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The Disparate Effects of Alzheimer's Disease and Huntington's Disease on Semantic Memory

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Alzheimer's disease (AD) and Huntington's disease (HD) impair performance on semantic memory tasks, but researchers disagree on whether AD and HD cause these impairments in the same manner. According to one view, AD disrupts the storage of semantic memories, whereas HD disrupts the retrieval of semantic memories. Dissenters argue that AD, like HD, disrupts retrieval. In this study, participants generated category exemplars (e.g., kinds of fruits) for 1 min, and response latencies were examined. Relative to healthy controls, the 12 AD patients produced a larger proportion of responses earlier in the recall period, consistent with the view that AD patients quickly exhaust their limited supply of items in storage. By contrast, the 12 HD patients produced a larger proportion of their responses late in the recall period, consistent with the view that HD slows retrieval.

Although both Alzheimer's disease (AD) and Huntington's disease (HD) are known to impair semantic memory, there remains widespread disagreement on whether AD and HD cause these impairments in the same manner. Specifically, this debate hinges on whether the semantic memory impairments in AD result from a storage deficit or a retrieval deficit. For instance, if an AD patient cannot name 10 kinds of fruits in 1 min, the underlying cause may be deterioration of items in storage or impaired retrieval. Regarding HD patients, however, most researchers agree that the semantic deficits result from a retrieval deficit. Therefore, either AD disrupts storage and HD disrupts retrieval, or both AD and HD disrupt retrieval. In an attempt to distinguish between these two possibilities, the present study compares the performance of AD patients and HD patients on a task in which these two views predict qualitatively distinct outcomes.

The deficits of storage and retrieval are hereinafter labeled as deficits of *storage loss* and *retrieval slowing,* respectively, but both terms are defined broadly. Specifically, a storage loss deficit exists if the recall failure results from any deterioration in either the representations or associations within semantic memory. A retrieval slowing deficit exists if the recall failure results from any abnormality in the retrieval process. Of note, if an item can be recalled in one task but not in another, the deficit is either one of storage or retrieval, depending on whether the recall failure resulted from a degraded representation or an abnormal retrieval process.

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Storage Loss in AD

Many of the empirical findings in favor of a storage loss deficit in AD rely on tasks that require participants to name exemplars from a given category. In this so-called category fluency task, the participant attempts to name all of the members of a given category (e.g., kinds of fruits) during a 1-min time period. In a study by Randolph, Braun, Goldberg, and Chase (1993), for instance, AD patients named members of a large category (e.g., animals) with or without the presence of four subcategory names (pets, jungle animals, water animals, and farm animals). Though these subcategory names increased response total in both healthy participants and HD patients, their presence had no effect on the response total of the AD patients. As argued by Randolph et al., the ineffectiveness of these subcategory names implicates a deterioration of the stored associations between the representations of the subcategory names and the category names.

Further evidence for the detrimental effects of AD on semantic storage is given by the types of items produced during a category fluency task. For instance, AD patients produce a disproportionately small number of subordinate responses (Martin & Fedio, 1983; Tröster, Salmon, McCullough, & Butters, 1989). As argued by these authors, this response pattern is parsimoniously explained by a bottom-up loss of semantic information, and, as Tröster et al. observed, "there is no obvious reason for a *general* retrieval

deficiency to have a more detrimental effect upon specific exemplars than upon general category labels" (p. 501). More decisively, perhaps, Chertkow and Bub (1990) reported that the items that are not produced during a category fluency task tend to be the same items that cannot be accessed by more direct means. Because these failures are specific to particular items and not specific to particular paradigms, this result is also well explained by a storage deficit.

In several studies of category fluency, participants have also been asked to generate words that begin with a specified letter—the letter fluency task (Butters, Granholm, Salmon, Grant, & Wolfe, 1987; Mickanin, Grossman, Onishi, Auriacombe, & Clark, 1994; Monsch et al., 1994). These researchers found that AD patients, relative to healthy controls, exhibited greater impairment in category fluency than in letter fluency. This finding is consistent with the view that AD deteriorates semantic associations because a category fluency task depends intrinsically on these associations, whereas the letter fluency task does not.

