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Semantic Priming Effects in a Patient with a Semantic Short-Term Memory Deficit

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ABSTRACT

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Some researchers have argued that short-term memory (STM) deficits are due to overly rapid loss of activation of the nodes in the lexical-semantic system. The current study investigated the activation and decay of semantic representations for a patient (ML) with a semantic STM deficit using a semantic priming paradigm. Experiment 1 used a traditional paired presentation task to examine priming at two SOAs (350ms & 900ms). Experiment 2 and 3 used a single presentation task to reduce the use of strategies at long SOAs. ML demonstrated normal priming effects at short and long SOAs when priming primarily reflected automatic spreading activation. The results suggest that ML has normal activation and decay in the lexical-semantic system and support a model with STM buffers separate from the lexical-semantic system. Experiment 4 explored ML and older control subjects' use of strategies in a priming task. The sources of ML's STM deficit were discussed.

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1. Introduction

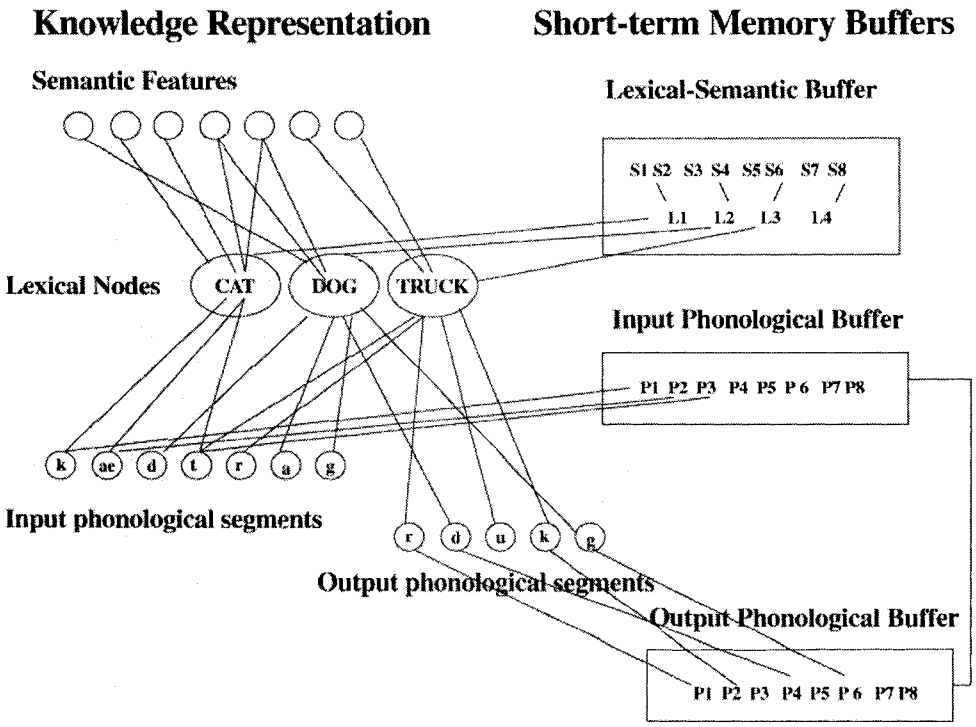
Martin and colleagues (Martin & Freedman, 2001; Martin & Lesch, 1996; Martin, Shelton & Yaffee, 1994) have reported several patients who showed a deficit in maintaining semantic information in short-term memory (STM). These patients, like other STM patients, remember fewer items on memory span tasks than age-matched control subjects. Two patients reported in a previous study, ML and GR, had word spans of 2.7 and 3.2 words, while age-matched normal subjects had spans of about 4 to 6 (Martin & Freedman, 2001). However, for these patients, their word span is similar to nonword span; that is, they do not show the advantage in remembering words over nonwords (Martin & Lesch, 1996), which neurally intact control subjects and other STM deficit patients usually show. They also show impairments in other tasks that require holding semantic information in STM, e.g. their performance in a category probe task is worse than in a rhyme probe task (Martin & Lesch, 1996). The pattern of deficits observed in these patients is opposite to that shown by another type of patient identified with phonological STM deficits. The phonological STM deficit patients have a smaller nonword span than word span, and perform better in category probe task than in rhyme probe task (Martin & Freedman, 2001).

Models of Short Term Memory

Martin, Lesch, and Bartha (1999) developed a model of STM (Figure 1) to account for different patterns observed in semantic STM patients and phonological STM patients. As depicted in Figure 1, the lexical knowledge network represented on the left-hand side is similar to that proposed by Dell and colleagues in their model of word production (Dell, 1986; Dell & O'Seaghdha, 1992). It represents the lexical system and

the phonological and semantic information in the long-term knowledge. On the right side, there are separate buffers to store lexical-semantic and phonological information. As phonological STM is not the focus of this study, the distinction between phonological and semantic buffers will not be discussed further here.

Figure 1. Model of Short-term Memory (R. Martin et al., 1999)



In perceiving a word, phonological or orthographic information is mapped to the lexical node of that word, which then activates the semantic information connected to that node. From one perspective, STM of words could be based on the same system used in spoken or written word perception. N. Martin and Saffran (1992) reported an aphasic patient NC whose dysphasia (characterized by making semantic errors in single word repetition) and STM impairment both recovered during the same time period. The authors argued that STM storage and word processing are both based on the same knowledge

representation system. They proposed a common cause for word processing deficit and STM deficit - activation of nodes does not persist in the lexical system as it does for control subjects. That is, reduced memory span does not come from a smaller mental space to store the remembered items or processes other than activation or persisting of activation in the lexical-semantic system. STM deficits could merely reflect deficits in the lexical-semantic system.

Different from this view, Martin et al. (1999) proposed a model with short-term memory buffers that are separate from the lexical-semantic system. Martin and Lesch (1996) have argued that the patients' semantic STM deficits are not due to a disruption of semantic information or difficulty accessing it, but instead reflect impairments of the semantic STM buffer. One piece of evidence against deficits in the lexical-semantic system is that the semantic STM patients were normal on many aspects of semantic processing for single words, yet showed very reduced capacity (Martin & Lesch, 1996). For instance, they performed normally in picture naming tasks (Martin et al., 1994; Martin & Lesch, 1996) and a single word/picture matching task (Freedman & Martin, 2001).

Although this evidence suggests that the semantic STM deficit patients have an intact lexical-semantic system, only explicit tasks such as single word repetition and comprehension tasks have been used to assess performance. Both accuracy and reaction time measures have been used, with accuracy often being within normal range. A more fine-grained assessment of performance is available from overall reaction time measures, but these are sometimes hard to evaluate due to the patients' motor deficits or due to the decision components involved in various tasks like lexical decision or synonymy

judgments. Automatic activation and decay in the lexical-semantic system have not been assessed for the semantic STM deficit patients. Semantic priming has been shown to be a powerful tool to investigate the lexical-semantic system in healthy subjects as well as people with dementias, aphasia, schizophrenia, and Alzheimer's disease (Moss & Tyler, 1995; Hagoort, 1997; Barch, Cohen, Servan-Schreiber, Steingard, Steinhauer & van Kammen, 1996). In semantic priming, differences in reaction time to related and unrelated prime-target pairs are evaluated, which allows one to investigate the size of priming effects as a function of various task manipulations. Thus, such measures allow one to avoid some of the problems in examining mean reaction time per se. The primary goal of the current study was to use semantic priming tasks to investigate the activation and decay of information in the lexical-semantic system.

