Dissociating Hippocampal versus Basal Ganglia Contributions to Learning and Transfer

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Abstract

■ Based on prior animal and computational models, we propose a double dissociation between the associative learning deficits observed in patients with medial temporal (hippocampal) damage versus patients with Parkinson's disease (basal ganglia dysfunction). Specifically, we expect that basal ganglia dysfunction may result in slowed learning, while individuals with hippocampal damage may learn at normal speed. However, when challenged with a transfer task where previously learned information is presented in novel recombinations, we expect that hippocampal damage will impair generalization but basal ganglia dysfunction will not. We tested this prediction in a group of healthy elderly with mild-to-moderate hippocampal atrophy, a group of patients with mild

Parkinson's disease, and healthy controls, using an "acquired equivalence" associative learning task. As predicted, Parkinson's patients were slower on the initial learning but then transferred well, while the hippocampal atrophy group showed the opposite pattern: good initial learning with impaired transfer. To our knowledge, this is the first time that a single task has been used to demonstrate a double dissociation between the associative learning impairments caused by hippocampal versus basal ganglia damage/dysfunction. This finding has implications for understanding the distinct contributions of the medial temporal lobe and basal ganglia to learning and memory. ■

INTRODUCTION

The medial temporal (MT) lobe and the basal ganglia are thought to play distinct roles in learning and memory. Traditionally, the MT lobe has been associated with declarative memory function in humans, while the basal ganglia are associated with procedural or habit learning in animal and humans. Humans with damage to the MT lobes, including the hippocampus, are often spared on a variety of tasks, which seem to involve incrementally acquired learning of habits or skills. For instance, individuals with MT damage can learn as quickly as healthy controls in paradigms ranging from delay eyeblink classical conditioning (Gabrieli et al., 1995; Woodruff-Pak, 1993) to category learning (Maddox et al., 1999; Squire & Knowlton, 1995). By contrast, humans with damage to the basal ganglia often show deficits on these forms of habit learning. For example, Parkinson's disease (PD) devastates dopaminergic neurons in the substantia nigra compacta, disrupting basal ganglia processing. Parkinson's patients are slow to acquire conditioned eyeblink

responses (Sommer, Grafman, Clark, & Hallett, 1999; Daum, Schugens, Breitenstein, Topka, & Spieker, 1996), probabilistic classification (Shohamy et al., 2002; Knowlton, Mangels, & Squire, 1996), and categorization (Maddox & Filoteo, 2001; Reed, Squire, Patalano, Smith, & Jonides, 1999). Together, these findings suggest that the basal ganglia, but not the hippocampus, play a critical role in stimulus–response-based habit learning (e.g., White, 1997; Knowlton & Squire, 1993; Mishkin, Malamut, & Bachevalier, 1984).

However, while the hippocampus may not be critical for learning simple stimulus–response-based learning, it does appear to be critical for some forms of more complex learning, such as the ability to transfer when familiar stimuli are presented in novel recombinations (e.g., Myers et al., 2002; Eichenbaum, Mathews, & Cohen, 1989). For example, in one recent study, nondemented elderly individuals with hippocampal atrophy (HA) revealed by neuroimaging were able to learn an eight-pair concurrent visual discrimination as quickly as nonatrophied controls, but were selectively impaired when challenged by a transfer test in which familiar stimulus features were presented in novel recombinations (Myers et al., 2002). This suggests that the hippocampus and related MT areas may be important for the ability to generalize learned information.

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Table 1. Acquired Equivalence Paradigm in Humans (Collie et al., 2002)

Acquisition Stage 1: Shaping	Acquisition Stage 2: Equivalence Training	Acquisition Stage 3: New Consequents	Transfer Phase: Equivalence Testing
		A1→X1	
A1→X1	A1→X1	A2→X1	A2→X2?
	A2→X1	A1→X2	
		B1→Y1	
B1→Y1	B1→Y1	B2→Y1	B2→Y2?
	B2→Y1	B1→Y2	

Note that transfer phase interleaved trials with the previously learned information as well as the novel pairs.

