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LEXICAL-SEMANTIC PRIMING AND DEMENTIA

Linda S. Carozza^(A,B,D,E,F)

Communication Sciences and Disorders, St. John's University, New York City,
New York, USA

SUMMARY

Alzheimer's disease is marked by loss of semantic memory. The neuropsychological experimental task known as priming (wherein recognition or recall is enhanced by prior exposure) is a pure measure of semantic memory. The study of the nature of the underlying semantic memory impairment in dementia has progressed through the contribution of the priming literature. This article will review past and current trends in priming research specifically related to Alzheimer's disease. The methodologies and findings of previous experimental literature will be discussed in attempts to demonstrate the controversies and scientific evidence-building regarding the language deterioration in dementia of the Alzheimer's type.

Key words: semantic memory, Alzheimer's disease, attention, memory

INTRODUCTION

In 1995, Carozza conducted an experiment to examine the cognitive processing operations of 18 healthy elderly (HE) and 12 Alzheimer's disease (AD) subjects in the mild clinical stages. A semantic priming test was administered to assess the semantic lexical activations in both automatic and controlled processing operations.

At the time Carozza conducted her research, it was reported that relatively little conclusive evidence was documented regarding the relative roles of attention and memory processing in the lexical-semantic impairment of AD. Temporal boundaries that reflected attention-dependent and non-attention dependent processing had indications in the literature, particularly in healthy young subjects. The research design was based on the previous successful procedural use of priming techniques with HE and AD subjects. A lexical decision task was implemented to investigate the effects of normal aging and neuropathological damage of AD on the subjects' semantic priming abilities. Carozza found that HE subjects demonstrated priming of a facilitative nature at both automatic and controlled temporal processing boundaries, whereas AD subjects demonstrated priming due to inhibition at long controlled temporal processing boundaries.

The purpose of the present study is to review the relevant research that has taken place in the area of lexical priming and dementia.

BACKGROUND

In the past 20 years, there has been much debate over the nature of the memory disorder associated with dementia of the Alzheimer type (DAT). The overall memory decline appears to be significantly related to the breakdown in the ability to form semantic associations, in which distinct operating mechanisms interrelate to serve our normal daily functions. Science attempts to systematically delineate the substrate systems, without accounting for interrelations, for purposes of clear explanation of the memory deficit.

In keeping with this notion, Salmon and his colleagues (1988) examined the ability of DAT subjects to form semantic relationships under "automatic," i.e., non-attention-dependent conditions and "effortful," or attention-dependent conditions. Their DAT subjects exhibited a priming deficit on a stem-completion task, which is a paradigm of associative priming. The DAT subjects also exhibited a significantly depressed ability to produce semantic pairs, which they interpreted as related to processing disorders in both domains, automatic and effortful processing.

Although priming had been cited as aberrant in DAT, there had been many contradictory reports in the literature since the 1980s. An early study by Ober and Shenaut (1988) reported an abnormal priming effect compared to typical priming results, providing evidence that DAT involved an increased susceptibility to lateral inhibition in the semantic network. This was followed by a re-

port by Chertkow, Bub, and Seidenberg (1989), in which semantic priming in Alzheimer's disease (AD) subjects was found to be specifically impaired in a lexical decision task. The authors reported greater semantic priming in the AD subjects relative to age-matched normals, a phenomenon known as "hyperpriming." This finding was not consistently reported in the literature; however, it has been associated with degradation of the lexical activation centers, due to the fact that targets are "over stimulated" in response to a loss of integrity of semantic features. The authors stated that these unexpected results called for greater investigation into the changes in semantic memory structure associated with dementia.

The literature indicated that there were priming behavior differences in dementia that appeared related to semantic memory loss or disorganization. The role of attention in the priming results was investigated by Nebes, Brady, and Huff (1989). Prior studies pointed to the failure of automatic priming in the sense that the behavior was not under conscious control of the subjects. The study aimed to shed light on the role of attention-dependent processes. If priming were due to only automatic processes, then there would be no priming under attention-dependent conditions. Their findings were that subjects exhibited priming effects under both conditions, indicating that priming in dementia may be associated with both automatic (non-attention-dependent) and attention-dependent operations. However, demented subjects exhibited greater semantic priming than either normal group on both tasks, which may be related to underlying semantic memory organization issues. In addition, the literature suggests that when only automatic conditions are considered, spared priming may be seen in AD subjects, but it is difficult to ascertain if the obtained effect is due to facilitory or inhibitory factors and implications may be obscured.