A Review of Semantic Priming

Semantic priming is one of the most basic effects in language processing (for a review, see McNamara, 2005). The presentation of the first word (the prime) reduces the time needed to process a second word (the target) which is semantically associated with the prime. Semantic priming effects can be observed in naming, lexical decision or semantic judgment tasks. For example, in a traditional lexical decision task, subjects respond faster to a word *cat* after seeing *dog* than after seeing an unrelated word *rug* that immediately precedes *cat*. Researchers have identified two different components that contribute to the processing advantage of a target word after seeing a semantically related prime: automatic spreading activation and controlled or strategic processing (Posner & Snyder, 1975; Neely, Keefe & Ross, 1989).

Automatic spreading activation is thought to be a basic process in the

lexical-semantic system. Perceiving the prime (e.g. *cat*) activates its lexical node and its associated semantic representation. Then activation spreads in the lexical-semantic system and activates adjacent nodes of related words (e.g. *dog*), facilitating their later identification (Collins & Loftus, 1975). Automatic activation can also spread to nodes that are indirectly related to the prime. This leads to the mediated priming effect (McNamara & Altarriba, 1988). For example, the word *winter* can prime *swim* through the mediating node *summer*. The spread of activation has a rapid onset (Fischer & Goodman, 1978). It is independent of attention or awareness (Ortells, Daza & Fox, 2003), and facilitates responses by pre-activating nodes representing related words (Collins & Loftus, 1975). Since observing a priming effect is a sign of activation of the lexical-semantic information, it provides a way to investigate the activation and persistence of activation in the lexical-semantic system. If priming effects are obtained at a shorter lag and a longer lag, the change in the magnitude of priming effect could reveal how activation in the lexical-semantic system changes over time.

Another source of the priming effect is the use of strategies (Neely et al.,1989). Two types of strategies have been identified: expectancy generation and post-lexical checking. Expectancy generation occurs when subjects read the prime and generate a possible target or a set of targets. When the target is in the pre-generated set, recognition of the target is faster. When the target is not in the pre-generated set, recognition is slower than in a neutral condition where the prime is simply 'XXXXX' or 'BLANK'. Thus, subjects show an inhibition effect for unrelated primes. Expectancy generation involves generation of possible targets during the interval between the prime and the target, which is manipulated by varying stimulus onset asynchrony (SOA). Thus

compared to automatic spreading activation, it has a slower onset (Neely, 1977; Hutchison, Neely & Johnson, 2001). The post-lexical checking strategy involves subjects' assessing the semantic relation between the prime and the target before making a decision to the target. Because the presence of a relation between the prime and target signifies that the target is a word, subjects can use detection of a relation to aid their "word" decision, and therefore respond faster to the target.

Several facts support the existence of strategies in semantic priming tasks. For one, the size of the semantic priming effect increases as the proportion of the associated word pairs increases, which is termed the relatedness proportion (RP) effect (Neely, 1991; Bodner & Masson, 2003). If priming were completely automatic the amount of priming should stay constant across all proportions of associated word pairs. The RP effect is explained by assuming that when the relatedness proportion is high, subjects are more likely to notice the relation between prime and target, and therefore are more motivated to engage in strategies such as expectancy generation. Another piece of evidence for strategy use is backward priming. Backward priming refers to priming for prime-target pairs which are not associated in a forward direction, but are associated in a backward direction (e.g. prime: *hop*; target: *bell*, Seidenberg, Waters, Sanders & Langer, 1984). This effect reveals post-lexical checking because the relation between the prime and the target becomes obvious to the subject only after the target is processed. The post-lexical checking effect is related to the nonword ratio, which is the proportion of nonword targets in the stimuli. The higher the nonword ratio, the more useful the post-lexical checking strategy, and the greater the backwards priming effect (Neely et al., 1989)

Although the primary goal of the current study was to investigate automatic

priming, the use of strategies is of interest for two reasons. First, strategies easily confound the results in experiments that claim to investigate automatic spreading activation. Priming tasks that aim to tap automatic processes require strict manipulation of stimuli and experimental procedures. Inappropriate manipulations may evoke strategies use and contaminate the results. Second, the ability to engage in strategies may provide information about other aspects of cognition. Hutchison (2007) investigated the relation between RP effect and attentional control (AC) in college students. Attentional control was measured by a composite score that consisted of performance in an operation span task (Turner & Engle, 1989; Kane & Engle, 2003), an antisaccade task (Kane, Bleckley, Conway & Engle, 2001), and a Stroop task (Spieler, Balota & Faust, 1996). Hutchison reported a significant linear correlation between the RP effect and the AC score. He concluded that subjects with higher AC are better at engaging strategies. Hamilton and Martin (2005) have reported that a semantic STM patient, ML showed an exaggerated interference effect in a Stroop task in a study on inhibition and demonstrated excessive proactive interference in a short-term recognition memory task. In contrast, ML performed normally on the antisaccade task and on a spatial version of the Stroop task. Thus, they concluded that he had a deficit in inhibition, which is considered to be one aspect of attentional control, though this deficit was limited to the verbal domain. Thus, it would be valuable to determine if semantic STM patients could engage strategies in a semantic priming task, which involves verbal materials.

Therefore, in this study I had two goals. The primary goal was to investigate whether semantic STM patients have deficits in activation and decay in the lexical-semantic system. If N. Martin and Saffran's claim (1992) is true that STM storage

is based on the activation and decay of nodes in the lexical-semantic system, then semantic STM patients may show deficits in automatic activation in the lexical system. Even if activation is normal, they may show overly rapid decay of activation relative to control subjects. This would be reflected in a weaker priming effect at a longer lag when that priming is due to automatic spreading activation. If no differences in activation or decay between patients and normal subjects are observed, the findings will support the model of Martin et al. (1999), which separates STM buffers from the lexical-semantic system. The second goal of this study was to explore whether semantic STM patients can engage strategy in priming tasks. A deficit in engaging strategies would result in weaker priming effect relative to controls in a task that maximally promotes strategy use.

The design of the experiments in the current study was based on the following findings. First, a high RP raises the possibility that subjects will use a strategy, i.e. expectancy generation. A lower RP would limit the likelihood of using this strategy and increase the likelihood that the priming effect reflects automatic spreading activation. Second, researchers have found that semantic priming due to automatic spreading activation is observable at a short SOA (as short as 40ms, Fischler & Goodman, 1978), while semantic priming due to strategies is observable at longer SOAs (e.g. 400ms and longer, Neely, 1977; Hutchison, Neely & Johnson, 2001; but see Hutchison, 2007). Third, using a single presentation procedure, rather than paired presentation, can reduce the influence of strategies because there is no obvious pairing of prime and target (Shelton & Martin, 1992; McNamara & Altarriba, 1988). Finally, using a high nonword ratio can promote using backward checking. One can test for the presence of backward checking

by including pairs that are related only in a backward direction and determine if backward priming is obtained.

In the current study, the first three experiments were designed to investigate automatic spreading activation. In Experiment 1, a paired presentation lexical decision task was used. The experiment included a short SOA of 350ms and a long SOA of 900ms. A low relatedness proportion (that is, the proportion of word pairs that are related among all word pairs) was used, that is, 20%. Despite the low relatedness proportion in Experiment 1, there was some evidence that control subjects engaged in strategies. Thus, in Experiments 2 and 3, a single presentation lexical decision task was used (Shelton & Martin, 1992; McNamara & Altarriba, 1988). Also, the relatedness proportion was reduced even further, to 17%. When the proportion of related word pairs among all pairs of adjacent items is considered, this value was less than 6%. Experiment 4 was designed to investigate the use of strategies. A paired presentation procedure with a long SOA was used. The stimuli had a high relatedness proportion of 67% and included some pairs that were related only in a backward direction.