A final set of relevant studies have attempted to dissociate the performance of AD and HD patients on tasks that are believed to distinguish between the deficits of storage and retrieval. As noted above, it is widely believed that HD disrupts retrieval. In fact, several studies have shown that HD impairs recall while sparing recognition, and, given that recall requires retrieval and recognition does not, these findings provide strong evidence for the view that HD disrupts retrieval (Brandt, 1985; Butters, Wolfe, Granholm, & Martone, 1986; Butters, Wolfe, Martone, Granholm, & Cermak, 1985; Caine, Ebert, & Weingartner, 1977; Wilson et al., 1987).

Dissociations between AD and HD have been found in several different tasks. For instance, though AD disrupts category fluency and spares letter fluency, HD disrupts both types of fluency (Butters et al., 1987; Monsch et al., 1994). Given that both fluency tasks require retrieval and only the category fluency task requires intact storage, this dissociation is consistent with the view that AD disrupts storage and HD disrupts retrieval. A similar dissociation is given by the naming task studies in which participants attempt to name a pictured object. In these naming task studies, AD patients, but not HD patients, commit errors that are typically semantic in nature (Hodges, Salmon, & Butters, 1990, 1991; but see Barbarotto, Capitani, Jori, Laiacona, & Molinari, 1998). Two additional dissociations of AD and HD have been observed in the Wisconsin Card Sorting Test (Paulsen et al., 1995) and the Dementia Rating Scale (DRS; Salmon, Kwo-on-Yuen, Heindel, Butters, & Thal, 1989).

Retrieval Slowing in AD

Of the evidence for the view that retrieval slowing causes the semantic memory impairments in AD, much relies on semantic priming paradigms. In one such task, a string of letters, or target, is presented shortly after the presentation of a prime word, and the participant must decide whether the target is a word or not. This lexical decision is made as quickly as possible, and response time (RT) is measured. In trials in which the target is a word, the prime and the target are either semantically related (e.g., nurse–doctor) or unrelated (e.g., bread–doctor). In healthy participants, RT is faster in the related condition than in the unrelated condition, and the magnitude of the difference serves as the measure of priming. As typically interpreted, priming occurs because the prime activates its corresponding representation within semantic memory, and that leads to the subsequent activation of its semantically related representations. Therefore, if the prime and target are semantically related, the presentation of the prime produces activation of the target before the target is presented (unless the target appears too soon). In short, priming is believed to reduce RT by facilitating the lexical decision, and priming cannot occur unless the associations within semantic memory are intact. However, priming occurs in AD patients, and this finding is thus interpreted as evidence that is inconsistent with the loss of associations within semantic memory. The presence of this "normal" priming in AD patients has been observed in numerous studies (Nebes, Brady, & Huff, 1989; Nebes, Martin, & Horn, 1984; Ober, Shenaut, Jagust, & Stillman, 1991; Shenaut & Ober, 1996; but see Bushell & Martin, 1997; Heindel, Salmon, & Butters, 1990). In addition to the lexical-decision task, researchers have reported results from semantic priming tasks that require word naming (Nebes et al., 1989) and sentence completion (Nebes, Boller, & Holland, 1986).

Further evidence for a retrieval slowing explanation is given by studies that incorporate a semantic memory task without a prime. For instance, Nebes and his associates have used sentence-completion tasks in which participants must supply the last word of a given sentence (Nebes et al., 1986) or decide whether a given sentence completion is sensible or not (Nebes & Brady, 1991). The difficulty of this task increases with the increase in the number of acceptable final words, and both AD patients and healthy controls exhibit a similar increase in RT as difficulty increases. This similarity in impairment is interpreted as evidence for a preserved semantic memory organization in AD patients. Finally, in a study of category fluency, the extent of the decline in response totals for AD patients was found to be uniform across different categories (Cronin-Golomb, Keane, Kokodis, Corkin, & Growdon, 1992). This, too, is not readily explained by a storage deficit because the loss of associations within semantic memory would presumably occur in some regions before others.

In summary, a survey of the literature reveals considerable disagreement on whether semantic memory impairments in AD are caused by a storage loss deficit or a retrieval slowing deficit. Furthermore, it is clear that this conflict cannot be resolved by attributing these semantic impairments to deficits of both storage and retrieval because the above-cited evidence for a retrieval deficit is explicitly interpreted as evidence for spared storage. In addition, the literature review reveals that the evidence for a retrieval slowing deficit in AD differs from that for storage loss in AD. Most of the evidence for a retrieval deficit relies on RT studies, whereas the evidence for a storage loss deficit relies heavily on the category fluency task. The present study incorporates both the category fluency task and the measure of RT.