If so, then we would expect similar patterns of spared initial learning but impaired generalization in individuals with HA on a range of learning and transfer tests. For example, acquired equivalence is a phenomenon in which prior training to treat two stimuli as equivalent increases generalization between them—even if those stimuli are superficially very dissimilar (e.g., Bonardi, Rey, Richmond, & Hall, 1993; Hall, Ray, & Bonardi, 1993; Grice & Davis, 1960). In one such study, Bonardi et al. (1993) demonstrated acquired equivalence in pigeons. First, the pigeons were trained to peck at a keylight, where stimulus orderings A1–X1, A2–X1, B1–Y1, and B2–Y1 all predicted food availability. In effect, antecedents A1 and A2 were "equivalent" in terms of their pairing with X1, while B1 and B2 were also "equivalent" in their pairing with Y1. Next, the pigeons were trained to peck to A1 but not B1; finally, the pigeons were tested for response to A2 and B2. The birds tended to respond strongly to A2 but not to B2. Apparently, the birds had learned equivalencies between stimuli with identical consequents: Given that A1 was rewarded and A2 was "equivalent" to A1, the birds expected that A2 would also be rewarded. Similar effects have been shown in humans (Spiker, 1956) and rats (Honey & Hall, 1991; Hall & Honey, 1989).

This type of acquired equivalence task seems to require flexible generalization, of the kind hypothesized to depend on hippocampal region mediation (Myers & Gluck, 1996; Eichenbaum et al., 1989). Suppose that an individual learned the A1–X1 and A2–X1 pairings, but this learning was hyperspecific—applying only to the trained pairings, without any equivalence being formed between A1 and A2. Then, although initial learning may look the same, subsequent learning about A1 should not transfer at all to A2. This would suggest that hippocampal damage or atrophy might spare learning during the acquisition phases but impair the ability to transfer.

Conversely, although basal ganglia damage may impair the ability to acquire simple stimulus associations, it should not directly affect hippocampal processing.

Thus, patients with basal ganglia dysfunction due to PD may be expected to show slower learning of the initial associations in the acquisition phases—but, once this information is acquired, there may be normal or near-normal transfer.

If these predictions hold, acquired equivalence may be a paradigm that allows double dissociation, within a single task, between the qualitative pattern of learning impairments following damage to the hippocampal region versus the basal ganglia.

To test this proposed dissociation, we used a computer-based acquired equivalence task, shown in Table 1 (Collie et al., 2002; Myers, Shohamy, Schwartz, & Gluck, 2000). In this version, there are three acquisition stages, antecedent stimuli are represented on the screen as cartoon faces, and consequents are represented as different colored cartoon fish. Two antecedent stimuli A1 and A2 were associated with the same consequent stimulus X1, while two antecedent stimuli B1 and B2 were associated with consequent Y1. Next, A1 was associated with a new consequent X2 while B1 was associated with a new consequent Y2. Finally, a transfer phase tested whether patients would show acquired equivalence and associate A2 with X2 and B2 with Y2, even though these particular stimulus pairings had never been trained.

We administered this acquired equivalence task to a group of nondemented elderly individuals with HA documented on neuroimaging ($n = 12$), a group of individuals with PD ($n = 12$), and appropriate matched controls ($n = 24$). We expected to find spared acquisition, but impaired transfer, in the individuals with HA; conversely, we expected slow acquisition, but spared transfer, in the Parkinson's patients.

