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Memory interference in multiple sclerosis

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Abstract

To explore verbal memory impairments associated with multiple sclerosis (MS), we compared proactive and retroactive interference effects on the California Verbal Learning Test (CVLT; Delis et al., 1987) in a sample of 83 community-residing individuals with MS and 80 healthy participants. Individuals with MS demonstrated normal accumulation of proactive interference (PI), but attenuated release from PI relative to healthy individuals. Furthermore, accumulation of retroactive interference (RI) at short-delay free recall (SDFR) was intensified for those with MS as compared to healthy participants. Interestingly, accumulation of RI predicted long-term memory (LTM) only for participants with MS. These findings suggest that individuals with MS may experience particular difficulty when required to use semantic properties of information flexibly to facilitate verbal LTM. (*JINS*, 2005, *11*, 737–746.)

Keywords: Verbal learning, Memory disorders, Semantics, Neuropsychology, Cognition, Demyelinating autoimmune diseases

INTRODUCTION

Diverse neurocognitive deficits affect approximately fifty percent of individuals with multiple sclerosis (MS) (DeSousa et al., 2002; Rao et al., 1991). Long-term memory (LTM) impairment is one of the most consistently reported findings in MS (Beatty, 1993; Beatty et al., 1989; Bobholtz & Rao, 2003; DeLuca et al., 1998; Rao, 1986; for a review see Thornton & Raz, 1997), and appears to be an ecologically valid indicator of daily functioning (Higginson et al., 2000). Over the past decade a clearer understanding of LTM function in MS has emerged. Initially, LTM deficits in MS were attributed to a selective retrieval failure (e.g., Armstrong et al., 1996; Rao et al., 1989, 1993), but recent evidence has also implicated acquisition and encoding processes (DeLuca et al., 1994, 1998; Demaree et al., 2000; Gaudino et al., 2001). Variability in the observed magnitude of LTM impairment (Thornton & Raz, 1997) may reflect the diversity of memory operations that have been investigated (e.g., Thornton et al., 2002), as well as disease-related variables (Arnett et al., 1999a, 1999b; Kessler et al., 1992; Thornton & Raz, 1997).

To further characterize MS-related memory functioning, the present study evaluates the contributions of interference to LTM impairment. The normative pattern of loss of information from the memory store (Brown, 1958; Peterson & Peterson, 1959) often reflects interference between previously learned and new information (Keppel & Underwood, 1962). Proactive interference (PI) occurs when prior learning impedes retention of subsequent materials, and retroactive interference (RI) refers to the decrement in retention of previously learned information caused by subsequent learning (Postman & Underwood, 1973). Interference is heightened when old and new information is taxonomically related. During encoding, interference between related words is assumed to reflect active use of the information's semantic properties (Wickens, 1970).

One paradigm used to elicit PI presents lists of words from the same semantic category, for one learning trial each. This is followed by a final trial of words drawn from a new

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Table 1. Selected studies of proactive interference effects

Investigators	Sample	Method	Results on Indices of Proactive Interference
Blusewicz et al., 1996	Chronic alcoholism *vs.* controls	Wickens	Increased accumulation, normal release
Dobbs et al., 1989	Review of 3 studies old *vs.* young	Wickens	Mixed: equivocal evidence of increased accumulation
Belleville et al., 1992	Probable DAT vs. controls	Wickens	Reduced accumulation and release
Cushman et al., 1988	Probable DAT *vs.* controls	Wickens	Reduced accumulation and release
Randolph et al., 1992	Schizophrenia *vs.* controls	Wickens	Reduced release
Squire, 1982	Korsakoff's *vs.* affective disorders *vs.* controls	Wickens	Reduced release in KS, other groups comparable
Winocur et al., 1981	Korsakoff's *vs.* controls	Wickens	Reduced release in KS
Beatty et al., 1989	MS *vs.* controls	Wickens	Comparable accumulation and release
Rao et al., 1993	MS *vs.* controls	Wickens	Comparable accumulation and release
Johnson et al., 1998	MS *vs.* controls	Wickens	Comparable accumulation and release
Sagar et al., 1991	Parkinson's disease *vs.* controls	Wickens	Comparable accumulation and release
Goldstein et al., 1989	TBI *vs.* controls	Wickens	Comparable accumulation and release
Numan et al., 2000	TBI *vs.* controls	CVLT	Reduced accumulation and release
Vanderploeg et al., 2001	TBI *vs.* controls	CVLT	Reduced accumulation and release
Kareken et al., 1996	Schizophrenia *vs.* controls	CVLT	Reduced accumulation (not release)
Sitskoorn et al., 2002	First episode schizophrenia *vs.* controls	CVLT	Reduced release
McDonald et al., 2001	Frontal *vs.* temporal resection patients	CVLT*	Frontal resection patients showed reduced release

Note. AD = Alzheimer's disease; CVLT = California Verbal Learning Test; DAT = Dementia of the Alzheimer's Type; HD = Huntington's disease; KS = Korsakoff's syndrome; MS = multiple sclerosis; TBI = traumatic brain injury.
*Release from Proactive Inference (RPI) was calculated using a simple unshared : shared ratio.

semantic category. Performance on this Wickens (1970) type paradigm declines linearly over learning trials as interference between semantically related items increases. Recall rebounds ("release" from PI) when the new semantic category is introduced. In clinical samples, intensified accumulation (but relatively preserved release) of PI has been associated with diminished working memory (WM) capacity (e.g., Blusewicz et al., 1996), while normal accumulation and poor release from PI has been attributed to concurrent mnemonic and executive deficits (Cermak et al., 1974; Randolph et al., 1992; Squire, 1982).

Despite high lesion burden in frontal regions (Sperling et al., 2001) and impairment on both "frontal" tasks of WM (Pelosi et al., 1997) and executive abilities (Foong et al., 1997), patients with MS have shown normal accumulation and release from PI on Wickens-type paradigms (Beatty et al., 1989; Johnson et al., 1998; Rao et al., 1993). However, some investigators suggest that this paradigm may be insensitive to subtle interference abnormalities (Dobbs et al., 1989). Given that divergence between MS patients and controls is most evident under cognitive challenge (D'Esposito et al., 1996; Legenfelder et al., 2003), the potential insensitivity of the Wickens paradigm might obscure subtle group differences in susceptibility to interference. The California Verbal Learning Test (CVLT; Delis et al., 1987) has also been used to evaluate interference (Kramer & Delis, 1991). The CVLT differs from Wickens-type paradigms in that accumulation and release from PI occur simultaneously. Specifically, List B of the CVLT concurrently presents items from the same semantic categories as List A, inducing PI, and items from new semantic categories, inducing release from PI. Compared to Wickens-type paradigms, the simultaneous processing inherent to the CVLT may increase its sensitivity.

