A cognitive examination of compulsive checkers' working memory and inhibitory performance

Ben Harkin

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School of Psychology
College of Science and Engineering

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Abstract

Checking is one of the most common symptoms observed in Obsessive Compulsive Disorder (OCD) with 50-80% of patients (Antony, Downie, & Swinson, 1998; Henderson & Pollard, 1988; Rasmussen & Eisen, 1988) and an additional ~15% of the general population demonstrating subclinical checking compulsions (Stein et al., 1997). A common finding is that checking actually impairs the memory of those items checked (van den Hout & Kindt, 2003a, 2003b), even though the mechanism underlying checking-related memory impairment has remained elusive. This is a shortcoming that we presently address in a series of short-term memory experiments and attentional tasks comparing high and low checkers (see VOCI; Thordarson et al., 2004). Generally, our memory tasks required stimuli to be remembered in their locations, which was designed to engage the episodic buffer (EB) of working memory (WM) (Baddeley, 2000). The key manipulation was to present an intermediate probe (between encoding and recall) in the form of a resolvable or misleading challenge which questioned an aspect of the encoding set; this was either present or absent, respectively. As expected, misleading probes specifically (Exp. 1, 2, extreme meta-comparison 3 & 9; Harkin & Kessler, 2009; 2011a; Harkin, Rutherford, & Kessler, 2011) and intermediate probes generally (Exp. 4; Harkin & Kessler, 2011a) tap into the inhibitory impairments of high (not low) checkers, which hampers EB functionality and impairs their memory. Indeed, it was only during misleading trials that high checkers made more unnecessary eye movements specifically to empty locations (Exp. 5; Harkin & Kessler, subm). Furthermore, for ecologically valid stimuli high checkers were impaired in inhibiting attention to threatening 'ON' states (Tasks 6 & 7; Harkin & Kessler, in press) and in their ability to recall if an appliance was 'ON' or 'OFF' (Exp. 8; Harkin, Rutherford, & Kessler, 2011). High checkers' intact performance on baseline no-probe-1 trials excludes a capacity-based explanation of their WM impairments. Overall, confidence measures revealed a general task-independent impairment which was attenuated by an intermediate probe. These findings were then used to create a classification system based upon **E**xecutive-Functioning, **B**inding Complexity and Memory **L**oad (EBL) to explain otherwise discrepant findings from 58 memory studies (Harkin & Kessler,

2011b). Thus, the contribution of this research is not only to (Exp. 1-9) indicate an actual mechanism (i.e., episodic buffer of WM) of memory impairment in checking/OCD but it also provides a new research platform on which to base where we *will* and *will not* observe memory impairments in OCD participants. The conclusion summarizes the main findings with respect to the development and maintenance of OCD symptoms, highlights limitations and provides solutions to these through future research.

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Acknowledgments

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1. Introduction: Repeated checking impairs memory, but how?

Obsessive-Compulsive Disorder (OCD) is characterized by repetitive, intrusive, impulses and thoughts that are experienced as inappropriate and anxiety provoking. The lifetime prevalence of OCD is between 1.5% and 3% (Stein et al., 1997), making it a debilitating and relatively common disorder. OCD patients experience intrusive thoughts (obsessions) that they feel compelled to neutralize through ritualistic behaviours (compulsions). Checking compulsions are most commonly observed in OCD with 50-80% of patients reporting this subtype (Antony, Downie, & Swinson, 1998; Henderson & Pollard, 1988; Rasmussen & Eisen, 1988) and an additional ~15% of the general population demonstrating subclinical checking compulsions (Stein et al., 1997).

Despite the commonality of checking little is understood about the mechanisms which mediate this phenomenon (Cuttler & Graf, 2007). One prominent theory is that checkers are compelled to check to compensate for impairments of memory. For example, in a meta-analysis of checkers memory performance, Woods et al. (2002) concluded that not only do they have objectively verifiable impairments in working and episodic memory but they also suffer from a subjective impairment in memory (i.e., they lack confidence in their ability to remember). Checkers may objectively fail to remember if they performed an action, such as switching the iron off, and/or they may subjectively lack confidence in their ability to remember (Cuttler & Graf, 2007; Sher, Mann, & Frost, 1984). Thus, poor memory and/or lack of confidence appears to fuel ritualistic checking, yet as they check and re-check to increase certainty this paradoxically decreases memory accuracy and confidence (Rachman & Shafran, 1998; Radomsky & Alcolado, 2010). For example, van den Hout and Kindt (2003b) asked participants to repeatedly turn on, off or check a computer simulation of a six burner gas stove for 20 trials after which they were asked to report the vividness, detail and memory confidence for their last check of the stove. In the checking, compared to the control-condition checkers had a significant decrease in the three aforementioned metacognitive measures and the authors concluded that checking breeds doubt, not certainty (van den Hout & Kindt, 2003b, 2004). Further, the same research group (van den Hout & Kindt, 2003a) showed that repetitively checking the same stimulus resulted in a shift in the nature of their memory recollections from being detailed and vivid (remember judgment) to being hazy, indefinite and unclear (know judgment) (Tulving, 1985). This they proposed was similar to the memory-ambivalence reported by clinical checkers (Reed, 1985).

Therefore, while these authors reported the outcome of checking, the exact mechanism of memory change was not stated. However, Radomsky and Alcolado (2010) provided a more specific indication of the domain specificity and the mechanism through which checking impaired memory. They asked participants to either *mentally* check their memory of an electrical stove or *physically* check an electrical stove. Mental checking required participants to "... imagine your hand manipulating the knobs, just like you would see yourself doing so in a real physical check" (Radomsky & Alcolado, 2010, p. 347). Memory accuracy was then determined with respect to the question: "Which three knobs did you check on the last trial?" (p. 347). The observed impairments were modality-specific: Repeated mental checking only impaired memory and metamemory for mental but not physical checks. Whereas, repeated physical checking only impaired memory and metamemory for physical but not mental checks. Domain specificity is further substantiated with compulsive staring resulting in distrust in perception not memory (van den Hout et al., 2008; van den Hout et al., 2009), whereas checking memory produced distrust in memory not perception (see Dek et al., 2010).

1.1. The story so far: Core features of checking-related memory impairment

In light of the aforementioned points and in agreement with Rachman's (2002) suggestion that any cognitive theory of pathological checking must account for such memory problems, we highlight the following key aspects of checking related memory impairment.

(1) Checking is domain specific, as only the cognitive processes (memory, metamemory) that are the object of checking (physical vs. mental; Radomsky & Alcolado, 2010) are negatively affected (Dek et al., 2010). This is in agreement with Nelson and Narens (1994) who proposed that the reactivation of memory traces can never provide an entirely veridical representation of the original input. That is, our memories will never be as vivid and ‘‘true’’ as the original experience and so unnecessarily checking, manipulating, and/or interacting with them appears to *further* impair the veridicality of their contents. In certain domains compulsive checkers might be overly aware of these natural shortfalls of memory traces, strongly experiencing doubt and starting to check the same memory trace over and over again, yet, without the possibility to enhance certainty.

(2) The contents of memory are sensitive to interference from internal (mental) and external (physical) sources (Radomsky & Alcolado, 2010). For example, misleading/intrusive information can be generated internally in the form of intrusive thoughts (‘‘I think I left a burner on’’) or can be provided by external prompting (i.e., experimentally directed mental checking; Radomsky & Alcolado, 2010). Kikul et al. (2011) measured the impact of cognitive self-consciousness (CSC: focus on their thoughts and mental strategies during encoding) and dual-task manipulations upon the subsequent recall of a complex visual stimulus for OCD patients and healthy controls. OCD patients' memory was impaired in the CSC and the dual-task conditions, whereas in controls only the dual-task condition resulted in impaired memory. This suggests that as the external CSC manipulation was congruent with the symptomatology of the OCD group, their internal focus on their thoughts attenuated performance in the primary memory task. More specifically, Omori et al. (2007) reported a negative correlation between poor inhibition and impaired memory in OCD-checkers but not OCD-washers. Suggesting that an inability to inhibit (i.e., stop thinking about/ignore) internally generated and/or externally threatening stimuli mediates checkers' memory performance. Indeed, inhibition is said to be required for the successful performance of a task during the simultaneous presence of task-irrelevant-stimuli, responses and (possibly) thoughts (Friedman & Miyake, 2004). Further, it was observed that a checking OCD subgroup had poorer memory compared to cleaners and controls (Cha et al., 2008) with checking and obsessional severity (e.g., "Did I check correctly?") associated with increased anxiety and poorer organization in early memory encoding (Jang et al., 2010). From this body of evidence we suggest that checkers have a cognitive profile which if pressured in the correct manner – i.e., experimentally and/or real-life – will negatively influence their memory performance. Yet, if the stimulus domain does not induce intrusive thoughts or there is no suitable external challenge then no performance deficit will be observed.