RESULTS

Behavioral Results: Acquisition Phase

Figure 1 shows the total errors to criterion in the acquisition phase for each group. Analysis of variance (ANOVA) confirmed a significant effect of group,
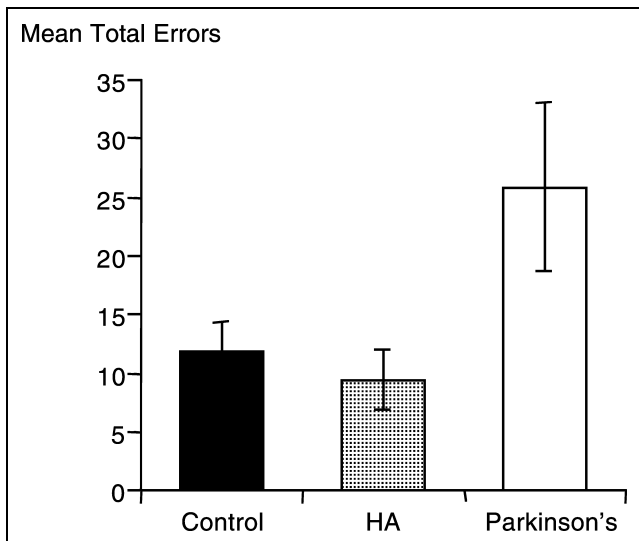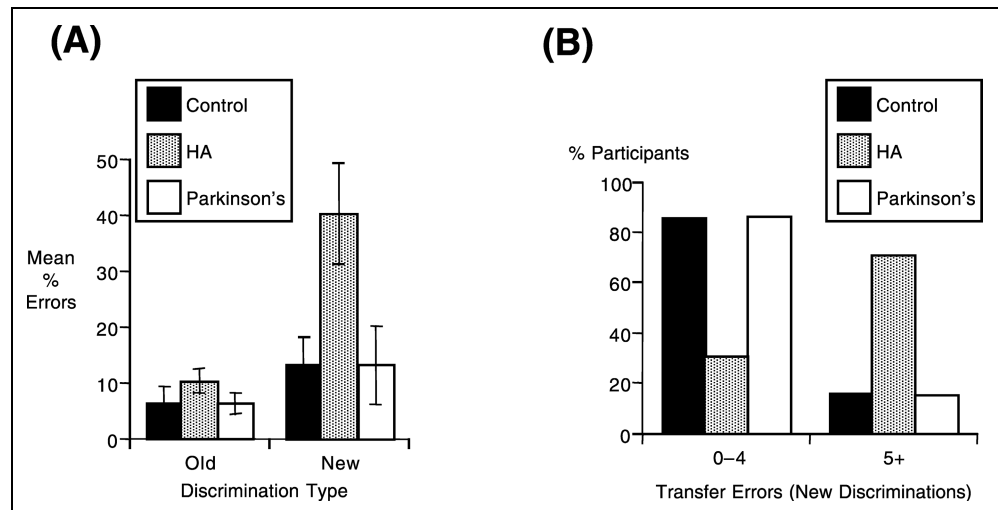
Figure 1. Total errors to criterion (±*SEM*) in the acquisition phase (Stages 1–3).

$F(2,45) = 4.04$, $p = .024$. Tukey's HSD tests revealed that the PD group made significantly more errors than either the control group ($p = .042$), or the HA group ($p = .038$); the control group and the HA group did not differ ($p > .500$).

In the control group, four participants completed Acquisition Stages 1 and 2 but failed to complete Acquisition Stage 3 within the maximum allowed trials. One participant in the HA group and one in the PD group showed this same pattern. Additionally, one HA and one PD participant failed to acquire any of the initial discriminations. In total, 20 of the control participants (83.3%) acquired all the initial discriminations, compared with 10 HA participants (83.3%) and 8 PD patients (66.7%). This completion rate did not differ among groups, $\chi^2(2) = 1.52$, $p > .50$.

Behavioral Results: Transfer Phase

Transfer performance was evaluated only in those participants who successfully mastered all the initial discriminations (i.e., who reached criterion in Acquisition Stage 3). In the transfer phase, subjects received test-only trials (no feedback) on the original discriminations trained in Stages 1–3 as well as the novel pairs.

One PD patient who had reached criterion in Acquisition Stage 3 subsequently went on to make 14 errors on the previously trained pairs in the transfer phase, more than four times as many errors as the PD group average; transfer data from this individual were excluded. Thus, transfer data were analyzed from 20 control, 10 HA, and 7 PD participants. As shown in Figure 2A, there were no significant differences between groups on performance on the previously trained pairs, ANOVA, $F(2,34) = .59$, $p > .50$.