To investigate this possibility, the terms *Wickens, interference, PI, release from PI, clinical, verbal memory, memory impairment*, and *CVLT* were entered into PsychInfo and Medline. Studies were retrieved if they included a Wickens-type paradigm with repeated presentations of a supraspan word list and a final release trial, or if CVLT interference effects were calculated using some comparison of shared and unshared items.[a]

Table 1 shows that abnormal interference effects on Wickens-type paradigms were reported in some, but not all, clinical samples with expected memory dysfunction. Specifically, patients with Parkinson's disease, traumatic brain injury, and MS did not differ from healthy controls in susceptibility to or release from PI. While studies have not systematically examined inference effects on the CVLT in MS, abnormal PI effects appear widespread on the CVLT in other patient groups. This pattern suggests that the CVLT may be more sensitive than Wickens-type paradigms to these effects.

Although increased susceptibility to PI has not been detected in MS using Wickens-type paradigms, susceptibility to PI appears to be an important determinant of verbal WM in healthy individuals (Lustig et al., 2001). Furthermore, as some evidence indicates that WM capacity con-

[a]Those studies using a traditional Brown-Peterson short-term memory task with presentation of groups of three or four words followed by a counting or sequencing distracter were not evaluated, as interference between semantically related words was not the primary focus of those studies.

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tributes to LTM dysfunction in MS (e.g., DeLuca et al., 1994; Litvan et al., 1988; Thornton et al., 2002), it may be fruitful to directly evaluate MS-related interference effects and their relationship to LTM with a more sensitive measure, such as the CVLT.

An interesting parallel line of inquiry is whether MS participants experience full recall recovery when interference ceases (i.e., preserved release from PI). Individuals with MS appear more likely to generally encode semantic information, and their memory lacks both semantic (Carroll et al., 1984) and contextual (Thornton et al., 2002) distinctiveness. If the loss of semantic distinctiveness is robust, it should generalize across memory paradigms. Consequently, MS participants might show diminished sensitivity to taxonomic shifts under demanding conditions, as evidenced by reduced release from PI. Finally, while individuals with MS have shown RI on the CVLT (Kessler et al., 1992; Troyer et al., 1996), these studies did not include control groups, precluding the evaluation of whether those with MS were disproportionately susceptible to this type of interference.

Based on the aforementioned rationale, we investigated whether individuals with MS exhibit differential sensitivity to the semantic properties of stimulus items and abnormal interference effects on the CVLT. Furthermore, we assessed the extent to which interference predicts delayed memory in MS. This later issue is interesting in light of the relationship between susceptibility to interference on the CVLT and LTM dysfunction reported in other clinical samples (Vanderploeg et al., 1994). Given MS-related losses in semantic and contextual distinctiveness (e.g. Carroll et al., 1984; Thornton et al., 2002) and WM deficits (e.g. Litvan et al., 1988), individuals with MS might be particularly susceptible to PI and RI on the CVLT, a vulnerability that would negatively affect delayed memory.

METHODS

Research Participants

All participants were fully informed of the nature of the study and consented to participate. Eighty-three participants with MS were recruited from a local registry of the National Multiple Sclerosis Society, and had received a diagnosis of multiple sclerosis from their physicians. Participants were recruited from the community, not from medical facilities or treatment programs. Exclusion criteria included: age over 60 years, self-reported medical or psychiatric disorders other than MS that would be expected to affect memory (e.g., active substance abuse, seizure disorders, head injury), and sensory deficits precluding participation in the assessment. Multiple sclerosis participants were well matched to a sample of 80 healthy controls who had volunteered from community sites and a university participant pool (see Table 2). Previously, we reported on a subsample of the current participants (Thornton et al., 2002).

Participants with MS completed a questionnaire addressing disease onset, course, and severity. Seventy-two percent of participants reported a diagnosis of definite MS from their physician, with fewer individuals reporting diagnoses of probable (18%) or possible (10%) MS. Those participants reporting possible MS described at least one discrete symptom exacerbation, as well as neurological signs consistent with MS.

Table 2. Demographic variables

	MS			Controls		
Variable	%	*M*	*SD*	%	*M*	*SD*
Age		42.5	8.4		40.8	7.2*
Education		15.1	2.6		14.7	2.0*
Gender (% female)	71			74		
BDI Score[a]		10.3	7.0		4.3	3.7**
Partial BDI Score[a]		6.6	5.4		2.7	2.7**

Note. *$p = ns$; **$p < .001$.
[a]Beck Depression Inventory (BDI) data were not available for 1 individual with MS and 4 individuals in the control sample.

Participants were classified on the basis of self-reported symptom onset and course in the following manner: 27% reported a sudden onset of symptoms with subsequent asymptomatic remission, 46% reported a fluctuating course with periods of remission, and 27% described a slow worsening of symptoms. Furthermore, most participants (70%) had not experienced symptom exacerbation within the past month. Using a self-report rating scale containing items comparable to those on the Expanded Disability Status Scale (EDSS; Kurtzke, 1965), MS participants also rated their own level of physical disability (EDSS equivalent $x = 3.1$, $SD = 2.2$). This self-rating instrument has been associated ($r = .82$, $p < .001$) with experimenter ratings of ambulation (Thornton et al., 2002), and other researchers have reported strong correlations between self-ratings and neurological exams in MS patients (Solari et al., 1993; Verdier-Taillefer et al., 1994).

Lastly, participants completed the Beck Depression Inventory (BDI, Beck et al., 1961), a 21-item self-report questionnaire of a broad range of depressive symptoms (see Table 2). This measure was scored using the traditional method and a "partial" scoring technique eliminating items directly assessing physical functioning and appearance (items 14, 15, 17, 20, & 21, see Thornton et al., 2002). This modification addresses the overlap between MS symptoms and the somatic features of depression tapped by the BDI (consistent with the recommendations of Minden & Schiffer, 1990). Using traditional BDI scores, there was a trend towards higher levels of depressive symptoms in those reporting current exacerbation of neurological symptoms [$F(3,67) = 2.4$, $p = .08$], but when partial BDI scores were used this difference was nonsignificant [$F(3,67) = 1.9$, $p = ns$]. No other differences in traditional or partial BDI scores were found for self-reported MS subtype, disease onset and course, or physical disability level (all ps $= ns$).

