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COGNITIVE INHIBITION IN OBSESSIVE-COMPULSIVE DISORDER: APPLICATION OF A VALENCE-BASED NEGATIVE PRIMING PARADIGM

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Abstract. We used a negative priming paradigm to test for deficits in cognitive inhibition in patients with obsessive-compulsive disorder (OCD), and to examine whether they exhibit greater inhibitory deficits when lexical targets are threat-related than when they are neutral. The results indicated that OCD patients, relative to healthy control participants, exhibited only marginally significant ($p < .10$) deficits in negative priming at short (100 ms), but not long (500 ms), stimulus onset asynchronies. There was no evidence that OCD patients exhibited disproportionate difficulty inhibiting negative words, nor was there any evidence that negative priming deficits differed between OCD checkers and OCD noncheckers.

Keywords: Negative priming, Tipper paradigm, cognitive inhibition, obsessive-compulsive disorder.

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Introduction

Experimental psychopathologists have used cognitive psychology methods to elucidate mechanisms producing intrusive thoughts in obsessive-compulsive disorder (OCD; McNally, 2000). Applying variants of Tipper's (1992) negative priming paradigm, Enright (1996) found that OCD patients, especially checkers, exhibit deficits in cognitive inhibition. People with OCD, however, do not report difficulty inhibiting distracting information in general, but rather report difficulty inhibiting specific disturbing thoughts. Therefore, we endeavoured to replicate Enright's findings and to test whether OCD patients exhibit disproportionate negative priming deficits for threat words relative to neutral words.

Method

Participants

Twenty-six OCD patients (13 women) and 19 healthy controls participated (11 women). The groups did not differ significantly (*ps* > .05) in either years of age (OCD: $M = 39.5$, $SD = 12.6$ versus control: $M = 38.7$, $SD = 14.2$) or education (OCD: $M = 16.0$, $SD = 2.8$ versus control: $M = 17.1$, $SD = 2.5$).

Materials and procedure

MacLab software, running on a Macintosh Powerbook, presented stimuli and recorded vocal response latencies. Each trial began with a 500 ms exclamation point replaced by a white fixation cross that remained at center screen for 500 ms. The cross was replaced by a priming pair that remained on the screen for either 100 ms (short stimulus onset asynchrony [SOA]) or 500 ms (long SOA). Short SOA trials presumably tap a more automatic process than do long SOA trials (Enright, 1996). The priming pair consisted of a threat word and a neutral word (except on nonlexical priming trials when it consisted of a *oooo* and *xxxx*). One word appeared in green letters and the other word appeared in red letters. The top word appeared just above center screen and the bottom word appeared just below center screen. The colours of the members of the priming pairs were counterbalanced for position (above and below center screen) across all stimulus presentations. Participants were instructed to attend to the red word and to name quickly the target word that replaced the priming pair. The target word appeared at center screen in red letters.

All stimuli appeared in lower case letters. For nonlexical priming trials, the priming pair comprised two nonlexical stimuli (i.e., *oooo* and *xxxx*). On half of the nonlexical priming trials, the *ooo* was red and the *xxxx* was green, whereas on the remaining trials the colours were reversed. For positive and for negative priming trials, the priming pair comprised one of the following: *chorus* and *poison*, *singer* and *hazard*, *conduct* and *danger*, *ballet* and *disease*, *rhythm* and *cancer*. Selected by OCD therapists, the threat words reflected common contamination concerns and the neutral words reflected musical themes. On positive priming trials the (red) target was the same word as the red member of the priming pair, whereas on negative priming trials, the (red) target was the same word as the green member of the priming pair. Each threat word always appeared with the same neutral word.

Half of the participants in each group received all short SOA trials before the long SOA

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trials, whereas the remaining participants received the long SOA trials before the short SOA trials. For each SOA type, there were 10 trials each for positive priming/neutral target, positive priming/threat target, negative priming/neutral target, and negative priming/threat target. There were 20 trials each for nonlexical priming/neutral target and nonlexical priming/threat target because we combined trials consisting of a green *oooo*/red *xxxx*. We generated four random sequences of stimulus presentation, and balanced sequences across participants and across groups. Because of unequal group sizes (n = 26 and n = 19), counterbalancing was perforce less than complete.

Results

For each participant, we calculated mean response latencies for short and long SOA trials separately. For each SOA type, we calculated mean response latencies for neutral targets when preceded by a nonlexical priming pair, by a positive priming pair, and by a negative priming pair. We calculated the same values for threat targets. To control for overall differences in naming speed, we calculated priming indices and conducted all analyses on these. That is, for short and long SOAs separately, we computed, for each participant, a negative priming index for threat (or neutral) targets by calculating the difference between the mean response latency for nonlexical priming trials having threat (or neutral) targets and the mean response latency for negative priming trials having threat (or neutral) targets.

Do OCD patients exhibit impaired negative priming overall relative to control participants? To test this hypothesis, we first averaged the negative priming indices for threat and neutral targets within each group prior to computing a planned contrast that provided marginal support for this hypothesis for 100 ms trials, $t(43) = 1.45$, $p < .10$, $r = .22$, but not for 500 ms trials, $t(43) = 0.46$, *ns*, $r = .07$. Repeating the aforementioned analyses, we found no evidence that OCD checkers (n = 8) exhibited less negative priming than noncheckers (n = 18) for either 100 ms or 500 ms trials, $ts(24) < 1$, *ns*.

Do OCD patients exhibit disproportionately defective inhibition for threat cues relative to neutral cues, compared to control participants? For neither short nor long SOA trials was there any evidence for this hypothesis, $t(43)$s < 1.

Discussion

OCD patients exhibited marginally significant negative priming deficits at short SOAs, but no greater for threat than for neural words. Checkers were not especially impaired.

Procedural differences may account for the tepid corroboration of Enright's (1996) results (e.g., members of the priming pair did not overlap on the screen). Also, because of the high-speed presentation on the 100ms SOA trials, participants had no time to read aloud the red member of the priming pair. Accordingly, we cannot be certain they always attended to the red word. Furthermore, the target appeared alone. Had it appeared with a distractor, greater priming effects might have occurred in the control relative to the OCD group. On the other hand, an exceptionally rigorous study recently revealed no evidence of negative priming deficits in OCD patients, implying the effect may not be that robust (MacDonald, Antony, MacLeod, & Swinson, 1999).

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