The focus of the present study was patient ML, who has been reported in several previous studies as showing a semantic STM deficit (Freedman & Martin, 2001; Martin & He, 2004; Martin & Lesch, 1996). As indicated in the patient background section, ML has nonfluent speech and good comprehension on clinical exam and thus might be classified as a Broca's aphasic. A number of previous studies have investigated automatic lexical-semantic access in Broca's aphasia, but these studies have not reached a consensus. Some researchers reported no priming effects in Broca's aphasics (Milberg & Blumstein, 1981; Milberg, Blumstein & Dworetzky, 1987). On the other hand, Hagoort

(1997) reported significant priming effects at both an SOA of 300ms and 1400ms in Broca's aphasics. The different findings across different studies may have resulted because the patients tested in the different labs were different in terms of their underlying cognitive deficits. Another possibility is that patient latencies are likely to be more variable than controls and a null effect may reflect a lack of statistical power. Thus the current study used relative large number of trials (80 key targets in Experiment 1, 3 &4) to determine whether an effect is significant at the single subject level.

Patient Background Information

ML

ML is a 64 year-old, right-handed male who suffered a cerebrovascular accident (CVA) in 1990, resulting in a left frontal and parietal infarction. He completed 2 years of college coursework.

He has a reduced memory span. In a list recall test, he recalled 77% lists correct for two-word lists and 10% lists correct for three-word lists. He shows a pattern consistent with a semantic STM deficit, e.g. he shows no advantage for word recall over nonword recall (Martin & Lesch, 1996) and his performance was worse on category than rhyme probe recognition tasks (Freedman & Martin, 2001).

His repetition of single words is excellent (96% correct) and he has good semantic processing on untimed tasks of single word comprehension and production. His score on the Peabody Picture Vocabulary Task (a word comprehension task; Dunn & Dann, 1997) is above the mean of control subjects (Hamilton & Martin, 2005). On the Philadelphia Naming Test (Roach, Schwartz, Martin, Grewal & Brecher, 1996), ML performed 98% correct, above the mean of control subjects (96% correct). Also, he performed normally

on living–nonliving judgment task (Martin & He, 2004).

ML has good comprehension of conversational speech, though he shows difficulty in comprehending sentences which require the maintenance of several unintegrated word meanings (Martin & He, 2004). His narrative production is marked by pausing, hesitations, word-finding difficulties, and reduced phrase length (Hamilton & Martin, 2005). ML can be categorized as a Broca's aphasic, though it should be noted that his speech is not classically agrammatic as he shows a normal level of production of function words and inflectional markers.

As discussed earlier, Hamilton and Martin (2005) reported that ML showed impaired performance on tasks requiring inhibition of irrelevant verbal information (Stroop task and a recent-negatives task), but no impairment on tasks not requiring manipulation of verbal material (a nonverbal spatial Stroop task and an antisaccade task).

DS

DS is a 55 year-old, right-handed female with 17 years of education. She completed a Bachelor of Commerce degree in Accounting. Her MRI indicates chronic left fronto-parietal convexity and subcortical left frontal white matter infarct and basal ganliaonic lacune.

DS has a reduced memory span. In a list recall test, she recalled 70% lists correct for two-word lists and 0% lists correct for three-word lists. She has a pattern consistent with a phonological STM deficit. Her performance on a nonword list recall test was worse than word list recall, with 50% lists correct on two-item lists and 0% lists correct on three-item lists. Her performance on rhyme probe task was worse than category probe task. She made 38% errors on three-item lists in rhyme probe task and only 25% errors

on four-item lists in category probe task.

She has no problem in repetition of single words (99% correct) and has good comprehension and production of single words. She performed 98% correct on a single picture/word matching test and 94% correct on the Philadelphia Picture Naming Test. She has good auditory comprehension of conversations. She communicates well but has some persistent word retrieval deficits.

DS was tested to provide a comparison with patient ML. That is, if ML showed evidence of abnormal activation or decay of semantic information in the priming task, it would be interesting to compare his performance with that of a patient with a phonological STM deficit. She is predicted to show a pattern similar to control subjects on semantic priming as she has no semantic deficits or semantic STM deficits. DS was only tested in Experiment 1.

2. Experiment 1: Semantic Priming at Two SOAs (350ms and 900ms) with Paired Presentation

In this experiment, a paired presentation lexical decision task was used. In a paired presentation task, a target is always paired with a prime, but no response to the prime is needed. The prime disappears from the screen automatically. This is the most frequently used semantic priming paradigm. In this experiment, two SOA conditions (350ms vs. 900ms) were constructed in order to obtain priming effects at an earlier and a later lag. A relatively low relatedness proportion (20%) was used in order to increase the likelihood that any observed semantic priming would reflect automatic spreading activation. Previous studies have shown that priming effects due to automatic spreading

activation decreases over time, and are often diminished at an SOA of 700ms or longer (Neely, 1977). Therefore, control subjects were expected to show a decrease in priming from the short SOA to the long SOA. Patient DS was expected to show a similar pattern as control subjects.

For the semantic STM deficit patient ML, a normal priming effect at the short SOA will imply normal activation in the lexical-semantic system. If he also shows a normal priming effect at the long SOA, and there is no evidence of a rapid decrease compared to control subjects, then this will imply that the activation does not decay faster for him. Together, both findings would imply no deficit in activation and decay in the lexical-semantic system.. Of course, if priming is reduced at the short SOA or if it shows a rapid decline from the short to the long SOA, the findings would indicate deficits in activation and/or persistence in the lexical-semantic system..

Methods

Participants

The participants were ML, DS and 12 older control subjects. In all experiments, control subjects and patients were given $10/hour.

Design

This experiment had a 2 (semantic relatedness: related vs. unrelated) x 2 (SOA: 350ms vs. 900ms) design. Both semantic relatedness and SOA were manipulated within-subjects.

Materials

Eighty strongly forwardly associated word pairs were selected from the South Florida Word Association Norms (Nelson, McEvoy & Schreiber, 1998). The average

forward association was 0.424 (0.173); the average backward association was 0.190 (0.195). They served as the related pairs in this experiment. The unrelated trials were created by re-pairing the related targets with different primes. Another 60 unrelated word pairs were selected as filler trials. 100 word-nonword pairs served as negative response trials.

The 80 related pairs and 80 unrelated pairs were divided into four lists, across which relatedness and SOA were counterbalanced. Each subject saw four lists, each consisting of 20 related word pairs, 80 unrelated pairs (60 filler trials and 20 trials re-paired from related trials) and 100 word-nonword pairs. The filler trials and negative response trials were the same in four lists. The proportion of related pairs among all word pairs was 20%. The proportion of nonword targets was 50%.

Procedure

Each subject was seated approximately 60 cm away from a VGA monitor. First, each subject read the experiment instructions from the instruction sheet. Subjects were informed that they would see a fixation point '+' at the center of the screen in each trial, followed by a word that was presented briefly in lower case. Soon after the word disappeared, they would see a letter string presented in upper case that was either a word or not a word. They were instructed to make a positive response if they thought the string was a word by pressing 'Yes' (spacebar on the keyboard), and to do nothing if they thought it was not. This go-no-go procedure was used so that the patients did not have to map the responses to two different buttons. One patient, DS, was inefficient in responding with two fingers.

