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Building Episodic Connections: Changes in Episodic Priming With Age and Dementia

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Previous studies of associative encoding that used explicit retrieval tasks have shown both age- and dementia of the Alzheimer type (DAT)-related declines, but such results may be biased by group differences in explicit retrieval. In the present experiment, the authors assessed implicit associative encoding for 25 younger adults (ages 18–25), 73 healthy older adults (ages 59–91), and 65 adults with DAT (ages 59–91) during a speeded word-naming task using an episodic priming measure. Episodic priming refers to the facilitation in responding to a target word after repetition of both words in a prime–target pair, in comparison with simple repetition of the target word with a new prime on each presentation. In contrast with other studies of implicit associative encoding that did not use an implicit episodic priming measure, the present study found both age- and DAT-related declines in associative encoding under conditions of massed learning trials.

One important aspect of human learning is the buildup of associations between events that tend to co-occur in the environment. Many aspects of learning and memory decline both with dementia of the Alzheimer type (DAT) and with healthy aging (e.g., Kausler, 1994; Nebes, 1992), and it has been proposed that one important decline in learning and memory for both groups is a decline in the ability to encode new associations in memory (e.g., Granholm & Butters, 1988; MacKay & Burke, 1990; Ruch, 1934). Support for this view comes from studies that used traditional explicit or direct memory tests (e.g., Duchek, Cheney, Ferraro, & Storandt, 1991; Granholm & Butters, 1988; Kausler, 1994); the evidence is less clear when implicit or indirect tests were used (e.g., Light, La Voie, & Kennison, 1995; Monti et al., 1997; Moscovitch, Winocur, & McLachlan, 1986).

Cued recall of paired associates after intentional learning of associations has held an important historical role in the assessment of associative learning (e.g., Underwood, 1957). Sizeable age- and DAT-related deficits in paired-associates learning are well documented in the literature (e.g., Balota, Duchek, & Paullin, 1989; Gilbert, 1941; Ruch, 1934; Salthouse, Kausler, & Saults, 1990). However, it is not always clear if group differences in this type of explicit task reflect encoding of new associations or processes acting at retrieval (e.g., Burke & Light, 1981; Thomson & Tulving, 1970). Two kinds of memory retrieval have become commonly recognized: *explicit retrieval,* involving conscious recollection of events and facts, and *implicit retrieval,* which is not mediated by conscious recollection (Graf & Schacter, 1985). Because of its reliance on explicit retrieval processes, group differences in the paired-associates task may be due to differences in explicit retrieval processes rather than to differences in encoding new associations (e.g., Burke & Light, 1981; Howard, 1993). A second problem with the paired-associates task is the potential influence of intentional learning strategies above and beyond the influence of repetition (Kausler, 1994).

The present study assessed the encoding of associates using a combined incidental learning–implicit retrieval task that has been previously shown to be sensitive to age-related differences in the encoding of associative information while minimizing strategic encoding effects and explicit retrieval processes (Spieler & Balota, 1996). More specifically, we used an implicit version of what is often referred to as an *episodic priming* task (e.g., Durgunoglu & Neely, 1987; McKoon & Ratcliff, 1979; Neely & Durgunoglu, 1985).

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Episodic Priming

Episodic priming (e.g., McKoon & Ratcliff, 1979; Spieler & Balota, 1996) refers to a facilitation in responding to a target item after repeated pairing with an associate as compared with responding to a particular item that repeats but is paired with a new associate on each presentation. In this way, episodic priming is an indicant of the extent to which repeated pairing of items facilitates performance above and beyond simple repetition of the target (i.e., above and beyond repetition priming), and it can be viewed as a measure of the strength of association between repeatedly paired associate and target items. Explicit retrieval processes are typically minimized in an episodic priming paradigm by having participants respond to some aspect of the test item (e.g., speeded naming or lexical decision) that does not directly require effortful retrieval of the event of prior exposure. By minimizing retrieval processes, episodic priming paradigms provide a more direct estimate of encoding deficits in groups such as healthy older adults (e.g., Burke & Light, 1981; Howard, 1993) and individuals with DAT who also exhibit sizable retrieval deficits (e.g., Holly, 1991). It is also worth noting that episodic priming tasks are typically continuous in nature in that the same trial is usually used both as a learning trial (e.g., third presentation of an associate and a target word) and as a test trial (e.g., time to name target word). Therefore, storage requirements are minimized by immediate testing of repetitions.

Group Differences in Episodic Priming

Studies of implicit memory for preexisting associations have consistently found relative preservation of preexisting semantic associations in healthy older adults in the 55- to 80-year-old age range (e.g., Balota & Duchek, 1988, 1989; Burke, White, & Diaz, 1987; Duchek & Balota, 1993). By contrast, studies of explicit memory have consistently found age-related breakdowns in the ability to encode associations in the same age range (e.g., Gilbert, 1941; Howard, Fry, & Brune, 1991; MacKay & Burke, 1990; Ruch, 1934). However, several studies have failed to find age-related declines in the encoding of new associations using episodic priming tasks (e.g., Balota & Duchek, 1989; Howard, Heisey, & Shaw, 1986; Rabinowitz, 1986). Other studies that used implicit measures such as compound word–nonword naming (e.g., Light et al., 1995) and sequence learning (e.g., Howard & Howard, 1989, 1992) have also failed to find age-related declines in the encoding of associations.

More recently, Spieler and Balota (1996) have demonstrated that older adults (M = 72.4 years, SD = 5.1 years) will produce reduced implicit episodic priming effects under conditions in which associates do not require overt responses. This study compared episodic priming performance in younger and older adults by using a word-naming task and found that older adults produced statistically equivalent episodic priming when participants named both the associates and targets. In a second experiment in which participants only named the target items, younger adults produced episodic priming but older adults (M = 71.8 years, SD = 4.3 years) did not. Spieler and Balota (1996) argued that this result was due to removal of an associative motor learning component when participants no longer name the associate. It is also possible that older adults process the contextual information (i.e., the associate) differently when they are not required to make an explicit response to associates (e.g., Light et al., 1995). The present study used a modified version of the Spieler and Balota task to examine episodic priming in healthy aging and DAT. One goal of the present research was to replicate the finding of equivalent episodic priming effects across young adults (18–30 years) and younger old adults in the 60- to 80-year-old age range when both the targets and associates are named. We also wanted to determine whether older old adults (ages 80+) would demonstrate a relative preservation in episodic priming under these same conditions.