(3) Points 1 and 2 converge on the recent perspective that memory impairment in OCD/checking are not attributable to a general mnestic deficit but rather are secondary to executive dysfunction (Bannon, Gonsalvez, & Croft, 2008; Greisberg & McKay, 2003; Olley, Malhi, & Sachdev, 2007; Omori et al., 2007; Penades et al., 2005). This explains the domain-specificity of checkers' memory impairment: Disrupted memory only occurs when a memory task or real life event taps into a dysfunctional component of the executive, i.e., failure to suppress intrusive information.

1.2. The present story: The central role of working memory

From the aforementioned points it is clear that in specific instances checkers' memory is impaired, however, the question as to what stage memory traces are interfered with remains unresolved (Coles, Radomsky, & Horng, 2006). Is the effect purely confined to episodic long-term memory or does it operate already at an earlier stage? Interference may occur within episodic representations in short-term Working-Memory (WM) and affect their transfer into Long-Term-Memory (LTM). For example, the familiar checkers question of "Did I turn ALL the burners off?" could arise seconds after leaving the kitchen and could strongly affect how they remember the state of the stove hours later.

A body of research indicates that memory impairments of OCD patients can occur at the level of WM and that this is attributed to deficits in executive control and not memory capacity *per se*. For example, Van der Wee et al. (2003) used a spatial variant of the *n*-back WM task with four levels of load. It was only at the highest load level (3-back) that patients with OCD significantly differed from controls with errors of 48% *versus* 25%, respectively. They argued that OCD patients may over-scrutinize their performance or have a deficit in supervisory (i.e., executive) processes, as opposed to deficits in maintenance or manipulation, which suggests that general capacity limitations are not responsible for the results. We propose that the stability of executive-memory impairment at higher levels of task complexity is further supported by its presence across a range of WM tasks, for example, the spatial WM task (Purcell et al., 1998a, 1998b), paired association learning (Morein-Zamir et al., 2010) and the corsi block tapping task (Boldrini et al., 2005; Moritz et al., 2003; Zielinski, Taylor, & Juzwin, 1991; Zitterl et al., 2001). In these instances OCD memory impairments are not attributable to capacity *per se* (i.e., intact at lower load levels; see also Ciesielski et al., 2007; Henseler et al., 2008) but rather represent a failure of executive functioning to match increasing task demands in terms of strategic resource organization. However, we highlight the following limitation of this conclusion: As load linearly increases in these tasks and OCD impairment occurs at higher load level, one cannot fully rule-out the role of impaired capacity. In compromise, for example, on the n-back task we suggest that load (i.e., increasing visuospatial information) and executive control (i.e., maintaining and sorting through that visuospatial information) are closely interrelated, making it difficult to tease apart which cognitive processes plays the (if at all) dominant role. Thus, one of the aims of the present experiments is to provide a clearer delineation between intact capacity and memory impairment driven by primary executive dysfunction.

In sum, the literature reveals that poor memory performance of OCD generally and checking specifically is explained by an interaction between deficits of inhibition, overactive performance monitoring (Veale et al., 1996), and impairments in WM. Whereby, an inability to ignore/inhibit irrelevant internal/external stimuli, likely triggers an existing preponderance to monitor/examine the contents of WM and impairs the veridicality of their contents (see Salkovskis, 1999; Veale et al., 1996). Importantly, these three factors have been identified as candidate endophenotypes of OCD, which implicates them at the neurocognitive level as central – impairment in first-degree relatives (heritability) and state independence – to the illness of OCD (Chamberlain et al., 2007; Delorme et al., 2007; Menzies et al., 2007; Riesel et al., 2011) and checking specifically.

1.2.1. A unifying framework: Baddeley's (2000) model of working memory

Despite the likelihood that WM representations are the target for compulsive checkers' concerns (Shimamura, 2000), the specific relationship between WM performance and checking is poorly understood (see Woods et al., 2002 for review). Here we propose that checkers' executive impairments – i.e., failing to inhibit irrelevant stimuli and/or repeatedly questioning the veridicality of memory representations (cf. Nelson and Narens, 1994) – will reduce performance already at the stage of WM. Not only does this highlight the centrality of executive dysfunction in impaired OCD/checkers' memory performance but it also implies the sensitivity of memory *per se* to interference. Based on these considerations, we propose Baddeley's (2000) model of WM as a unifying framework for explaining: (1) generally, deficits of executive control and memory in compulsive checking/OCD and (2) specifically, the mechanism underlying poor memory, i.e., executive dysfunction interferes with fragile attention-dependent bindings maintained in the EB (EB) (see fig. 1) (Harkin & Kessler, 2009). To reiterate, Baddeley's (1986) original model included a central executive, phonological loop and visuospatial sketchpad and was deemed separate from long-term memory (LTM) (Baddeley & Hitch, 1974). While this simple model explained a range of data (e.g., phonological similarity; Baddeley, 1966), it could not account for all experimental phenomena. For example, the visuospatial sketchpad, a capacity limit of 4 units was observed for the maintenance of individual features (colors <u>*or*</u> orientations) as well as for integrated objects with colors <u>*and*</u> orientations (Luck & Vogel, 1997). In addition, the original separation between WM and LTM was unsupportable because: (1) chunking in verbal WM is aided by existent information in LTM (e.g., Ericsson & Kintsch, 1995),

(2) patients with disturbed phonological loop functioning are impaired in long-term language learning (Baddeley, Papagno, & Vallar, 1988), and (3) bindings in visuospatial WM influence long-term visuospatial learning (Logie, Brockmole, & Vandenbroucke, 2009).

1.2.2. The centrality of the episodic buffer

The complementary nature of WM-LTM processes, in addition to efficient chunking and binding, hinted at a distinct cognitive resource, one that could integrate information from a variety of sources (e.g., phonological, color, location, smell) into a single memory episode. The so-called "binding problem" (e.g., Treisman, 1996) refers to the fact that information presented in visual scenes rarely consists of isolated features. Rather, features pertain to objects, objects to locations, and objects are further embedded into episodes together with a plethora of contextual information. A parallel processing architecture like the human brain needs mechanisms for tracking "what goes with what" in order to generate and maintain bindings between multiple features (Hinton, McClelland, & Rumelhart, 1986). Therefore, accurate memory (WM and LTM) requires the encoding, maintenance and retrieval of bindings between various aspects of a multimodal episode (Allen, Baddeley, & Hitch, 2006). Baddeley (2000), therefore, extended his classic 1986 WM model to include an "EB" that allowed for multimodal, temporarily integrated representations and served as an interface with episodic LTM. Based on this development, we proposed (Harkin & Kessler, 2009) that an executive dysfunction (e.g., unsuppressed intrusive thoughts/stimuli) might strongly impair the consolidation of representations in the so-called EB of WM, impairing memory over the short- and possibly long-term (Harkin & Kessler, 2009: see fig. 1).

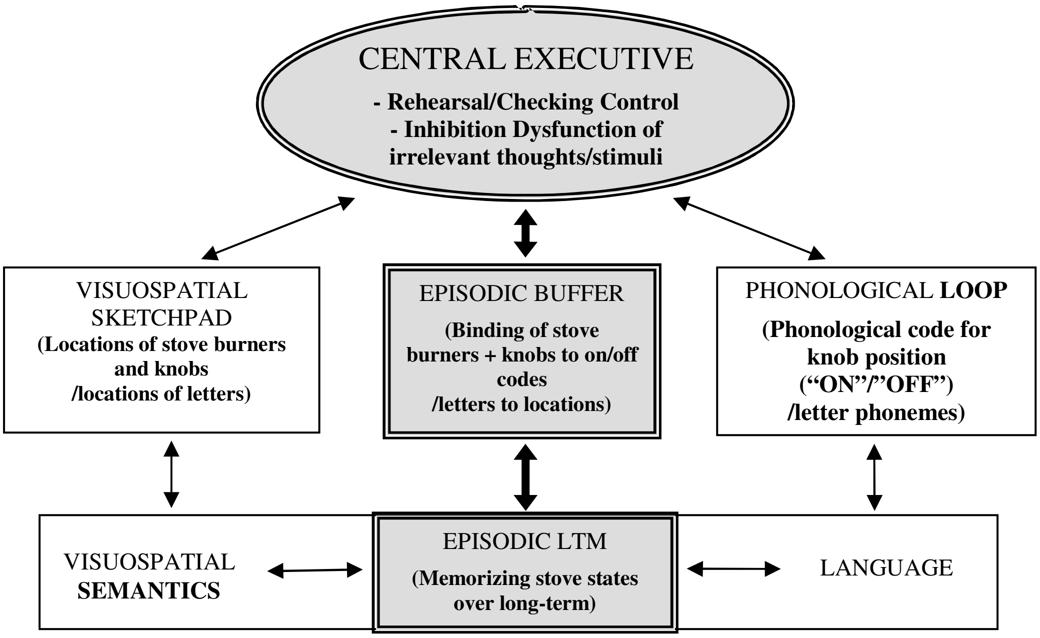

Figure 1. Adaptation from Baddeley (2000) as originally proposed by Harkin & Kessler (2009). The grey parts of the WM framework highlight the components and their interactions which we propose to be involved in compulsive checking. A specific central executive dysfunction (inhibition of irrelevant thoughts/stimuli) interferes with binding of the episodic buffer disrupting memory performance over the short-term and potentially the long term. Further explanations in the text.