All stimuli were presented using DMDX (Forster & Forster, 2003). On each trial,

a fixation point was first presented in the center of the screen for 800ms, followed by the prime word in lowercase, for 300ms. After 50ms or 600ms (for SOAs of 350 or 900 ms), the target letter string appeared and stayed on the screen until a response was made or after 2500ms for controls and 3000ms for the patient. 1000ms after the subject made a response, or after the timeout, the next trial began. Patient ML and DS responded with left hand because of difficulty with their right hand. Control subjects were required to use their non-dominant hand. Each list took 20 to 30 minutes. ML and the control subjects completed one list per week for four weeks. In every session they completed 20 practice trials before the experimental trials.

Results and Discussion

Because subjects only made a positive response when the target was a word, the word trials on which the subjects did not give a response were coded as an error and were excluded from the response time analysis. For each subject, response times outside three standard deviations from the condition means were deleted during response time analysis. The percentage of deleted trials was 0.7% trials for controls, 2% for ML and 1.3% for DS. The results are presented in Table 1.

Table 1. Experiment 1: Mean Response Times and Priming Effects

	SOA = 350ms			SOA = 900ms		
	Unrelated	Related	priming	Unrelated	Related	priming
controls	691	657	35	681	644	37
patient ML	922	849	72	888	858	30
patient DS	926	828	98	831	705	126

Controls

The average response time for control subjects ranged from 507 ms to 831 ms with a mean of 668 ms. The mean priming effect for controls was 35 ms (range 4ms – 104ms) at the 350 ms SOA and 37 ms (range -3ms – 87ms) at the 900 ms SOA. The controls' data were analyzed by subjects ($F1$) and by items ($F2$), with SOA and relatedness as repeated measures factors in both. The analyses revealed a main effect of relatedness (priming effect), $F1(1,11) = 25.52$, $p < .001$, $MSE = 361$; $F2(1,79) = 65.12$, $p < .001$, $MSE = 1{,}145$. The SOA effect was marginal in the analysis by subjects, $F1(1,11) = 4.37$, $p = .061$, $MSE = 603$, and significant in the analysis by items, $F2(1,79) = 9.15$, $p = .003$, $MSE = 1{,}618$. Response time was shorter at the long SOA than the short SOA. The interaction between SOA and relatedness was not significant, $F1(1,11) = 0.17$, $p = .686$, $MSE = 112$; $F2(1,79) = 0.15$, $p = .695$, $MSE = 1{,}153$). Thus, contrary to expectations, there was no sign that the priming effect decreased across the SOA conditions for the control subjects.

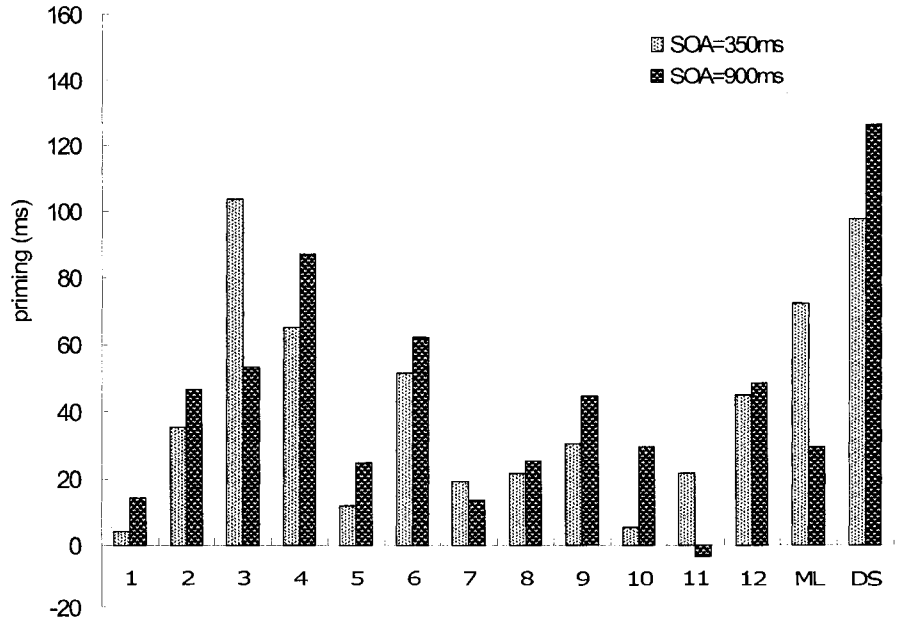
ML

ML's average response time was 905ms. At the short SOA, ML showed a priming effect of 72 ms; at the long SOA, ML's priming effect was 30 ms. ML's response time data were analyzed by items using a two-way repeated measure ANOVA. ML showed significantly faster times for the related than the unrelated condition, $F(1,63) = 4.38$, $p = .041$, $MSE = 45{,}250$. The SOA main effect was not significant, $F(1, 63) = 0.09$, $p = .765$, $MSE = 65{,}874$. The relatedness by SOA interaction was marginally significant, $F(1,63) = 3.60$, $p = .062$, $MSE = 39{,}015$, showing a tendency for a decrease in priming from the short SOA to the long SOA.

DS

The phonological STM patient DS's average response time was 822ms. At the short SOA, DS showed a priming effect of 98ms. At the long SOA, DS showed a priming effect of 126ms. DS's data were analyzed by items using a two-way repeated measure ANOVA. DS showed significantly faster response times for related than unrelated targets, $F(1,65) = 17.00$, $p < .001$, $MSE = 41{,}748$. She also showed a significant SOA main effect, $F(1,65) = 14.56$, $p < .001$, $MSE = 58{,}901$, with a shorter response time at the long SOA. The relatedness by SOA interaction was not significant, $F(1,65) = 0.50$, $p = .482$, $MSE = 50{,}924$, indicating that the priming effects at short SOA and long SOA were not significantly different.

Figure 2. Individual Priming Effects at Short and Long SOAs



The priming effects for individuals at the two SOAs are displayed in Figure 2. To our surprise, instead of showing decrease in the priming effect, 9 out of 12 control subjects showed greater effects at the long SOA than at the short SOA. Only 3 subjects showed a decrease in the priming effect.

To further evaluate whether ML's decrease of priming effect was abnormal compared to the control group, the decrease in the priming effect from the short SOA to the long SOA for each subject was calculated and entered into a modified t-test suggested by Crawford and Garthwaite (1998) (Crawford & Howell, 1998; Crawford & Garthwaite, 2002). This test takes the control group as a sample and tests whether a single case is different from the control group. Crawford and Howell (1998) showed that this test is more appropriate than the traditional method which treats control group as a population, especially when the size of the group is small. Using this test, the decrease of priming effect for ML was significantly larger than that from the control group, $t(11) = 2.054$, $p = .032$, one-tailed.

At first glance, these data would seem to suggest that ML has a faster decay of lexical-semantic information. A priming effect comparable to that of control subjects at a short SOA indicates that ML has normal lexical access, but the decrease in priming at the longer SOA seems to support a prediction following from N. Martin and Saffran (1992)'s claim of an abnormally rapid loss of activation. However, from the individual data, we can see that most control subjects and the phonological STM patient DS showed an increase in the size of priming effect from the short SOA to the long SOA. This contradicts the assumption that the priming effect in the current design only reflects automatic activation, as the effect should decay over time (Neely, 1977). It suggests that

some other process was involved and it influenced the pattern of priming observed for the controls and DS in this experiment. Most likely, the control subjects might have engaged a strategy such as expectancy generation, which would have been most effective at the long SOA (Neely et al.,1989; Hutchison, Neely & Johnson, 2001). Expectancy generation might have inflated the priming effect at the long SOA. There is some evidence that strategic effects may be obtained using a paired presentation lexical decision task, even with a low RP (Shelton & Martin, 1992). Therefore, it is possible that the procedure used in Experiment 1 was not sufficient to prevent strategy use at a long SOA. If that is the case, ML's pattern may reflect normal spreading activation and decay, but a failure to adopt strategies which would enhance the priming at the long SOA.

