Journal of the International Neuropsychological Society (2003), **9**, 830–838.

DOI: 10.1017/S1355617703960024

Exploring semantic memory by investigating buildup and release of proactive interference in healthy older adults and individuals with dementia of the Alzheimer type

KRISTI S. MULTHAUP,[1] DAVID A. BALOTA,[2] AND MARK E. FAUST[3]
[1]Davidson College, Davidson, North Carolina
[2]Washington University in St. Louis, Missouri
[3]Department of Psychology, University of South Alabama, Mobile, Alabama

(Received June 11, 2001; Revised July 24, 2002; Accepted October 16, 2002)

Abstract

There is debate regarding the integrity of semantic memory in dementia of the Alzheimer type (DAT). One view argues that DAT is associated with a breakdown in semantic memory; the other argues that DAT is associated with predominantly preserved semantic memory and a breakdown in retrieval. The classic release from proactive interference (RPI) paradigm was used to shed light on this debate. Individuals with early-stage DAT ($n = 36$) and healthy older adult controls ($n = 45$) participated in an RPI paradigm. Each trial was a Brown–Peterson task in which participants read three-word lists, counted (for 0, 3, 6, or 9 s), and recalled the words. Both groups showed significant proactive interference (PI), but the size of the PI was significantly smaller in the DAT group. The group difference in PI may be due to the faster forgetting rate in the DAT group. Both groups showed significant RPI and there was no group difference in size when RPI was considered in terms of PI levels. Both groups showed PI and RPI in prior list intrusions. The DAT group's significant buildup and release of PI based on semantic categories suggest predominantly preserved semantic memory activity, at least, in early-stage DAT individuals.
(*JINS*, 2003, *9*, 830–838.)

Keywords: Semantic memory, Alzheimer's disease, Forgetting rate, Release from proactive interference

INTRODUCTION

Research in the past few decades has clearly shown that individuals diagnosed with dementia of the Alzheimer type (DAT) show deficits on a variety of semantic-memory tasks. Of current debate is the underlying cause of this set of performance deficits. Some researchers (e.g., Bayles et al., 1999; Norton et al., 1997; Salmon et al., 1999a; for a review see Salmon et al., 1999b) argue that semantic memory breaks down in the course of DAT. Other researchers (e.g., Balota & Duchek, 1991; Balota et al., 1999a; for reviews see Nebes, 1992; Ober & Shenaut, 1995) have argued that the semantic-memory network remains intact at least in early-stage DAT, although intentional retrieval from it may break down. Key data used to support this latter view are that under conditions of automatic activation (as opposed to controlled attentional activation), DAT-related performance changes are not typically found. Note that this debate is not about late-stage dementia where it is likely that all aspects of cognition succumb to the disease, but rather the debate concerns the earlier stages of DAT.

Researchers exploring semantic memory in DAT groups have used a variety of measures including category fluency (e.g., Salmon et al., 1999a), general knowledge questions (e.g., Norton et al., 1997), similarity judgments (e.g., Ober & Shenaut, 1999), and semantic priming (e.g., Balota & Duchek, 1991). A classic measure of the use of semantic information that has not been prominent in the current debate is the release from proactive interference paradigm (e.g., Wickens, 1972). Proactive interference (PI) is demonstrated when word-list recall drops from trial to trial, particularly when the lists are from the same semantic category (e.g., each list contains different color names). The prior lists interfere with the ability to recall the current list. In fact, prior-list words are sometimes produced instead of

Reprint requests to: Kristi S. Multhaup, P.O. Box 7000, Davidson College, Davidson, NC 28035-7000. E-mail: krmulthaup@davidson.edu

the current-list words; these are called prior-list intrusions. Release from proactive interference (RPI) is demonstrated when a set of lists from the same semantic category (e.g., colors) is followed by a word list from a new semantic category (e.g., professions) and recall for the words on this last list is higher than the recall of the several prior lists, often rising close to recall level of the first list presented (e.g., Wickens, 1972). For PI to develop and for RPI to occur in this case, participants must encode the semantic aspects of word lists that are presented to them.

We explored DAT individuals' performance in the RPI paradigm for several reasons. First, RPI is a well-established method of measuring semantic memory, as well as interference. Furthermore, Wickens (1970) argued that when participants complete the RPI task, they encode features of words (e.g., semantic category) automatically. This aspect of the RPI task is particularly important because it allows us to study semantic memory in DAT with a relatively pure measure; that is, with little influence from attentional control processes which have been argued to be impaired in DAT groups relative to healthy older adult controls (e.g., Balota et al., 1999a, 1999b; see Balota & Faust, 2001, for a review). Other tasks used to explore semantic memory in DAT (e.g., general knowledge questions, similarity judgments) are likely to be more susceptible to the influence of attentional control processes, thus complicating the interpretation of those data. Second, predictions can be made about the current debate regarding the mechanisms that underlie DAT-related deficits. If the semantic network is still predominantly intact in DAT individuals, then DAT groups should show buildup of PI. However, if the semantic network is largely degraded in DAT, DAT individuals should not show a buildup of PI. Similarly, if semantic memory is predominantly intact, RPI should be found for DAT individuals. By contrast, if semantic memory is largely degraded in DAT individuals, then this group should not show RPI, partly because they would not show PI. As discussed in the next section, the literature regarding these predictions is equivocal.

Proactive Interference (PI) in DAT

Recall measures of PI

Previous reports of research on PI in DAT with the typical word-list task (Belleville et al., 1992; Cushman et al., 1988; Wilson et al., 1983) suggest that DAT individuals do not show a buildup of PI in their recall performance. However, interpretation of these results is compromised by issues such as low sample sizes and floor performance of DAT groups. For example, Cushman et al. (1988) and Belleville et al. (1992) reported that their DAT groups did not show PI effects, but both studies had very small DAT groups ($n = 13$ and 10, respectively). Because of the variability often found in DAT groups, studies with small groups of DAT individuals are particularly open to Type II errors. As another example, Wilson et al. (1983) asked participants to do an immediate free recall of 12-word lists. The authors argued that PI did not build up in their patient group. However, the mean number of words recalled by the DAT group was between 1 and 1.5 (see Figure 1 in Wilson et al., 1983). With performance so close to floor it is very difficult to detect PI.