Time Course of Recall From Semantic Memory

There have been numerous investigations into the time course of recall from semantic memory in healthy adults (Baddeley, Lewis, Eldridge, & Thomson, 1984; Bousfield & Sedgewick, 1944; Graesser & Mandler, 1978; Gruenewald & Lockhead, 1980; Herrmann & Chaffin, 1976; Herrmann & Murray, 1979; Indow & Togano, 1970; Johnson, Johnson, & Mark, 1951; Metlay, Handley, & Kaplan, 1971; Rohrer, Wixted, Salmon, & Butters, 1995; Wixted & Rohrer, 1994). These category fluency studies have demonstrated that the measure of RT can reveal differences between groups that cannot be detected by the measure of response total. For instance, if 2 participants each produce five types of fruits in 1 min, their abilities still differ dramatically if, for example, 1 participant recalls the fifth and last exemplar 30 s before the other participant.

Likewise, the analysis of RTs in a category fluency task can determine whether performance is impaired by a storage deficit or a retrieval deficit, whereas the sole analysis of response totals cannot. Specifically, although both deficits reduce response totals, these two deficits predict opposite effects on the temporal locus of these responses. With storage loss, the reduction in the number of items within semantic memory allows storage loss patients to quickly complete their retrieval process because fewer exemplars remain, thereby increasing the proportion of their responses given early in the recall period. A retrieval slowing deficit, however, slows the retrieval of items from semantic memory, thereby increasing the proportion of responses given late in the recall period. Before the evidence for these effects is described, an example is presented.

Figure 1 illustrates these predicted effects of storage loss and retrieval slowing on the measures of response total and response latency. Figure 1A includes schematic diagrams that illustrate the effects of storage loss and retrieval slowing on semantic memory, and Figure 1B illustrates the predicted effects of both deficits on response latency. Each of the recall periods in Figure 1B includes the response latencies for a single trial. Thus, as shown, the recall period for each deficit includes 5 responses and the recall period for each control includes 10. It must be noted that latency is measured from the beginning of the recall period, not the previous response. As consistent with the above-described predicted effects of a storage deficit and a retrieval deficit, Figure 1B reveals that the bulk of the storage loss responses are shifted to the left, whereas the bulk of the retrieval slowing responses are shifted to the right.

The direction and the magnitude of these shifts are best portrayed by measuring the change in mean response latencies. In Figure 1B, for example, the mean response latency for the storage loss responses equals 20 s, which is simply the mean of the five response latencies, $(4 + 9 + 14 + 23 + 50)/5 = 20$. Likewise, the mean response latencies for the retrieval slowing responses equals 30 s, and the mean response latency for both sets of control responses equals 25 s. Thus, these values illustrate the *predicted effects of a storage deficit and a retrieval deficit on* the measure of mean response latency. In Figure 1B, as in every figure in this article, mean response latencies are indicated by a vertical marker along the horizontal axis.