In order to address this possibility, Experiment 2 was conducted using another paradigm, which would be unlikely to encourage strategies, to investigate automatic priming at a long SOA.

3. Experiment 2: Automatic Priming at Long SOA with Single Presentation

In a paired presentation lexical decision task, prime-target pairs are quite obvious to the subjects. In Experiment 1, many subjects said they were aware of the relation between the primes and targets. A relatedness proportion of 20% in a paired presentation procedure may have not been sufficient to limit the awareness of prime-target relation. So some control subjects could use a strategy to aid to their response at the long SOA. In the following experiment, a single presentation semantic priming task was used.

The single presentation semantic priming task was first designed by McNamara and Altarriba (1988) to assess automatic spreading activation in priming. In this task, there is no obvious pairing of prime and target and subjects make lexical decisions to each of them. Shelton and Martin (1992) evaluated this task and provided evidence that the single presentation task could limit semantic priming to automatic processes. In their first experiment, they compared mediated priming and inhibition effects obtained between single presentation task (with a relatedness proportion of 9%) and paired presentation task (with a low relatedness proportion of 9% and a high relatedness proportion of 82%) using the same key related pairs. As mentioned in the introduction, a mediated priming effect results from automatic spreading activation; an inhibitory effect results from engaging expectancy generation. Shelton and Martin (1992) reported that mediated priming and no inhibitory effect were observed in single presentation condition, supporting the conclusion that this task taps automatic processes. Their data also showed that strategies were not totally eliminated in paired presentation task even with a low relatedness proportion, because of the presence of inhibitory effect in this condition compared to the single presentation condition. In their second experiment, they found that post-lexical checking was not present in the single presentation task. Priming for the backwardly associated pairs was not significant in the single-presentation condition and it was also significantly less than that obtained in both paired presentation conditions. Significant backward priming was obtained in both of the paired presentation conditions. These experiments provided convincing evidence that the single presentation task would be more likely to reduce strategy and tap automatic priming.

Thus, in Experiment 2, the single presentation task was used to investigate priming for ML and controls. The SOA in this experiment was quite long as it depended on the time to respond to each item and the interval between a response and the onset of next trial. In Experiment 2, a response-to-stimulus interval (RSI) of 200 ms was used. Therefore the SOA was response time plus 200ms.

Methods

Participants

Patient ML and twelve older control subjects were tested. Seven subjects had also participated in Experiment 1, which was conducted 2 months before.

Design

The only factor in this experiment was relatedness. As in Experiment 1, it was manipulated within-subjects.

Materials

Forty semantically associated word pairs were selected from the South Florida Word Association Norms (Some word pairs appeared in Experiment 1. Experiment 2 was conducted 2 months after Experiment 1.) The unrelated trials were created by re-pairing the related targets with different primes. These experimental pairs were then divided into two subsets (target frequency and association strength matched), across which the relatedness were counterbalanced. Then they were embedded into two lists consists of words and nonwords. There were 20 related word pairs in each list, but there were no obvious indication of primes and targets. Each subject saw two lists of stimuli consisting of 165 words and 135 nonwords presented sequentially in pseudorandom orders, with one week separating presentation of the two lists. The relatedness proportion was 17%. The

proportion of related pairs was less than 6% when considering all possible pairs of adjacent items.

Procedure

On each trial, a word or a nonword appeared in the center of the screen. Subjects were instructed to make a decision to each, and they were not informed of the existence of prime - target relations. To follow the paradigm of McNamara and Altarriba (1988) and Shelton and Martin (1992), the go-no-go response was not used in this experiment. Subjects were required to press 'Yes' if the string was a word and 'No' if it was not, using two fingers of their non-dominant hand. The letter string stayed on the screen until a response was made or to a maximum of 3 s. After a decision was made, the screen was blank for 200ms before the next item appeared. Each list took about 20 minutes to complete. ML and the control subjects completed one list per week for two weeks. Before each list, they started with a practice session consisting of 20 trials.

Results and Discussion

For all subjects, when an error was made on the prime or the target of the key pairs, the response time for that target was excluded from analysis. For the control subjects, 0.2% trials were deleted and for ML, 6% of trials were deleted. Furthermore, any response time shorter than 300ms and longer than 2500ms were removed for control subjects. For ML, any response time shorter than 300ms and longer than 3000ms where deleted. Finally, response times outside three standard deviations from the cell mean were excluded. In total, 2.5 % of the control data and 8.8% of the patient data were excluded from the following analyses. The mean response time data for each condition are presented in Table 2.

Table 2. Experiment 2: Mean Response Times and Priming Effects

	Unrelated	Related	priming
controls	723	683	40
patient ML	1284	1085	199

Controls

The mean response time for control subjects was 703 ms, with a range of 545ms - 929ms. The control subjects showed a priming effect of 40ms with a range of 7ms – 124ms. Using a paired t-test with relatedness as a within-subject factor, the control subjects showed a significantly faster times in the related than the unrelated condition, $t1(11) = 3.91, p = .002, SE = 10.34$; $t2(39) = 3.94, p < .001, SE = 9.76$.

ML

The average response time for ML was 1184ms, longer than that of all the control subjects. Patient ML showed a priming effect of 199 ms which reached significance, $t(32) = 2.06, p = .048, SE = 75.30$. The priming effect of ML appears to be outside the range of control subjects (7ms – 124ms). However, researchers (Giffard, Desgranges, Kerrouche, Piolino & Eustache, 2003) have shown that a numerically big priming effect (hyperpriming) may be due to long response times and large variance that is associated with the long response time, instead of reflecting abnormality in the level of activation in lexical-semantic system. Considering ML's long response time in this experiment, the ratio of priming effect to the average response time of targets under unrelated condition and the effect size (*Cohen's d*) for control subjects and ML were calculated.

The ratio of priming effect to the average response time of targets under unrelated condition was used to reduce the influence of response time on the priming effect. Control subjects had a mean of 0.05 (range 0.01 – 0.14). The ratio for ML was 0.16, slightly outside the range of controls. The effect size (*Cohen's d*) was used to control the influence of variance on priming effect. The control subjects had a mean effect size of 0.24 (range 0.08 - 0.56). ML's effect size was 0.36, within the range of the control subjects. This suggests that ML's large priming effect resulted from a large variance. When the variance is controlled, the effect size is within the range of the control subjects. The highest effect sizes from the control data were 0.34, 0.36 and 0.56. Thus, ML's priming effect size was still at the high end.

In Experiment 2, the control subjects made few errors (11 control subjects made 0% errors and one made 2.5%). ML made more errors (6%) than any controls subjects, with an error rate of 5% (two trials) in the unrelated condition and 7.5% (three trials) in the related condition.

In this experiment, the SOAs were quite long. The mean SOA for controls was 903 ms, whereas that for ML was 1384 ms (given his longer reaction times). Therefore, with a paradigm that limits the use of strategy and taps automatic activation, ML showed a significant priming effect at a long SOA. Thus, data from this experiment suggest that ML does not show overly rapid decay of semantic information. If anything, his priming effect at the long SOA in this experiment was larger than that for many control subjects.