By contrast, Binetti et al. (1995) argued that their DAT group *did* show buildup of PI across trials, based on the lack of a significant Trial × Group (control, DAT) interaction. However, the recall difference between Trials 1 and 4 was 1.94 for controls and .21 for the DAT group (out of 12 possible). The lack of significance for a relatively large group difference (roughly 25% drop of the Trial 1 level of recall by Trial 4 for the controls compared with a roughly 7% drop for the DAT group) suggests that there was a lack of power in the analysis or that the DAT group was approaching floor performance. Thus, the argument for significant PI buildup in DAT based on the Binetti et al. (1995) data is weak at best (see also Kopelman, 1991, for a similar pattern with $n = 16$ for Alzheimer and control groups).

Overall, these prior studies suggest that research that involves both a relatively large group of DAT individuals and list lengths that are more manageable for DAT individuals (to reduce floor performance) is needed in order to obtain a clearer picture of whether or not DAT individuals show a buildup of PI.

Intrusion measures of PI

In contrast to the often reported lack of PI in DAT with recall measures, past studies, including some of the recall studies described above (e.g., Belleville et al., 1992; Butters et al., 1987; Wilson et al., 1983), have used intrusions as a measure of PI (*increased* intrusions from prior trials is the pattern that suggests PI). Belleville et al. found a higher prior-list intrusion rate for their DAT group than their control group. Similarly, Butters et al. found that, on a task in which a series of stories were presented and recalled, the DAT group showed significantly more prior-story intrusions than did controls (see also Helkala et al., 1989). By contrast, Wilson et al. found that their DAT group showed fewer prior-list intrusions, although their DAT group was at floor in recall of the lists which may contribute to this pattern. Overall, with the intrusion measure, there is evidence that DAT individuals seem to experience more interference from prior trials than controls do. This is in stark contrast to the lack of PI often reported for DAT groups when recall is the dependent measure. Thus, the intrusion data also suggest the need to further explore PI in DAT because these data suggest that PI may build up in DAT individuals after all.

Release from Proactive Interference (RPI) in DAT

Several of the studies of PI in DAT have also examined RPI. Cushman et al. (1988) reported no RPI for their DAT group, which is not surprising, given that the DAT group had not shown a buildup of PI. Binetti et al. (1995) reported that their DAT group showed RPI, but given that it is unclear that their DAT group showed buildup of PI, it is hard to interpret what a release from PI would mean (see also Belleville et al., 1992, and Kopelman, 1991). Thus, the current data leave us unable to draw conclusions about RPI in DAT individuals.

The Present Research

To increase our power to detect buildup and release of PI, as well as any group differences between the DAT and healthy older adult groups, we used a relatively large sample. To keep the DAT group off floor performance we used the traditional short three-word lists (Wickens, 1970; see also Freedman & Cermak, 1986). Also to minimize floor and ceiling effects, we varied the difficulty of the postlist distraction activity. This allowed us to better measure PI and RPI, rather than any differential effects due to differences in distractor task difficulty across groups. In addition, in order to explore the rate of loss of information in control and DAT individuals' performance, we used a Brown–Peterson design with four delays (0, 3, 6, and 9 s). This is an important aspect of the design because any possible group differences in the buildup of PI may be related to forgetting rate. If one group has a higher forgetting rate than the other, that group should also show less buildup of PI because the forgotten information will not be present to contribute to PI. Indeed, there is evidence in the literature suggesting that DAT groups show higher forgetting rates than controls (e.g., Dannenbaum et al., 1988; Larrabee et al., 1993; Salmon et al., 1989). Thus, it is particularly important to obtain a measure of forgetting rate in order to interpret any possible group differences in PI.

METHOD

Research Participants

Ninety-six participants were drawn from the Washington University Medical School Alzheimer's Disease Research Center, St. Louis, Missouri. A physician screened participants for neurologic, psychiatric, or medical disorders and medications with the potential to cause dementia or other memory problems. The criteria for a diagnosis of DAT conform to the National Institute of Neurological and Communicative Disorders and Stroke—Alzheimer's Disease and Related Disorders Association (NINCDS—ADRDA) criteria (McKhann et al., 1984) and have been described in detail elsewhere (e.g., Morris et al., 1988). When these criteria were used, reported diagnostic accuracy for Alzheimer's disease (AD) has been high (e.g., 96%, Alzheimer's disease confirmed in 102 of 106 consecutive autopsies in DAT individuals; Berg & Morris, 1994).

Dementia severity of participants was staged according to the Washington University Clinical Dementia Rating (CDR) Scale (Berg et al., 1998; Hughes et al., 1982; Morris, 1993). The CDR is based on a 90-min interview conducted by a board-certified physician with both the patient and the collateral source. The interview covers cognitive functioning in areas of memory, orientation, judgment and problem solving, community affairs, hobbies, and personal care. Each interview is videotaped and, for purposes of reliability, is reviewed by a second physician. A CDR scale score of 0 indicates no cognitive impairment, a score of .5 indicates questionable or very mild dementia, a score of 1 indicates mild dementia, a score of 2 indicates moderate dementia, and a score of 3 indicates severe dementia. CDR scores of .5 have been found to accurately indicate the earliest stages of DAT at the Washington University Alzheimer's Disease Research Center (Morris et al., 1991).