Baddeley (2003) later emphasised the parallels between his EB and the concept of a "global workspace" (Baars, 2002; Dehaene & Naccache, 2001), which is a formal neuro-cognitive approach to conscious/aware processing. In short, this embraces the notion that compulsive checking and associated executive impairments affect the current stream of consciousness (e.g., Salkovskis, Forrester, & Richards, 1998). Thus, intrusive thoughts that doubt the veridicality of memory traces (e.g., "Did I REALLY turn all the burners off?") could therefore be more detrimental for compulsive checkers because they cannot easily inhibit these thoughts from affecting ongoing conscious processing (cf. Bannon, Gonsalvez, & Croft, 2008; Salkovskis, Forrester, & Richards, 1998). This concurs with the finding that 'not just right' obsessions significantly correlated with checking, control and some elements of perfectionism (Coles et al., 2003).

Furthermore, Miyake et al. (2000) observed that various facets of executive control are interconnected, which neatly explains the manner in which executive dysfunction impairs memory functioning. Their latent variable analysis identified three major control functions of the central executive: (1) *inhibition*: resist disruption from task-irrelevant stimuli, (2) *shifting*: shift attention between different yet task-relevant options, and (3) *updating*: "updating and monitoring of WM representations" (p. 56). Specifically, their analysis revealed that while these were relatively independent constructs, they were also interdependent, which implies they all rely to some extent upon the attentional resources of the central executive (Eysenck et al., 2007). Therefore, it follows that an inability to ignore irrelevant stimuli may potentially reduce the attention allocated to the concurrent updating of information presently maintained in the EB of WM. We, therefore, propose the EB as the focal point for memory impairments in OCD/checking: EB functionality (binding) is vulnerable to interference through executive dysfunction (e.g., failure to inhibit intrusive thoughts/stimuli). In other words, interference from executive dysfunction reduces the veridicality of multimodal bindings within the EB, attenuating memory performance.

1.2.3. Episodic buffer bindings' sensitivity to interference

While there is some debate regarding the exact mechanism for binding multimodal features together into a representation (i.e., object-unit hypothesis; Luck & Vogel, 1997 *versus* independent-unit hypothesis; Wheeler & Treisman, 2002), researchers tend to agree that attentional effort (executive control) is required for their generation and maintenance (Delvenne & Bruyer, 2006; Fougnie & Marois, 2009; Hyun, Woodman, & Luck, 2009; Makovski, Sussman, & Jiang, 2008; Rudner & Ronnberg, 2008; Wheeler & Treisman, 2002). Thus, critically for the present thesis, the EB is assumed to be controlled by the central executive" (Baddeley, 2000, p. 421) which is consistent with Wolters and Raffone's (2008) tri-partite definition of executive functioning: (1) *Attentional Control*: top-down selective activation of task-relevant representations and suppression of task-irrelevant stimuli and responses, (2) *Maintenance*: holding task-relevant information in an active state, and (3) *Integration*: flexibly bind and manipulate information from multimodal sources, in the service of controlling task execution. Therefore, memory impairments occur if distraction is sufficient to interfere with attentional control specific to the maintenance and integration of bindings in the EB (Elsley & Parmentier, 2009). For example, Wheeler and Treisman (2002) measured WM recall with single- and whole-probe displays and reported a binding impairment specific to the whole-probe condition. They argued that as binding is dependent upon sustained attention so the presentation of a whole-probe withdraws attention to those bindings simultaneously maintained in WM. Also greater attentional resources – as measured by a larger N2pc ERP amplitude – was observed for the binding of colors to locations than individual colors (Hyun, Woodman, & Luck, 2009). Fougnie and Marois (2009) tested the role of attention in binding using an attentionally demanding Multiple Object Tracking (MOT) task, which involved tracking through space relevant targets among irrelevant distractors, all of which are moving. The MOT was presented between the encoding set of a separate memory task (color, shape, color and shape, conjunctions of color and shape) and the memory probe at the end of the trial. Only memory for feature bindings (conjunctions of color and shape) was impaired and was specific to the attentive tracking of the MOT as similar attenuation was not observed for a static distractor. They hypothesized that attention iteratively refreshes multimodal representations in WM: it is only when a distractor sufficiently interferes with attention that there is a failure to maintain features in a bound manner (e.g., Harkin & Kessler, 2009, 2011a; Harkin, Rutherford, & Kessler, 2011; Kessler & Kiefer, 2005; Mather et al. 2006). This dove-tails nicely with findings showing that the more emotionally engaging a given distractor is to an individual (or group) the more it interferes with attention-dependent bindings (Dolcos & McCarthy, 2006; Johnson et al., 2005; Mather, 2007; Mather et al., 2006). Mather et al. (2006), for example, presented pictures of high, medium and low arousal in various locations. Picture-location accuracy decreased as arousal increased. Interestingly, depression scores were negatively correlated with picture-location accuracy for negative images. Emotional arousal, therefore, interfered with binding accuracy at a global (all subjects) as well as an individual (depressed) level. In this reasoning, complex representations that are salient to a checking/OCD individual/group will likely result in memory impairments (i.e., Cha et al., 2008; Jang et al., 2010).

1.3. The aim of the present experiments: Targeting the episodic buffer

With these points in mind, the present series of experiments set out to: (1) *engage* the EB using stimuli which require multimodal conjunctions between phonological (letters) or visual (kitchen appliances) and spatial (locations) features and (2) *hamper* EB functionality by presenting an intermediate probe that was relevant to the executive impairments of high but not low checkers during the WM retention interval.

1.3.1. Primary experimental manipulations

Specifically, we presented an intermediate probe (between the encoding set and memory task) in the form of two types of external challenge which resulted in two main trial types:

(1) *Resolvable Trials*: probing an aspect of the encoded set (identity/location) where a correct response is possible, i.e., it is resolvable. For example, asking for the color of an item at a location where <u>*one was*</u> presented.

(2) *Misleading Trials*: probing an aspect of the encoded set (identity/location) where a correct response is impossible, i.e., it is irresolvable. For example, asking for the color of an item at a location where <u>*none*</u> was presented.

1.3.2. Primary experimental predictions

In relation to these experimental manipulations we make a *strong* and a *weak* hypothesis with respect to the memory performance of high compared to low checkers.