Studies of implicit memory for preexisting associations that use tasks such as word-associate generation (e.g., Huff, Mack, Mahlmann, & Greenberg, 1988) or category-exemplar generation (e.g., Monti et al., 1996; Salmon, Butters, & Chan, 1999) have shown adverse effects of DAT on associative information and have led to network models that propose disruption of associations in semantic memory (e.g., Chan, Salmon, & Butters, 1998). Moreover, controlled aspects of lexical priming from preexisting associations appear to be disrupted (e.g., Balota & Duchek, 1991; Balota, Watson, Duchek, & Ferraro, 1999), whereas automatic aspects of lexical priming appear relatively well preserved in DAT (e.g., Ober, Shenaut, Gregory, Jagust, & Stillman, 1991; Ober, Shenaut, Gregory, & Reed, 1995).

Studies of explicit memory consistently find DAT-related breakdowns in the ability to encode associations (e.g., Duchek et al., 1991; Granholm & Butters, 1988); breakdowns in the ability of individuals with DAT to store new associations, above and beyond the ability to explicitly encode them, have also been reported (e.g., Larrabee, Youngjohn, Sudilovsky, & Crook, 1993). Studies of implicit memory for new associations have also often found declines in individuals with DAT (e.g., Balota & Duchek, 1991; Ferraro, Balota, & Connor, 1993). By contrast, all studies to date have failed to find a DAT-related change in implicit episodic priming performance (e.g., Ergis, Van der Linden, & Deweer, 1998; Monti et al., 1997; Monti, Gabrieli, Wilson, & Reminger, 1994; Moscovitch et al., 1986). However, there is reason to view the state of knowledge regarding episodic priming and DAT as being somewhat unsettled. One hurdle to the interpretation of DAT-related changes in associative encoding is that some of these studies have not distinguished between repetition priming and episodic priming.

For example, the Monti et al. (1994) study used a sentence-rereading task that seemed to show episodic priming across repetitions, but proper controls for the effect of repetition of target words as opposed to repetition of associations were not included in this study. Recently, Monti et al. (1997) had participants reread scrambled sentences in either a constant order or a varying order. Although these researchers reported no statistically significant findings pertaining to age- or DAT-related changes in episodic priming,

an informal visual inspection of the third repetition of their same and differently ordered conditions (i.e., episodic priming) yielded a trend for episodic priming to decrease across groups (i.e., approximate *M*s = 15 ms, 9 ms, and 6 ms, for the young, healthy old, and DAT, respectively). Ergis et al. (1998) reported finding no DAT-related declines in episodic priming. Neither the healthy older controls nor the individuals with DAT produced significant episodic priming effects, whereas the younger adults did. Finally, the sentence-reading task that has been used to assess episodic priming in DAT may allow for compensatory cognitive mechanisms, such as comprehension and syntactic constraints, to mask DAT-related declines in episodic priming. Moscovitch et al. (1986) controlled for the effects of repetition priming for target words and reported episodic priming effects that were equivalent for young adults (*M* = 20–23 years across three experiments), community-dwelling older adults (*M* = 68–71 years), and memory-impaired older adults (*M* = 51–64 years). However, the memory-impaired groups were mixtures of individuals with DAT and other etiologies.

Because of these weaknesses, a major goal of the present research was to assess episodic priming with an indirect test that has been found to yield episodic priming in healthy older adults and that separates episodic priming and repetition priming effects. This latter requirement is important because a wide range of studies have found equivalent repetition priming effects in DAT and older adults (e.g., Balota & Ferraro, 1996; Gabrieli et al., 1999; Nebes, 1992; Ober & Shenaut, 1988). Therefore, it is important to control for the effects of repetition priming (i.e., repetition of the target items per se) when attempting to assess the encoding of new associations.

Method

Participants

Seventy-one individuals with DAT and 74 healthy older adults were recruited from the Washington University Medical School Alzheimer's Disease Research Center (ADRC). The healthy older adults and the individuals with DAT were all seen by a physician and completed a battery of psychometric tests approximately once a year. An additional 25 younger adults (ages 25 years or less) were recruited from the Washington University community and were paid $10 for their effort. This was done to better equate the benefits younger adults received from the testing with those that the healthy older adults and individuals with DAT received (e.g., occasional free lunches and free visits to a physician). Healthy older adults and DAT individuals were all screened by a physician for neurologic, psychiatric, or medical disorders with the potential to cause dementia. The inclusionary and exclusionary criteria for a diagnosis of DAT have been described in detail elsewhere (e.g., Morris, McKeel, Fulling, Torack, & Berg, 1988) and conform to those outlined in the National Institute of Neurological and Communicative Disorders and Stroke and Alzheimer's Disease and Related Disorders Association (NINCDS–ADRDA) criteria (McKhann et al., 1984). Diagnostic accuracy for Alzheimer's disease has been reported to be high (e.g., 96%; Alzheimer's disease confirmed in 102 of 106 consecutive autopsies in DAT individuals; Berg & Morris, 1994) when these criteria are used. All participants were native English speakers.

Dementia severity for each DAT individual who was recruited from the Washington University ADRC was staged in accordance with the Washington University Clinical Dementia Rating (CDR) scale (Hughes, Berg, Danziger, Coben, & Martin, 1982; Morris, 1993). According to this scale, a score of 0 indicates no cognitive impairment; a score of 0.5 indicates very mild dementia; a score of 1 indicates mild dementia; and a score of 2 indicates moderate dementia. At the Washington University ADRC, a CDR 0.5 rating has been found to accurately indicate the earliest stages of DAT (Morris et al., 1991).

Data from 6 DAT individuals and from 1 healthy older adult were removed because of error rates that were greater than the 25% error cutoff set for this experiment (all individuals removed from consideration produced more than 30% errors). The resultant sample of 65 individuals with DAT included 36 individuals with very mild DAT (i.e., CDR 0.5) and 29 individuals with mild DAT (i.e., CDR 1.0). The younger adults had a mean age of 20.6 years (*SD* = 1.8, *n* = 25), the healthy older adults (ages 59–91) had a mean age of 77.6 years (*SD* = 8.9, *n* = 73), and the individuals with very mild to mild DAT (ages 59–91) had a mean age of 76.0 years (*SD* = 6.1, *n* = 65). To compare age-related effects in the present study with those of Spieler and Balota (1996), we split our healthy older adult group into two subgroups using the age of 80 years as a cutoff score. This allowed direct comparison of our younger old group with their older adult groups. Moreover, because it has become common in the literature on cognitive changes in healthy older adults to distinguish between younger old and older old with a cutoff of 75–80 years of age (e.g., Balota & Ferraro, 1996), the results of the analyses of these subgroups in the present study will be directly comparable to many studies in the cognitive aging literature. The younger healthy older adults (ages 59–80) had a mean age of 70.8 years (*SD* = 6.1, *n* = 39), and the older healthy older adults (ages 80+) had a mean age of 85.5 years (*SD* = 3.5, *n* = 34).