One healthy older adult and eight DAT participants had incomplete data and were not included in the analyses. Six DAT participants were excluded because they recalled on average less than one word per trial, making it unlikely that any patterns of interference and/or release could be detected on three-item lists (i.e., these participants were considered to be at floor performance). Thus, the data are reported for 81 participants: 45 healthy older adults and 36 early-stage DAT individuals (24 very mild DAT and 12 mild DAT individuals). The healthy older adults (21 females, 24 males) had a mean age of 77.91 years (SD = 8.50) and the DAT group (10 females, 17 males) had a mean age of 74.53 years (SD = 8.89). The healthy older adults had a mean of 13.84 years of education (SD = 3.06) and the DAT group had a mean of 13.47 years (SD = 2.98).

Participants were administered a 2-hr battery of psychometric tests by psychometricians who were unaware of the participants' CDR ratings. Table 1 shows selected psychometric test data by group. Memory was assessed with the Wechsler Memory Scale (WMS; Wechsler & Stone, 1973) Associates and Logical Memory subscales. General intelligence was measured with the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955) Information and Digit Symbol subscales. Lexical retrieval was assessed with the Word Fluency Test (Thurstone & Thurstone, 1949) and the Boston Naming Test (Kaplan et al., 1983). As expected, on all tests the DAT group performed more poorly than the healthy older adult group.

Materials

Stimuli were 12 categories of words taken from Battig and Montague (1969; see Appendix). Three-word lists were created by randomly selecting words from a given category. Each three-word list served in Trials 1–4 a similar number of times across participants. To avoid idiosyncratic list effects, two different sets of three-word lists were constructed and counterbalanced across participants. A given word did not appear more than once for a given participant. Nonshift blocks contained four trials of word lists from the same semantic category (e.g., four lists of color names). Shift blocks contained three trials of word lists from the same semantic category ("preshift" lists) and the fourth trial was a word list from a different semantic category ("shift

Table 1. Scores on selected psychometric tests by group

Tests	Group: Healthy controls (n = 45)	DAT individuals (n = 36)	$F(1,79)^a$
WMS Associate Learning			
M	14.01	9.72	21.80*
SD	4.33	3.81	
WMS Logical Memory			
M	9.22	5.03	26.98*
SD	3.67	3.54	
WAIS Information			
M	20.96	15.00	29.94*
SD	4.25	5.55	
WAIS Digit Symbol			
M	47.29	35.67	22.24*
SD	8.97	13.15	
Word Fluency (S & P)			
M	33.02	24.47	13.65*
SD	12.10	7.61	
Boston Naming			
M	55.67	46.03	20.21*
SD	3.86	13.74	

Note. DAT = dementia of the Alzheimer type; WMS = Wechsler Memory Scale; WAIS = Wechsler Adult Intelligence Scale.
[a]From group main effect.
* $p < .001$.

list"; e.g., three preshift lists of color names followed by a shift list of professions). Each category was rotated through the nonshift, preshift, or shift positions across participants. Each participant saw each of the 12 categories as a nonshift category, a preshift category, or a shift category. Each participant received four nonshift blocks and four shift blocks; the order of nonshift blocks or shift blocks first was counterbalanced across participants.

There were four retention intervals used between presentation and recall of the words (0, 3, 6, and 9 s). The order of retention intervals within a block (e.g., 0 s on Trial 1, 6 s on Trial 2, 3 s on Trial 3, and 9 s on Trial 4) was varied across participants and across blocks of the same shift type, that is, there was a different ordering of retention intervals for each of the nonshift blocks; shift blocks had the same pattern as nonshift blocks for a given participant. Stimuli were presented with an IBM AT-compatible computer that employed a VGA graphics card on a standard VGA monitor in 640 × 350 pixel mode.

Procedure

Controls and AD individuals probably would not find a given distractor task equally difficult, making ceiling or floor effects likely for one of the groups if the same distractor task was used for each participant. Thus, we tailored the difficulty of the distractor task in the word recall task to each participant's ability by doing a pretest counting task (see also Mistler-Lachman, 1977; Schonfield et al., 1983). Participants were shown a three-digit number in a box in the center of the computer screen. They said the number out loud and then started to count backwards by three (controls, very mild DAT individuals) or two (mild DAT individuals). If participants correctly reported 5 numbers in 15 s on two trials, they were assigned that counting task in the delay intervals of the word recall phase. If they were unable to do so, the counting task was completed again with a less difficult task until the participant met the counting criteria (the difficulty levels were backwards three, backwards two, backwards one, forward one). This difficulty level was then used in the delay intervals in the word recall task which immediately followed this counting task.

Throughout each trial of the word recall task, a white rectangle (approximately 3.2 cm high × 8.9 cm wide) was present. All characters were white (approximately 1.3 cm high × 0.6 cm wide). The trial sequence was as follows: warning signal (+) for 1000 ms, blank box for 750 ms, Word 1 which the participant read aloud and then the experimenter pressed the space bar to continue, Word 2 with the same procedure as with Word 1, Word 3 with the same procedure, retention interval of 0, 3, 6, or 9 s (after Brown, 1958; Peterson & Peterson, 1959). If the retention interval was 3, 6, or 9 s, a random three-digit number was presented, the participant read the number aloud and counted from it according to the rule established during the pretest. After the retention interval the recall cue (? ? ?) was presented for 10 s, followed by a blank box for 5 s.[a] The sessions were tape-recorded and the participants' responses were transcribed from the tapes for scoring.

There were four practice trials for the recall task, one at each retention interval. Participants needing more practice were given another set of four practice trials. There were 32 experimental trials (four nonshift blocks and four shift blocks, each with four trials).

RESULTS

Forgetting Rates

The top panel of Figure 1 contains the recall percentages for each group, for each shift type, and for each delay (collapsed across trial). Forgetting rates can be compared in the top panel by examining each group's recall percentages across delay intervals. Shift and nonshift trials are separated in Figure 1 for the purposes of later analyses. However, shift and nonshift were collapsed for the recall analysis because the distinction between nonshift and shift is a sham for Trials 1–3. Recall scores were entered into a group (controls, DAT individuals) × retention interval (0, 3, 6, 9 s) × trial number (1, 2, 3, 4) analysis of variance (ANOVA). All main effects were significant, $F > 29.14$, $p < .001$, but they were qualified by interactions.