Figure 2A also shows performance on the novel "transfer" pairs. There were significant differences between the groups, ANOVA, $F(2,34) = 5.17$, $p = .01$. Pairwise Tukey's tests confirmed significant differences between the control and HA groups ($p = .01$) but not between the control and PD groups ($p > .50$); the difference between HA and PD groups approached significance ($p = .059$). Among the control group data, there appeared to be a bimodal distribution, with 17 controls making 0–2 errors, and three controls making six or more errors. Using a pass–fail criterion of at most four errors, 17 controls (85%), 3 HA (30%), and 6 PD patients (85.7%) satisfied this criterion. Figure 2B shows these data; the group difference was significant, $\chi^2(2) = 10.64$, $p < .01$.

Given that the HA group made more errors on the transfer phase, it is possible that they simply forgot the Phase 1 information more quickly than the other two groups; if so, this should be reflected in a regression toward chance performance as the transfer phase

Figure 2. Transfer phase performance. (A) Mean percent errors (±*SEM*) on the old (previously trained) and new discriminations. (B) Percent of participants in each group who make few (<5) or many (≥5) errors on the new discriminations.

progressed. To investigate this possibility, we examined the transfer phase data, divided into three blocks of 16 trials (one example of each of the eight discriminations in each left–right arrangement). A repeated-measures ANOVA on total errors to the previously trained pairs across the three transfer blocks revealed no effect of group, $F(2,33) = 1.98$, $p = .15$, or block, $F(2,66) = .68$, $p > .50$, and no Group × Block interaction, $F(4,66) = .43$, $p > .50$.

DISCUSSION

This study predicted a double dissociation in learning deficits between individuals with HA and Parkinson's patients with basal ganglia dysfunction, using an acquired equivalence task. In the acquisition, PD patients were slower than controls or HA participants. Among individuals who had acquired all six discriminations in Phase 1, there were no group differences in transfer phase performance on previously trained pairs, indicating that all groups could retain this information when tested without feedback. However, PD patients were significantly worse than controls on the novel pairs, while HA participants did not differ from controls. Thus, the results of this study are consistent with a double dissociation: On this acquired equivalence task, basal ganglia dysfunction but not HA disrupts initial learning; conversely, HA but not basal ganglia dysfunction disrupts transfer.

One factor which might have contributed to the results is age: The HA group was significantly older than either the PD group or the control group (see Participants, below). It is possible that some effect of aging—independent of HA—could selectively impair transfer performance on this test. Additionally, HA is associated with cognitive decline, even in healthy elderly (Golomb et al., 1996). Although the HA group in the present study was screened for cognitive impairments, it is possible that mild cognitive decline—too subtle to be picked up in standard neuropsychological screening—did exist in this group relative to nonatrophied control participants. However, in a prior study, Collie et al. (2002) found that performance on the acquisition stages of the acquired equivalence task was impaired in older individuals with cognitive decline—but these individuals were not impaired at the transfer phase relative to age-matched healthy controls. This is in fact the opposite pattern from that observed in our HA group, which was unimpaired at initial learning but subsequently impaired at transfer to novel pairs. This suggests that cognitive decline alone cannot account for the pattern of results observed in our HA participants.

It is worth stressing that the amount of hippocampal injury in the HA group was quite limited. Prior studies examining pathologic changes affecting the hippocampus in older adults with minimal or mild cognitive impairment have demonstrated low densities of neuritic plaques and neurofibrillary change that are far less than that observed in patients with Alzheimer's disease (Price & Morris, 1999). Nevertheless, a large body of prior work demonstrates that mild HA based on subjective rating in cognitively intact elderly subjects correlates well with clinical measures of secondary memory performance (e.g., de Leon et al., 1997; Golomb et al., 1993) and can predict the subsequent emergence of dementia (de Leon et al., 1993). A validation of these ratings with respect to quantitative magnetic resonance (MR) volumetry of the hippocampus and other MT lobe structures has been reported (Convit et al., 1993; de Leon et al., 1993). Additionally, the cross-sectional cognitive correlations obtained using subjective ratings have been reproduced employing MR volumetry in a sample of cognitively normal older subjects selected using identical criteria to the current study (Golomb et al., 1994). The relationship of the subjective rating system to postmortem neuropathologic observations has also been published (Narkiewicz et al., 1993).