Psychometric Test Performance

In addition to the experimental task, all of the individuals with DAT and all of the healthy older adults who were recruited from the ADRC participated in a 2-hr battery of psychometric tests as part of a larger longitudinal study of cognitive performance in DAT. Further details on the full set of tests administered in the battery are available elsewhere (see Rubin et al., 1998). We chose a subset of these tests to be reported in the present study that were consistent with prior published reports from our group (e.g., Balota & Ferraro, 1996; Faust & Balota, 1997) and that focused on language, memory, and intelligence (see Table 1). The main purpose for reporting these results was to document memory and intellectual declines in the DAT group. It should be noted that some participants did not finish some of the tasks, and, therefore, sample size varies somewhat across tasks. Memory was assessed with the Wechsler Memory Scale (WMS; Wechsler & Stone, 1973) Associates subscale (paired-associates learning) and the WMS Logical Memory subscale (surface-level story memory). Measures of general intelligence were the Information and Digit Symbol subtests of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955). Participants also received the Word Fluency Test in which they were required to name as many words as possible that began with a specified letter (*P* or *S*) in a 60-s interval (Thurstone & Thurstone, 1949). Participants also received the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983). As shown in Table 1, the performance of the DAT group was less than that of the healthy old group on all tests. Because the young were recruited from another source, they did not participate in the psychometric battery.

Table 1
Mean and Standard Deviations of Age, Education, and Scores on Selected Psychometric Tests for Healthy Older Adults and Dementia of the Alzheimer's Type (DAT) Individuals

Measure	Group				
	Younger old adults (n = 39)	Older old adults (n = 36)	Very mild DAT (n = 37)	Mild DAT (n = 29)	F
WMS Associates					
M	16.01	12.33	9.41	6.48	50.61
SD	3.46	3.29	3.51	2.10	
WMS Logical Memory[a]					
M	10.13	7.64	5.31	1.71	65.32
SD	2.52	2.37	3.35	1.02	
WAIS Information[a]					
M	22.41	20.42	15.84	9.82	53.08
SD	4.17	4.05	4.41	4.81	
WAIS Digit Symbol[b]					
M	47.87	38.69	34.26	23.75	25.99
SD	12.00	9.90	10.80	10.20	
Word Fluency[a]					
M	30.82	30.00	22.46	17.68	13.57
SD	10.56	10.27	9.25	8.51	
Boston Naming Test[a]					
M	55.56	52.50	47.05	34.57	32.15
SD	5.03	5.12	11.55	13.12	

Note. For all Fs, $p < .001$. WMS = Wechsler Memory Scale; WAIS = Wechsler Adult Intelligence Scale.
[a] These tests included 28 mild DAT. [b] WMS Digit Symbol included 35 very mild DAT and 24 mild DAT.

Apparatus

All stimuli were presented by an IBM AT-compatible computer fitted with a video graphics array (VGA) graphics card on a standard VGA monitor in 640 × 350 pixel mode. Participants viewed displays at an approximate distance of 60 cm. The basic display consisted of a centrally presented white square with a blue border (approximately 8 cm or 7.6 degrees of visual angle per side) on a dark background. Words were presented in lowercase and were black in color. Words were centered in the white square and were presented in a large, highly visible font (e.g., lowercase *a* was approximately .6 cm or .57 degrees of visual angle in width and approximately .8 cm or .76 degrees of visual angle in height). Words ranged from approximately 2.8 cm or 2.7° of visual angle in width to approximately 5.5 cm or 5.2° of visual angle in width. Naming latency for each word was measured using a microphone attached to a Gebrands Model G1341T voice-operated relay that was interfaced with the parallel port of the computer. Verbal responses were recorded on audiotape so that errors could be marked in each participant's data file.

Materials

The stimuli were a subset of words used by Spieler and Balota (1996). There were 416 one- and two-syllable words, with at least five occurrences per million (Kučera & Francis, 1967), ranging from three to six letters that were presented to each participant. From these words, 213 semantically unrelated (as determined by D. S.) word pairs were formed. Each trial consisted of a word pair being presented one word at a time on the computer monitor. Each word pair consisted of a prime (first presented) word followed by a target (second) word. All words were presented once to each participant unless otherwise noted.

Two types of repetition of word pairs were included in the experiment (see Table 2). In the target repetition condition, the target word was repeated with a new prime across all presentations of the pair. In the episodic repetition condition, both the prime and target were repeated across all presentations of the pair. The occurrence of a particular word as a prime or a target or in the target or episodic repetition conditions was counterbalanced across participants. Word pairs were presented five times within a block of trials; the first four presentations were learning trials and the final presentation was the test trial. The four learning trials were either massed together with relatively few (0–3) intervening trials or were spaced out with a greater number (8–12) of intervening trials. The fifth presentation, or test trial, was either presented after a shorter (0–3 intervening trials) or a longer (18–23 intervening trials) delay. Table 2 presents an example of each type of word-pair repetition (i.e., target repetition only or episodic repetition of both prime and target) along with the position of each presentation in the overall order of trials (i.e., massed vs. spaced learning, and shorter vs. longer delay for the fifth or test trial).

Table 2
Example Conditions

Presentation	Spaced trial no.	Massed trial no.	Repeated episodic	Repeated target
1	12	12	dog–chair	tree–chair
2	20	15	dog–chair	car–chair
3	30	17	dog–chair	bag–chair
4	42	20	dog–chair	knife–chair
Shorter delay test	45	23	dog–chair	can–chair
Longer delay test	62	43	dog–chair	can–chair

Because of the large number of cross-trial contingencies, we used a single list structure. Thus, each trial was assigned a specific combination of presentation (i.e., first through fifth), learning spacing (i.e., massed vs. spaced), and test spacing (i.e., shorter vs. longer delay). However, word-pair repetition type (i.e., target vs. episodic) was independent of the trial order and was counterbalanced across participants. The multiple constraints of the trial list structure required insertion of eight filler trials. The final list structure for each block consisted of 248 trials, with each participant completing two blocks, and with each block using different words but in the same basic trial order. The resultant 496 trials yielded 12 observations per cell in the design.