Procedures

The California Verbal Learning Test (CVLT; Delis et al., 1987) was administered as part of larger neuropsychologi-

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cal batteries. The CVLT is a verbal list-learning test in which a series of 16 shopping items (List A) is repeated over five learning trials. Participants are directed to recall as many items as they can after each trial. After the learning trials, a new 16-item shopping list (List B) is recited once, and participants are required to recall as many of the new items as they can remember. Then, without repetition of the list by the administrator, participants are directed to recall items from List A (Short Delay Free Recall; SDFR). This is followed by a semantic cueing trial (e.g., "Tell me the items from the first list that were herbs and spices"). Following a 20-minute delay, free and cued recall trials are administered again. Next, participants are required to discriminate List A items ("yes") from List B items and other distracters ("no") presented in a recognition format.

Interference

Interference measures were calculated based on the procedure outlined by Kramer and Delis (1991). One-half of the items on both List A and List B are "shared" (i.e., they are members of the same semantic categories—fruits and spices/herbs), while the other half of the items are "unshared" (they are members of distinct semantic categories—tools and clothing for List A, fish and kitchen utensils for List B). Recall of shared and unshared items on the first trial of List A (List A-1) is compared with recall of shared and unshared items on List B. Reduced recall of shared items on List B relative to shared item recall of List A-1 reflects accumulation of PI, while improved recall of unshared items on List B relative to unshared items on List A-1 indicates release from PI. Retroactive interference is reflected by decreased post-interference recall (SDFR) relative to Trial 5 of List A, with greater declines expected for shared than unshared item recall.

In accordance with Kramer and Delis (1991), weighted averages of shared and unshared words recalled on Trial 1 were calculated for each participant in order to control for proportion of shared words recalled across trials and corresponding differences in the opportunity to accumulate interference (weighted average of shared category items = number of items recalled on Trial 1 × [total number of shared category items recalled on Trials 1–5 ÷ total number of words recalled on Trials 1–5]). Similarly, in assessing RI, SDFR of shared and unshared items were compared to weighted averages of List A-5 recall to control for the relative proportion of shared and unshared items learned.

Individual participant interference scores

To evaluate individual differences in interference effects and the relationship between these effects and LTM, we created individual difference scores. The importance of controlling for baseline performance to avoid possible confounds when calculating trial-to-trial difference scores to represent interference effects has been previously noted (Torres et al., 2001). Given that the items on the CVLT are of equivalent recall difficulty, the average of shared and unshared items recalled on the baseline trial (List A-1 for PI and List A-5 for RI) provides the most stable estimate of how many shared or unshared items each individual *should* be able to recall on any trial. This estimate can be compared with actual shared or unshared item recall on Trial B and SDFR for PI and RI, respectively. We therefore calculated difference scores in the following manner:

$$\text{Accumulated PI} = \frac{\left[\frac{(\text{Shared Trial A-1 Recall} + \text{Unshared Trial A-1 Recall})}{2}\right] - \text{Trial B Shared Recall}}{\left[\frac{(\text{Shared Trial A-1 Recall} + \text{Unshared Trial A-1 Recall})}{2}\right]}$$

$$\text{Release from PI} = \frac{\left[\frac{(\text{Shared Trial A-1} + \text{Unshared Trial A-1})}{2}\right] - \text{Trial B Unshared Recall}}{\left[\frac{(\text{Shared Trial A-1} + \text{Unshared Trial A-1})}{2}\right]}$$

$$\text{Accumulated RI} = \frac{\left[\frac{(\text{Shared Trial A-5 Recall} + \text{Unshared Trial A-5 Recall})}{2}\right] - \text{SDFR Shared Recall}}{\left[\frac{(\text{Shared Trial A-5 Recall} + \text{Unshared Trial A-5 Recall})}{2}\right]}$$

These calculations reflect relative rather than absolute interference-related changes in recall. The rationale for using relative interference scores is derived from the need to account for general LTM ability. Absolute (raw) interference difference scores have the inherent disadvantage of being strongly influenced by baseline recall. For example, participants with higher List A-1 recall will produce greater raw interference scores merely because of their strong initial recall. In contrast, individuals with poor List A-1 recall truncate the extent to which their recall will decline when under the influence of interference. Consequently, relative interference scores accounting for initial recall level were constructed in the same manner as retention scores, which have been used as an index of forgetting in other research paradigms.

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Learning strategies

As the CVLT possesses a latent semantic structure, we assessed the recognition and use of this structure with the semantic clustering index. As increased recall itself results in increased clustering, the index was calculated according to an observed ÷ expected ratio as described in the manual (see Delis et al., 1987). We also examined other aspects of learning, including rates of perseverations and intrusions, recall consistency, and recognition discriminability to further characterize this sample.

RESULTS

Overall Verbal Memory

Multivariate analysis of variance (MANOVA) indicated that MS participants recalled significantly fewer items across all five learning trials [$F(1,159) = 16.3, p < .001$], at short-delay free recall [$F(1,159) = 18.9, p < .001$] and cued recall [$F(1,159) = 17.6, p < .001$], and at long-delay free recall [$F(1,159) = 12.2, p \leq .001$] and cued recall [$F(1,159) = 18.7, p < .001$]. Although there were no group differences in semantic and serial clustering, participants with MS showed reduced middle region recall [$F(1,159) = 5.5, p < .05$] and lower recall consistency [$F(1,159) = 21.5, p < .001$]. In addition, MS participants generated more perseverations during free and cued recall trials [$F(1,159) = 4.4, p < .05$] and more cued recall intrusions [$F(1,159) = 6.3, p < .05$]. Lastly, recognition discriminability was lower for MS participants [$F(1,159) = 7.9, p < .01$].

Interference Analyses

For each interference analysis presented next, data was evaluated for distributional normality. In the PI interference data, this inspection revealed the presence of seven participants (1 MS and 6 controls) who generated scores in one or more of the recall trials that were either outlying or extreme (at least two *SD*s from the mean). As the scores of these individuals approached either the floor or ceiling of each recall condition, their inclusion would constrain recall associated with PI. In addition, their removal improved the distributional properties of List B recall, reducing skew by approximately half a standard deviation with minimal effect on kurtosis. Consequently, the seven participants were removed from the PI analyses reported later.