In this experiment, we used a response-stimlus interval (RSI) of 200ms. The letter string disappeared from the screen when a response was made and after 200ms the next letter string appeared. In Experiment 1, the interval between a response and the onset of

the next trial was 1000ms. In Experiment 2, patient ML's average response time was substantially slower in Experiment 2 than in Experiment 1 and the difference between his mean RT and that of controls was much larger in Experiment 2 than Experiment 1. This is probably due to the shorter RSI used in Experiment 2 and the consequent difficulty that ML may have had in switching attention from one trial to the next within that short interval. In addition, although traditionally researchers manipulate SOA to observe the onset and decline of priming effects, the RSI may also be important while investigating the decrease of priming effect. When the prime continues to be present on the screen, it may somewhat maintain the activation of the lexical-semantic information. The decay of activation may be more observable when there is a longer period after the prime disappears and before the onset of the target. Considering these issues, Experiment 3 was conducted.

4. Experiment 3: Automatic Priming at long SOA and long RSI with Single Presentation

Experiment 3 used a single presentation lexical decision task with a RSI of 500ms. This could provide another means of assessing the persistence of automatic priming for patient ML and control subjects. If ML's long reaction times in Experiment 2 were due to the short time period between response to one stimulus and appearance of the next, the longer RSI should result in ML's reaction times being closer to those of controls, making comparison of priming effects more straightforward. The longer RSI could also allow us to determine whether priming persists over a long delay even when the prime is no longer present.

Methods

Participants

Patient ML and eleven age-matched control subjects were tested.

Design

The only factor in this experiment was relatedness. Relatedness was manipulated within-subjects.

Materials

The 80 key trials from Experiment 1 were used as the key trials in this experiment. The unrelated trials were created by re-pairing the primes to different targets. The stimulus lists were constructed in the same way as in Experiment 2. The number of key trials doubled in this experiment in order to increase the likelihood of detecting a priming effect at a long RSI. Thus, twice the filler words and nonwords were included in the stimuli to keep the same relatedness proportion. Each subject saw two lists of stimuli consisting of 330 words and 270 nonwords presented sequentially in pseudorandom order, with one week separating presentation of the two lists. The relatedness proportion was the same as in Experiment 2 (17%). The proportion of related pairs was less than 6% when considering all possible pairs of adjacent items.

Procedure

The procedure was the same as in Experiment 2 except that after a decision was made, the screen was blank for 500ms before the next item appeared.

Results and Discussion

The data were treated in the same way as in Experiment 2. 2.2% response time data of control subjects and 4.4% response time data of ML were excluded.

Table 3. Experiment 3: Mean Response Times and Priming Effects

	Unrelated	Related	priming
controls	601	607	7
patient ML	714	726	12

Controls

The mean response time for control subjects was 604ms (range 469ms – 719ms). The response time data and priming effects are presented in Table 3. When the data from control subjects were analyzed with a paired t-test with relatedness as a within-subject factor, the relatedness effect was not significant either by subjects, $t1(10) = 1.37$, $p = .202$, $SE = 3.95$, or by items, $t2(79) = 0.99$, $p = .327$, $SE = 5.97$.

ML

The average response time for ML was 720ms, slightly outside of the range of the control subjects. The priming effect for ML was 12ms, which did not reach significance, $t(72) = 0.52$, $p = .606$, $SE = 25.24$. The priming effect of ML was within the range of control subjects (-6ms – 34ms). Also, the ratio of priming effect by response time under unrelated condition was 0.017, within the range of control subjects (-0.013 – 0.050). The effect size (*Cohen's d*) of ML was .061, also within the range of the control subjects (-0.061 - 0.264). When the raw priming effect for ML and control subjects were analyzed using the modified t-test (Crawford & Garthwaite, 1998), there was no evidence that ML was different from the control group, $t(10) = 0.40$, $p = .698$, two-tailed.

In Experiment 3, with a 500ms RSI and a mean SOA of 1104 ms, the control subjects showed no significant priming effect. With a 500ms RSI and a mean SOA of 1220ms, ML's priming effect also did not reach significance. The results for Experiment 3 suggest that the activation in the lexical-semantic system has decayed to a non-significant level for both control subjects and ML. Thus, these data provide further evidence that semantic priming under automatic conditions does decay over time. Consequently, they provide further support for the argument that the increase in priming from 350 to 900 ms SOA for many controls in Experiment 1 was due to strategic effects.

In this experiment, with a RSI of 500ms, both control subjects and patient ML responded faster than in Experiment 2. Control subjects showed a decrease in response time of 99ms while ML showed a decrease of 464ms from Experiment 2 to Experiment 3. The exaggerated decrease suggests that ML may have some difficulty in switching from making a response to one trial to attending to the next trial.

Combining Experiment 1 short SOA and Experiment 3

Although Experiment 1 used paired presentation, which seems to have encouraged strategy use, it is unlikely that older control subjects could engage strategies at the short SOA of 350ms, considering the expectancy generation requires a relatively long SOA (Neely, 1977; Neely, 1991). Thus, it is reasonable to assume that at the short SOA, priming primarily reflected automatic activation. In Experiment 3, in which single presentation was used, the data presumably also reflected automatic activation. As 10 subjects and ML completed both Experiment 1 and Experiment 3, and the key trials used in these experiments were the same, the data from Experiment 1 short SOA and Experiment 3 were compared. Each subject's decrease in the priming effect from the

short SOA in Experiment 1 to Experiment 3 was calculated. From these data we can gain a picture of the decay of activation for control subjects and patient ML. (Although this comparison is not straightforward, given the many differences in experiment design between Experiments 1 and 3, the change in priming may still provide some information about expected decay across SOA.) On average the control subjects showed a decrease of 33ms in priming effect, with a range of 0ms- 113ms. ML showed a decrease of 60ms in priming effect. When ML was analyzed against the 10 control subjects using the modified t-test (Crawford & Howell, 1998), ML's decrease was not significantly larger than the control subjects, $t(9) = 0.718$, $p = .248$, one-tailed. Therefore, there is no evidence that ML has a faster decay rate in the lexical-semantic system.

To summarize, the results from the first 3 experiments provide evidence that ML's semantic STM deficit is not due to deficits of activation in the lexical-semantic system, given his normal priming effect at the short SOA in Experiment 1, nor due to overly rapid decay of lexical-semantic information, given the results of Experiment 2 and 3. Thus, the results are inconsistent with the claim that STM deficits result from impairments in the lexical-semantic system, rather than that activation in the nodes does not persist. Instead they support a model which attributes semantic STM deficits to mechanism outside the lexical-semantic system.

In Experiment 1, ML showed a greater decrease in the priming effect at the long SOA compared to control subjects, while most control subjects actually showed an increase in the priming effect. We suspected that the control subjects might have used strategies which inflated the priming effect at the long SOA. ML's decreased priming

effect might have resulted from failure to use strategies. The results from Experiment 3 supported that the control subjects used strategies in Experiment 1. The previous analyses also demonstrated that ML has normal activation and decay in the lexical-semantic system. These results further suggest that ML's decreased priming effect in Experiment 1 was due to not using strategic processes as control subjects did. Experiment 4 was conducted to further explore using strategies in priming for ML and control subjects.

5. Experiment 4: Strategic Priming

Unlike priming due to automatic spreading activation, strategy use involves controlled processing, presumably tapping attentional processes (Neely, 1977; Hutchison, 2007; Posner & Snyder, 1975). Researchers (Kane & Engle 2003; Engle, 2002) have argued that there is a close relation between attentional control and working memory or STM. Engle (2002) argued that "executive attention", which is the central executive (Baddeley & Hitch, 1974) in control of attention, has a crucial influence on working memory. It also modulates STM performance through supporting the use of grouping/chunking skills and coding strategies (Engle et al., 1999). Executive attention is also argued to modulate inhibition (Engle, 1996), which is a component of the executive function in the model proposed by Miyake and colleagues (2000). Given previous evidence of ML's difficulty in engaging in executive processes possibly related to attention control (i.e., his difficulty with inhibiting irrelevant information; Hamilton & Martin, 2005, 2007), I was interested in determining if he could use strategic processes in priming.