One clear limitation of the use of subjective ratings to assess HA is that it does not take into account age-related volume loss in other brain regions, such as the cortex (particularly prefrontal cortex). It is possible that shrinkage to one or more other brain regions may be confounded with HA, or may exacerbate its effects on behavior. Investigation of these possibilities remains a subject for further research.

However, the current findings are consistent with an earlier study showing that nondemented elderly individuals with mild-to-moderate HA are unimpaired at learning an eight-pair concurrent visual discrimination, but are severely impaired at a subsequent transfer stage involving a shift of irrelevant features (Myers et al., 2002). In both cases, it appears that mild HA does not impair the ability to learn simple stimulus associations. However, HA may affect "how" those associations are learned. Healthy individuals appear to learn these discriminations in a manner that supports subsequent generalization when familiar stimulus objects are presented in novel recombinations (as in the present task) or with altered features (as in the Myers et al., 2002 task). Hippocampal damage may cause individuals to learn the initial associations in a more specific way, making them less able to transfer when challenged to apply this learning in a new context (see also Myers & Gluck, 1996; Gluck & Myers, 1993).

The opposite pattern appears in Parkinson's patients. The Parkinson's patients are slower to learn the initial discriminations than either controls or HA participants; however, once they do, they can perform subsequent generalizations as well as controls. This finding is consistent with prior suggestions that the basal ganglia are important for incrementally acquired associative learning (e.g., White, 1997; Knowlton & Squire, 1993; Mishkin et al., 1984), as well as with prior studies suggesting that Parkinson's patients may differ from

controls in their ability to learn a probabilistic category learning task, lagging behind control performance even after 3 days of training (Shohamy, Myers, Onlaor, & Gluck, 2002).

One open question is why Parkinson's patients should show slower initial learning in Phase 1: Given that the hippocampal system is generally assumed to underlie declarative memory, could not the PD patients use this system to simply memorize the information in Phase 1? Our current data suggest that controls (with functioning hippocampal systems) do not simply memorize the six associations after a single exposure to each, but require several passes through the training set (refer Figure 1) and continue to make a small proportion of errors on this information during the transfer phase (refer Figure 2). Thus, it would appear that healthy controls tend to approach Phase 1 not by memorizing the association pairs, but as an incremental learning task, presumably mediated by the basal ganglia. This would be consistent with functional imaging studies showing basal ganglia activation (and hippocampal deactivation) during learning of related association tasks (e.g., Poldrack et al., 2001). PD patients may be forced to adopt the alternate strategy of relying on their hippocampus, and the task may simply be too hard (too many associations with too much overlap) for this strategy to be efficient, or they may continue to rely on their damaged basal ganglia. In either case, their learning would be impaired.

The current data do not distinguish these possibilities. The obvious conclusion here is that simply noting "how fast" a subject learns a task does not necessarily provide any information about "how" the subject is encoding that information—or which brain structures are being used (see also Shohamy et al., 2002). This is one reason why transfer performance may be a more informative index than simply measuring trials to criterion. Functional imaging studies of patients and controls would also be helpful in this context.

The current findings nevertheless suggest that while the basal ganglia are indeed critical for simple associative learning, the hippocampal system is also normally involved in this type of learning—even though its contribution may not be strictly required. The hippocampal contribution may alter how these associations are learned, and whether the learning can subsequently generalize when familiar information is presented in novel contexts.

To our knowledge, this study represents the first time that such a dissociation between basal ganglia and hippocampal function has been observed within a single task. Further studies are obviously indicated to determine whether this proposed double dissociation holds generally across a variety of acquisition and transfer tasks. Nevertheless, this initial result suggests that the two brain systems do make distinct contributions to learning, and that these contributions can be differentially affected by damage to one or the other brain system.