In the RI data, recall scores of one MS and three control participants were identified as outlying or extreme. (One of these controls had also been eliminated from the PI analyses). Removal of these individuals improved the distribution of short-delay recall scores, reducing skew by half a standard deviation with minimal effect on kurtosis. These four outliers were therefore removed from the RI analyses reported later, as well as an additional control participant who was missing SDFR data.[b]

Proactive interference effects

Proactive interference effects were analyzed using a $2 \times 2 \times 2$ mixed factorial analysis of variance (ANOVA), with List (List A-1 or List B) and category (shared or unshared) as within-subject variables, and group (MS or controls) as the between-subjects factor. A significant main effect for group [$F(1,154) = 6.0, p < .05$] reflected superior overall item recall in the control group. Both groups remembered fewer items on List B than List A-1 [$F(1,154) = 21.1, p < .001$], and all participants demonstrated accumulation of PI and release from PI [$F(1,154) = 268.7, p < .001$]. More importantly, the analyses revealed a List × Category × Group interaction [$F(1,154) = 4.1, p < .05$] indicating differential recall patterns across the PI factors for participant groups. The divergent PI pattern was decomposed by examining group differences in (1) recall of *shared* items in List A-1 *versus* List B, (2) recall of *unshared* items in List A-1 *versus* List B, as well as (3) recall of *shared versus unshared* items on List A-1, and (4) recall of *shared versus unshared* items on List B, using the original mixed factorial model.

In terms of accumulation of PI across the Lists, both groups showed a similar recall decrement for shared items from List A-1 to List B [$F(1,154) = 215.5, p < .001$], that did not interact with group [$F(1,154) = 0.3, p = ns$]. Both groups also made significant gains in recall of unshared items from List A-1 to List B [$F(1,154) = 54.0, p < .001$], demonstrating release from PI. Interestingly, this result was moderated by a significant List × Group interaction [$F(1,154) = 4.7, p < .05$]. Figure 1 shows that, relative to control participants, MS participants exhibited attenuated gains in their recall of unshared items from List A-1 to List B.

The group differences in PI effects were further defined by the number of shared and unshared items recalled on each list. On the initial List A-1 trial, unshared items were more readily recalled than shared items [$F(1,154) = 8.5, p < .01$]; this pattern did not vary with Group [$F(1,154) = .7, p = ns$]. The superior recall of unshared items persisted through List B, becoming even more compelling [$F(1,154) = 318.4, p < .001$]. In terms of differential release from PI, the List B data revealed that control participants showed greater facility in the recall of unshared relative to shared items than did MS participants [$F(1,154) = 5.6, p < .05$]. Indeed, the List B data of Figure 1 shows that control participant recall was superior only for the unshared items [$F(1,154) = 9.9, p < .01$], and not for the shared items [$F(1,154) = .5, p = ns$]. This differential pattern of shared

[b]Although elimination is not an uncommon treatment for outliers, numerous authors (e.g., Allison & Gorman, 1993) suggest running analyses with and without outliers to evaluate their effect on the results. With the exception of stabilizing a trend towards greater susceptibility to RI in our MS participants, the removal of these outliers did not affect the direction or significance of our results for PI and RI analyses.

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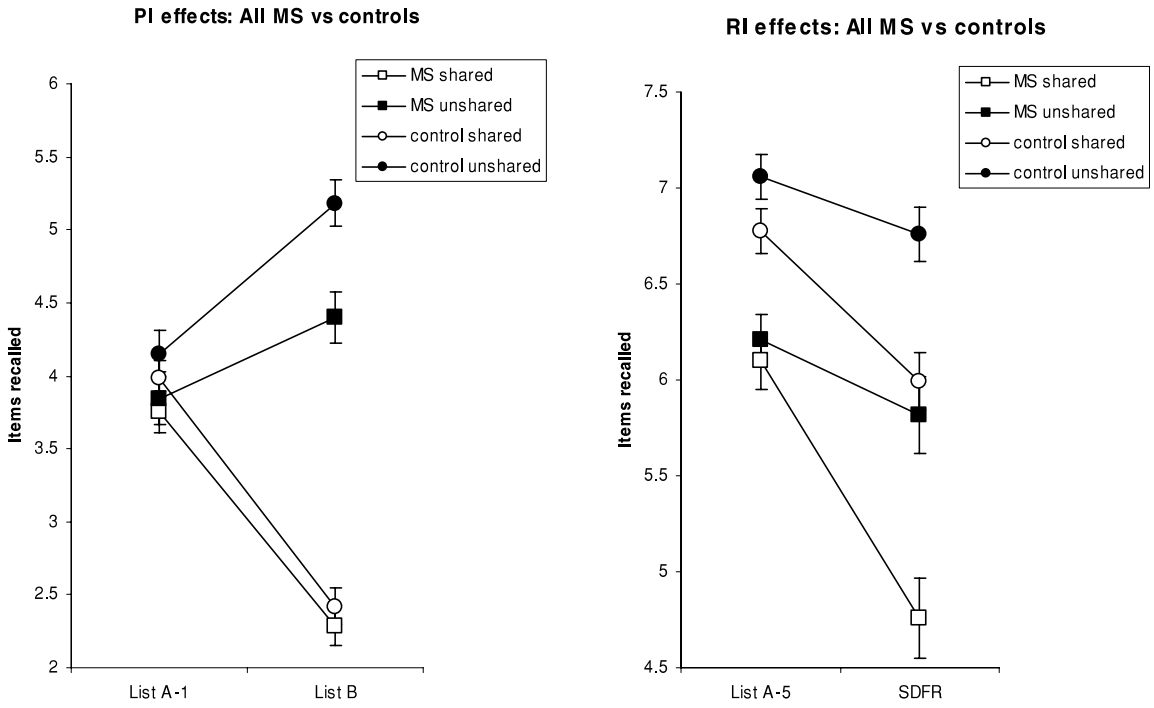

Fig. 1. Interference effects in the full multiple sclerosis (MS) sample. (PI = proactive interference; RI = retroactive interference; SDFR = short-delay free recall).

versus unshared recall provides further evidence for attenuated release from PI in MS participants.