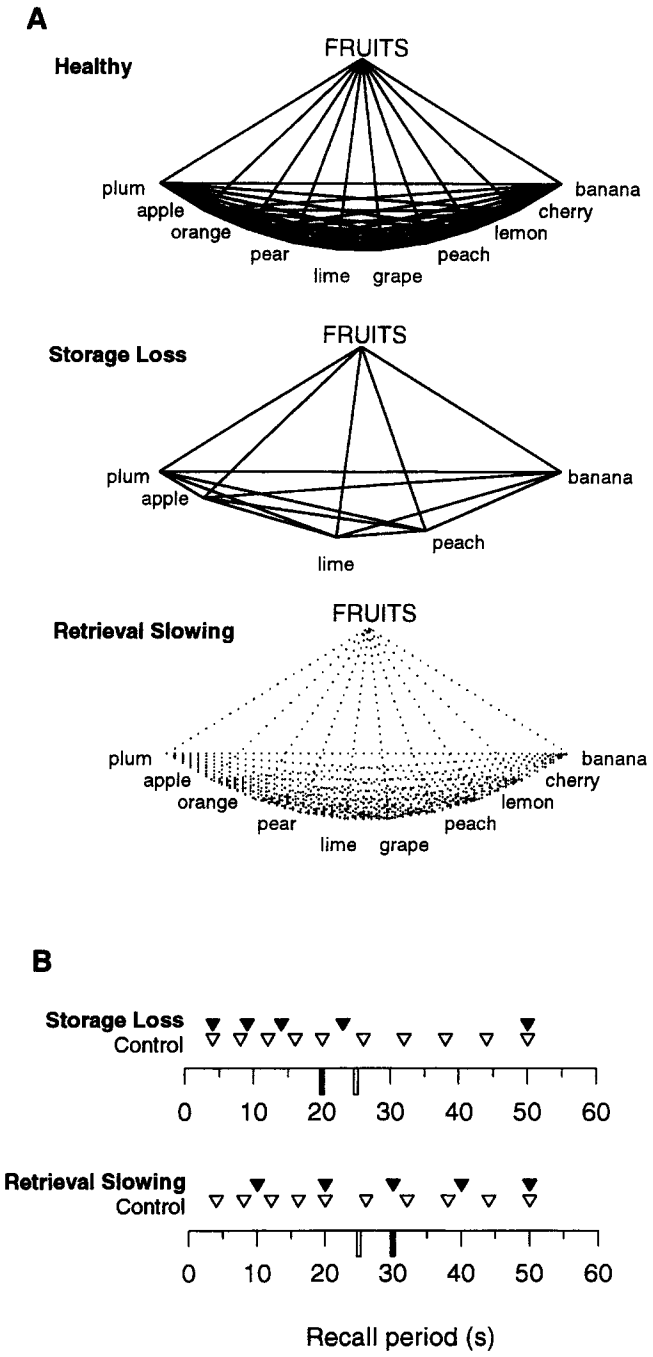

Figure 1. Idealized semantic memory networks for healthy, storage loss, and retrieval slowing patients (A) and idealized response latencies given in a single trial of category fluency for a storage loss patient, a retrieval slowing patient, and their respective controls (B). The dementia data points are represented by solid symbols, whereas control data points are represented by open symbols. Vertical markers along the horizontal axis indicate the mean response latency for the responses of each participant.

The purported effects of storage loss and retrieval slowing on mean response latency is supported by prior empirical

investigation. Specifically, Rohrer et al. (1995) attempted to simulate both a storage loss deficit and a retrieval slowing deficit in healthy adults. In the first experiment, college students generated category exemplars from either a small or large category. Because smaller categories have fewer exemplars than large categories, the small category condition was intended to simulate a storage loss deficit, and, as expected, mean response latency was shorter in this simulated storage loss condition. In the second experiment, college students generated category exemplars with or without a concurrent dual task. The dual-task condition was intended to simulate a retrieval slowing deficit, and, as expected, mean response latency was greater in this simulated retrieval slowing condition. Thus, as consistent with the predicted effects illustrated in Figure 1B, storage loss decreased mean response latency and retrieval slowing increased mean response latency.

These findings, of course, lead directly to the question of interest in the present study, as shown in Figure 2. Specifically, if AD disrupts storage and HD slows retrieval, then AD should decrease mean response latency and HD should increase mean response latency. More precisely, mean response latency for AD patients should be significantly shorter than that for older controls, and mean response latency for HD patients should be significantly greater than that for middle-aged controls. In Figure 2A, these predictions are illustrated by a single trial of idealized response latencies given by 1 participant from each of these four groups. In Figure 2B, these same predictions are shown for data from multiple trials by the presentation of a response latency distribution. In these distributions, response latencies are grouped into 10-s bins, and the relative frequency for each bin is given by its corresponding data point. That is, each data point represents the proportion of responses produced in that particular 10-s interval. As shown by the idealized data in Figure 2B, for instance, the AD patients produced about 40% of their responses during the first 10-s interval. In sum, both the mean response latency and the distribution of response latencies are left shifted by AD and right shifted by HD. This is the predicted result for the present study.