Accuracy – Strong Hypothesis

Compulsive checkers have been reported to show a deficit in inhibiting intrusive thoughts and distracting stimuli (e.g., Olley, Malhi, & Sachdev, 2007; Omori et al., 2007; Savage et al., 2000). As inhibitory functioning is associated with the ability resist interference from distractors (Friedman & Miyake, 2004), it follows that impaired inhibitory functioning will reduce the ability to accurately maintain task goals when confronted by externally task-irrelevant stimuli (see Eysenck et al., 2007). We therefore expect that the presence of a misleading but irrelevant probe-1 question will especially interfere with the WM representations of higher checkers. This, we argue, is analogous to the process of having just completed a task (e.g., turning off the stove) and then almost immediately starting to check the maintained WM representations for their veridicality (see above; Radomsky & Alcolado, 2010). Thus, checking the contents of memory is likely to be driven by a thought or an external stimulus that is task-related but irrelevant to the successful recall of the memory trace, e.g., external misleading cue: "Was that letter there?" leading to the thought: "I am unsure!" This assertion is supported by the observation that OCD checkers are poorer at tolerating uncertainty (i.e., misleading probe-1 letter) than OCD non-checkers and controls (Tolin, Woods, & Abramowitz, 2003), and that an inability to tolerate uncertainty is associated with subsequent checking and repeating rituals (Lind & Boschen, 2009; Tolin, Woods, & Abramowitz, 2003). Misleading intermediate probes may induce checkers to 'check another time' in an attempt to 'be sure', however, as we have seen this only serves to further undermine memory at the level of accuracy and confidence. As a result, we expect that high checkers will have poorer memory for misleading but not resolvable trials in comparison to low checkers. We suggest that for a misleading probe, checkers are more likely to repeatedly compare the visually presented probe to the contents of the memorised set, yet, frustratingly without success. At the representational level this would lead to a competition between a strong visual stimulus and weaker, memorised bindings. The stronger this competition is (lack of suppression of misleading information) and the more often this competition is repeated (checking) the more strongly the originally encoded memories are weakened – ultimately resulting in a performance deficit on the actual memory test (probe-2) (see Simplified Comparison Hypothesis: Makovski, Sussman, & Jiang, 2008).

Accuracy – Weak Hypothesis

For high checkers, an intermediate probe (resolvable or misleading) will be experienced as *generally* distracting (executive impairment) which will result in the withdrawal of attention from attention-dependent bindings underlying the encoding set. (Gajewski & Brockmole, 2006; Wheeler & Treisman, 2002). This will result in high checkers having poorer memory performance for misleading *and* resolvable trials in comparison to low checkers.

Measuring working memory capacity

For either the weak or the strong hypothesis, and in agreement with previous findings (e.g., Ciesielski et al., 2007; Henseler et al., 2008) we predict that WM capacity will not explain these group differences. To support this we will include trials without an intermediate probe (no-probe-1 trials) which will measure WM functioning under ideal conditions. Thus, for these trials we expect that high checkers memory performance will not to differ from low checkers. In doing so we will provide a clearer demarcation of the conditions where primary executive dysfunction results in secondary memory impairment in OCD-checking and that WM capacity is not responsible (c.f., Cha et al., 2008; Omori et al., 2007). However, we do expect to potentially observe memory impairments in no-probe-1 trials in our latter experiments, i.e., 7 and 8. As these experiments use stimuli concordant with the symptoms of high checkers, this may evoke anxiety, which as discussed previously (see Dolcos & McCarthy, 2006; Johnson et al., 2005; Mather, 2007; Mather et al., 2006) may generally interfere with attention to bindings irrespective of an intermediate probe. It is important to reiterate, that this is not evidence of a general impairment in the WM capacity of high checkers, as the absence of impairment in no-probe-1 trials in ours (Exp. 1-4) and others experiments at low load levels (e.g., Ciesielski et al., 2007; Henseler et al., 2008; van der Wee et al., 2003, 2007) argues against this.

Reaction Times

We will measure reaction times (RTs) in relation to the memory task. We do not expect that RTs will differ between high and low checkers on this measure. If this is the case then we will be able to rule out a speed-accuracy trade-off with respect to high checkers poorer memory performance, i.e., they will *not* be faster than low checkers.

Confidence

We will also measure confidence after the memory task (probe-2). We justify this by observing the centrality of doubt regarding memory performance in checking (for reviews see Woods et al., 2002 and Muller & Roberts, 2005). With the literature showing two prominent effects: (1) checking impairs confidence (e.g., van den Hout & Kindt, 2003a) and (2) poorer confidence motivates checking (e.g., Alcolado & Radomsky, 2011). Tolin et al. (2001) reported that with repeated exposures to threatening stimuli, OCD patients showed a progressive decline in memory confidence across trials compared to anxious and non-anxious controls. More specifically, after 1-week, OCD checkers had poorer confidence in memory for threatening stimuli compared to non-checking OCD patients. This suggests that poor memory confidence is a characteristic of OCD in general, but is particularly pronounced amongst checkers in the long-term. A finding that is concordant with the plethora of evidence showing that repeated checking reduces memory confidence specific to the domain of checking (Dek et al., 2010; Radomsky & Alcolado, 2010; Radomsky, Gilchrist, & Dussault, 2006; van den Hout & Kindt, 2003a, , 2003b) with this metamemory effect occurring in as few as two checks (Coles, Radomsky, & Horng, 2006). Considering this alongside the evidence that OCD checkers have less confidence in their memories compared to OCD non-checkers and controls (MacDonald et al., 1997; McNally & Kohlbeck, 1993; Sher, Frost, & Otto, 1983); low memory confidence in checkers may contribute to the self-perpetuating mechanism of further checking and reduced confidence (Rachman, 2002). This agrees with the recent research demonstrating that poor memory confidence predicts repeated checking. For example, Nedeljkovic and Kyrios (2007) showed that low trait memory confidence was associated with severity of checker's obsessional symptoms (i.e., "Did I turn it off?") and higher-order executive processes related to memory (i.e., attention/concentration) (Nedeljkovic & Kyrios, 2009a). In a similar manner, Cougle, and colleagues reported that checkers doubted their own memory abilities, lacked confidence in their memory for OCD stimuli, and that confidence correlated with memory accuracy (Cougle, Salkovskis, & Thorpe, 2008; Cougle, Salkovskis, & Wahl, 2007). This suggests that low memory confidence may be a risk factor for checking especially in a context of uncertainty (Tolin et al., 2003), a suggestion confirmed by Alcolado and Radomsky (2011) who showed that manipulating confidence (positive vs. negative false feedback) influenced subsequent urges to check. Those who received false feedback (low memory confidence condition) had stronger urges to check than those who received positive feedback (high memory confidence condition). In addition, Nedeljkovic and Kyrios (2007) proposed that *covert* checking (i.e., comparing misleading P1 to contents of WM) and poor metamemory are particularly detrimental for tasks that are dependent upon the maintenance of internal representations, i.e., the type of WM task used in the current experiments.

In the context of the present experiments, checking implies a lack of confidence in the veridicality of the reactivated WM information that is detrimental without the original sensory information to check against, or even with competing new sensory information present. As a result, we expect high checkers will have generally poorer memory confidence relative to low checkers; a difference that will be further mediated by the presence of an intermediate probe. In addition, if high checkers are specifically unable to ignore a misleading probe – due to impairments of inhibitory functioning – not only will they have poorer memory in a misleading context but they will also reveal poorer memory confidence relative to low checkers.

1.4. Creating high checking and low checking groups

All of the present experiments require us to determine the checking tendency of each individual participant. To this end we used the checking subscale of the Vancouver-Obsessional Compulsive Inventory (VOCI; Thordarson et al., 2004). The VOCI consists of 55 items that comprise 6 subscales: Contamination (12 items), Checking (6 items), Obsessions (12 items), Hoarding (7 items), Just Right (12 items), and Indecisiveness (6 items). Each item is rated 0 (not at all), 1 (a little), 2 (some), 3 (much), or 4 (very much) in response to the prompt: "How much is each of the following statements true of you?" The VOCI possesses excellent inter-item reliability in student, community, OCD, and clinical control populations (Cronbach's alpha: 50.96, 0.90, 0.94, and 0.98, respectively). Although participants completed all items of the VOCI, only the checking subscale was used to create a high and a low checking group.

1.5. The structure of the present thesis

The present experiments will measure memory performance of high compared to low checkers in novel WM tasks – fulfilling the aforementioned design criteria – and a specific measure of inhibitory (i.e., executive) functioning.

Chapter 2. Checkers' show robust and consistent impairments in a misleading context: A simple working memory task

Experiments 1, 2 and an extreme group meta-comparison provide our first attempt to interfere with the WM performance of high but not low checkers (see Harkin & Kessler 2009). Simply, we present letters in locations and measure the impact of a misleading versus resolvable intermediate (probe-1) upon the WM and metamemory performance of high and low checkers. In line with our strong hypothesis, we expect that high checkers will be unable to ignore a misleading intermediate probe which will then impair memory performance on the subsequent WM (probe-2) task. We do not expect to observe difference in basic WM capacity (no-probe-1 trials).