Retroactive interference effects

Retroactive interference effects were analyzed using a 2 × 2 × 2 mixed factorial ANOVA, with List (List A-5 or SDFR) and category (shared or unshared) as within-subject variables, and group (MS or controls) as the between-subjects factor. A significant main effect for group [$F(1,156) = 24.6$, $p < .001$] indicated diminished recall on both trials for MS participants. The List × Category interaction [$F(1,156) = 45.7$, $p < .001$] reflected the presence of RI effects for both groups. More importantly, a List × Category × Group interaction [$F(1,156) = 4.6$, $p < .05$] showed differential recall patterns across RI factors between participant groups. This interaction was decomposed by evaluating group differences in (1) recall of *shared* items in List A-5 *versus* SDFR, (2) recall of *unshared* items in List A-5 *versus* SDFR, as well as (3) recall of *shared versus unshared* items on List A-5, and (4) recall of *shared versus unshared* items on SDFR, using the original mixed factorial model.

A substantial decrement in recall of shared items from List A-5 to SDFR [$F(1,156) = 120.1$, $p < .001$] was observed for all participants, but this result was moderated by a significant List × Group interaction [$F(1,156) = 8.2$, $p < .01$]. Figure 1 illustrates that compared to control participants, those with MS experienced a more precipitous decline in recall of shared items from List A-5 to SDFR. In terms of unshared item recall, both groups showed a decline from List A-5 to SDFR [$F(1,156) = 15.7$, $p < .001$] that did not differ between groups [$F(1,156) = .3$, $p = ns$]. Lastly, unshared items were more readily recalled than shared items on List A-5 [$F(1,156) = 7.5$, $p < .01$] and at SDFR [$F(1,156) = 66.2$, $p < .001$], a pattern that did not vary between groups for either List A-5 [$F(1,156) = 1.4$, $p = ns$] or SDFR [$F(1,156) = 1.6$, $p = ns$].[c]

Interference scores and long-term memory

Interference difference scores were calculated according to the methodology specified in the Procedures section. To evaluate the relationship between these scores and LTM, linear regression was carried out separately in MS participants and controls to identify predictors of long-delay free recall (LDFR). Six independent variables were entered into the model in the following order: block 1, demographic variables (age and education); block 2, partial BDI score; block 3, interference indices (accumulated PI, release from PI, and accumulated RI). Long-delay free recall was the dependent variable in these analyses. As indicated in Table 3, age and partial BDI score, but none of the interference

[c]To ensure that our findings were robust to the possible confounding effects of disease-related variables such as MS subtype, comorbid depression, medication use, and recent symptom exacerbation, the previous analyses were repeated in subsamples of participants reporting they had received a diagnosis of Definite MS ($N = 59$), those with Total BDI scores below 20 ($N = 71$), those not currently taking anxiolytics ($N = 73$), and those reporting symptom remission of at least one month ($N = 57$). A BDI cutoff score of 20 was chosen to maximize sensitivity and specificity, in accordance with the recommendations of Lykouras et al. (1998) for neurological patients. In all analyses, the patterns of PI and RI effects were comparable with those reported in the larger group (all *ps* < .05), with the exception of PI effects in the symptom remission group, where interference scores were almost identical to the larger sample, but reduced statistical power attenuated significance of the between group interaction ($p < .10$).

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Table 3. Predictors of long-delay free recall (LDFR) in MS and control groups

	MS			Controls		
Variable	R^2	R^2 Change	Std. β	R^2	R^2 Change	Std. β
Block 1	.02	.02		.076	.076	
Age			−.074			−.278*
Education			.014			.030
Block 2	.02	.001		.154	.078*	
Partial BDI Score			−.067			.287*
Block 3	.392	.371**		.199	.045	
Accumulated PI			−.040			.082
Release from PI			−.001			−.056
Accumulated RI			−.621**			−.181

Note. BDI = Beck Depression Inventory; MS = multiple sclerosis; PI = proactive interference; RI = retroactive interference.
* $p < .05$; ** $p < .001$.

indices, predicted LDFR in healthy participants. In contrast, only accumulated RI predicted LDFR in the MS group.

DISCUSSION

Consistent with past research (e.g., Bobholtz & Rao, 2003; Rao, 1986), we found diminished verbal long-term memory in MS participants relative to healthy controls. Although prior studies have reported similar findings using the CVLT (Kessler et al., 1992; Troyer et al., 1996), they have not described corresponding patterns of interference effects, nor their contribution to LTM. Our findings indicate that while individuals with MS appear no more susceptible to accumulation of PI than healthy individuals, they demonstrate attenuated release from PI and intensified RI. These results appear to be germane to ultimate LTM, as accumulated RI predicted long-delay free recall in people with MS, but not in healthy participants. This divergence in interference-memory patterns suggests that there are unique MS-related processing vulnerabilities that may mediate retention of information. The nature of these vulnerabilities is detailed in the following discussion of the pattern of PI and RI effects.

Retroactive Interference Effects

The current results indicate heightened susceptibility to RI in MS when semantically associated material is processed in memory. An intriguing subsequent observation is that RI contributes substantially to the eventual recall of MS participants. After controlling for potential confounding variables, vulnerability to RI accounted for approximately 37% of the delayed recall variability of MS participants, but did not reliably predict memory in healthy individuals. Apparently, individuals with MS have specific difficulties retaining and retrieving old information after acquiring new, related information. In aging, this deficit has been attributed to compromised source monitoring (Hedden & Park, 2003) and frontal lobe dysfunction (Glisky et al., 2001). Similarly, intensified RI in MS may indicate a difficulty using source and contextual information to facilitate retention and retrieval of information.

Although two prior investigations reported SDFR decrements (i.e., RI) on the CVLT in MS samples (Kessler et al., 1992; Troyer et al., 1996), neither study evaluated shared and unshared recall patterns, nor conducted control group comparisons. Another study reporting no differences between MS and control participants in postinterference recall used procedures (i.e., RAVLT) that did not manipulate the semantic relationships between items (Minden et al., 1990). Given that susceptibility to RI should be strongest for shared item recall (Kramer & Delis, 1991) these null findings may be artifactual. Indeed, the current study demonstrated no group differences in the pattern of unshared item recall across the final learning and postinterference trials.