Because these predicted effects of AD and HD on mean response latency are in opposite directions, any difference in the dementia levels of these two groups is not problematic. Specifically, any change in the dementia level of either patient group may change the effect's magnitude, not its direction. By contrast, if the predicted effects had instead differed by a matter of degree, any observed difference could be alternatively explained by a difference in the group's dementia level. Though such confounds can be addressed by

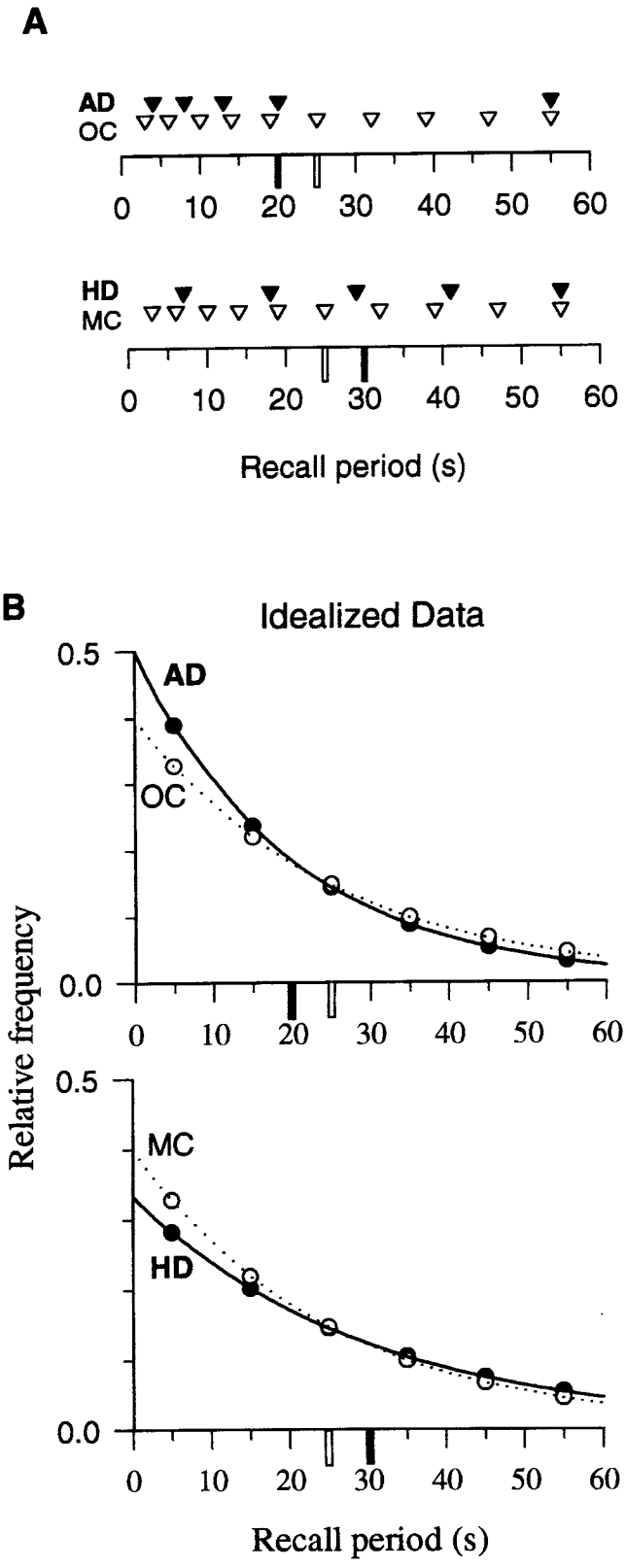

Figure 2. Idealized response latencies given in a single trial of category fluency for an Alzheimer's disease (AD) patient, Huntington's disease (HD) patient, and their controls (A) and idealized relative frequency distributions (10-s time interval) for the AD study and the HD study (B). The dementia data are represented by solid data points, solid boxes, and solid lines. Vertical markers along the horizontal axis indicate values of mean response latency. OC = older controls; MC = middle-aged controls.

an attempt to match patient groups on a standardized test score, the qualitative nature of this study eliminates the need to match. Nevertheless, we present a post hoc comparison of these groups on the basis of a subset of the patients, and these subgroups are matched on severity.

Of these two predicted outcomes in this study, the decrease in mean response latency for AD patients has been reported previously by Rohrer et al. (1995). Some have argued, however, that this finding by itself does not rule out the possibility of a retrieval slowing explanation of AD impairments because a retrieval slowing deficit might have also reduced mean response latency. For instance, the reduced mean response latency of AD patients may simply be an artifact of their reduced response totals. That is, because AD patients produce fewer responses than healthy older controls, the mean response latency of AD patients is necessarily reduced because it simply requires more time to recall more responses. In order to rule out this rival hypothesis, it must be demonstrated that a retrieval slowing dementia, such as that which accompanies HD, does, in fact, decrease response total while increasing mean response latency. This finding, therefore, would both reveal the effect of HD on the time course of recall from semantic memory as well as complete the argument for a storage loss explanation of the semantic memory impairments in AD.