Chapter 3. Checkers' memory impairments persist in more complex working memory experiments

Experiments 3 and 4 attempt to increase the group differences observed in Experiments 1 and 2 (see Harkin & Kessler, 2011a). In Experiment 3 we increase the complexity of the encoding set (letters in locations) by presenting the letters in different colours. We expect that increasing the binding load of the encoding set will increase its sensitivity to interference, which may boost the memory impairments of high checkers observed in Experiment 1 and 2. Then, in Experiment 4 we attempt to increase the strength of interference caused by the intermediate probe by presenting it as a strong visuospatial at a resolvable or misleading location. We predict that this strong visuospatial distractor (relative to Exp. 1, 2, and 3) will definitively tap into checkers' impairments in inhibiting irrelevant stimuli and so boost memory impairments. In this instance, high checkers may be generally distracted by such an intermediate probe leading to poorer WM performance in misleading and resolvable trials alike (i.e., supporting the weak hypothesis).

Chapter 4. Do checkers actually check?: An eye movement study

In Experiment 5, we address a primary methodological limitation of our previous experiments (see Harkin, Miellet, & Kessler, subm). Specifically, we cannot say with certainty that high checkers do actually check the contents of WM when presented with a misleading intermediate probe. To this end, we use eye tracking as a means of measuring fixation number and fixation duration across three critical periods of our original WM paradigm (i.e., Exp. 1, 2; Harkin & Kessler, 2009). We focus on these eye movement measures as these mimicked the symptoms of checking, i.e., fixation number related to unnecessary checking and fixation duration similar to perseveration. Simply, we expect that in misleading trials high checkers' inhibitory impairments for misleading information results in them checking (longer looking at) the contents of WM in a manner which is unnecessary (specifically in misleading trials) and uninformative (empty locations), in comparison to low checkers and resolvable trials. Checking empty locations will provide specific evidence that in a context of uncertainty (misleading trials) high checkers' attempt to remove it by examining locations were no additional task-relevant information is present.

Chapter 5. Using ecologically valid stimuli to address previous experimental concerns

In Tasks 6 and 7 (see Harkin & Kessler, in press) and Experiments 8 and 9 (see Harkin, Rutherfored, & Kessler, 2011) we again attempt to tap more strongly into the executive impairments of checkers by using stimuli (i.e., electrical kitchen appliances) that are more concordant with their symptoms. Please note that the reference to Tasks 6 and 7 (as opposed to Experiments 6 and 7) is to avoid confusion which arose in explaining and discussing the counterbalanced design which was used in this case. Tasks 6 and 7 attempt to determine if checkers do in fact suffer from executive impairments in a novel application of the Inhibition of Return (IOR; Posner, Cohen, & Rafal,

1982) paradigm. If so, this will provide explicit evidence that checkers suffer inhibitory impairments for stimuli that are specific to their symptomatic concerns. Experiments 8 and 9 then require participants to memorise the location of the same (Tasks 6 and 7) electrical kitchen appliances presented on a kitchen countertop. Between encoding and the memory task we again presented a spatial location probe as it previously (Exp. 4) revealed strong and robust group differences. We then tested memory by asking if an appliance had been 'ON' or 'OFF' (Exp. 8) or if it was correctly located (Exp. 9). By using such symptom specific stimuli, high checkers may possibly reveal novel and potentially larger WM impairments relative to low checkers.

Chapter 6. The role of working memory in compulsive checking and OCD: A systematic classification of 58 experimental findings

Then using evidence from the previous experiments (1-9) and that of the existing OCD literature we provide a classification system which allows us to position individual OCD memory experiments and explain why they did or did not report memory impairments (see Harkin & Kessler, 2011b). This classification system moves away from the classic verbal *versus* visual distinction and issues of basic capacity. Rather, we extend the argument that memory impairments in OCD are secondary to executive dysfunction and highlight that the following three main factors which underlie memory impairment in OCD: (1) **E**: executive functioning efficiency, (2) **B**: binding complexity of stimuli used and (3) **L**: overall load of task upon WM resources. We use this EBL classification to explain otherwise discrepant findings from 58 studies.

Chapter 7. Conclusion: Overview, clinical implications, limitations and future research, and contribution to OCD memory research

Finally, we conclude with an overview of our current experimental findings as they relate to our primary hypotheses, followed by a discussion of the manner in which checkers' attention/WM impairments contribute to the maintenance and development of their symptoms. Then limitations of the research are identified and, when appropriate, avenues of future research are proposed as a solution. Finally, the contribution of our findings and theories are then discussed with respect to OCD memory research as a whole.

2. Checkers' show consistent and robust memory impairments in a misleading context: A simple working memory task

The following three experiments were our first attempt to measure the impact of an irrelevant intermediate probe upon subsequent WM performance of low and high checkers (see Harkin & Kessler, 2009). We presented 4 letters randomly in 6 possible locations (encoding set); with the primary memory task (probe-2) requiring the participant to determine if an individual letter was correctly or incorrectly located with respect to the original encoding set. We chose an easy primary task (4 letters in 6 locations) to avoid group differences due to differences in WM capacity at high load (see Van der Wee, 2003; Purcell et al., 1998a, 1998b) and we included a control condition without intermediate probe (no probe-1) to obtain a baseline indication of capacity. The checking manipulation between the encoding set and the memory task was induced by presenting a probe that was potentially misleading in its form. Participants were asked explicitly <u>where</u> a specific letter had been, while this letter, e.g. "T" or "K" (see fig. 1), either was (hence, resolvable) or <u>was not</u> (hence, misleading) part of the encoded set. For the latter, we expect that questioning the location of a letter that is not solvable will tap into checkers established executive impairments in inhibition (Olley, Malhi, & Sachdev, 2007; Omori et al., 2007) the inherent irresolvability of misleading information will induce a degree of repeated checking of the veridicality of the encoded representations especially in high checkers (Veale et al., 1996). This is in agreement with the observation that intolerance of uncertainty (i.e., aversion to uncertainty about the presence/absence of probe-1) mediates checking in OCD (Lind & Boschen, 2009; Tolin et al., 2003). Thus, high checkers will have impaired memory performance in a misleading but not a resolvable context compared to low checkers and these differences will not be due to capacity, i.e., no group difference on no-probe-1 trials. We expect that confidence – measured after the memory task – will be poorer in high compared to low checkers and that this will be mediated by the presence of an intermediate probe. We tested these hypotheses in two experiments (Exp. 1: low vs. high checkers; Exp. 2: replication of Exp. 1) and an extreme group meta-comparison (using high checkers from Exp. 1 and 2 which scored in the clinical range according to the VOCI; Thordarson et al., 2004).

2.1. Experiment 1

2.1.1. Method

Participants

40 Participants (mean 22.7 years: 12 male, 26 female) from the University of Glasgow gave written informed consents. British Psychological Society ethical requirements were met, including that of participant debriefing. A median split of VOCI checking scores was used to obtain two groups: low (mean = 1.11, SD = 1.10) and high (mean = 9.53, SD = 5.49) “checkers”.

Stimuli and Procedure

Participants sat 90cm from a 19’’ computer screen ran at 800x600 resolution with their head on a chin rest. Stimuli were capital letters in font Arial, size 18 and were presented against a black background within a 2 (columns) by 3 (rows) matrix covering an area of 300x420 pixels. After 1000ms fixation, 4 letters were presented randomly in 4 of the 6 possible locations and participants had 2000 ms to encode the identity and the location of each letter (see fig. 2). After 500 ms, the probe-1 question requested the location of a specific letter. Participants indicated the location through a 2x3 spatially mapped keypad and were instructed to respond within 4000 ms (to keep the WM delay constant). Whether the probe-1 letter had or had not been part of the encoded set created the resolvable versus misleading (irresolvable) trials. In a baseline condition probe-1 was omitted to measure WM performance on the primary task under ideal conditions.

A 1000 ms interstimulus interval (ISI) separated probe-1 and probe-2. Since baseline trials did not include the intermediate probe-1 a black screen was shown for 5500 ms between encoding and probe-2 (equaling the ISI between encoding and probe-2 on the other trial types). Probe-2 was the actual memory test for each trial and required participants to indicate if a letter was correctly located with respect to the originally encoded set. In all trials the probe-2 letter had been part of the encoded set in terms of identity while the probe location was correct only on 50% of the trials.