For this purpose, I used conditions which were highly likely to evoke the use of strategies. This experiment used a paired presentation priming task with a long prime-target SOA and a high relatedness proportion of 67% to maximize the use of expectancy generation. Also, in this experiment, word pairs with a backward association were included in the stimuli in order to detect if the control subjects and patient ML used post-lexical checking. As Experiment 1 has shown that older control subjects could possibly use strategies, and also previous studies (Burke, White & Diaz, 1987) have demonstrated that older subjects could engage expectancy generation, the control subjects were expected to show a priming effect in the forward association condition, and possibly the backward association condition. In addition, with a high relatedness proportion, the control subjects were expected to show bigger priming effects compared to the long SOA condition in Experiment 1. If the control subjects show this effect or the backward priming effect, while ML does not, this will support the view that control subjects had used strategies and ML might have problem doing that. It will also suggest that ML may have deficit in controlled processing.

Methods

Participants

Patient ML and 11 age-education matched control subjects were tested.

Design

The factors in this experiment were direction of association (forward vs. backward) and relatedness (related vs. unrelated). Both factors were manipulated within-subjects.

Materials

The 80 related pairs and 80 unrelated pairs (re-paired from the 80 related pairs) from Experiment 1 were used as the forwardly associated key trials. In addition, 40 backwardly-associated word pairs (e.g. *scout – boy*) were selected from Peterson and Simpson (1989). They were re-paired to form 40 unrelated pairs. The key trials were equally divided into two sets that half related pairs appeared in one set and the unrelated pairs with the same targets appeared in the other. Another 120 related word pairs were created as filler trials. 200 word-nonword pairs were created as the "no" response trials. Each subject completed two lists consisting of 60 unrelated pairs, 180 related pairs and 200 'no' response pairs, with a RP of 67%, and nonword ratio of 45.5%.

Procedure

The procedure was the same as in Experiment 1, except that there was only a long SOA of 900ms. Each subject completed two lists in two weeks, with an interval of at least one week. In each period, they started with 20 practice trials.

Results

Incorrect responses were excluded from the analyses of response time data. Response times shorter than 300ms or longer than 2500ms were excluded. Response times outside three standard deviations from the condition means were excluded. In total, 2.5% trials for controls and 2.9% trials for patient ML were excluded. The results are presented in Table 4.

Table 4. Experiment 4: Mean Response Times and Priming Effects

	forward			backward		
	Unrelated	Related	priming	Unrelated	Related	priming
control	658	619	39	652	645	7

ML	643	603	40	621	663	-42

The response time data were analyzed by subjects, with direction of association and relatedness as within-subject factors, and by items, with direction of association as a between-subject factor and relatedness as a within-subject factor. There was a significant relatedness main effect, $F1(1,10) = 15.95$, $p = .003$, $MSE = 358$; $F2(1,118) = 22.74$, $p < .001$, $MSE = 1{,}269$. Control subjects responded significantly faster under the related conditions than the unrelated conditions, with a mean difference of 23ms. The main effect of direction of association was not significant, $F1(1,10) = 2.43$, $p = .150$, $MSE = 480$; $F2(1, 118) = 2.40$, $p = .124$, $MSE = 3{,}117$. The relatedness by direction interaction was significant, $F1(1,10) = 13.95$, $p = .004$, $MSE = 204$, and $F2(1,118) = 11.37$, $p = .001$, $MSE = 1{,}269$, which was due to greater priming in the forward association direction than in the backward association condition.

For the forward association conditions, control subjects on average responded 39ms faster in the related condition than in the unrelated condition. A paired t-test showed that this effect was significant, $t1(10) = 7.34$, $p < .001$, $SE = 5.30$; $t2(79) = 7.11$, $p < .001$, $SE = 5.58$. The range of the priming effect for the control subjects is 18ms – 69ms. For the backward association conditions, the control subjects responded 7 ms faster under the backwardly associated condition than under the unrelated condition. This effect was not significant by subjects, $t1(10) = 0.78$, $p = .453$, $SE = 8.61$, nor by items, $t2(39) = 0.84$, $p = .406$, $SE = 8.11$. Thus, there was no significant backward priming observed for control subjects.

ML's mean response time for each condition is presented in Table 4. On the forward association trials, ML showed a priming effect of 40ms, which reached significance, $t(73) = 2.16$, $p = .034$, $SE = 19.89$. The priming effect of ML was about at the mean of the control subjects, suggesting that ML performed normally on the forward association condition. For the backward association condition, ML was 42 ms slower under the backward-related condition than the backward-unrelated condition. This effect did not reach significance, $t(38) = 1.15$, $p = .257$, $SE = 37.39$. The negative 42ms effect that ML showed was slightly outside the range of control subjects (-35ms to 62 ms). When the backward priming effect of ML was tested against those of the control sample using the modified t-test (Crawford & Howell, 1998), ML was not different from the control group, $t(10) = 1.635$, $p = .130$, two-tailed.

The control subjects made less than 1% errors on the key trials. The average error rate was 0.57% on the forward-related condition, 1.25% on the forward-unrelated condition, 1.1% on the backward-related condition and 0.91% on the backward-unrelated condition. ML made 3.75% error in the forward-unrelated condition and 0% in other conditions.

Discussion

We had anticipated that control subjects would show a larger priming effect in this experiment than in the long SOA condition in Experiment 1 and would show evidence of backward priming – both of which would support the conclusion that they had used strategies. However, the 39 ms priming effect in the forward direction was similar in size to that observed at 900 ms in Experiment 1 (37ms). Also, the backward priming effect was very small and non-significant. However, we had argued that it

appeared that most control subjects were using strategies in Experiment 1 at the long SOA; thus, the failure to observe a larger priming effect here may be due to the fact that strategies were already in effect in Experiment 1. The failure to show a backward priming effect suggests that controls were not using a post-lexical checking procedure, but does not rule out the possibility that they were using expectancy generation.

With respect to the priming data for ML, there was evidence that the priming effect was larger and more consistent in this experiment than in Experiment 1. The 30ms priming effect he showed at the long SOA in Experiment 1 was not significant and had an effect size (*cohen's d*) of 0.06. In Experiment 4, ML's priming effect of 40ms was significant, and the effect size (*cohen's d*) was 0.26, indicating much less variance in the size of the priming effect across items. The results from the two experiments suggest that ML can use expectancy generation in a priming task, but he is likely to do so only when there is a high relatedness proportion and the prime-target relation is quite obvious.

The results from Experiment 4 also suggest that both ML and control subjects did not use post-lexical checking. Since there is little literature on backward priming in older adults and patients, it is hard to draw conclusions on whether it is the case that older adults never use post-lexical checking to aid to their performance, or the manipulation used in this experiment was not sufficient to evoke using this strategy.