There was a Group × Retention Interval interaction, $F(3,237) = 13.41$, $p < .001$, reflecting a faster forgetting

[a]The recall cue was three question marks separated by spaces and centered on the screen.

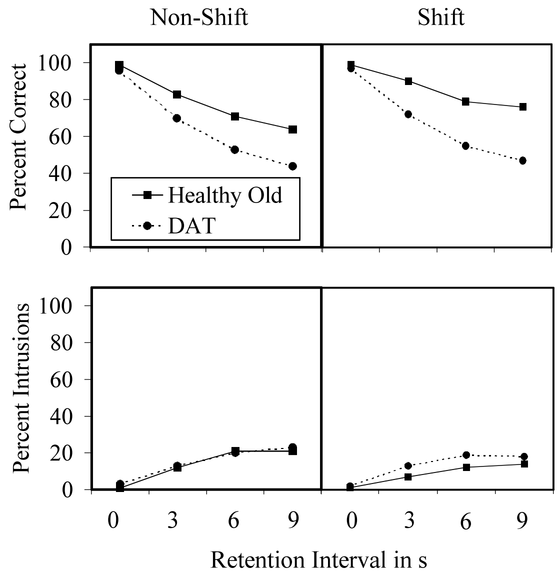

Fig. 1. Percent recall (top panel) and intrusions (bottom panel) for nonshift and shift blocks by group and retention interval, collapsed across trial number.

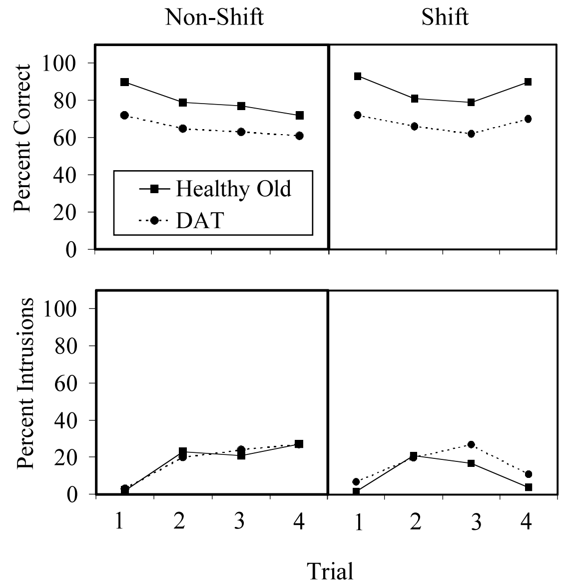

Fig. 2. Percent recall (top panel) and intrusions (bottom panel) for nonshift and shift blocks by group and trial number, collapsed across 3-, 6-, and 9-s retention intervals.

rate for the DAT group compared with the controls (see the top panel of Figure 1). There was a significant Retention Interval × Trial interaction, $F(9,711) = 3.72$, $p < .001$, reflecting the relatively low forgetting rate in Trial 1. No other interactions approached significance, $F < 1.39$.

The primary purpose of the 0-s delay was to provide even the most memory impaired participants with some successful trials. As can be seen in Figure 1 (percent correct recall in the top panel), the DAT individuals could do the task; in fact, they were at ceiling performance in the 0-s delay condition. Because both groups were at ceiling in this condition, the 0-s data were not included in the PI and RPI analyses of the recall data, nor were they included in the intrusion analyses.

PI in Recall

The top panel of Figure 2 contains the recall percentages for each group, for each shift type, and for each trial (collapsed across 3-, 6-, and 9-s retention intervals). The measure of PI was the difference score [(the combined nonshift Trial 1 and shift Trial 1 recall data) − nonshift Trial 4 recall data]. Note that in the present study shift type (nonshift, shift) was a within-subjects variable rather than a between-subjects variable as it often has been in past research (e.g., Wickens, 1970); thus collapsing across shift type was not problematic in the present study. We combined the nonshift and shift Trial 1 data because, as noted earlier, the distinction between shift and nonshift exists only in Trial 4; this is a sham distinction in all other trials. Table 2 shows that both groups showed significant PI, $t(44) = 7.81$, $p < .001$ and $t(35) = 3.99$, $p < .001$, for the control and DAT groups, respectively (also see the top panel of Figure 2). There was a significant group difference in PI, $t(79) = 2.34$, $p = .022$, reflecting less buildup of PI in the DAT group than in the control group. This significant, yet reduced, PI in the DAT group is addressed in the Discussion.

RPI in Recall

The measure of RPI was the ratio of [Trial 4 shift recall data − Trial 4 nonshift recall data] divided by the PI measure described above (see Wickens, 1972). A ratio of 1 suggests that Trial 4 recall levels returned to the Trial 1 recall levels, that is, a complete release from PI. By contrast, a ratio of 0 suggests that there was no release from PI. Because the RPI measure involves a ratio with PI in the denominator, participants who showed no buildup of PI had 0 in the denominator and thus were not included in this analysis (three controls and six DAT individuals). As seen in the top panel Figure 2, both groups showed significant RPI,

Table 2. Proactive interference (PI) and release from proactive interference (RPI) estimates by group

Group	PI	RPI (ratio)
	Recall Data	
Healthy controls	26%	1.07
DAT individuals	14%	1.06
	Intrusion Data	
Healthy controls	25%	.90
DAT individuals	20%	.84

Note. PI = [(the combined nonshift Trial 1 and shift Trial 1 recall data) − nonshift Trial 4 recall data] (see text for further discussion). RPI = [Trial 4 shift recall data − Trial 4 nonshift recall data] divided by the PI measure (see text for further discussion). DAT = dementia of the Alzheimer type.