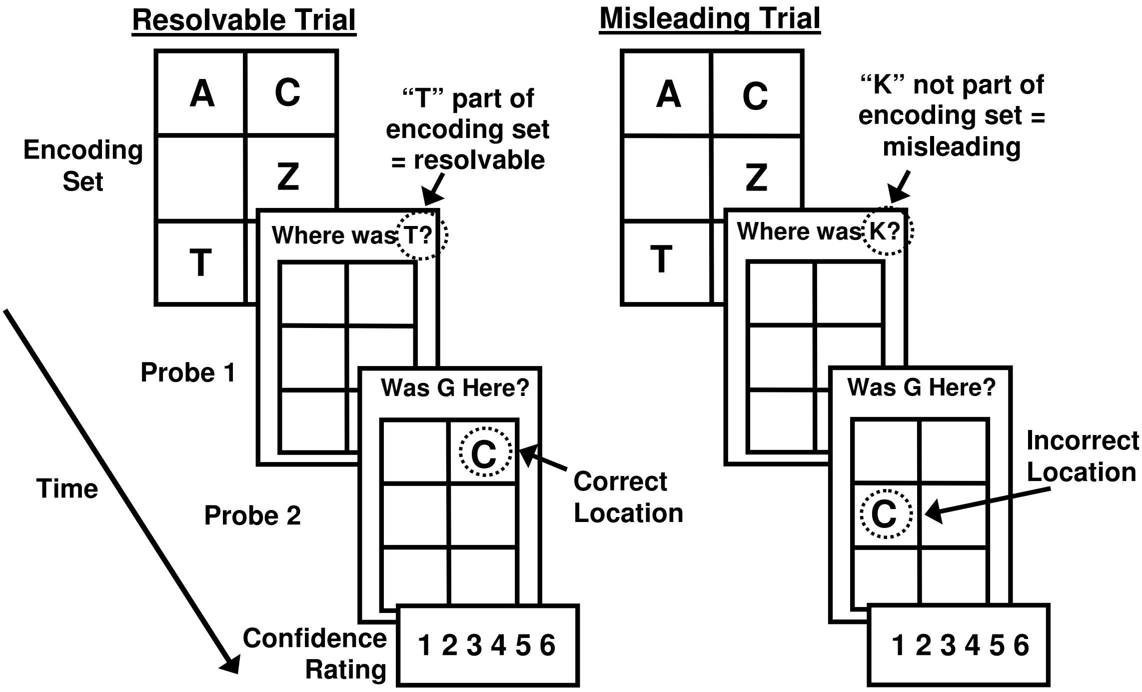

Figure 2. Schematic procedure of resolvable and misleading trials. A set of 4 letters presented randomly in 4 out of 6 possible locations had to be encoded within 2 seconds. Encoding was then followed after 500 ms by a first probe letter (probe-1) which was either part or not part of the encoded set, i.e., was resolvable or misleading. Subsequently participants had to indicate if the probe-2 letter was correctly or incorrectly located with respect to the encoded set, which was the actual memory test. Finally confidence in the probe-2 response had to be indicated on a scale from 1 (highly certain) to 6 (highly uncertain). Further explanations in the text.

Finally, a scale was displayed prompting participants to indicate their degree of confidence in their probe-2 response (6 levels: 1=totally certain to 6=totally uncertain). Three self-paced breaks were included and the experiment lasted approximately 90 minutes. The resolvable block comprised 180 trials with 120 resolvable trials, 40 misleading trials, and additional 20 baseline trials (no probe-1). Correspondingly, the misleading block (180 trials) was made up of 40 resolvable trials, 120 misleading trials, and again 20 baseline trials. The sequence of these blocks was counterbalanced across participants in order to avoid order effects.

Design

A two (*group*: low vs. high checkers) by two (*block type:* mostly resolvable vs. mostly misleading block) by three (*probe-1 trial type*: resolvable, misleading, no probe-1) mixed design was employed with *group* as the between- and *block* and *probe-1* as the within-subjects factors.

2.1.2. Results

MANOVA's for a 2x2x3 design were carried out for reaction times, accuracy and confidence on probe-2 responses due to violations of the sphericity assumption (Mauchley's tests). As our theoretical predictions focused on the effect of checking induced by resolvable vs. misleading probe-1 trials we also conducted 2x2x2 ANOVA's removing the no-probe-1 trials. The datasets of two participants were not used in further analysis as accuracy was at chance levels in at least one condition. All other participants performed well above chance level in all conditions (> 70% accuracy).

Probe-2 reaction times

The MANOVA (2x2x3) for probe-2 latencies revealed a main effect of trial type ($F(2,72)=10.65$, $p<0.001$) and the ANOVA for the reduced 2x2x2 design (without no-probe-1 trials) also revealed a main effect of trial type ($F(1,36)=9.46$, $p<0.004$). This indicates that the misleading trials were the slowest (Table 1).

Table 1: Reaction times for all participants for resolvable, misleading and no probe-1 trial types.

Trial Type	**Resolvable**	**Misleading**	**No Probe-1**
RT (msec)	1782.021	1896.743	1982.715

Probe-2 accuracy

The MANOVA (2x2x3) for probe-2 accuracy revealed a main effect of block ($F(1,36)=5.64$, $p<0.03$) which was indicative of generally less accuracy in the misleading block than the resolvable block. The main effect of trial type was also significant ($F(2,35)=3.53$, $p<0.04$) and indicated the greater accuracy in the no-probe 1 trials relative to the resolvable and misleading trials. Importantly, the simple effect for no-probe 1 trials (baseline) revealed no significant difference between high- and low-scorers ($F(1,36)$, $p<0.5$, $p>0.48$) indicating that WM capacity was comparable between groups.

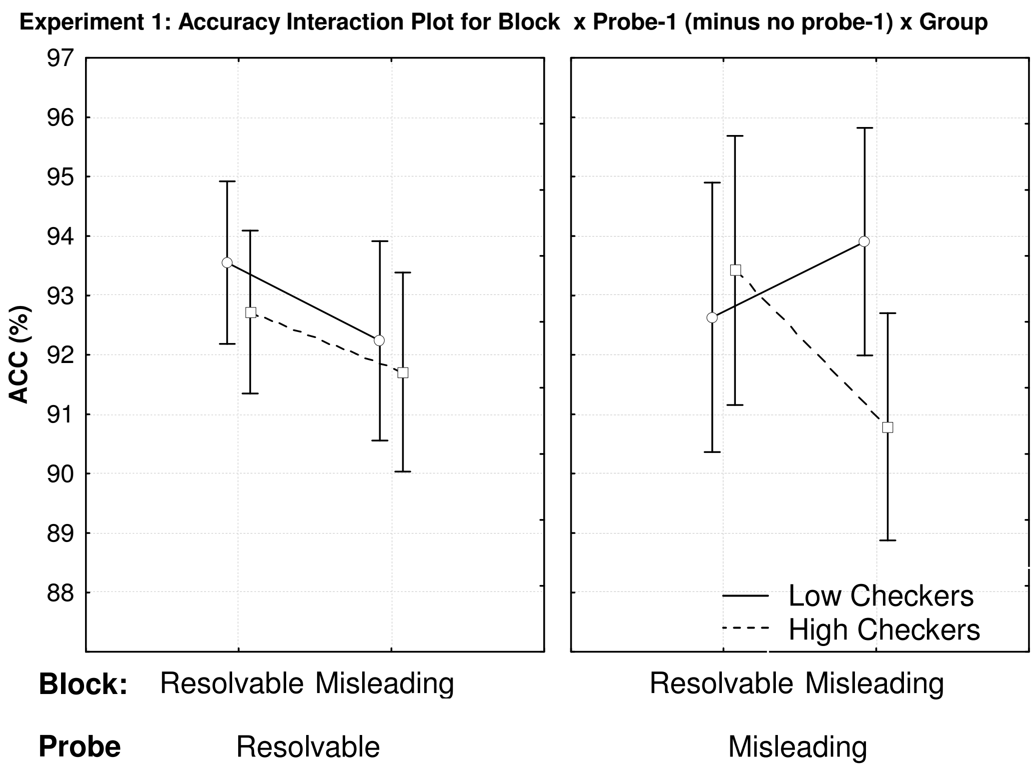

Figure 3. Experiment 1: Group (low vs. high checkers) x block (resolvable vs. misleading) x trial type (resolvable vs. misleading) interaction plot for probe-2 accuracy. Vertical bars denote standard errors.

Next, we removed the no-probe-1 trials to focus on the more relevant resolvable and misleading trials. The ANOVA for the reduced 2x2x2 design revealed a significant 3-way interaction (see fig. 3) for group x block x trial type ($F(1,36)=4.35$, $p<0.05$). To clarify which conditions generate this complex interaction we split the analysis into two more simple 2-way ANOVAs of group x block, for resolvable and misleading trials separately (see left and right plots in fig. 3). Only the interaction for misleading probe-1 trials (right plot in fig. 3) reached significance ($F(1,36)=5.98$, $p<0.02$), suggesting that accuracy (on the subsequent probe-2) for misleading trials differed significantly between blocks and between checking groups. Most interestingly, this difference appears to exist only within the misleading block (low checkers: 93.90 vs. high checkers: 90.79) supporting our strong hypothesis that high checkers memory performance is more impaired in the misleading compared to the resolvable context.