Method

Participants

The AD study included 12 AD patients and 12 demographically matched older controls (OC), and the HD study included 12 HD patients and 12 demographically matched middle-aged controls (MC). The data from the AD study have been reported previously (Rohrer et al., 1995), but the analyses of those data presented herein are novel.

The AD patients were tested and diagnosed at the Alzheimer's Disease Research Center of the University of California, San Diego. The diagnosis of AD was given by two senior staff neurologists, according to both the criteria for primary degenerative dementia put forth by the American Psychiatric Association (1994) and the criteria for probable Alzheimer's disease as established by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association task force (McKhann et al., 1984). In addition, other possible causes of dementia were ruled out by medical, laboratory, and neuropsychological testing. Both neurologists were unaware of the patient's performance in the present experiment. The OCs were either volunteers recruited through newspaper advertisements or spouses of patients. Control participants with a learning disability, serious neurologic or psychiatric illness, or history of alcohol or drug abuse were excluded. Written informed consent was obtained from each participant or his or her caregiver, and all participants were native English speakers.

The HD patients were participants of the Huntington's Disease Program at the University of California, San Diego. The diagnosis of HD was made by a senior neurologist on the basis of a positive family history of the disease, the presence of involuntary choreiform movements, and the presence of dementia, according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994). The MCs were recruited by advertisements and were paid for their participation.

In both the AD and HD studies, the patient group and the respective control group were matched on age, years of education, and sex, as listed in Table 1. Naturally, neither study produced a statistically significant difference between age (both $ts < 1$), years of education (both $ts < 1$), or sex (both χ^2s < 1). As discussed earlier, however, there was a significant difference between the DRS scores of AD patients ($M = 100$, $SE = 4.6$) and the DRS scores of HD patients ($M = 119$, $SE = 3.5$), $t(22) = 3.98$, $p < .001$.

Materials

Four categories were included in the present analyses: countries in Europe, fruits, musical instruments, and vegetables. These four categories appeared in an order that was uniquely randomized for each participant. It is important to note that seven categories were analyzed in the original AD study by Rohrer et al. (1995), but three of these categories are excluded here because they were not used in the HD study (pets and farm animals, U.S. Presidents, and wild animals).

Procedure

In the beginning of each session, participants received instructions and then completed one practice trial (insects). After the practice trial, the remainder of the experiment was paced by a computer program. At the beginning of each 60-s trial, the participant saw the category name on an index card and simultaneously heard the experimenter read the category name aloud. After each response, the experimenter immediately tapped a computer key. Although such hand timing yields response latencies that are overestimated by a fraction of a second, this lag is effectively meaningless because every response latency is overestimated by approximately the same amount. Moreover, this small loss of temporal resolution is inconsequential because the response latencies were grouped into 10-s bins before analysis.

Results

Response Total

Not surprisingly, both the AD group and the HD group produced significantly fewer responses than their respective control groups, $t(22) = 7.40$, $p < .001$, and $t(22) = 3.57$, $p < .005$, respectively, as listed in Table 2. These response totals excluded both repetitions and extracategory intrusions. Both kinds of errors were rare, however, as all four participant groups produced an average of less than one of each kind per trial.

Table 1
Demographic Characteristics of Each Participant Group

	Age (years)		Education (years)		Sex
Participant group	*M*	*SE*	*M*	*SE*	M:F
Alzheimer's disease	75.1	1.5	14.3	2.1	7:5
Older control	74.6	2.2	14.3	1.0	7:5
Huntington's disease	47.2	2.0	13.2	0.46	5:7
Middle-aged control	49.2	3.5	13.6	0.38	5:7

Note. M = male; F = female.