6. General Discussion

In this study, three experiments were conducted to investigate automatic activation and decay in the lexical-semantic system in a patient with semantic STM deficits and one experiment was conducted to explore using strategies in the patient and

older control subjects. Experiment 1 demonstrated that patient ML was normal in activating semantic-lexical representation. The result from Experiment 2 suggested that at a long SOA, activation was not weaker for patient ML compared with control subjects when a procedure tapping automatic priming was used. Experiment 3 demonstrated that at a long prime-target SOA, and with a long RSI, activation decayed for both the control subjects and patient ML. Combining results from Experiment 1and 3, we found no evidence that the activation in lexical-semantic system decayed faster for ML. Experiment 4 was developed to investigate if ML could use strategies in a priming task. The results provided some evidence that ML is able to use expectancy generation, when there is a high proportion of related trials.

The first three experiments focused on the question whether semantic STM deficits are due to impairment in the lexical-semantic system. Specifically, N. Martin and Saffran (1992) have argued that STM storage is based on the same mechanism as word processing - the activation of nodes in lexical-semantic system. The data from the current study clearly do not support this view, as patient ML showed normal activation in the lexical-semantic system and a decay pattern similar to that of the control subjects. Together with previous findings that ML has normal performance in naming and picture/word matching tasks (Martin & Freedman, 2001; Martin & Lesch, 1996), ML demonstrated intact single word processing in both explicit tasks and implicit task.

The results of the current study are thus more consistent with a model which does not assume that the activation and decay of nodes in the lexical-semantic system accounts for STM deficits. The model of Martin et al. (1999) is more consistent with the current findings in that it has proposed STM buffers separate from the lexical-semantic system.

Impairments in semantic STM are attributed to some disruption of the semantic STM buffer instead of the lexical-semantic system. Martin and colleagues (1994) have proposed that the STM buffers store the output of word processing. For the semantic STM buffer, the representations stored are semantic codes derived from word processing.

These codes in the buffer are used in explicit word recall. Impairment in STM may result from reduced capacity for the retention of codes and increased interference between the items stored in the buffer, which can be caused by brain damage (Martin & Lesch, 1996). Some other researchers have argued that attentional or control processes can also influence STM performance (Cowan, 1993; Kane & Engle, 2003). As mentioned earlier, Engle and colleagues (1999) have proposed that executive attention can modulate STM performance by supporting using grouping/chunking skills and coding strategies. The using of coding strategies could probably increase the efficiency of the semantic STM buffer. This gives some suggestions for future research. One direction could be to investigate how control processes influence STM or whether the STM deficits in patients are related to impaired control processes.

The current study has provided evidence that the semantic STM deficits are not due to problems in the lexical-semantic system. Above, I discussed some possible sources of semantic STM deficits outside the lexical-semantic system based on the model of STM proposed by Martin and colleagues (1999). Clearly, further research is needed to investigate the mechanisms of STM. Patients with STM deficits will continue to be a valuable source of data.

There are several limitations in the current study. First, the experiments were conducted across about twelve months. Many control subjects were not able to participate

in all the experiments. Some new control subjects participated in the next experiment. Therefore, some of the control subjects had different familiarity to the experimental paradigm and some stimuli. This makes it hard to compare across experiments. Second, different paradigms were used between experiments. In Experiment 1, all subjects responded in a go-no-go fashion, while in other experiments a Yes-and-No response paradigm was used. In the future it would be better if all experiments use the same response method. Third, in this study, 11 to 12 control subjects were tested in each experiment. This might be sufficient in providing a comparison to the patient ML. But it might not be enough for observing some effects in the control subjects. For instance, the relatedness proportion effect, that is priming effect increases as the relatedness proportion increases, is usually observed with large numbers of subjects. Fourth, this study focused on one semantic STM patient ML. DS was only tested on Experiment 1. It is important to test more patients in the future – both those with semantic and phonological STM deficits – to determine if the results hold up for other patients. It is possible that for some subset of patients with semantic STM deficits, overly rapid decay is at work. If so, the different types of STM deficits may give rise to different patterns of performance in other language and memory tasks.

7. Conclusions

To conclude, the current study demonstrated that the impairment of semantic STM for patient ML is not due to a problem in slow activation or rapid decay in the lexical-semantic system. Further research is needed to investigate the mechanism underlying the deficits of semantic STM patients and the mechanism involved in

maintaining information in the STM buffer. The control processes that have been shown to influence STM performance should be further examined in other tasks, including priming tasks to examine the generality of semantic STM patients' control deficits across various verbal tasks. Of course, stronger evidence for the use of control processes by control subjects than was obtained in Experiment 4 would be critical for evaluating patient performance. Despite these remaining questions regarding the nature of control processes acting on lexical and short-term memory representations, the present experiments provide strong evidence against the notion that ML's semantic STM deficit arises from deficits in the lexical-semantic system per se. Of course, it will be important to replicate these findings in additional patients showing his STM pattern and to extend the findings for patients like DS who show a phonological STM deficit.

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Appendix A: Forwardly Associated Word Pairs for Experiment 1, 3 and 4

Prime	Target	Prime	Target
orchard	apple	razor	blade
march	april	fighter	boxer
beauty	beast	paint	brush
sweep	broom	alarm	clock
table	chair	washer	dryer
circus	clown	vase	flower
reef	coral	spoon	forks
demon	devil	ghoul	ghost
court	judge	shame	guilt
tiger	lions	famine	hunger
clam	oyster	chain	links
spinach	popeye	death	lives
jigsaw	puzzle	belt	pants
seafood	shrimp	guardian	parent
cigar	smoke	venom	poison
web	spider	pint	quart
monument	statue	cup	saucer
bathroom	toilet	honey	sweet
grain	wheat	victory	winner
handbag	purse	curse	witch

Prime	Target	Prime	Target
length	width	cycle	bikes
knight	armor	rug	carpet
shore	beach	camel	desert
tumor	cancer	trench	ditch
sofa	couch	argue	fight
swear	curse	jam	jelly
mule	donkey	orange	juice
dresser	drawer	tarzan	jungle
roman	empire	major	minor
bride	groom	thread	needle
penny	nickel	niece	nephew
doctor	nurse	sound	noise
salt	pepper	stone	rocks
socks	shoes	dressing	salad
bath	shower	buyer	seller
grief	sorrow	athlete	sport
guitar	string	step	stair
pole	vault	zebra	stripe
server	waiter	saber	sword
wrist	watch	racket	tennis

Appendix B: Associated Word Pairs for Experiment 2

Prime	Target	Prime	Target
meat	steak	pizza	cheese
server	waiter	mule	donkey
length	width	raisin	grape
famine	hunger	sheriff	deputy
orange	juice	sofa	couch
ape	monkey	thread	needle
cards	poker	lion	tiger
grief	sorrow	doctor	nurse
cup	saucer	buyer	seller
jigsaw	puzzle	seafood	shrimp
salt	pepper	grain	wheat
tumor	cancer	pint	quart
beauty	beast	washer	dryer
fool	idiot	belt	pants
web	spider	bride	groom
athlete	sport	niece	nephew
handbag	purse	penny	nickel
saber	sword	seashore	shell
circus	clown	bath	shower
curse	witch	demon	devil

Appendix C: Backwardly Associated Word Pairs for Experiment 4

Prime	Target	Prime	Target
fire	camp	market	flea
wars	star	ache	stomach
water	duck	top	roof
bag	sleeping	liberty	statue
mask	catcher	light	lamp
button	belly	book	check
wood	termite	clothes	washing
hole	doughnut	string	puppet
person	famous	air	fan
board	bulletin	foot	bare
dry	towel	cloth	table
belt	seat	yard	barn
aid	first	dishes	dirty
control	birth	blue	denim
legs	crab	butter	peanut
seek	hide	shadow	eye
stop	bus	bite	mosquito
bucket	ice	truck	dump
lost	maze	image.	mirror
cake	cheese	moon	harvest