Throughout this literature, there is a problem with the use of various priming methodology as a possible cause of discrepant results. Glosser and Friedman (1991) used a threshold oral reading task. They attempted to differentiate the lexical versus semantic aspects of priming.

Healthy elderly patients had priming effects for both associatively and semantically associated, whereas AD subjects showed effects for only associatively related words, but not for word pairs that shared semantic features. These results confirm that semantic processing is impaired in AD, but suggest differential impairment and may reflect independent networks that are vulnerable to the neurological damage associated with the disease.

The dissociative parameters of priming were examined by Keane and colleagues (1991). In a comparison of word completion and perceptual priming, AD patients demonstrate differences, with word completion priming being impaired, reinforcing the notion of the primary semantic component of their abnormal priming behavior, and thereby implicating different brain regions (temporo-parietal lobe) as compromised in AD.

In a separate study by Gabrieli et al. (1994), the AD patients had impaired word-completion priming with intact incomplete-picture priming, a form of perceptual priming; this dissociation supported prior research as cited above. The various studies were accumulating to suggest evidence for neuropsychological differences in patient groups that inferred differential brain damage supported by priming results.

Nebes, R.D. (1994) has been one of the major contributors to the area of Alzheimer's disease research. He reported that studies of obtained semantic priming effects (facilitation of lexical processing by a prior semantic context) could be interpreted to infer intact semantic-memory structure in Alzheimer's disease, and clarified that the priming produced by single-word primes may reflect the facilitation of preexisting lexical associations and not intact semantic memory in AD.

The work of Carozza in 1995 as part of her doctoral dissertation tested two attention dependent variables, an interstimulus interval of 1500 ms as compared to an interstimulus interval of 500 ms. Healthy elderly subjects demonstrated significant priming effects at both intervals, whereas AD subjects demonstrated no priming at the shorter interval and priming effect (hyper-priming) effect at the longer interstimulus interval. Results were attributed to possible difficulties in redirection of lexical search, in keeping with certain earlier reports.

In 1998, Fleischman and Gabrieli studied repetition priming in normal aging and Alzheimer's disease. While repetition priming is a kind of implicit memory that has initially been believed to be immune to the effects of aging and greatly reduced in Alzheimer's disease, findings have been controversial. The variation in priming abilities that occurs with normal aging and AD is attributed to three factors, namely, measurement issues, individual differences among participants, and dissociable processes. The measurement issues and methodological differences of these studies continue to be a subject that is examined in this literature. Furthermore, the difficulty in exact matching of patients with dementing processes is an additional confound. Nevertheless, there is evidence that priming is dissociable by task.

Glosser and colleagues (1998) went on to examine lexical semantic and associative priming in Alzheimer's disease. Semantic memory impairment was investigated in patients with probable Alzheimer's disease using a threshold oral word reading task. Healthy elderly controls showed significant priming for associatively related nouns, such as "tempest-teapot" and also for nouns semantically related either because both designate basic-level exemplars of a common superordinate category i.e., cousin-nephew, or because the target names in the superordinate category of the prime as in daughter-relative. AD patients, however, revealed preserved priming of lexical associates but impaired priming of certain semantic relationships (no priming between words designating coordinate exemplars within a category, despite preserved priming of the superordinate category label). Findings suggest that

part of the semantic deficit in AD is due to disruption of semantic knowledge that affects relationships among basic-level concepts, more than the relationships between these concepts and their corresponding superordinate category of membership. This has relevance for the nature of semantic degradation and deterioration of semantic organization.

Nakamura et al. (2000) AD studied priming in Japanese patients. The AD subjects exhibited priming whereas other experimental groups did not. The small number of patients in the study was a limitation, although the findings still tended to support those of larger scale studies. What was becoming clearer, however, was the fact that normal priming was not an indicator of intactness and required further analysis via experimental design.

Research by Bell, Chenery and Ingram (2001) demonstrated that subjects with dementia of the Alzheimer's type showed no automatic priming, but did exhibit hyperpriming. They indicate that this effect has implications for disorder in the spread of activation in related brain areas. Their paradigm manipulated relatedness proportion and stimulus onset asynchrony, allowing them to detect dissociations. Demented subjects performed differently, with larger facilitation effect than the normal expectations.