Probe-2 confidence

The MANOVA (2x2x3) for confidence in probe-2 responses revealed a significant main effect of trial type ($F(2,35)=8.67$, $p<0.001$) with misleading probe-1s inducing the least confidence in subsequent probe-2 responses. In addition, however, there was a significant 3-way interaction of group x trial type x block ($F(2,35)=4.16$, $p<0.03$). Figure 4 shows that high checkers indeed show decreased confidence compared to low checkers, but that this difference is not consistent across trial types and blocks, i.e., a quite similar pattern is observed for the two groups for resolvable trials in both blocks (left in fig. 4) while a more dissimilar pattern is revealed for misleading and no probe-1 trials (middle and right in fig. 4).

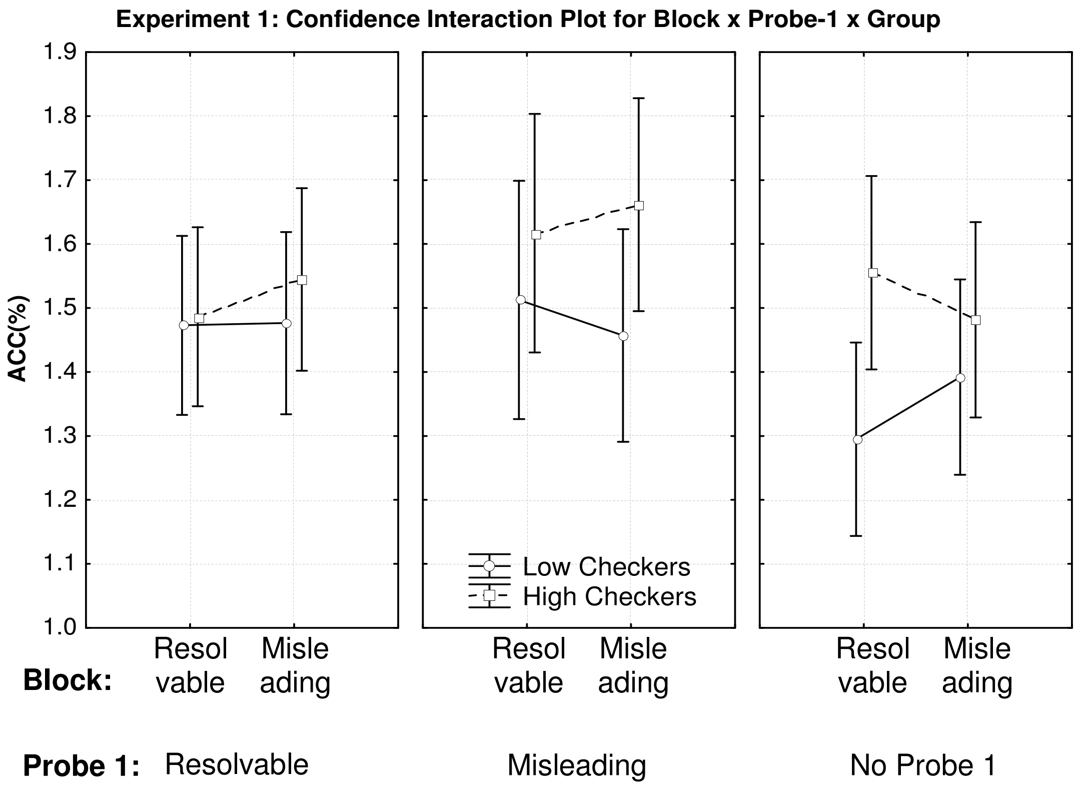

Figure 4. Experiment 1: Group (low vs. high checkers) x block (resolvable vs. misleading) x trial type (resolvable, misleading, or no P1) interaction plot for confidence in probe-2 responses. The scale ranged from 1 (highly certain) to 6 (highly uncertain), i.e., lower values reflect higher confidence. Vertical bars denote standard errors.

The ANOVA for the reduced 2x2x2 design (without no-probe 1 trials) failed to reveal any significant results, suggesting that the MANOVA results were substantially driven by the difference between misleading and no-probe1 trials (see fig. 4 middle and right graphs).

Finally we directly compared confidence between resolvable and misleading trials for equal frequencies to further understand the role of the misleading trials in the significant 3way interaction of the MANOVA. That is, we compared the 120 resolvable trials of the resolvable block to the 120 misleading trials of the misleading block. “Group” was included as a second factor. This 2x2 ANOVA revealed a significant group x trial type interaction ($F(1,36)=8.56$, $p<0.006$) that is shown in Figure 5. This interaction further substantiates the difference in confidence ratings between groups observed for misleading trials within the misleading block (middle graph fig. 4), where high checkers had less confidence than low checkers.

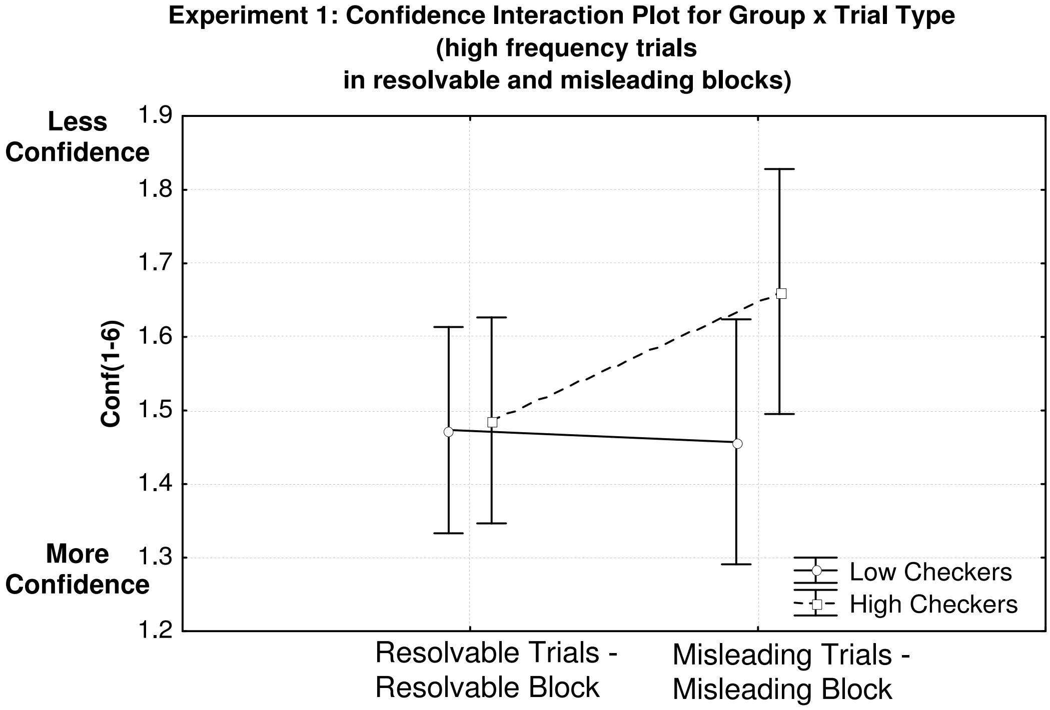

Figure 5. Experiment 1: Group (low vs. high checkers) x trial type (resolvable trials in the resolvable block vs. misleading trials in the misleading block) interaction plot for confidence in probe-2 responses. To re-iterate, lower values reflect higher confidence. Vertical bars denote standard errors.

2.1.3. Discussion of Experiment 1

We found evidence in reaction time (RT) and confidence ratings (CR) data, suggesting that our manipulation was successful in inducing checking, although the WM task was very easy (all conditions revealed mean accuracies over 90%). This effect was most evident in RTs, where across blocks and groups probe-2 responses were performed faster after a resolvable probe-1 than after a misleading probe-1. This effect was supported by the confidence ratings, where misleading trials led to lower confidence than resolvable and no-probe-1 trials. Furthermore, high checkers had less confidence than low checkers on misleading trials compared to resolvable trials when the respective trial types where blocked together. Finally and most importantly we observed group differences in performance accuracy for misleading trials within the mostly misleading block as reflected in a significant interaction of group, trial type and block. This suggests that checkers cannot easily ignore a misleading cue even if the experimental context emphasizes the irrelevance of the cue (i.e., strong hypothesis). Since this is the result with the potentially strongest impact on our understanding of compulsive checking we wanted to ensure its reliability. In a replication study we presented the misleading block only and focused on the group differences for the misleading trials.