$t(41) = 9.38$, $p < .001$ and $t(29) = 4.21$, $p < .001$, for the control and DAT groups, respectively. There was no reliable difference in RPI for the DAT and control groups (see Table 2). While the absolute difference in [Trial 4 shift recall data − Trial 4 nonshift recall data] is larger for the control group (24%) than for the DAT group (13%), when this difference is expressed as a proportion of the initial PI, the groups' proportions are virtually identical and remarkably close to 1 which is complete release from PI (the group means of each individual's proportion is 1.07 and 1.06 for controls and the DAT group, respectively). In other words, the difference in shift and nonshift recall in Trial 4 appears to decline in the DAT group compared with the control group when the absolute difference is examined, but most, if not all, of this decline seems to be accounted for by the decline in PI in the DAT group compared with the control group.

Prior-List Intrusions

Intrusions can be from prior lists in the study or from outside of the list (extralist intrusions). Because we are particularly interested in PI (the influence of prior information on the ability to remember current information), we begin with the prior-list intrusions which made up 88% of total intrusions. The bottom panels of Figures 1 and 2 show intrusion data that matches the recall data in top panels. The recall data and the prior-list intrusion data are not complementary because of the extralist intrusions and trials on which participants failed to recall three words.

PI in prior-list intrusions

As in the recall data, the PI measure was the difference score for [(the combined nonshift Trial 1 and shift Trial 1 intrusion data) − nonshift Trial 4 intrusion data]. Table 2 shows that both groups showed significant PI, $t(44) = 8.23$, $p < .001$ and $t(35) = 6.51$, $p < .001$, for the control and DAT groups, respectively (also see the bottom panel of Figure 2). There was not a significant group difference in PI, $t(79) = 0.97$, $p = .335$ (see bottom panel of Figure 2).

RPI in prior-list intrusions

Following the recall data, the measure of RPI was the ratio of [Trial 4 shift intrusion data − Trial 4 nonshift intrusion data] divided by the PI measure described in the previous section. Again, a ratio of 1 suggests complete release from PI and a ratio of 0 suggests no release from PI. As noted with the recall data, because the RPI measure involves a ratio with PI in the denominator, participants who showed no buildup of PI had 0 in the denominator and thus were not included in this analysis (seven controls and two DAT individuals). Both groups showed significant RPI, $t(37) = 14.59$, $p < .001$ and $t(33) = 6.58$, $p < .001$, for the control and DAT groups, respectively (see the bottom panel of Figure 2). There was no difference in RPI for the DAT and control groups, $t(70) = 0.45$, $p = .651$ (see Table 2). While the absolute difference in [Trial 4 shift recall data − Trial 4 nonshift recall data] is larger for the control group (23%) than for the DAT group (16%), when this difference is expressed as a proportion of the initial PI, the previous analysis indicates that there is no significant group difference in RPI (the group means of each individual's proportion is .90 and .84 for controls and the DAT group, respectively). As with the recall data, the difference in shift and nonshift recall in Trial 4 appears to decline in the DAT group compared with the control group when the absolute difference is examined, but most of this decline seems to be accounted for by the decline in PI in the DAT group compared with the control group.

Extralist Intrusions

As noted above, the vast majority of intrusions were from prior lists (417 for the DAT group and 424 for the controls), but there were also extralist intrusions which were either appropriate (e.g., giving a tree type when recalling trees) or inappropriate (e.g., giving a profession or a seemingly random word when recalling trees). The two groups had similar numbers of appropriate extralist intrusions (36 for the DAT group and 40 for the controls), but the DAT group (30) had more inappropriate extralist intrusions than the controls did (11).

DISCUSSION

Before returning to the issue of the mechanisms that underlie semantic-memory deficits in DAT groups compared with control groups, we will comment on the PI and RPI data which yield several interesting findings regarding the sensitivity of DAT individuals to PI and RPI. First, DAT individuals showed significant PI, although the amount of PI is smaller in DAT individuals than in controls when recall is the dependent measure. The present finding of PI in DAT supports Binetti et al. (1995) and Kopelman (1991) without depending on the lack of a Group × Trial interaction which is particularly open to power issues. Second, DAT individuals also showed significant RPI. While the amount of RPI looks smaller in DAT individuals than in controls when considering only Trial 4 shift and nonshift data, the groups show similar degrees of RPI when the release effect is considered in terms of the overall PI each group built up.

Implications Regarding the Measurement of RPI

Past research into PI and RPI in DAT (e.g., Belleville et al., 1992; Binetti et al., 1995; Cushman et al., 1988; Wilson et al., 1983) did not show convincing evidence for the existence of PI and RPI in DAT, as discussed in the Introduction. We were able to provide clear evidence of PI and RPI in DAT individuals, likely because we used a relatively large DAT sample in combination with the use of short lists and procedures to minimize floor performance on recall. An additional important point to consider is the potential problems that may arise when RPI is assessed without reference to the magnitude of preceding PI (i.e., when RPI is defined as recall on the last trial − recall on the second-to-last trial). When this method is used, researchers have reported RPI in DAT without clear evidence of PI (e.g., Belleville et al., 1992; Binetti et al., 1995). Such patterns are hard to interpret because it is unclear what participants were "released" from if there was no clear build up of PI before the "release." Importantly, this measure of RPI does not take into account PI, as the traditional Wickens measure does (e.g., Wickens, 1972). The Wickens RPI measure takes the measure of RPI often used by past cognitive neuropsychology researchers (e.g., Belleville et al., 1992; Binetti et al., 1995) and divides it by the level of PI (this was done in the present research). Clearly, the way RPI is measured can lead to very different conclusions regarding the presence of RPI and group differences in RPI. We believe that contextualizing release in terms of PI buildup is necessary to avoid the interpretive knot of RPI in the absence of PI, thus helping us to better understand potential group differences in RPI.

