

Visual anti-priming: Tests of amnesic patients and older controls

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INTRODUCTION

Neocortical areas that underlie visual object identification appear to utilize partially overlapping representations of objects (Haxby et al., 2001; Tanaka, 1993). We have hypothesized that **priming** for an object reflects changes in weighted connections between units that strengthen the object's distributed representation (Marsolek, 2003). Such changes are needed to counteract the detrimental interference from previous changes that strengthened the representations for other objects with which it is partially superimposed. This detrimental effect has been demonstrated in several studies with young adults and is referred to as **anti-priming** (Marsolek, Westerberg, & Deason, 2004).

While the demonstration of anti-priming is consistent with the theory that small changes in superimposed shape representations occur during object identification, another possibility is that the effect simply reflects episodic interference from previously viewed items. A participant may either attempt to draw from his or her previous encoding experiences or try to inhibit the influence of these experiences. This possibility was examined in the current study, in which anti-priming was tested in patients suffering from global amnesia.

METHODS

Nine amnesic patients and 10 matched controls (see Table 1) engaged in a 2 part experiment separated by a short break. In the first part, a baseline measure of familiar object recognition is obtained (uninfluenced by positive priming or anti-priming, and against which both priming and anti-priming is measured). In the second part, participants view an additional set of different visual objects, and after that they name that set of objects again (to measure positive repetition priming) intermixed with another set of new objects (to measure anti-priming).

Patient	Etiology	Age	Ed.	WAIS-III			WMS-III		
				VIQ	GM	AD	VD	WM	
1	Anoxia	75	18	113	75	80	72	102	
2	Anoxia	48	14	111	59	72	52	93	
3	Encephalitis	49	14	92	45	56	55	96	
4	Thalamic Stroke	63	12	84	73	67	84	99	
5	Anoxia	52	12	83	52	56	55	81	
6	Anoxia	44	14	90	45	53	52	91	
7	Anoxia	20	12	91	45	46	56	102	
8	Encephalitis	61	12	106	69	77	68	111	
9	Anoxia	51	17	134	70	67	75	88	

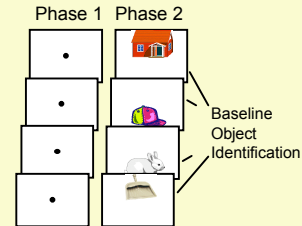
Patient Mean 51 13.9 100 59 64 63 96
Control Mean 55.4 13.7 106

Table 1 - Note - Age and education is in years; WAIS-III, Wechsler Adult Intelligence Scale, 3rd edition [Wechsler, 1997 #1179], VIQ = verbal IQ, Wechsler Memory Scale, 3rd edition [Wechsler, 1997 #1279] - GM = general memory index.

Part 1 – Baseline level of identification

•In phase 1, participants judged whether they liked or disliked each of 50 **auditorily** presented common objects (3 sec).

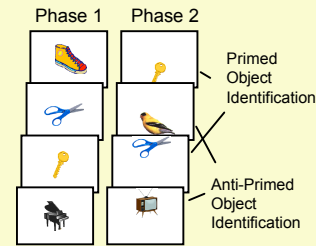
•In phase 2, participants named 100 items that were briefly (33.4 msec) presented either above or below center fixation.



Part 2 – Primed and anti-primed level of identification

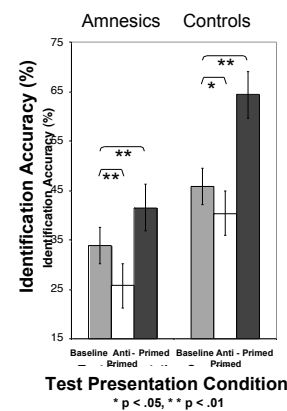
•In phase 1, participants judged whether they liked or disliked each of 50 **visually** presented common objects (3 sec).

•In phase 2, participants named 100 items that were briefly (33.4 msec) presented either above or below center fixation. Fifty of these items were repeated from phase 1 and 50 were novel items.



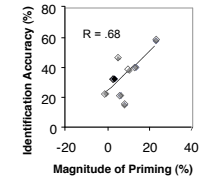
RESULTS

The results are consistent with the hypothesis that anti-priming does not reflect influences from episodic memory. While identification levels were lower for amnesic patients overall ($F(1,17) = 8.03, p < .05$), both patients and controls displayed significant priming and anti-priming ($F(2,34) = 66.8, p < .001$). In addition, there was a significant interaction with group ($F(2,34) = 5.14, p < .05$), this reflected a greater level of priming in controls versus patients with no difference in the level of anti-priming.



The finding of a lower level of priming in amnesia is not consistent with some of the literature on priming in amnesia. However, the current study used a fixed presentation rate that was not titrated to optimized the range of identification on an individual basis. When the level of priming was normalized to the overall identification rate, there was no difference in priming between amnesics and controls ($F < 1$).

This relationship between priming and overall identification was supported by a significant correlation within the patient group.



CONCLUSIONS

• Inconsistent with the hypothesis that the previously identified anti-priming phenomenon reflects interference from episodic memory, anti-priming was found to be intact in patients suffering from global amnesia.

• Since amnesic patients are not impaired at object recognition, this would be expected if anti-priming reflected the detrimental effect of the weight changes between distributed nodes that occurs with repetition priming.

• While the level of priming in patients was lower than controls, the level of anti-priming did not differ between groups. This is also consistent with the model, since priming in this task only reflects the benefits of a single previous exposure while anti-priming reflects the cumulative effect of exposure to several different objects during visual encoding.

• Future work will focus on testing the level of anti-priming in patients with damage to occipital/temporal regions associated with object identification.

ACKNOWLEDGEMENTS

This work was supported by a career development award from NIMH – K23 MH64004 and by NINDS grant NS26985, both to Boston University.