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An experimental manipulation of metacognition: A test of the metacognitive model of obsessive-compulsive symptoms

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ABSTRACT

The metacognitive model of obsessive-compulsive symptoms [Wells, A. (1997). *Cognitive therapy of anxiety disorders: A practice manual and conceptual guide*. Chichester, UK: Wiley] assigns a necessary causal role to metacognitive beliefs in the development of symptoms. The current study tested the model by evaluating the effects of experimentally manipulating such beliefs. A 2 × 2 factorial design was used. Thirty-two students with high and 32 students with low obsessional symptoms were subject to an experimental (metacognitive belief induction) or control (no metacognitive belief induction) condition. All participants underwent fake EEG recordings and were informed that the EEG could sense hypothalamus activity caused by having thoughts related to drinking. Participants in the experimental condition were told that if such thoughts were detected they may be exposed to an aversive noise. Controls were told that they may hear an aversive noise but this would be unrelated to the thoughts they had. Results showed a significant interaction effect between level of obsessional symptoms and belief induction. Analysis of this effect demonstrated that in the high obsession group, participants in the experimental condition had significantly more intrusions about drinking, time spent thinking about these intrusions and discomfort from them, than controls. There were also significant main effects on some measures, such as effort to control intrusions about drinking, with higher scores in the experimental condition irrespective of levels of obsessional symptoms. Results support the metacognitive model.

Introduction

The metacognitive model of obsessive-compulsive (o-c) symptoms (Wells, 1997, 2000; Wells & Matthews, 1994) proposes that intrusive thoughts or mental experiences are misinterpreted because of metacognitive beliefs. Wells (1997, 2000) extended Rachman's (1993) term of Thought–Action Fusion, originally used to refer to a cognitive distortion, when labelling these beliefs as: Thought–Event Fusion, Thought–Action Fusion and Thought–Object Fusion. They concern the idea that thoughts and/or feelings can directly cause events to occur in the world or signal that situations must exist, can directly cause unwanted behaviour/actions, or can contaminate or fuse with objects. Once activated, these beliefs lead to worry about the occurrence of intrusions and elevated discomfort. The discomfort and threat prime beliefs about rituals which then guide the use of neutralizing and coping behaviours. These responses backfire and increase symptoms as well as the stability of metacognitive beliefs. (For a review of evidence for the model and an in-depth contrast between the metacognitive and other Obsessive-Compulsive Disorder (OCD) models see Fisher, 2009.)

Thought-fusion beliefs have a central causal role in the development of o-c symptoms in the metacognitive model. A key feature of the model is that irrespective of the content of both intrusions and beliefs about the self or the world, o-c symptoms are caused by a small set of specific metacognitions concerning the power and significance of thoughts and how to react to them. Therefore, thought-fusion beliefs can concern neutral thoughts and can lead these to become the source of o-c problems. For example, thinking of a certain number (e.g., the number 3) could become the focus of an obsession if a person believes its occurrence is likely to cause harm to self or others (an example of thought–event fusion).

The Thought Fusion Instrument (TFI; Wells, Gwilliam, & Cartwright-Hatton, 2001) was developed to measure the three domains of fusion-related metacognitive beliefs and it has been used to test the model. Studies have demonstrated a significant and positive association between the TFI and o-c symptoms cross-sectionally (Gwilliam, Wells, & Cartwright-Hatton, 2004; Myers,

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Fisher, & Wells, 2009a; Myers & Wells, 2005; Solem, Myers, Fisher, Vogel, & Wells, 2010) and prospectively (Myers, Fisher, & Wells, 2009b).

Support for the causal role of thought-fusion metacognitive beliefs comes from experiments; these have mainly looked at thought–**event** fusion and most have examined the effects of inducing it (but see Fisher and Wells (2005) for a study that supports the role of thought-fusion by showing the effects of **reducing** these beliefs). Studies that have examined the effects of the experimental induction of thought–event fusion were originally conceived as inducing thought–action fusion in line with the conceptualisation of Rachman et al. (Rachman, 1993; Rachman, Thordrarson, Shafran, & Woody, 1995; Shafran, Thordarson, & Rachman, 1996). However, the metacognitive model with its taxonomy of fusion beliefs interprets these studies as manipulating thought–**event** fusion as they are concerned with the fusion of thoughts with events namely harm happening to others. Most of these studies (e.g., Bocci & Gordon, 2007; van den Hout, Kindt, Weiland, & Peters, 2002; Rachman, Shafran, Mitchell, Trant, & Teachman, 1996) have attempted to induce thought–event fusion using the "sentence paradigm" devised by Rachman et al. (1996). This consists of participants completing the sentence "I hope _____ is in a car accident", by putting a friend or relative's name in the blank. Participants are then asked to visualise the accident. Studies using the sentence paradigm have shown it induces OCD-like symptoms i.e. an urge to neutralize and anxiety. These findings have been interpreted (Shafran & Rachman, 2004) as implying that the paradigm is inducing thought-fusion and that this is producing symptoms.