Release from Proactive Interference

Previous reports of normal accumulation and release from PI in MS using Wickens-type paradigms (Beatty et al., 1989; Johnson et al., 1998; Rao et al., 1993) are in contrast with the current findings of attenuated release from PI on the CVLT. As opposed to the singular categorical shift on release trials in Wickens-type paradigms, two simultaneous events occur during release on the CVLT. Specifically, successful release from PI occurs when participants register the distinctiveness of unshared List B items, while simultaneously inhibiting interference between shared words from List A and to-be-recalled List B categorical associates. During this ongoing semantic interference, participants must *simultaneously* register a categorical shift, a process requiring processing resources in healthy individuals (Moscovitch, 1994). Normal accumulation and attenuated release from PI may reflect a selective deficit in the ability to register and respond to the semantic properties of stimulus items *flexibly* in order to facilitate encoding under taxing conditions. A decreased response to semantic shifts in the stimulus set parallels the loss of semantic distinctiveness of memory material (Carroll et al., 1984) and substantially reduced incidental seman-

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tic categorization in LTM (e.g., Arnett et al., 1997) previously reported in MS samples.

Reduced ability to register distinctiveness in the memory trace could increase susceptibility to RI. Persons with MS might preferentially register gist (general) information, rather than contextual and semantic details (see Goldstein et al., 1992; Thornton et al., 2002). Encoding of general attributes may make previously learned information more vulnerable to interference from new entries of a general form, activating similar, superordinate general target attributes. Conversely, activation of specific and unique stimulus attributes registering distinctiveness might preserve old information from the interfering effects of new information (e.g., Hunt & McDaniel, 1993). Diminished release from PI in MS may therefore reflect a reduced ability to appreciate the specific (unique) semantic information conveyed in new words.

Attenuated release from PI has often been attributed to information processing deficits superimposed on memory impairment (Freedman & Cermak, 1986; Squire, 1982). Intact set-shifting (Parkin & Lawrence, 1994) and number of categories completed on the Wisconsin Card Sorting Test (WCST; Binetti et al., 1995) have been reported as correlates of release from PI in the elderly and early Alzheimer's patients, respectively.

Interestingly, CVLT List B recall has also been selectively associated with executive abilities in neurological patients (Vanderploeg et al., 1994), and reduced release from PI following frontal *versus* temporal lobe resection has been reported in epilepsy samples (McDonald et al., 2001). Furthermore, Troyer and colleagues (1996) found that the relationship between neurological impairment, age, and CVLT performance was mediated by total errors on the WCST in their MS sample. Based on these findings, attenuated release from PI could be characterized in terms of reduced mental flexibility leading to diminished encoding efficiency.

Limitations

There are several methodological limitations to consider when placing our results within the wider context. An important limitation regarding diagnostic classification was that MS participants did not undergo a neurological examination. While this may be problematic in correctly identifying participants as meeting Poser et al. (1983) or McDonald et al. (2001) criteria for MS, it seems improbable that participants would erroneously report receiving a diagnosis of MS from their physician. Nonetheless, the validity of the diagnosis, especially for participants reporting probable or possible MS, is questionable. To address this limitation, we replicated the pattern of effects in a subsample of those individuals who had reported a diagnosis of definite MS.

Secondly, our participant sample was not well characterized medically. It would have been ideal to access complete medical records of MS participants to address this limitation. Nevertheless, MS patients appear to be generally capable of making accurate judgments of their symptomatology and daily functioning. Other researchers have noted good agreement (72%–86%) between neurologist and patient ratings of both MS symptoms and performance of activities of daily living (ADLs) (Gulick et al., 1997).

Finally, several confounding factors could affect the pattern of interference indices. To address this issue, the current findings were replicated in participant subsamples who were (1) less apt to be significantly depressed, (2) whose medications are not specifically antagonistic to memory, and (3) who were not experiencing symptom exacerbation. In these analyses, the overall pattern of results held.

Summary and Conclusions

In a community-based MS sample, attenuated release from PI and intensified RI were observed on the CVLT. For MS participants only, RI emerged as a strong predictor of LTM; thus, it appears to represent a central feature of the MS-related memory deficits. We also propose that these abnormal interference effects in MS may be mediated by limitations in the contextual and semantic richness of encoding and/or by decreased mental flexibility and executive functions. Future studies will be necessary to clarify more precisely the mechanisms underlying abnormal interference effects in MS.

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REFERENCES

Allison, D.B. & Gorman, B.S. (1993). Some of the most common questions asked of statistical consultants: Our favourite responses and recommended readings. *Genetic, Social & General Psychology Monographs*, *119*, 155–186.

Armstrong, C., Onishi, K., Robinson, K., D'Esposito, M., Thompson, H., Rostami, A., & Grossman, M. (1996). Serial position and temporal cue effects in multiple sclerosis: Two subtypes of defective memory mechanisms. *Neuropsychologia*, *34*, 853–862.

Arnett, P.A., Higginson, C.I., & Voss, W.D. (1999a). Depression in multiple sclerosis: Relationship to working memory capacity. *Neuropsychology*, *13*, 546–556.

Arnett, P.A., Higginson, C.I., & Voss, W.D. (1999b). Depressed mood in multiple sclerosis: Relationship to capacity-demanding memory and attentional functioning. *Neuropsychology*, *13*, 434–446.

Arnett, P.A., Rao, S.M., Grafman, J., Bernardin, L., Luchetta, T., Binder, J.R., & Lobeck, L. (1997). Executive functions in multiple sclerosis: An analysis of temporal ordering, semantic encoding, and planning abilities. *Neuropsychology*, *11*, 535–544.

Beatty, W.W. (1993). Memory and "frontal lobe" dysfunction in multiple sclerosis. *Journal of the Neurological Sciences*, *115(Suppl)*, S38–S41.

Beatty, W.W., Goodkin, D.E., Beatty, P.A., & Monson, N. (1989). Frontal lobe dysfunction and memory impairment in patients

with chronic progressive multiple sclerosis. *Brain and Cognition*, *11*, 73–86.

Beck, A.T., Ward, C.H., & Mendelson, M. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, *4*, 561–571.

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

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

Blusewicz, M.J., Kramer, J.H., & Delmonico, R.L. (1996). Interference effects in chronic alcoholism. *Journal of the International Neuropsychological Society*, *2*, 141–145.

Bobholtz, J.A. & Rao, S.M. (2003). Cognitive dysfunction in multiple sclerosis: A review of recent developments. *Current Opinion in Neurology*, *16*, 283–288.

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

Carroll, M., Gates, R., & Roldan, F. (1984). Memory impairment in multiple sclerosis. *Neuropsychologia*, *22*, 297–302.

Cermak, L.S., Butters, N., & Moreines, J. (1974). Some analyses of the verbal encoding deficit of alcoholic Korsakoff patients. *Brain and Language*, *1*, 141–150.