Implications for Understanding Semantic-Memory Deficits in DAT Groups

As described in the Introduction, the present data were collected to yield insight into the ongoing debate about the integrity of semantic memory in DAT individuals. We proposed that if the semantic network is still predominantly intact in DAT individuals, they should show both build up of PI and RPI. However, if the semantic network has largely degraded in DAT, the DAT group should not show PI, much less RPI. We found significant PI and RPI in DAT individuals. In addition, we found that the buildup of PI was significantly lower in the DAT group than in the control group in the recall data. The group difference in PI could be used to argue that semantic memory has degraded in DAT individuals. However, the significant buildup of PI and subsequent RPI in the DAT group is inconsistent with the idea of gross deterioration and reorganization of semantic memory in DAT individuals. Thus we see at least two possible interpretations of these data.

The first possibility is that the group difference in PI is due to a faster forgetting function in the DAT group compared with the control group (see Figure 1; cf. Dannenbaum et al., 1988; Larrabee et al., 1993; Salmon et al., 1989). If DAT individuals are less likely to remember prior information, then they should suffer from less PI than controls because the DAT individuals have less prior information available to create PI. Because the Trial 4 shift lists release participants from PI by definition, the differential forgetting rate across groups is not an issue for RPI, and thus this account is consistent with similar levels of RPI in the DAT and control groups. The present data emphasize the need to look at forgetting rates in conjunction with PI measures, particularly when comparing groups.

If this interpretation of the group difference in PI is correct, one may expect fewer prior-list intrusions from the DAT group. However, guessing on the present task could increase the prior-list intrusion rate and may underlie the similar prior-list intrusion rates for the present DAT and control groups. The lack of a group effect in intrusion rate in the present data, as opposed to larger intrusion rates in DAT compared with control groups in past research (Belleville et al., 1992; Butters et al., 1987; Helkala et al., 1989), may be due to the present participants knowing that there were three items per list. The present participants rarely offered more than three words during the recall of a word list. It may be that under conditions where participants recall nearly all of the items (lists of 3 words), intrusions will not occur more frequently in DAT individuals, but under conditions in which participants recall a relatively small proportion of the items (e.g., when recalling an entire story, when recalling relatively longer word lists), intrusions will occur more frequently in DAT individuals.

A second possible explanation of the group difference in PI is that the semantic-memory network remains preserved only for the very earliest stage of DAT. Note that our DAT sample included individuals with both very mild DAT and mild DAT diagnoses. While there were too few participants to analyze these groups separately, it is of interest that in the DAT group, the Boston Naming Test scores correlated with our measure of PI, $r(35) = .34$, $p < .05$, and in a median split on the Boston Naming Test scores of the DAT individuals, all but three of the top half of the participants were diagnosed with very mild DAT. Similarly, the correlation between the Boston Naming Test scores and our measure of RPI was positive, but it was not significant with the smaller sample size (see the Results section for details), $r(29) = .28$, $p < .14$. Thus, it is possible that as DAT progresses there will be increased contribution of semantic-memory deterioration, which in turn can produce decreased PI and RPI. Of course, even in this situation, one must be very sensitive to potential scaling issues, wherein the more severe forms of DAT may be approaching floor performance.

The pattern of significant PI and RPI in the present DAT group also adds to comparisons of DAT with other memory-impaired groups. For example, the present DAT pattern distinguishes DAT from Korsakoff's syndrome for which reported evidence suggests buildup of PI (a drop in recall of roughly 20% between Trial 1 and Trial 5), but no RPI by any measure (Janowsky et al., 1989). More research needs to be done to compare the present DAT pattern with frontal lobe and nonalcoholic patients. While evidence for both buildup of PI and RPI has been found in both of those groups (Janowsky et al., 1989; see Freedman & Cermak, 1986 for a distinction between relatively good and poor memory frontal patients), it is unclear whether the levels of PI and RPI found in these groups are equal to levels found in controls, or may be reduced as in the case of PI in the present DAT group. A particularly fruitful comparison would be the performance of DAT and frontal lobe groups given that source monitoring deficiency is one source of interference effects (e.g., Kane & Hasher, 1996), both groups show deficits on source memory tasks (see Janowsky et al., 1989, as cited in Shimamura, 1995, for frontal data; see Multhaup

& Balota, 1997, for DAT data), and DAT has been increasingly associated with frontal lobe deficiencies (e.g., Balota & Faust, 2001; Morris et al., 1996; Morrison et al., 1986; Parasuraman & Haxby, 1993).

In summary, the present study provides evidence that early-stage DAT individuals show significant PI and RPI. Their PI is reduced compared with controls in recall, but this may be due to faster forgetting rates in the DAT group. PI was similar across groups in intrusion rates. DAT individuals show similar levels of RPI as controls do, by both recall and intrusion rate measures, when initial PI is taken into account in measuring RPI. The buildup and release of PI in the present task is based on automatically encoding the semantic category to which the words belong (Wickens, 1970). Thus, when the semantic-memory system is assessed with a technique that does not rely on attentional control processes, early-stage DAT individuals (perhaps primarily those diagnosed with very mild DAT) appear to have predominantly intact semantic memory (cf. Balota et al., 1999a; Nebes, 1992; Ober & Shenaut, 1995).

ACKNOWLEDGMENTS

This research was supported by NIA grants AG00030 and RO1 AG10193. Portions of these data were presented at the Thirty-Seventh Annual Meeting of the Psychonomic Society (1996, Chicago). We thank Kris Kuhlemeier and Renée Frederickson for their help in collecting data, and Jennifer Caldwell and Debbi Berry for their help in coding data. Special thanks to John C. Morris, Director, Washington University Alzheimer's Disease Research Center (ADRC) Clinical Core, and the physicians associated with the ADRC for their expertise in providing diagnostic and dementia severity ratings and to Martha Storandt, J. Philip Miller, and Betsy Grant for providing psychometric data on the participants.

REFERENCES

Balota, D.A. & Duchek, J.M. (1991). Semantic priming effects, lexical repetition effects, and contextual disambiguation effects in healthy aged individuals and individuals with senile dementia of the Alzheimer type. *Brain and Language*, *40*, 181–201.