Naming Task

We chose a modified version of an implicit episodic priming task used by Spieler and Balota (1996) that had been modified in turn from an explicit episodic memory task used by Balota, Duchek, and Paullin (1989). The task involved naming of pairs of words presented one at a time on a computer monitor. The first word of each pair was the prime, and the second word of each pair was the target. The target words could repeat either with a consistent preceding prime (i.e., episodic repetition; dog–chair, dog–chair) or with a new preceding prime on each presentation (i.e., target repetition; tree–chair, dog–chair; see Table 2). The extent to which target words were named faster in the episodic priming condition, where both primes and targets repeated, than in the target repetition condition, where only the target repeated, was taken as a measure of the associative strength between the successively presented words in each pair (i.e., episodic priming). This procedure allowed for assessment of the buildup of associations online because learning trials simultaneously acted as test trials, and also controlled for the effects of repetition of the target word in each pair (i.e., repetition priming).

In addition to the episodic priming manipulation, we varied the spacing of the repetitions during a learning phase of four repetitions of word pairs. Word pairs were either massed with few intervening trials or spaced with far more intervening trials. This manipulation was included because of the well-established finding in the list-learning literature that spaced repetition items are generally remembered better than massed repetition items (see Crowder, 1976, for a review). Thus, episodic priming was assessed (via differential learning curves for target repetition trials and episodic repetition trials) across repetitions for massed and spaced repetition items.

However, the massed and spaced trials are not directly comparable during the learning phase (i.e., the first four presentations) because retention delay is distinguished from spacing during learning. By definition, the retention interval between one spaced presentation of a word pair and another is greater than the delay between one massed presentation of a word pair and another. We therefore included a fifth test trial for each word pair and added a manipulation of delay (i.e., shorter vs. longer) that was completely crossed with the spacing of the trials during the learning phase.

Procedure

When being scheduled for yearly psychometric testing, individuals with DAT and healthy older adults were recruited for participation in the current study. Every effort was made to schedule the testing for the present project either during a separate block of time during the same day or within a few days after psychometric testing. All individuals with DAT and healthy older participants were tested in the present study within 8 weeks of their latest psychometric testing. All participants completed a battery of two naming tasks consisting of first a picture-naming task that was part of another project and then the present word-naming task. None of the object names used in the picture-naming study were used in the present naming task. Total testing time was 1 to 1.5 hr per participant including all break, testing, and practice intervals.

The task was explained to participants verbally. We instructed participants to "Name each word aloud into the microphone as fast as you can while making only a few errors." Participants completed two blocks of 248 trials each. Each block took approximately 20 min and included a brief 1-min rest break halfway through. Participants performed another unrelated task taking approximately 15 min between the two blocks of trials. There were 20 practice trials and participants were encouraged to ask questions if they had any. None of the individuals with DAT or healthy older adults had participated in other studies using reaction time and computer presentation of stimuli within the preceding 12-month period.

A centrally presented filled white square with a blue border was displayed against a dark background throughout a block of trials. Each trial began with the presentation of a ready signal, consisting of three plus signs with intervening spaces for 300 ms in the center of the square followed by the presentation of a blank square again for 400 ms. The prime word was then presented, centered horizontally and vertically in the white square, until 250 ms after detection of a vocal response; it was followed by the blank square for 500 ms. Then the target word was presented until 250 ms after detection of a vocal response. Thus, the response-stimulus interval was 750 ms. After the response to the second (target) word, the blank square was presented for a 1,500 ms intertrial interval. A timeout deadline of 5,000 ms was imposed for each word. If the participant did not respond within this time interval, an error was recorded, the word was removed, and the task continued.

Results

Naming latencies were removed from further analysis for trials in which the participant (a) was still responding to the prime when the target word was presented, (b) made a response within 200 ms of target presentation, (c) made no response prior to a 5,000-ms timeout, or (d) misread the target item (as determined from audiotape recordings). We then computed an overall mean and standard deviation for each participant and removed any responses more deviant than 2.5 *SD* from each participant's overall mean. The early, timeout, and outlier responses resulted in removal of 3.8%, 4.9%, 5.4%, 4.8%, and 6.9% of the experimental trials for the young, younger old, older old, very mild DAT, and mild DAT groups, respectively. The exclusion of misreading errors resulted in removal of an additional 0.9%, 1.1%, 1.7%, 1.9%, and 3.0% of the experimental trials for the young, younger old, older old, very mild DAT, and mild DAT groups, respectively. Using the remaining correct responses, we calculated the mean for each participant in each experimental condition and submitted these values to further statistical analyses as described later.

Recently, there has been an increased recognition of the problem of interpretation of response latency effects when one group is generally slower than another group (e.g., Faust, Balota, Spieler, & Ferraro, 1999). Mean latencies for healthy older adults and individuals with DAT are often linearly related to younger adult and healthy older adult

means, respectively (e.g., Cerella, 1985; Myerson, Hale, Wagstaff, Poon, & Smith, 1990; Nebes & Brady, 1992; Nebes, Brady, & Reynolds, 1992), in such a manner as to bias the slower group to produce larger effects in mean response latencies. To evaluate the results for such a bias, we correlated each participant's overall mean latency with his or her episodic priming effects on the fifth test trial (i.e., yielding four priming effects for each individual due to the crossing of two types of learning and two test delays). None of the correlations between overall mean latency and episodic priming effects were statistically significant and all were less than .12 in absolute value.

As a further precaution, we performed a linear regression transformation (see Faust et al., 1999, for details) of mean response latencies that resulted in a linear rescaling of all individuals to have the same overall mean latency. This procedure has been shown to effectively correct for large-scale linear individual (and by extension group) differences in general cognitive speed (Faust et al., 1999). The resultant transformed naming latencies were analyzed in the same manner as the untransformed latencies, and no appreciable differences in the pattern of significant effects were found. We therefore only report on the analyses of untransformed latencies.