METHODS
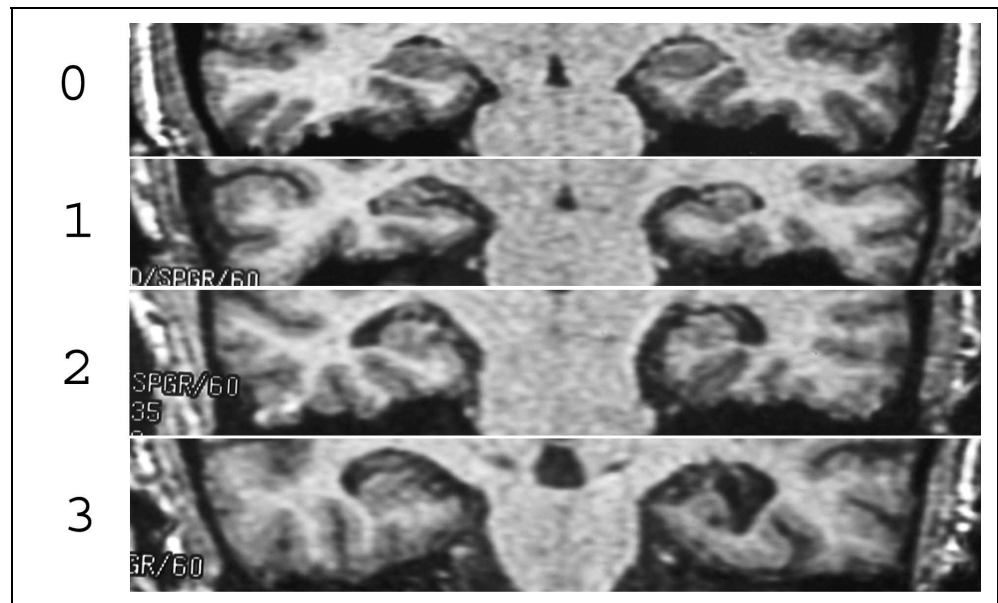
Participants

Hippocampal Atrophy

Twenty-four elderly individuals (age range 53–83 years) were recruited via the NYU Aging and Dementia Research Clinic in New York City. These individuals were participating in other ongoing research at the clinic that involved neuropsychological assessment and neuroimaging. These participants were screened for the absence of dementia, depression, or other neurological or psychiatric conditions that could contribute to memory impairment. All participants were required to score at least 26 on the Mini-Mental State Exam (Folstein, Folstein, & McHugh, 1975), as an estimator of intact cognitive function and absence of dementia; the group mean was 28.96 (SD 1.3).

These participants were also given a comprehensive cognitive battery (Kluger, Ferris, Golomb, Mittelman, & Reisburg, 1999). This battery included the Global Dementia Scale (GDS; Reisberg, Ferris, de Leon, & Crook, 1982), which rates subjects on a seven-point scale, with a score of 4 or higher indicating progressive degrees of dementia. All subjects in the current study were required to score 3 or lower, consistent with a lack of dementia. Additionally, the cognitive battery included the paragraph delay recall test (PDR) of the Guild Memory Test (Gilbert, Levee, & Catalano, 1968); impairment on this test has previously been shown to correlate with HA (Golomb et al., 1994) and to predict cognitive decline in nondemented elderly (Kluger et al., 1999).

Each participant also received MR imaging via a GE 1.5 T MR scanner including a 3-D spoiled gradient recalled acquisition (SPGR) sequence. Diagnostic screening excluded any evidence of infarct, hydrocephalus, intracranial mass, or moderate to severe white matter lesions. From the coronal SPGR scan, 3 mm axial reformats were created parallel to the long axis of the hippocampus. Coronal 1.3 mm reformats were also generated perpendicular to the long axis of the hippocampus.

Based on neuroimaging assessment, participants were divided into two groups based on the presence (HA) or absence (noHA) of HA, following the procedure outlined by Myers et al. (2002). The coronal temporal lobe images were used to supplement the axial images in the final assignment of an HA score. Figure 3 shows example images for four subjects to illustrate gradations of HA. After rating, the HA group contained 12 individuals: four females and eight males; the noHA group also contained 12 individuals: six females and six males. Participants in the HA group averaged 72.3 years of age (SD 7.2), while the noHA group averaged 63.9 years (SD 7.4); this difference was

Figure 3. Coronally reformatted T1-weighted MRI images (SPGR) of four subjects highlighting the inferior temporal lobe anatomy to illustrate gradations of HA. 0 = no atrophy (noHA), 1 = questionable or mild HA, 2 = mild to moderate HA, 3 = moderate to severe HA.