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

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). *The California Verbal Learning Test*. San Antonio, TX: Psychological Corporation.

DeLuca, J., Barbieri-Berger, S., & Johnson, S.K. (1994). The nature of memory impairments in multiple sclerosis: Acquisition versus retrieval. *Journal of Clinical and Experimental Neuropsychology*, *16*, 183–189.

DeLuca, J., Gaudino, E.A., Diamond, B.J., Christodoulou, C., & Engel, R.A. (1998). Acquisition and storage deficits in multiple sclerosis. *Journal of Clinical and Experimental Neuropsychology*, *20*, 376–390.

Demaree, H.A., Gaudino, E.A., DeLuca, J., & Ricker, J.H. (2000). Learning impairment is associated with recall ability in multiple sclerosis. *Journal of Clinical and Experimental Neuropsychology*, *22*, 865–873.

DeSousa, E.A., Albert, R.H., & Kalman, B. (2002). Cognitive impairment in multiple sclerosis: A review. *American Journal of Alzheimer's Disease and Other Dementias*, *17*, 23–29.

D'Esposito, M., Onishi, K., Thompson, H., Robinson, K., Armstrong, C., & Grossman, M. (1996). Working memory impairments in multiple sclerosis: Evidence from a dual-task paradigm. *Neuropsychology*, *10*, 51–56.

Dobbs, A.R., Aubrey, J.B., & Rule, B.G. (1989). Age-associated release from proactive interference: A review. *Canadian Psychology*, *30*, 588–595.

Foong, J., Rozewicz, L., Quaghebeur, G., Davie, C.A., Kartsounis, L.D., Thompson, A.J., Miller, D.H., & Ron, M.A. (1997). Executive function in multiple sclerosis. *Brain*, *120*, 15–26.

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

Gaudino, E.A., Chiaravalloti, N.A., DeLuca, J., & Diamond, B.J. (2001). A comparison of memory performance in relapsing-remitting, primary progressive, and secondary progressive multiple sclerosis. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *14*, 32–44.

Glisky, E.L., Rubin, S.R., & Davidson, P.S. (2001). Source memory in older adults: An encoding or retrieval problem? *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *27*, 1131–1146.

Goldstein, F.C., Levin, H. S., & Boake, C. (1989). Conceptual encoding following severe closed head injury. *Cortex, 25*, 541–554.

Goldstein, F.C., McKendall, R.R., & Haut, M.W. (1992). Gist recall in multiple sclerosis. *Archives of Neurology*, *49*, 1060–1064.

Gulick, E.E., Cook, S.D., & Troiano, R. (1997). Comparison of patient and staff assessment of MS patients' health status. *Acta Neurologica Scandinavica*, *88*, 87–93.

Hedden, T. & Park, D.C. (2003). Contributions of source and inhibitory mechanisms to age-related retroactive interference in verbal memory. *Journal of Experimental Psychology: General*, *132*, 93–112.

Higginson, C.I., Arnett, P.A., & Voss, W.D. (2000). The ecological validity of clinical tests of memory and attention in multiple sclerosis. *Archives of Clinical Neuropsychology*, *15*, 185–204.

Hunt, R.R. & McDaniel, M.A. (1993). The enigma of organization and distinctiveness. *Journal of Memory and Language*, *32*, 421–445.

Johnson, S.K., DeLuca, J., Diamond, B.J., & Natelson, B.H. (1998). Memory dysfunction in fatiguing illness: Examining interference and distraction in short-term memory. *Cognitive neuropsychiatry*, *3*, 269–285.

Kareken, D.A., Moberg, P.J., & Gur, R.C. (1996). Proactive inhibition and semantic organization: Relationship with verbal memory in patients with schizophrenia. *Journal of the International Neuropsychological Society*, *2*, 486–493.

Keppel, G. & Underwood, B.J. (1962). Proactive inhibition in short-term retention of single items. *Journal of Verbal Learning & Verbal Behavior*, *1*, 153–161.

Kessler, H.R., Cohen, R.A., Lauer, K., & Kausch, D.F. (1992). The relationship between disability and memory dysfunction in multiple sclerosis. *International Journal of Neuroscience*, *62*, 17–34.

Kramer, J.H. & Delis, D.C. (1991). Interference effects on the California Verbal Learning Test: A construct validation study. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, *3*, 299–302.

Kurtzke, J.F. (1965). Further notes on disability evaluation in multiple sclerosis, with scale modification. *Neurology*, *15*, 654–661.

Legenfelder, J., Chiaravalloti, N.D., Ricker, J.H., & DeLuca, J. (2003). Deciphering components of impaired working memory in multiple sclerosis. *Cognitive and Behavioral Neurology*, *16*, 28–39.

Litvan, I., Grafman, J., Vendrell, P., Martinez, J.M., Junque, C., Vendrell, J.M., & Barraquer-Bordas, J.L. (1988). Multiple memory deficits in patients with multiple sclerosis: Exploring the working memory system. *Archives of Neurology*, *45*, 607–610.

Lustig, C., May, C.P., & Hasher, L. (2001). Working memory span and the role of proactive interference. *Journal of Experimental Psychology: General*, *130*, 199–207.

Lykouras, L., Oulis, P., Adrachta, D., Daskalopoulou, E., Kalfakis, N., Triantapyllou, N., Papageorgiou, K., & Christodoulou, G.N. (1998). Beck Depression Inventory in the detection of depression among neurological inpatients. *Psychopathology*, *31*, 213–219.

McDonald, C.R., Bauer, R.M., Grande, L., Gilmore, R., & Roper, S. (2001). The role of the frontal lobes in memory: Evidence from unilateral frontal resections for relief of intractable epilepsy. *Archives of Clinical Neuropsychology*, *16*, 571–585.

McDonald, W.I., Compston, A., Edan, G., Goodkin, D., Hartung, H.P., Lublin, F.D., McFarland, H.F., Paty, D.W., Polman, C.H., Reingold, S.C., Sandberg-Wollheim, M., Sibley, W., Thompson, A., van den Noort, S., Weinshenker, B.Y., & Wolinsky, J.S. (2001). Recommended diagnostic criteria for multiple sclerosis: Guidelines from the International Panel on the Diagnosis of Multiple Sclerosis. *Annals of Neurology*, *50*, 121–127.