A limitation of the sentence paradigm is that it does not directly manipulate thought–event fusion beliefs but rather attempts to induce pre-existing beliefs. A further limitation was raised by van den Hout et al. (2002). In their study they found that even when participants were prevented from neutralizing following the sentence paradigm, their anxiety levels decreased quickly. Van den Hout et al. suggested that thinking about an accident occurring to a loved one has an immediate impact on anxiety as would any thought relevant to a person's wellbeing. However, this dissipates quickly when it is fully processed and the person realises the thought is not important. Thus, they questioned the effectiveness of the paradigm at provoking thought-fusion. Alternatively, the paradigm may not lead to the activation of significant thought-fusion beliefs in non-clinical samples such as in van den Hout et al.'s study (Shafran & Rachman, 2004). This would limit the usefulness of the sentence paradigm in non-clinical studies.

Rassin, Merkelbach, Muris, and Spaan (1999) used a different, innovative paradigm to manipulate thought–event fusion experimentally. They gave participants a fake EEG recording session and told them that if they had the thought "apple" this would be picked up by the machine. The experimental but not the control group was informed that having the thought "apple" would lead to an electric shock being given to another participant which they could interrupt by pressing a button. They found that the experimental group had more "apple" intrusions, discomfort and put more effort into avoiding the target thought.

This study supports the role of thought–event fusion in OCD. However, it has several limitations particularly in testing the role of thought–event fusion as a belief proposed in the metacognitive model. These are:

1) The experimental group was told they could interrupt the signal causing the electric shock by pressing a button soon after having the "apple" thought. This complicates interpretation of the findings regarding the role of thought–event fusion as more than one independent variable is being manipulated in the experimental group. Both thought–event fusion beliefs about the importance attached to the apple thought, and beliefs about rituals concerning the effect of behaviours carried out in response to the thought (i.e. coping) are being manipulated.
2) This study used participants whose o-c symptom levels were not assessed. There is support for the theory that OCD exists on a continuum (see e.g., Belloch, Morillo, Lucerno, Cabedo, & Carrio, 2004; and review in Gibbs, 1996) and thus it is expected that inducing thought-fusion should produce OCD-like effects even in people with low symptomology. However, according to metacognitive theory these effects should be stronger in people with high levels of o-c symptoms or OCD. This is because people with high levels of o-c symptomology are likely to have pre-existing metacognitive beliefs including beliefs about rituals that influence interpretations and coping with intrusions. It is likely that inducing thought-fusion in people with high o-c symptoms will activate these pre-existing metacognitions and this will produce greater OCD-like effects. Studies utilising participants with high as well as low levels of o-c symptomology would allow the assessment of whether thought-fusion impacts differently on these groups.
3) In the study, only the experimental group was presented with the possibility of a negative event happening (the administration of an electric shock). This may be a confound in that the control group were not expecting any event that could cause distress.
4) To ensure the manipulation was believable, participants were mainly high school students, and psychology or medical students were excluded. This limits both the generalisability of the findings and the general utility of the paradigm. A paradigm is needed where the thought-fusion manipulation is believable to most adults.

In the present study we aimed to test the metacognitive model by examining the effects of experimentally manipulating thought–event fusion beliefs using a paradigm inspired by the Rassin et al. (1999) study but which addressed the limitations outlined above. Additionally, whereas most previous studies have examined the role of thought–event fusion beliefs concerning causing harm to others, the current study aimed to extend previous findings by examining a different form of thought–event fusion which can be present in OCD namely fusion beliefs concerning harm coming to the self. In the current study, participants with either high or low levels of obsessional symptoms were randomly assigned to an experimental or control group. All participants underwent fake EEG recordings and were informed that the EEG could sense hypothalamus activity caused by having thoughts related to drinking. Participants in the experimental (thought-fusion induction) condition were told that if such thoughts were detected they may hear an aversive high pitched and loud noise while the control group were told that they may hear this noise but this was not connected to the thoughts they had. The design of the present study aimed to address the limitations of the Rassin et al. study described earlier, as in the current study:

1) The experimental group was not provided with an explicit form of avoidance or coping. Thus, only thought–event fusion beliefs were directly manipulated.
2) Groups with high and low obsessions were used so allowing for a test of whether the manipulation affected these groups differently—as would be predicted by the metacognitive model.
3) Both groups were expecting the possibility of an unpleasant event i.e. loud white noise but thought–event fusion was only manipulated in the experimental group. Thus, unlike in Rassin's study both groups were aware that they may experience a distressing event.