Balota, D.A. & Faust, M.E. (2001). Attention in dementia of the Alzheimer's type. In F. Boller & S. Cappa (Eds.), *Handbook of neuropsychology: Vol. 6. Aging and dementia* (2nd ed., pp. 51–80). New York: Elsevier Science.

Balota, D.A., Watson, J.M., Duchek, J.M., & Ferraro, F.R. (1999a). Cross-modal semantic and homograph priming in healthy young, healthy old, and in Alzheimer's disease individuals. *Journal of the International Neuropsychological Society*, *5*, 626–640.

Balota, D.A., Cortese, M.J., Duchek, J.M., Adams, D., Roediger, H.L., III, McDermott, K.B., & Yerys, B.E. (1999b). Veridical and false memories in healthy older adults and in dementia of the Alzheimer's type. *Cognitive Neuropsychology*, *16*, 361–384.

Battig, W.F. & Montague, W.E. (1969). Category norms for verbal items in 56 categories: A replication and extension of the Connecticut category norms. *Journal of Experimental Psychology: Monograph*, *80*, 1–46.

Bayles, K.A., Tomoeda, C.K., & Cruz, R.F. (1999). Performance of Alzheimer's disease patients in judging word relatedness. *Journal of the International Neuropsychological Society*, *5*, 668–675.

Belleville, S., Peretz, I., Arguin, M., Fontaine, F., Lussier, I., Goulet, P., & Joanette, Y. (1992). Assessment of semantic processing in patients with Alzheimer's type dementia: The release-of-proactive-inhibition paradigm. *Neuropsychology*, *6*, 29–41.

Berg, L. & Morris, J.C. (1994). Diagnosis. In R.D. Terry, R. Katzman, & K. Bick (Eds.), *Alzheimer's disease* (pp. 9–25). New York: Raven Press.

Berg, L., McKeel, D.W., Jr., Miller, J.P., Storandt, M., Rubin, E.H., Morris, J.C., Baty, J., Coats, M., Norton, J., Goate, A.M., Price, J.L., Gearing, M., Mirra, S.S., & Saunders, A.M. (1998). Clinicopathological studies in cognitively healthy aging and Alzheimer's disease: Relation of histologic markers to dementia severity. *Archives of Neurology*, *55*, 326–335.

Binetti, G., Magni, E., Padovani, A., Capra, S.F., Bianchetti, A., & Trabucchi, M. (1995). Release from proactive interference in early Alzheimer's disease. *Neuropsychologia*, *33*, 379–384.

Brown, J. (1958). Some tests of the decay theory of immediate memory. *Quarterly Journal of Experimental Psychology*, *10*, 12–21

Butters, N., Granholm, E., Salmon, D.P., & Grant, I. (1987). Episodic and semantic memory: A comparison of amnesic and demented patients. *Journal of Clinical and Experimental Neuropsychology*, *9*, 479–497.

Cushman, L.A., Como, P.G., Booth, H., & Caine, E.D. (1988). Cued recall and release from proactive interference in Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, *10*, 685–692.

Dannenbaum, S.E., Parkinson, S.R., & Inman, V.W. (1988). Short-term forgetting: Comparisons between patients with dementia of the Alzheimer type, depressed, and normal elderly. *Cognitive Neuropsychology*, *5*, 213–233.

Freedman, M. & Cermak, L.S. (1986). Semantic encoding deficits in frontal lobe disease and amnesia. *Brain and Cognition*, *5*, 108–114.

Helkala, E.-L., Laulumaa, V., Soininen, H., & Riekkinen, P.J. (1989). Different error pattern of episodic and semantic memory in Alzheimer's disease and Parkinson's disease with dementia. *Neuropsychologia*, *27*, 1241–1248.

Hughes, C.P., Berg, L., Danziger, W.L., Coben, L.A., & Martin, R.L. (1982). A new clinical scale for the staging of dementia. *British Journal of Psychiatry*, *140*, 566–572.

Janowsky, J.S., Shimamura, A.P., Kritchevsky, M., & Squire, L.R. (1989). Cognitive impairment following frontal lobe damage and its relevance to human amnesia. *Behavioral Neuroscience*, *103*, 548–560.

Kane, M.J. & Hasher, L. (1996). Interference. In G. Maddos (Ed.), *Encyclopedia of Aging* (2nd ed., pp. 514–516). New York: Springer.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *Boston Naming Test scoring booklet*. Philadelphia, Pennsylvania: Lea & Febiger.

Kopelman, M.D. (1991). Frontal dysfunction and memory deficits in the alcoholic Korsakoff syndrome and Alzheimer-type dementia. *Brain*, *114*, 117–137.

Larrabee, G.J., Youngjohn, J.R., Sudilovsky, A., & Crook, T.H., III. (1993). Accelerated forgetting in Alzheimer-type dementia. *Journal of Clinical and Experimental Neuropsychology*, *15*, 701–712.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS–ADRDA Work Group under the auspices of the Department of Health and Human Service Task Force on Alzheimer's Disease. *Neurology*, *34*, 39–44.