Minden, S.L., Moes, E.J., Orav, J., Kaplan, E., & Reich, P. (1990). Memory impairment in multiple sclerosis. *Journal of Clinical and Experimental Neuropsychology*, *12*, 566–586.

Minden, S.L. & Schiffer, R.B. (1990). Affective disorders in multiple sclerosis: Review and recommendations for clinical research. *Archives of Neurology*, *47*, 98–104.

Moscovitch, M. (1994). Cognitive resources and dual-task interference effects at retrieval in normal people: The role of frontal cortex and medial temporal cortex. *Neuropsychology*, *8*, 524–534.

Numan, B., Sweet, J.J., & Ranganath, C. (2000). Use of the California Verbal Learning Test to detect proactive interference in the traumatically brain injured. *Journal of Clinical Psychology*, *56*, 553–562.

Parkin, A.J. & Lawrence, A. (1994). A dissociation in the relation between memory tasks and frontal lobe tests in the normal elderly. *Neuropsychologia*, *32*, 1523–1532.

Pelosi, L., Geesken, J.M., Holly, M., Hayward, M., & Blumhardt, L.D. (1997). Working memory impairment in early multiple sclerosis: Evidence from an event-related potential study of patients with multiple sclerosis. *Brain*, *120*, 2039–2058.

Peterson, L. & Peterson, M.J. (1959). Short-term retention of individual verbal items. *Journal of Experimental Psychology*, *58*, 193–198.

Poser, C.M., Paty, D.W., Scheinberg, L., McDonald, W.I., Davis, F.A., Ebers, G.C., Johnson, K.P., Sibley, W.A., Silberberg, D.H., & Tourtellotte, W.W. (1983). New diagnostic criteria for multiple sclerosis: Guidelines for research protocols. *Annals of Neurology*, *13*, 227–231.

Postman, L. & Underwood, B.J. (1973). Critical issues in interference theory. *Memory & Cognition*, *1*, 19–40.

Randolph, C., Gold, J.M., Carpenter, C.J., Goldberg, T.E., & Weinberger, D.R. (1992). Release from proactive interference: Determinants of performance and neuropsychological correlates. *Journal of Clinical and Experimental Neuropsychology*, *14*, 785–800.

Rao, S.M. (1986). Neuropsychology of multiple sclerosis: A critical review. *Journal of Clinical and Experimental Neuropsychology*, *8*, 503–542.

Rao, S.M., Grafman, J., DiGiulio, D., Mittenberg, W., Bernardin, L., Leo, G.J., Luchetta, T., & Unvervagt, F. (1993). Memory dysfunction in multiple sclerosis: Its relation to working memory, semantic encoding, and implicit learning. *Neuropsychology*, *7*, 364–374.

Rao, S.M., Leo, G.J., Bernardin, L., & Unverzagt, F. (1991). Cognitive dysfunction in multiple sclerosis: I. Frequency, patterns, and prediction. *Neurology*, *41*, 685–691.

Rao, S.M., Leo, G.J., & St. Aubin-Faubert, P. (1989). On the nature of memory disturbance in multiple sclerosis. *Journal of Clinical & Experimental Neuropsychology*, *11*, 699–712.

Sagar, H.J., Sullivan, E.V., & Cooper, J.A. (1991). Normal release from proactive interference in untreated patients with Parkinson's disease. *Neuropsychologia*, *29*, 1033–1044.

Sitskoorn, M.M., Nuyen, J., Appels, M.C., van der Wee, N.J., & Kahn, R.S. (2002). Release from proactive inhibition in schizophrenia and its potential as a genetic marker. *Journal of Clinical and Experimental Neuropsychology*, *24*, 67–81.

Solari, A., Amato, M.P., Bergamaschi, R., Logorscino, G., Citterio, A., Bochicchio, D., & Filippini, G. (1993). Accuracy of self-assessment of minimal record of disability in patients with multiple sclerosis. *Acta Neurologica Scandinavica*, *87*, 43–46.

Sperling, R.A., Guttman, C.R., Hohol, M.J., Warfield, S.K., Jakab, M., Parente, M., Diamond, E.L., Daffner, K.R., Olek, M.J., Orav, E.J., Kikinis, R., Jolesz, F.A., & Weiner, H.L. (2001). Regional magnetic resonance imaging lesion burden and cognitive function in multiple sclerosis. *Archives of Neurology*, *58*, 115–121.

Squire, L.R. (1982). Comparisons between forms of amnesia: Some deficits are unique to Korsakoff's syndrome. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *8*, 560–571.

Thornton, A.E., Raz, N., & Tucker, K.A. (2002). Memory in multiple sclerosis: Contextual encoding deficits. *Journal of the International Neuropsychological Society*, *8*, 395–409.

Thornton, A.E. & Raz, N. (1997). Memory impairment in multiple sclerosis: A quantitative review. *Neuropsychology*, *11*, 357–366.

Torres, I.J., Flashman, L.A., O'Leary, D.S., & Andreasen, N.C. (2001). Effects of retroactive and proactive interference on word list recall in schizophrenia. *Journal of the International Neuropsychological Society*, *7*, 481–490.

Troyer, A.K., Fisk, J.D., Archibald, C.J., Ritvo, P.G., & Murray, T.J. (1996). Conceptual reasoning as a mediator of verbal recall in patients with multiple sclerosis. *Journal of Clinical and Experimental Neuropsychology*, *18*, 211–219.

Vanderploeg, R.D., Crowell, T.A., & Curtiss, G. (2001). Verbal learning and memory deficits in traumatic brain injury: Encoding, consolidation, and retrieval. *Journal of Clinical and Experimental Neuropsychology*, *23*, 185–195.

Vanderploeg, R.D., Schinka, J.A., & Retzlaff, P. (1994). Relationships between measures of auditory verbal learning and executive functioning. *Journal of Clinical and Experimental Neuropsychology*, *16*, 243–252.

Verdier-Taillefer, M.H., Roullet, E., Cesaro, P., & Alperovitch, A. (1994). Validation of self-reported neurological disability in multiple sclerosis. *International Journal of Epidemiology*, *23*, 148–154.

Wickens, D.D. (1970). Encoding categories of words: An empirical approach to meaning. *Psychological Review*, *77*, 1–15.

Winocur, G., Kinsbourne, M., & Moscovitch, M. (1981). The effect of cuing on release from proactive interference in Korsakoff amnesic patients. *Journal of Experimental Psychology, Human Learning and Memory*, *7*, 56–65.