Learning Trials 1–4

Mean correct response latencies to name the target word in each word pair for the learning trials (1–4) for the young, younger old, and older old adults are presented in Figure 1. Mean correct response latencies for the total sample of healthy older adults (ages 59–91) and for the total sample of individuals with DAT (ages 59–91) are presented in Figure 2. Inspection of Figures 1 and 2 reveals three qualitative effects of interest: (a) response latency slowed with both age and DAT, (b) episodic priming was greater for massed as opposed to spaced learning for all groups, and (c) episodic priming decreased with both age and DAT for the massed learning condition. To evaluate these qualitative patterns in the data, we submitted mean response latencies to two separate Group (either young vs. younger old vs. older old or healthy old vs. DAT) × 2 (spacing; massed vs. spaced) × 4 (repetition) × 2 (repetition type; target vs. episodic) mixed model analyses of variance (ANOVAs), with group as a between-participants factor and repetition and repetition type as within-participant factors.

Overall analysis. Before reporting on main effects and interactions involving the repetition type factor, that is, episodic priming effects, we first report on effects not involving episodic priming. Mean response latencies slowed with age (M = 448 ms, SD = 56 ms; M = 560 ms, SD = 75 ms; and M = 595 ms, SD = 76 ms, for the young, younger old, and older old groups, respectively), $F(2, 95) = 32.74$, $p < .001$. Mean response latencies also slowed with DAT (M = 576 ms, SD = 77 ms, and M = 671 ms, SD = 118 ms, for the healthy older adult and DAT groups, respectively), $F(1, 136) = 31.52$, $p < .001$. Participants were faster to name target words as the number of repetitions increased, $F(3, 285) = 64.13$, $p < .001$; and $F(3, 408) = 39.59$, $p < .001$, in both the analysis of age effects and the analysis of DAT effects, respectively. There were significant effects of spacing, $F(1, 95) = 37.31$, $p < .001$; and $F(1, 136) = 4.94$, $p = .028$; Group × Spacing, $F(2, 95) = 3.42$, $p = .037$; and $F(1, 136) = 6.75$, $p = .010$; and Spacing × Repetition, $F(3, 285) = 38.40$, $p < .001$; and $F(3, 408) = 7.72$, $p = .001$, in the age and DAT analyses, respectively. The Group × Spacing × Repetition interaction was significant in the DAT analysis only, $F(3, 408) = 4.96$, $p = .002$. However, these interactions are mainly of interest when viewed in conjunction with the episodic priming effect (i.e., the effect of repetition type).

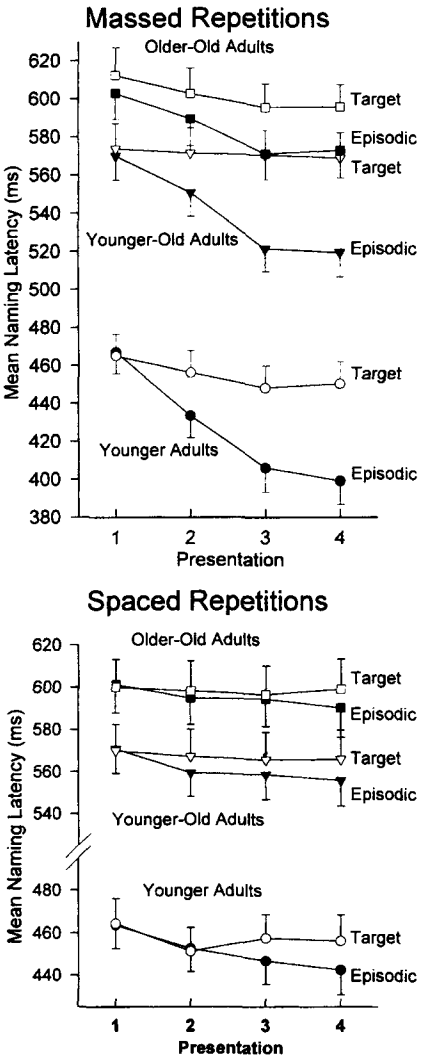

Figure 1. Mean response latency as a function of repetition type, spacing, and presentation for younger, younger old, and older old adults for the learning trials (i.e., first 4 presentations).

Age and episodic priming. Episodic repetition (i.e., repetition of both the target and the preceding associate) yielded faster responses (M = 536 ms, SD = 91 ms) than did target repetition (M = 551 ms, SD = 93 ms), indicating overall significant episodic priming, $F(1, 95) = 61.88$, $p <$

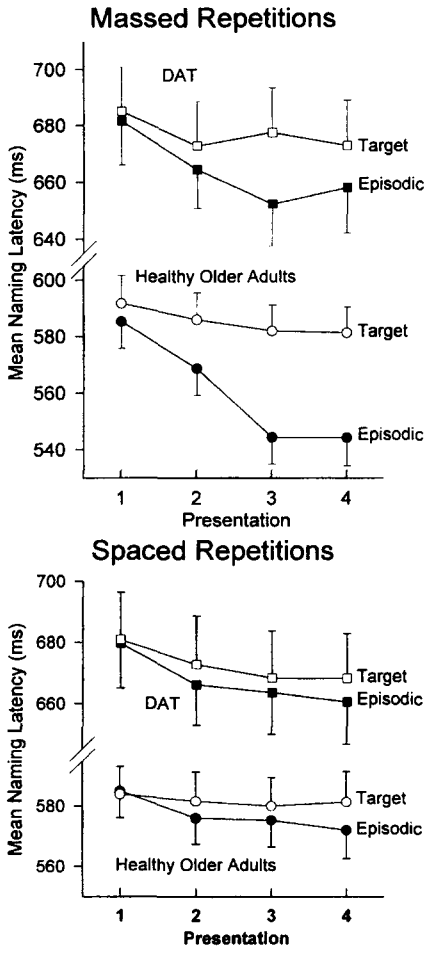

Figure 2. Mean response latency as a function of repetition type, spacing, and presentation for healthy older adults (ages 59–91) and for individuals with very mild and mild dementia of the Alzheimer type (DAT; ages 59–91) for the learning trials (i.e., first 4 presentations).

.001. Furthermore, the episodic priming effect interacted with spacing, $F(1, 95) = 32.08$, $p < .001$, indicating that episodic priming was greater for massed as opposed to spaced learning. Episodic priming also interacted with repetition, $F(3, 285) = 29.30$, $p < .001$, indicating that episodic priming increased with the number of presentations of the target word. There was a significant three-way Spacing × Repetition × Repetition Type interaction, $F(3, 285) = 17.82$, $p < .001$, indicating that episodic priming increased more across presentations of the target word for the massed as opposed to the spaced condition.