Padovan (2002) studied affective priming, i.e. the nature of the primes in terms of positive and negative emotional value. Her subjects revealed preserved automatic activation. This evidence indicated that properties of language may be differentially affected. Positive information may be more vulnerable to AD changes. This research suggested that AD subjects have reduced capacity for automatic priming and that their deficits lie in controlled recall. The patient groups of this and other studies exhibited variations in priming with resultant differing explanations of semantic memory intactness.

Manenti et. al. (2004) studied the effects of aging and Alzheimer's disease on semantic and gender priming. In this case, Italian words which were either masculine or feminine were used in an on-line reading test. In contrast to normal young and old subjects, AD patients had a significant interference and facilitation effects on congruent and incongruent semantic and grammatical information. The findings suggested that AD involves an impairment of inhibitory mechanisms. When AD patients performed tasks that involve some degree of controlled inhibition, they showed signs of impairment, but automatic tasks appeared to be unaffected. The use of so-called automatic versus controlled operations in experimental design was helpful in clarifying when effects occurred, thereby implicating various subsystems.

Recently, Cummings (2006) reported hyperpriming as due to cognitive slowing. The hyperpriming phenomenon in and of itself is an abnormal finding. The underlying disturbance or slowing in activations is still not clear in terms of an explanation, and hyperpriming has not been found consistently across studies, as has been demonstrated.

The fact that priming is a reliable factor for analysis in dementia, however, was underscored by the longitudinal work conducted by Verfaellie et al.

(2006). Their one subject showed normal priming in years 1 and 2 of his disease and then exhibited changes in priming results in years 3 and 4 of the dementing process.

The nature of the type of information that is destroyed was examined by Hajilou (2007), whose research proved that both structural and semantic knowledge is impaired in dementia. Therefore, when impaired priming is found, it is occurring on a number of levels, intertwined to the point that only the most sophisticated designs, if any, can identify the components.

Perhaps the most promising report in this paper is the one provided by Giffard and colleagues (2008). They examined the neural substrates of semantic memory deficits in early Alzheimer's disease by mapping the correlations between resting-state brain glucose utilization measured by FDG-PET and semantic priming scores in a group of 17 AD patients. The priming scores correlated positively with the metabolism of the superior temporal areas on both sides of the subjects' brains, especially the right side, and this correlation was shown to be specific to the semantic priming effect. This pattern of results suggests that a dysfunction of the right superior temporal cortex may contribute to early semantic deficits, characterized by the loss of specific features of concepts in AD. The relative contributions of both hemispheres of the brain to components of semantic memory structure are made clear by this investigation. The addition of correlative physiological studies to priming research is extremely promising in clarifying the findings in this area of study.

SUMMARY AND CONCLUSIONS

The work of two decades of research in Alzheimer's disease and its related semantic deterioration is described in an effort to elucidate the various findings that were obtained and the controversies that continue to exist, using the original work of Carozza (1995) as a central discussion point for comparison and contrast. The semantic memory impairment of dementia appears specific to dementia related to Alzheimer's disease. Priming behavior is aberrant in early disease stages, so that changes in priming may be considered an early disease marker. This is highly significant in that although laboratory diagnosis has certainly advanced to the point of approximately 90% certainty, science in this area is greatly assisted by skilled clinical assessment. Language changes exist that have characteristics which can be further clarified by manipulation of the priming methodology. As opposed to obscuring diagnoses, the variants of priming methods and the addition of variables such as repetition priming and form priming and relatedness proportion and timing variables demonstrate that the semantic memory network is exceedingly complicated in its organization. This is a potential explanation for the discrepant reports, in that researchers are looking at a complicated phenomenon from many different perspectives, such that only a specific aspect of operations is revealed. The fact that subjects may demonstrate priming is fur-

ther clarified when inhibition (unrelated primes causing a delay in response recognition) is considered, and furthermore when conscious and non-conscious operations are examined. Although this methodology has advanced considerably, it still can be stated that on line measures are not fully capable of elucidating operation failure. The combination of physiological measurement of metabolic activity studies and electrophysiological measurement has been invaluable in describing the underlying mechanisms for the abnormal priming behavior seen in these patients.

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Address for correspondence:

Linda S. Carozza, Ph.D.
St. John's University
St. John's College
Communication Sciences and Disorders
300 Howard Ave.
Staten Island, NY 10301
drlcarozza@aol.com