statistically significant [independent-samples t test, $t(22) = 2.80$, $p = .01$]. Average GDS score in the HA group was 2.3 (SD 0.7); average GDS score in the noHA group was 2.2 (SD 0.6), not significantly different from the HA group, $t(22) = .66$, $p > .50$. HA and noHA groups likewise did not differ on the PDR [HA mean score 7.0, SD 3.8; noHA mean score 9.0, SD 3.26; $t(22) = 1.45$, $p = .16$], or the Mini-Mental State Exam [HA mean score 29.3, SD 1.1; noHA mean score 28.7, SD 1.4; $t(22) = 1.10$, $p = .28$].

Parkinson's Disease

Twelve individuals (five females, seven males) with PD were recruited through the St. Barnabus Movement Disorders Clinic (West Orange, NJ), and from patients under care for PD at Columbia Presbyterian Hospital in New York City. Age in these patients ranged from 53 to 81 years (mean 66.6, SD 8.3). Degree of parkinsonism, as assessed by the Hoehn–Yahr (1967) scale, ranged from 2.0 (mild) to 3.0 (moderate). Duration since initial diagnosis of the disease ranged from 1 to 15 years (mean 6.4, SD 3.8). All but one of the Parkinson's patients were on dopaminergic medication (Sinemet) at the time of testing; the remaining patient uses holistic approaches to manage her symptoms. No patients were on anticholinergic medication.

All PD patients were required to score at least 26 on the Mini-Mental State Exam, as an estimator of intact cognitive function and absence of dementia; the group mean was 29.0 (SD 1.0). Only one patient scored below 28; this patient scored 27 and was the oldest in the group, at 81 years, but was otherwise comparable to the rest of the PD group in neuropsychological assessment and behavioral performance. Patients were also screened for absence of dementia, and required to score below 15 on the Beck Depression Inventory—II

(BDI-II; Beck, 1987); group mean was 6.3 (SD 3.4). The referring neurologist also screened patients for absence of other neurological or psychiatric disorder other than PD.

Twelve healthy control participants (seven females, five males) were recruited through the Memory Disorders Project at Rutgers University. These individuals ranged in age from 51 to 77 years (mean 66.1, SD 8.4). This did not differ significantly from the Parkinson's group [independent-samples t test, $t(22) = .15$, $p > .50$]. All healthy controls were screened for the absence of neurological or psychiatric disorders, including dementia, depression, and PD. The control group averaged 28.5 (SD 1.2) on the Mini-Mental State Exam, and 5.3 (SD 4.4) on the BDI-II. Neither measure differed significantly from the Parkinson's group (independent-samples t tests, all $p > .100$).

Control Group

No statistically significant differences were found between the noHA group or the healthy control group on any demographic, neuropsychological, or behavioral measures (all $p > .50$). Accordingly, data from these two groups were pooled into a single control group ($n = 24$). This control group had an average age of 65.0 years (SD 7.8), which differed significantly from the HA but not the PD group, ANOVA, $F(2,43) = 4.32$, $p = .02$; Tukey's post hoc pairwise tests revealed significant differences between control and HA groups ($p = .03$), but not between control and PD or HA and PD groups ($p > .10$).

Apparatus

Behavioral testing was automated on a Macintosh PowerBook 520c or 1400cs laptop computer with a color screen, using software programmed in the SuperCard

language. Testing took place in a quiet room, with the participant seated in front of the computer at a comfortable viewing distance. The keyboard was masked except for two keys, labeled "LEFT" and "RIGHT," which the participant could press to record a response.

Stimuli

Four drawings of faces (man, woman, girl, boy) served as the antecedent stimuli. The boy and woman had yellow hair while the girl and man had brown hair. Thus, each antecedent had three obvious, binary-valued features: age (adult vs. child), gender (male vs. female), and hair color (blond vs. brunette); each antecedent shared exactly one feature with each other antecedent. For each participant, the four face drawings were randomly assigned to be antecedents A1, A2, B1, and B2.

The consequents were four drawings of a fish colored red, orange, pink, and purple. For each participant, the colored fish were randomly assigned to be the consequents X1, X2, Y1, Y2.