The four-way Group × Spacing × Repetition × Repetition Type interaction was significant, $F(6, 285) = 3.30$, $p = .004$. As shown in Figure 2, the four-way interaction appears to be driven by the decrease in the size of the episodic priming effect with age for the massed learning trials. To further evaluate this hypothesis, we conducted a 3 (group) × 4 (repetition) × 2 (repetition type) mixed model ANOVA for the massed spacing trials only and found that the three-way interaction was significant, $F(6, 285) = 3.77$, $p = .001$, indicating that episodic priming did decrease with age for the massed spacing conditions. A similar test performed on this three-way interaction for spaced learning trials was not significant ($F < 1$). The Repetition × Repetition Type interaction was significant for the young adults, $F(3, 72) = 7.78$, $p < .001$, but not for the younger old or older old (both Fs < 1.5, ps $> .23$), indicating that episodic priming effects were present in the spaced learning trials for the young adults only.

DAT and episodic priming. Episodic repetition (i.e., repetition of both the target and the preceding associate) yielded faster responses ($M = 615$ ms, $SD = 107$ ms) than did target repetition ($M = 627$ ms, $SD = 112$ ms), indicating overall significant episodic priming, $F(1, 136) = 31.88$, $p < .001$. Furthermore, the episodic priming effect interacted with spacing, $F(1, 136) = 19.48$, $p < .001$, which indicates that episodic priming was greater for massed as opposed to spaced learning. Episodic priming also interacted with repetition, $F(3, 408) = 13.46$, $p < .001$, indicating that episodic priming increased with the number of presentations of the target word. There was a significant Spacing × Repetition × Repetition Type interaction, $F(3, 408) = 9.01$, $p < .001$, indicating that episodic priming increased more across repetitions for the massed as opposed to the spaced condition. There were no significant episodic priming effects in the spaced trials for either the healthy old or the DAT groups as evidenced by a lack of a Repetition × Repetition Type interaction for either group, $F(3, 216) = 2.43$, $p = .066$, and $F(3, 192) = .49$, $p = .685$.

There was a nonsignificant trend for the episodic priming effect in the massed condition to be reduced for the DAT group (i.e., a nonsignificant Group × Spacing × Repetition Type interaction), $F(1, 136) = 3.62$, $p = .059$. A post hoc t test revealed that episodic priming was larger for the healthy older adults ($M = 37$ ms, $SE = 6.1$ ms) than for the individuals with DAT ($M = 15$ ms, $SE = 6.9$ ms), $t(136) = 2.422$, $p = .017$, for the fourth presentation of the massed repetition condition.

Test Trials (Fifth Presentation)

The major purpose of the present study was to assess the encoding of new associates under conditions of varied spacing of presentations during learning. However, because episodic priming effects that were obtained by comparing the learning curves for target and episodic (i.e., both prime and target) repetition conditions (see Figures 1 and 2) do not distinguish between learning and test intervals, direct comparison of episodic priming effects during the massed and spaced learning trials is difficult. Because spaced trials are presented with more intervening trials, there is a longer retention interval between each point in the spaced learning curves than for the massed learning curves and, additionally, more opportunity for retroactive interference. We now turn to an analysis of the test (fifth presentation) trials for which retention delay and spacing during learning were completely crossed. Mean response latencies were submitted to two separate Group (either young vs. younger old vs.

older old or healthy old vs. DAT) × 2 (spacing; massed vs. spaced) × 2 (delay) × 2 (repetition type; target vs. episodic) mixed model ANOVAs, with group as a between-participants factor and delay and repetition type as within-participant factors. To avoid duplication with the overall analyses reported for the learning trials (1–4) and to maintain a focus on episodic priming effects, we report on only the main effect of repetition type (i.e., episodic priming) and the interactions involving repetition type.

Age and episodic priming. There was a main effect of repetition type, $F(1, 95) = 54.42$, $p < .001$, indicating a significant episodic priming effect. The episodic priming effect varied with delay, $F(1, 95) = 73.50$, $p < .001$; it was greater at the shorter delay as opposed to the longer delay. There was a trend for episodic priming to be smaller in the older old adults ($M = 15$ ms, $SE = 4.4$ ms) than in the younger old adults ($M = 32$ ms, $SE = 6.8$ ms) or in the younger adults ($M = 30$ ms, $SE = 4.2$ ms), but this Group × Repetition Type interaction, $F(2, 95) = 3.00$, $p = .055$, did not reach statistical significance.

Finally, there was a Group × Spacing × Repetition Type interaction, $F(2, 95) = 4.02$, $p = .021$, indicating that the relative magnitude of the episodic priming effect for the massed, as opposed to the spaced, learning conditions varied with age. As can be seen in Table 3, all three groups produced relatively equivalent episodic priming effects for the spaced conditions (collapsed across delay), $F(2, 95) = .78$, $p = .460$ ($M = 29$ ms, $M = 23$ ms, $M = 18$ ms, for the young, younger old, and the older old groups, respectively). However, episodic priming effects for the massed repetition conditions varied significantly across the three groups, $F(2, 95) = 4.78$, $p = .011$, with the older old producing significantly less massed episodic priming ($M = 10$ ms, $SE = 6.4$ ms) than the younger-old adults ($M = 41$ ms, $SE = 8.7$ ms; $p = .008$, Tukey post hoc test). Neither the Spacing × Delay × Repetition Type nor the Group × Spacing × Delay × Repetition Type interaction was significant (both $Fs < 1$).

DAT and episodic priming. There was a main effect of repetition type, $F(1, 136) = 34.01$, $p < .001$, indicating a significant episodic priming effect. The episodic priming effect varied with delay (i.e., a Delay × Repetition Type interaction), $F(1, 136) = 43.18$, $p < .001$; the effect was greater at the shorter delay as opposed to the longer delay (see Table 4). There was a Group × Repetition Type interaction, $F(1, 136) = 5.05$, $p = .026$, indicating that episodic priming was smaller for the individuals with DAT ($M = 11$ ms, $SE = 4.0$ ms) than for the healthy older adults ($M = 24$ ms, $SE = 4.3$ ms).