Table 2
Summary of Results

Participant group	Response total		Response latency	
	M	*SE*	*M*	*SE*
Alzheimer's disease	4.17	0.59	21.48	2.18
Older control	14.10	1.21	27.12	2.70
Huntington's disease	6.75	1.07	29.17	2.73
Middle-aged control	11.94	1.18	21.71	1.86

Response Latency

Before presenting values of mean response latency, two technical details must be described. First, the latency of each response represents the time elapsed since the first response rather than the time elapsed since the beginning of the recall period. By excluding the time prior to the first response, this paradigm provides a more accurate portrayal of the time course of retrieval because the time prior to the first response includes perception, initiation processes, category access, and, perhaps, additional prompting by the experimenter. Incidentally, this initial time period was omitted in the original AD study as well (Rohrer et al., 1995). Second, the reported values of mean response latency were estimated by fitting a simple exponential function to the response latency data for each group. This is done because the estimated value is relatively unaffected by the duration of the recall period, whereas the observed value can depend greatly on this duration (Gronlund & Shiffrin, 1986; Roediger, Stellon, & Tulving, 1977; Rohrer, 1996; Wixted & Rohrer, 1994). After these mean latencies are estimated, the statistical differences between these parameter estimates are obtained by a *t* test of parameter values (see Ratkowski, 1983). The measure of mean response latency by parameter estimation is used in virtually every study of free-recall latency.

In the present study, these mean response latencies were affected in the predicted direction, as shown in Table 2. Specifically, mean response latency for the AD group was significantly less than that of the OC group, $t(8) = 1.86, p < .05$, whereas the mean response latency for the HD group was significantly greater than that of the MC group, $t(8) = 2.01, p < .05$. In addition, a direct comparison of the AD and HD groups revealed that the AD group produced a significantly shorter mean response latency, $t(8) = 2.20, p < .05$. Finally, the mean response latency of the OC group was slower than that for the MC group, though this difference was not statistically different.

These effects on mean response latency are best illustrated by the relative frequency distributions in Figure 3. As in Figures 1 and 2, the values of mean response latency are indicated by the vertical markers along the horizontal axis. Because AD and HD produced opposite effects on mean response latency, the AD and OC distributions intersect differently than do the HD and MC distributions. That is, the AD distribution exceeds the OC distribution before the intersection, whereas the HD distribution exceeds the MD distribution after the intersection. In other words, the AD group, relative to the OC group, produced a greater proportion of their responses in the earlier part of the recall period, whereas the HD group, relative to the MC group, produced a greater proportion of their responses in the later part of the recall period.

Discussion

In this study, the AD group produced category exemplars with a mean response latency that was significantly shorter than that of the OC group, and the HD group produced category exemplars with a mean response latency that was significantly greater than that of the MC group. In addition, these effects on mean response latency were large, as the AD value dropped 21%, and the HD value grew 34%. These opposite effects are consistent with the view that the

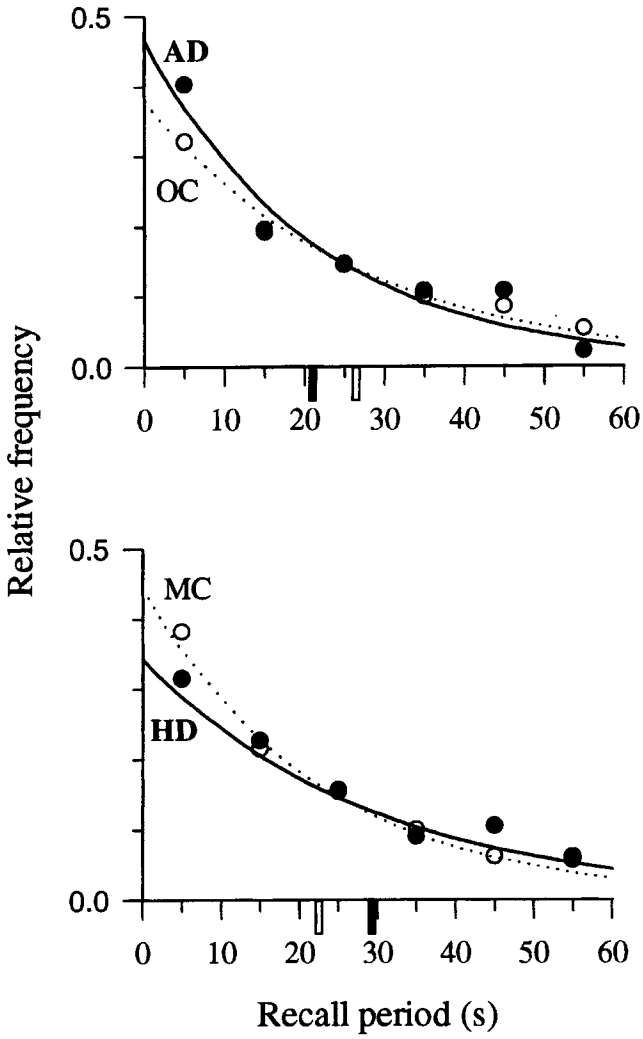

Figure 3. Observed relative frequency distributions (10-s interval) for the Alzheimer's disease (AD) study and the Huntington's disease (HD) study. Each line represents the least-squares fit exponential of the corresponding data. The dementia data are represented by solid data points, solid boxes, and solid lines. Vertical markers along the horizontal axis indicate the mean response latencies for each group. OC = older controls; MC = middle-aged controls.

