modalities. That is, the MTG is seated between primary auditory and secondary visual association cortex in addition to massive connectivity to both the medial temporal and frontal lobes (Beauchamp, 2005; Beauchamp, Argall, Bodurka, Duyn, & Martin, 2004; Beauchamp, Lee, Argall, & Martin, 2004; Binder et al., 2009; Binder et al., 2000; Kellenbach, Brett, & Patterson, 2001; Kellenbach, Hovius, & Patterson, 2005).

SD and AD as Lesion Models for Semantic Impairment

Patients with SD and AD share some degree of overlap in their distributions of canonical temporal lobe pathology, but there are also differences. Within the context of our proposed model of semantic memory, we predict that the extensive atrophy of lateral and inferior temporal cortex in SD profoundly impairs both multimodal conceptual representations and modality-specific visual association cortex. As such, SD patients would be expected to show relatively sweeping impairments across modalities, but this impairment should become especially evident for tasks that require access to visual feature knowledge (for recent disussion of abstract word advantages in SD see Bonner et al., 2009).

Relative to SD, AD presents a more heterogeneous lesion model for semantic impairment and one that we have argued is best described as a multifactorial loss of both content and process. Executive dysfunction is a pervasive symptom of AD, as is diffuse damage to multimodal regions of temporal cortex and more concentrated atrophy of portions of the posterior visual association cortex (Giannakopoulos et al., 1999; Grossman et al., 2004; Harnish et al., 2010; Lewis et al., 1987). As such, AD patients would be predicted to show some degree of simultaneous impairment within each of the three systems we have proposed as subserving semantic memory (i.e., modality-neutral, modality-specific, and processing). Moreover, patients with AD likely exhibit a high degree of variability in the severity of their impairment within each of these domains. Such intra- and interindividual variability may prove useful toward explaining why some AD patients behave in a manner consistent with degraded storage accounts of semantic impairment,

whereas other patients show modality advantages more consistent with semantic access or disconnection approaches.

Concluding Remarks

Contemporary theories of semantic memory can be broadly divided into two distinct camps, supporting modality-specific or modality-neutral storage of object knowledge. We recently advanced a theory that synthesizes relative merits of both approaches to semantic memory. In our current study, the contrastive performance of patients with AD and SD provides additional support for this model and highlights the role of semantic memory as an interactive multiple-component system that is susceptible to multiple types of disruption.

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