Table 3
Mean Naming Latencies and Standard Errors for the Target Word of Test Trials (Fifth Repetition) as a Function of Group, Spacing During Learning, and Delay

	Delay					
	Shorter		Longer		Overall	
Condition	*M*	*SE*	*M*	*SE*	*M*	*SE*
Younger adults (ages 18–24 years, $n = 25$)						
Massed repetitions						
Target	442	12	458	12		
Episodic	396	13	442	12		
Episodic-priming effect	46	6	16	6	31	5
Spaced repetitions						
Target	455	13	454	10		
Episodic	409	13	442	11		
Episodic-priming effect	46	5	12	5	29	5
Overall	46	5	14	5		
Younger old adults (ages 59–79 years, $n = 39$)						
Massed repetitions						
Target	567	13	574	13		
Episodic	511	14	548	12		
Episodic-priming effect	56	10	26	9	41	9
Spaced repetitions						
Target	567	13	562	13		
Episodic	533	14	550	12		
Episodic-priming effect	34	8	12	7	23	6
Overall	45	8	19	7		
Older old adults (ages 80–91 years, $n = 34$)						
Massed repetitions						
Target	592	12	593	13		
Episodic	566	14	598	14		
Episodic-priming effect	26	7	−5	8	10	6
Spaced repetitions						
Target	596	15	600	13		
Episodic	565	15	595	14		
Episodic-priming effect	31	8	5	7	18	6
Overall	29	5	0	5		

Discussion

The results of the present study yielded clear age- and DAT-related declines in episodic priming. Consistent with previous research using a similar version of this task (Spieler & Balota, 1996, Experiment 1), the results failed to reveal any age-related declines in episodic priming in younger old adults in the 60- to 80-year-old age range. By contrast, the present findings did yield an age-related decline in episodic priming in older old adults (ages 80–91) for the massed learning conditions in both the learning curve and test trial analyses. Moreover, the older old produced equivalent episodic priming on the test trials (5) for the spaced learning (1–4) trials (i.e., collapsed across delay). Overall, the results support an age-related breakdown in episodic priming that was carried out primarily in the massed learning conditions. However, because of a general lack of robust episodic priming effects for all groups (e.g., $M = 14$ ms, $SE = 5.3$ ms; $M = 9$, $SE = 3.8$; and $M = 8$ ms, $SE = 4.0$, for Trial 4 of the spaced learning condition for the young, healthy old, and DAT groups, respectively), no strong conclusions can be drawn regarding a relative preservation of episodic priming for spaced learning conditions.

The present results suggest that the ability to form new associations may be relatively well preserved up to the eighth decade of life. This result is consistent with longitu-

Table 4
Mean Naming Latencies and Standard Errors for the Target Word of Test Trials (Fifth Repetition) as a Function of Group, Spacing During Learning, and Delay

Condition	Delay: Shorter *M*	Delay: Shorter *SE*	Delay: Longer *M*	Delay: Longer *SE*	Overall *M*	Overall *SE*
Older adults (ages 59–91 years, n = 73)						
Massed repetitions						
Target	579	9	583	9		
Episodic	537	10	572	10		
Episodic-priming effect	42	7	12	6	27	6
Spaced repetitions						
Target	580	10	580	9		
Episodic	548	10	571	9		
Episodic-priming effect	33	6	9	5	21	4
Overall	37	5	10	5		
DAT (very mild to mild, ages 59–91 years, n = 65)						
Massed repetitions						
Target	667	17	676	17		
Episodic	645	14	671	15		
Episodic-priming effect	22	8	4	8	13	6
Spaced repetitions						
Target	675	17	660	14		
Episodic	649	15	670	15		
Episodic-priming effect	26	7	−10	7	8	4
Overall	24	6	−3	4		

Note. DAT = dementia of the Alzheimer type.

dinal studies suggesting that primary mental abilities may remain relatively stable until ages 75–80 (e.g., Schaie, 1995). One factor limiting such a conclusion is the possibility that motoric response priming may have provided support for newly formed associations, thereby reducing potential age-related differences in cognitive components of episodic priming (e.g., Spieler & Balota, 1996). Such response priming effects have been found to be relatively well preserved with age (e.g., Ferraro et al., 1993; Howard & Howard, 1989). The present results are consistent with recent studies that assess implicit episodic priming in older adults. Ergis et al. (1998) and Spieler and Balota (1996) reported age-related changes in episodic priming with age under some testing conditions, but Monti et al. (1997) did not. However, visual inspection of episodic priming effects in Monti et al. suggests a trend toward decline in episodic priming with both age and DAT. The present study can therefore be seen to add to an emerging finding of modest breakdowns in episodic priming with age.

The breakdowns in episodic priming appeared to be more general with respect to DAT. The analysis of learning trials yielded modest evidence for a DAT-related decrease in episodic priming during the massed learning trials, and the analysis of test trials yielded an overall decline in episodic priming for the DAT group. These findings, coupled with the fact that the DAT failed to produce significant episodic priming for any of the conditions with a long delay (i.e., the spaced learning and delayed test trials), suggest a general breakdown in episodic priming in DAT. It is worth noting that all of the groups (i.e., young, healthy old, and DAT) produced significant episodic priming at the shorter test delay for both learning conditions (all $ps < .003$ for six one-tailed t tests with Bonferroni corrected alpha set at .008). Therefore, none of the breakdowns that were observed should be viewed as total.

The present results are inconsistent with recent studies that fail to report significant DAT-related changes in episodic priming (e.g., Ergis et al., 1998; Monti et al., 1994, 1997). However, Monti et al. (1994) failed to control for repetition priming effects, Ergis et al. (1998) used a task that did not yield episodic priming in healthy older controls, and the results of Monti et al. showed a trend toward DAT-related changes in episodic priming.

The analysis of episodic priming effects for the test trials indicated that retention delay, independent of spacing during learning, was a major influence on the magnitude of episodic priming for all groups. This finding compromises the evaluation of group differences in the effect of delay; some of the groups (i.e., older old and DAT) failed to produce episodic priming for the delayed test trials, which led to possible floor effects. Therefore, the fact that younger adults, healthy older adults, and individuals with DAT all produced equivalent declines in episodic priming across delays for test trials may indicate that decay and interference processes weakened associative strength across delay equivalently for all groups; this result also might be due to floor effects in the DAT and older old.

A second issue involves the possible strategic use of repetition. Because of time limitations in testing the individuals with DAT, the present study did not include a prime-only repetition condition. Inclusion of prime-only repetition trials might reduce the likelihood that participants will explicitly use prime repetition, in and of itself, to predict a target repetition. However, Spieler and Balota (1996) included a prime-only repetition condition in their version of the episodic naming task and found that performance did not vary across repetition in this condition. In addition, the basic pattern of episodic priming effects reported in Spieler and Balota (Experiment 1) is equivalent to the present results for the young and the younger old groups.