semantic memory deficits in AD are caused by a storage deficit, whereas the semantic memory deficits in HD are caused by a retrieval deficit. Specifically, if AD disrupts the associations within semantic memory, the number of accessible items is decreased, and this reduced number of category exemplars is quickly exhausted. By contrast, if HD slows retrieval, response latencies should occur late in the recall period. These shifts in the distribution of response latencies necessarily shift mean response latency in the same direction. Thus, as observed, mean response latency is left shifted by AD and right shifted by HD.

Although the qualitatively distinct effects of AD and HD preclude the need to match AD and HD patients on the extent of dementia, a post hoc analysis was conducted to alleviate any concerns regarding this difference in the severity of the dementia. Specifically, mean response latency was reanalyzed for the three so-called mild AD patients, whose mean DRS score did not statistically differ from the mean DRS score for the original HD group (118.3 vs. 119.6). Nevertheless, the mean response latency for this mild AD group was significantly less than that for both the HD group and the OC group.

The difference between the mean response latencies of the two control groups was presumably due to aging-induced slowing, as the OC group was, on average, about 30 years older than the MC group. If such aging-induced slowing did exist, it worked against the significant difference in the mean response latencies of the AD and HD groups. Specifically, because the mean response latency for the older AD group was shorter than that of the younger HD group, aging should have attenuated this observed difference.

Although the reduction in mean response latency for AD patients is inconsistent with the view that their semantic memory impairments result from retrieval slowing, this finding is not inconsistent with the view that AD causes retrieval slowing, per se. Indeed, retrieval slowing among AD patients was evident in the present study as well, because both AD patients (and HD patients) produced responses with interresponse times (IRTs) that greatly exceeded, on average, both control groups. Such slowing is, of course, expected (see Nebes & Brady, 1992). Nonetheless, these slowed responses are entirely consistent with the prediction of a storage loss deficit because it is mean response latency, not mean IRT, that distinguishes between the storage loss explanation and a retrieval slowing explanation of semantic memory impairments in AD.

Arguably, the increased mean response latency of HD patients can be partly attributed to motoric slowing rather than retrieval slowing. That is, perhaps HD patients retrieved items into consciousness at a normal rate and then vocalized these responses very slowly. Evidence for such motoric slowing is given by findings in which HD patients exhibited slowed RTs in speeded tasks that included only a brief cognitive component (e.g., a choice-RT task with high stimulus–response compatibility; Willingham & Koroshetz, 1993). Although the presence of such motoric slowing cannot be definitively ruled out in the present study, the data suggest that motoric slowing did not contribute to the observed difference between the AD and HD groups. Specifically, if motoric slowing had affected the mean latency of the HD group, then the IRTs should have been especially slow. However, an analysis of the IRT distributions revealed that HD patients produced a greater proportion of brief IRTs than did AD patients. Specifically, the HD patients, compared with AD patients, produced a greater proportion of their IRTs in both the first 1-s bin (.08 vs. .06) and the second 1-s bin (.24 vs. .17). Even though neither difference was statistically significant, the direction of the difference is the opposite of that predicted by motoric output slowing.

Finally, it should be noted that the above analyses of response latency divulge two drawbacks in the current use of category fluency tasks in neuropsychological tests, such as the DRS. First, these tests typically include large categories, such as animals or supermarket items, and both memory patients and healthy participants cannot complete their recall of the large number of exemplars within 1 min. With these large categories, then, the observation of a less-than-normal response total may reflect either a reduction in the number of accessible items or a slowing of retrieval. This ambiguity detracts from the diagnostic value of these tests. Second, regardless of whether patients complete their recall or not, the measure of response total does not reveal the amount of time needed to complete the responses. The additional measure of response latency solves both problems.

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