Mistler-Lachman, J.L. (1977). Spontaneous shift in encoding dimensions among elderly subjects. *Journal of Gerontology*, *32*, 68–72.
Morris, J.C. (1993). Clinical dementia rating. *Neurology*, *43*, 2412–2414.
Morris, J.C., McKeel, D.W., Fulling, K., Torack, R.M., & Berg, L. (1988). Validation of clinical diagnostic criteria for Alzheimer's disease. *Annals of Neurology*, *24*, 17–22.
Morris, J.C., McKeel, D.W., Jr., Storandt, M., Rubin, E.H., Price, J.L., Grant, E.A., Ball, M.J., & Berg, L. (1991). Very mild Alzheimer's disease: Informant-based clinical, psychometric, and pathologic distinction from normal aging. *Neurology*, *41*, 469–478.
Morris, J.C., Storandt, M., McKeel, D.W., Jr., Rubin, E.H., Price, J.L., Grant, E.A., & Berg, L. (1996). Cerebral amyloid deposition and diffuse plaques in "normal" aging: Evidence for presymptomatic and very mild Alzheimer's disease. *Neurology*, *46*, 707–719.
Morrison, J.H., Scherr, S., Lewis, D.A., Campbell, M.J., Bloom, F.E., Rogers, J., & Benoit, R. (1986). The laminar and regional distribution of neocortical somatostatin and neuritic plaques: Implications for Alzheimer's disease as a global neocortical disconnection syndrome. In A.B. Scheibel, A.F. Wechsler, & M.A.B. Brazier (Eds.), *The biological substrates of Alzheimer's disease* (pp. 115–131). New York: Academic Press.
Multhaup, K.S. & Balota, D.A. (1997). Generation effects and source memory in healthy older adults and in adults with dementia of the Alzheimer type. *Neuropsychology*, *11*, 382–391.
Nebes, R.D. (1992). Cognitive dysfunction in Alzheimer's disease. In F.I.M. Craik & T.A. Salthouse (Eds.), *The handbook of aging and cognition* (pp. 373–445). Hillsdale, New Jersey: Erlbaum.
Norton, L.E., Bondi, M.W., Salmon, D.P., & Goodglass, H. (1997). Deterioration of generic knowledge in patients with Alzheimer's disease: Evidence from the Number Information Test. *Journal of Clinical and Experimental Neuropsychology*, *19*, 857–866.
Ober, B.A. & Shenaut, G.K. (1995). Semantic priming in Alzheimer's disease: Meta-analysis and theoretical evaluation. In P.A. Allen & T.R. Bashore (Eds.), *Age differences in word and language processing*. Amsterdam: North-Holland/Elsevier Science Publishers.
Ober, B.A. & Shenaut, G.K. (1999). Well-organized conceptual domains in Alzheimer's disease. *Journal of the International Neuropsychological Society*, *5*, 676–684.
Parasuraman, R. & Haxby, J.V. (1993). Attention and brain function in Alzheimer's disease: A review. *Neuropsychology*, *7*, 242–272.
Peterson, L.R. & Peterson, M.J. (1959). Short-term retention of individual verbal items. *Journal of Experimental Psychology*, *58*, 193–198.
Salmon, D.P., Granholm, E., McCullough, D., Butters, N., & Grant, I. (1989). Recognition memory span in mildly and moderately demented patients with Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, *11*, 429–443.
Salmon, D.P., Heindel, W.C., & Lange, K.L. (1999a). Differential decline in word generation from phonemic and semantic categories during the course of Alzheimer's disease: Implications for the integrity of semantic memory. *Journal of the International Neuropsychological Society*, *5*, 692–703.
Salmon, D.P., Butters, N., & Chan, A.S. (1999b). The deterioration of semantic memory in Alzheimer's disease. *Canadian Journal of Experimental Psychology*, *53*, 108–117.
Schonfield, A.E.D., Davidson, H., & Jones, H. (1983). An example of age-associated interference in memorizing. *Journal of Gerontology*, *38*, 204–210.
Shimamura, A.P. (1995). Memory and frontal lobe function. In M.S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 803–813). Cambridge, Massachusetts: MIT Press.
Thurstone, E.L. & Thurstone, T.G. (1949). *Examiner manual for the SRA Primary Mental Abilities Test*. Chicago, Illinois: Science Research Associates.
Wechsler, D. (1955). *Manual: Wechsler Adult Intelligence Scale*. New York: Psychological Corporation.
Wechsler, D. & Stone, C.P. (1973). *Manual: Wechsler Memory Scale*. New York: Psychological Corporation.
Wickens, D.D. (1970). Encoding categories of words: An empirical approach to meaning. *Psychological Review*, *77*, 1–15.
Wickens, D.D. (1972). Characteristics of word encoding. In A.W. Melton & E. Martin (Eds.), *Coding processes in human memory* (pp. 191–215). New York: John Wiley & Sons.
Wilson, R.S., Bacon, L.D., Fox, J.H., & Kaszniak, A.W. (1983). Primary memory and secondary memory in dementia of the Alzheimer type. *Journal of Clinical Neuropsychology*, *5*, 337–344.

APPENDIX

Categories of Words Used as Stimuli

Colors	Cloths	Musical instruments	Occupations	Trees	States	Furniture	Fish	Earth formations	Vegetables	Birds	Seasonings
blue	cotton	piano	lawyer	oak	Florida	table	trout	mountain	carrot	robin	salt
red	wool	drum	teacher	maple	Texas	bed	shark	hill	pea	sparrow	sugar
green	silk	trumpet	dentist	pine	Virginia	desk	perch	valley	corn	cardinal	garlic
yellow	linen	violin	carpenter	elm	Maine	lamp	salmon	river	bean	eagle	vanilla
orange	satin	clarinet	salesman	birch	Ohio	couch	cod	lake	potato	crow	cinnamon
black	velvet	flute	nurse	spruce	Iowa	dresser	carp	canyon	lettuce	canary	cloves
purple	burlap	guitar	plumber	walnut	Georgia	stool	pike	cliff	broccoli	wren	paprika
white	denim	saxophone	accountant	hickory	Oregon	bookcase	minnow	ocean	beets	parrot	oregano
pink	flannel	trombone	farmer	ash	Kentucky	cabinet	guppy	cave	squash	pigeon	nutmeg
brown	tweed	tuba	banker	poplar	Utah	chest	flounder	volcano	onions	dove	parsley
gray	canvas	harp	fireman	willow	Nevada	bench	marlin	plateau	cucumber	owl	ginger
tan	felt	banjo	secretary	cedar	Wyoming	rocker	halibut	desert	turnip	finch	mint