Another possibility is that the age- and DAT-related breakdowns in episodic priming for massed learning trials may be due to breakdowns in explicit short-term rehearsal processes. However, use of such a strategy should have been constrained by the large number of words used (416) and the large proportion of repetitions (i.e., 576 of 992 presentations were repetitions). Because both primes and targets were explicitly named with stimulus onset asynchronies of only 750–1,500 ms between words in the continuous computer controlled list, it seems unlikely that explicit rehearsal mechanisms would play a major role in this task.

Spaced Learning and Associative Encoding

The generally small episodic priming effects in the spaced learning and the longer delay test trials stand in

contrast to the generally robust episodic priming in all groups for the shorter test trials after spaced learning. One interpretation of this pattern of results is that proactive interference effects precluded large episodic priming effects at longer delays (e.g., Wickens, 1972). This suggests that the present task might be improved by holding the number of intervening trials between learning and test trials constant and varying temporal delay. It is also important to recognize that because each trial includes a learning component, the shorter delay test trials after spaced learning can be viewed as a mixture of four spaced learning gaps followed by a single massed learning gap. Therefore, the robust episodic priming in this condition might be due to the introduction of some massed learning into the sequence.

To control for the influence of the shorter delay between the fourth and fifth presentations in this condition and, incidentally, to control for potential prime-target awareness strategies, we compared the amount of episodic priming for the second presentation of a word pair of the massed learning condition with the amount of episodic priming for the fifth presentation in the spaced-learning/short-delay condition. These trials are directly comparable because in both cases the same number of intervening trials (i.e., 0–3) occurred between the final presentation in the learning sequence and the final test trial. The only differences between the two conditions were the spacing and number of presentations of the word pair during learning, that is, either one single prior presentation or four spaced prior presentations. The results of this analysis are presented in Figure 3. The young, healthy old, and DAT each produced a significant Learning Trials × Repetition Type interaction, $F(1, 24) = 10.07, p = .004$; $F(1, 74) = 4.20, p = .044$; and $F(1, 65) = 4.96, p = .036$, respectively, indicating that episodic priming effects were significantly larger for each of the groups after four spaced presentations than after a single presentation. Moreover, the amount of the increase in episodic priming effects was similar for each group and there was no three-way interaction including group (i.e., young vs. healthy old vs. DAT; $F < 1$). This result demonstrates that all groups produced equivalent significant increments in episodic priming because of additional repetition of word pairs in a spaced fashion. Therefore, the results are consistent with the hypothesis that the encoding of new associations for spaced learning situations is relatively preserved in healthy aging and DAT (see also Balota & Ferraro, 1996).

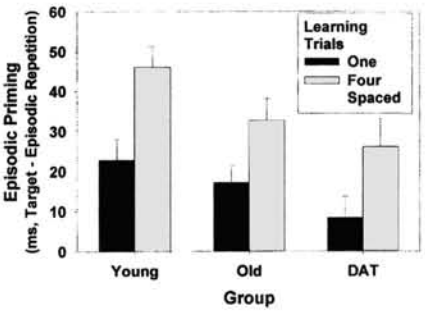

Figure 3. Episodic priming difference score (i.e., mean target repetition latency minus mean episodic repetition latency) for an immediate test trial, as a function of number of learning trials for young, healthy old, and dementia of the Alzheimer type (DAT) groups. The one learning trial condition (black bars) is the second presentation of the massed learning trials (i.e., the second presentation followed within three trials of the first). The four learning trial condition (gray bars) is the fifth presentation of the spaced learning trials with an immediate test delay (i.e., the fifth presentation followed within three trials of the fourth presentation of the spaced repetition sequence). See text for explanation of comparison.

Mechanisms of Associative Encoding of Words

The present results yielded clear age- and DAT-related declines in episodic priming for massed learning conditions. However, the general lack of robust episodic priming effects for spaced learning trials and delayed test trials made interpretation of the lack of significant group differences during spaced learning difficult. An additional analysis that controlled for the effects of delay (see Figure 3) provided some support for a relative preservation of associative encoding with age and DAT. This pattern of results suggests that the older old adults and individuals with DAT may experience a breakdown in a short-term binding mechanism that modulates the formation of new associations. Such a mechanism, when operating normally, would automatically associate pairs of events, in this case pairs of unrelated words. It is possible that the binding mechanism is dependent on the maintenance of information in an active state across time, and that limits on this maintenance process produced the group-related changes in performance in the massed repetition conditions. Although this account is consistent with other work on attention and maintenance deficits in these groups of individuals (see Balota & Faust, in press, for a review), further work is needed to better understand the nature of the constraints on this binding process that modulate simple speeded naming performance above and beyond simple repetition effects.

It has been argued that associative encoding of words might operate at any of three distinct levels (e.g., Goshen-Gottstein & Moscovitch, 1995a, 1995b; Monti et al., 1997; Spieler & Balota, 1996): conceptual, perceptual, and response. Monti et al. (1997) found some support for the involvement of perceptual and response components during associative encoding in a text-rereading task but little evidence for the involvement of conceptual processes above and beyond perceptual and response processes. Similarly, Spieler and Balota (1996) found a relatively small decline in episodic priming with age that was apparent only when differences in overall speed were controlled for, under conditions in which participants named both the prime and target word of an unrelated word pair. By contrast, in a second experiment in which participants named only the target word, Spieler and Balota found that episodic priming effects were markedly smaller for older adults than for younger adults. Indeed, there were no significant episodic priming effects for older adults in this second experiment. Spieler and Balota argued that their results indicated that older adults were able to compensate for breakdowns in

conceptual–perceptual encoding mechanisms through use of response associations (e.g., the execution of a motor program for naming the word *dog* primes the motor program for *rock*) in their first experiment but not in their second experiment. The present study has extended this work by demonstrating that even when response-encoding processes are of potential benefit, older old adults and individuals with very mild to mild DAT will produce decrements in episodic priming for word naming. These deficits are most pronounced under conditions of massed repetition and may indicate a deficit in maintaining context across short intervals in these groups. When combined with the results of Spieler and Balota and Monti et al. (1997), the present results suggest that implicit measures of episodic priming can be used to identify age- and DAT-related breakdowns in associative encoding.

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