The antecedents and consequents all appeared about 1 in. tall on the computer screen, with the subject seated at a comfortable viewing distance (approximately 18 in.).

Procedure

All participants signed statements of informed consent before the initiation of any behavioral testing. All research procedures conformed to the regulations established by the Federal Government and by Rutgers University.

At the start of the experiment, the following instructions appeared on the screen: "Welcome to the experiment. You will see drawings of people who each have some pet fish. Different people have different kinds of fish. Your job is to learn which kinds of fish each person has. At first, you will have to guess." The experimenter read these instructions aloud to the participant and then clicked the computer mouse button to begin the acquisition phase.
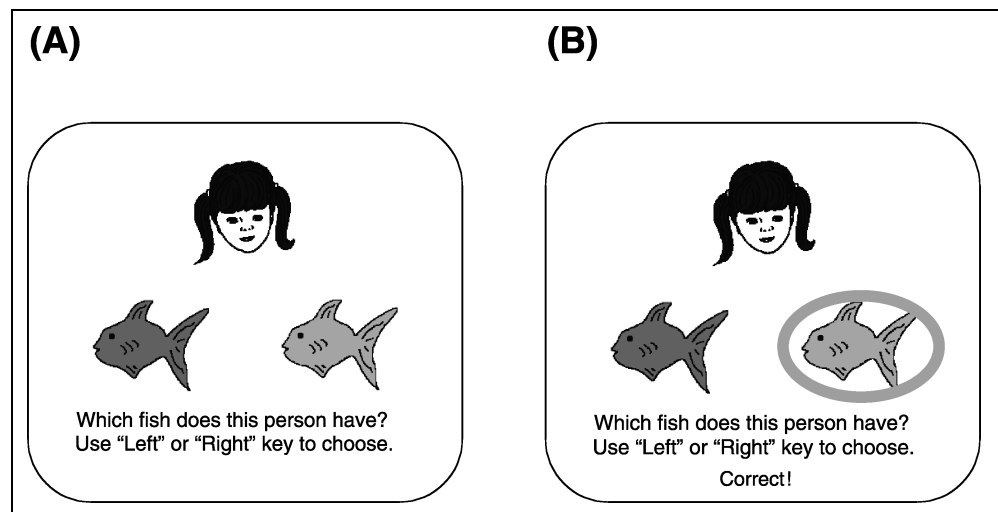
On each trial, the screen showed an antecedent (face) and two consequents (fish), as shown in Figure 4A, along with the prompt: "Which fish does this person have? Use the LEFT or RIGHT key to choose." The participant responded by pressing one of the two labeled keys. The selected consequent (fish) was circled, and corrective feedback was given (Figure 4B). In the case of an incorrect response, an alert beep also sounded.

There were three stages of acquisition, each with increasing numbers of trial types as shown in Table 1. Since the consequents could appear in either left–right ordering, there were 4 trial types in Acquisition Stage 1, 8 in Acquisition Stage 2, and 12 in Acquisition Stage 3. Each stage consisted of a maximum of eight blocks, each consisting of one instance of each trial type in random order. Acquisition Stages 1 and 2 terminated early if the participant reached criterion performance of eight consecutive correct responses; Acquisition Stage 3 terminated early if the participant reached criterion performance of 12 consecutive correct responses. The start of a new training stage was not signaled to the participant.

At the conclusion of Acquisition Stage 3, the following instructions appeared: "Good! In this part of the experiment, you will need to remember what you have learned so far. You will NOT be shown the correct answers. At the end of the experiment, the computer will tell you how many you got right. Good luck!"

The transfer phase followed. There were 16 trials: all six trial types from the acquisition phase plus the two new test trial types (A2→X2 or Y2, and B2→X2 or Y2), with the consequents in each possible left–right ordering. On each trial, the screen showed one face and two fishes; the fish chosen by the participant was circled, but no corrective feedback was given. Trial order was random for each participant.

Figure 4. Example screen events during one trial. (A) Stimuli appear. (B) Participant responds and corrective feedback is given.

On each trial, the computer recorded the antecedent and consequents shown, as well as the desired and actual responses.

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