2.2. Experiment 2 (replication of Experiment 1)

2.2.1. Method

Participants

40 volunteers (mean age 23.88: 14 male, 25 female) participated in this second study and a median split of the VOCI scores was used again to obtain a group of high (mean = 8.65, SD = 3.70) and a group of low (mean = 1.05, SD = 1.18) scorers on the checking scale.

Stimuli and Procedure

The same stimuli and procedure as in Experiment 1 were employed. The only change was that only the misleading block was presented (2/3 misleading trials).

2.2.2. Results

In order to test for a replication of the main finding of Experiment 1 we carried out hypothesis driven t-tests to compare probe-2 accuracy for high and low checkers. We expected high checkers to show again a lower performance for misleading probe-1 trials, which was supported by a significant t-test ($t(1,37)=2.276$, $p<0.029$) (fig. 6). The t-tests for the resolvable and no-probe-1 trials did not reach significance (both $t(1,37)<0.35$, $p>0.56$), supporting the notion that there were no general differences in WM capacity. This again supports the *strong* hypothesis which we proposed in section 1.4.2. With respect to confidence ratings (CR) numerically observed group differences did not reach significance (resolvable, misleading, no-probe-1: $p>0.28$).

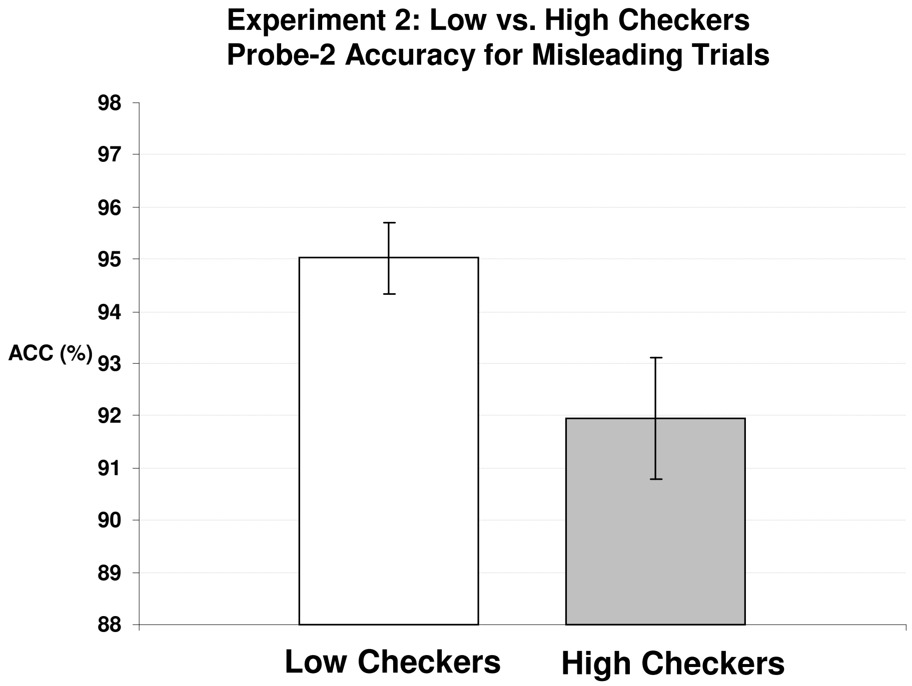

Figure 6. Experiment 2: Group comparison (low vs. high checkers) for probe-2 accuracy on misleading trials only. Vertical bars denote standard errors.

2.2.3. Discussion of Experiment 2

The accuracy outcome is a clear replication of the main finding in Experiment 1, allowing for a convincing conclusion that higher checking disposition is related to attenuated performance within the episodic part of WM if misleading information is provided. However, in both experiments we used a median split to create the two checking groups (low *versus* high). As a result, the high checking group in both experiments (Exp 1 = 9.53; Exp 2 = 8.65) scored below the clinical mean (15.6) of compulsive checkers on the checking subscale of the VOCI (Thordarson et al., 2004) making our conclusions tentative with respect to the clinical population. Therefore, we conducted a “meta-comparison” where we compared the extremely high checkers (mean score 15.8) to the lowest scorers (0.5) across both experiments.

2.3. Extreme Groups Meta-Comparison

We compared the data of the 10 participants with the highest scores on the checking subscale of the VOCI (mean = 15.8, SD = 2.57) to the data of the 10 lowest scoring participants (mean = 0.5, SD = 0.71) from both experiments. Critically, the high group scored in the clinical range for checking according the VOCI (Thordarson et al., 2004). Only the data from the misleading block were employed for participants drawn from Experiment 1 (n=6) to keep the data congruent to Experiment 2. Like in Experiment 2, hypothesis driven t-tests were conducted. Our strong hypothesis was again supported as only for misleading trials did *extreme* high checkers differ significantly from *extreme* low checkers ($t(1,18)=2.289$, $p<0.034$) (see fig. 7). Whereas, the t-tests for the resolvable ($t(1,18)=0.141$, $p>0.175$) and no-probe 1 ($t(1,18)=0.33$, $p>0.745$) trials did not reach significance, supporting the notion that the two extreme groups were comparable with respect to general WM capacity. Again, numerically observed group differences for confidence ratings did not reach significance (resolvable, misleading, no-probe-1: $p>0.39$).

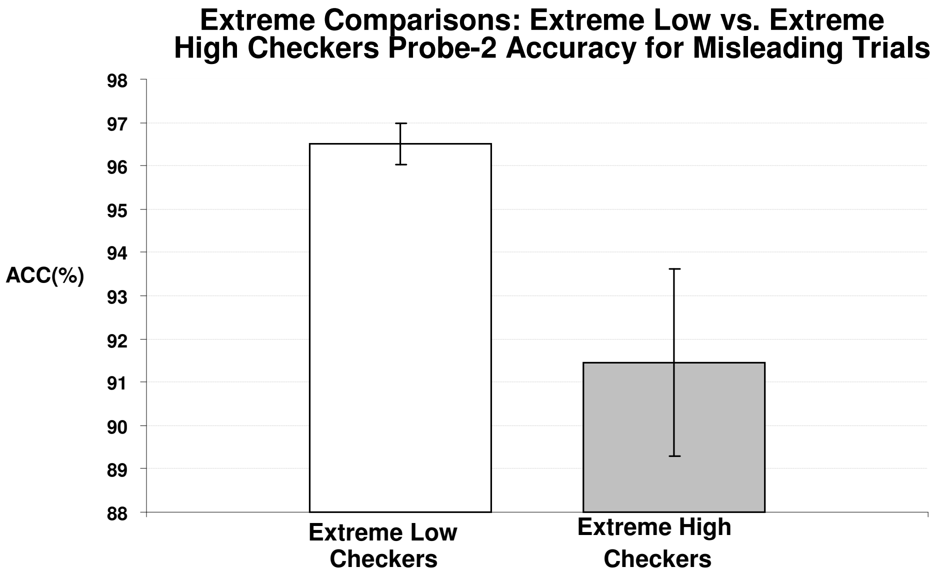

Figure 7. Extreme Scorers Meta-Analysis: Group comparison (extreme low vs. extreme high checkers) for probe-2 accuracy on misleading trials only. Vertical bars denote standard errors.

2.4. General Discussion

The aim of these first experiments was to show that internal WM representations, i.e., cross-modal bindings within the EB, can be affected by unproductive checking. Deteriorated WM performance was expected to be more pronounced in participants with a high checking predisposition. We found evidence in reaction time (RT) and confidence ratings (CR) data of Experiment 1 suggesting that our manipulation was successful in inducing a certain amount of detrimental checking in *all* participants. This effect was most evident in the RTs of Experiment 1 (Table 1), where across groups responses on the actual memory test (probe-2) were performed faster after a resolvable probe-1 than after a misleading probe-1. This effect was supported by the confidence ratings, where misleading trials led to lower confidence than resolvable and no-probe-1 trials. Importantly, this concurs with the classic attention-based WM rehearsal finding of Awh and Jonides (2001) who reported slower memory probe RTs when attention was previously directed to a different location (mismatch condition/misleading trials) compared to the same location (match condition/resolvable trials).

Regarding our group hypothesis Experiment 1 revealed less confidence for high checkers than low checkers in misleading trials but not in resolvable trials (fig. 5). However, in Experiment 2 and in the extreme groups meta-comparison only numerical differences were observed, possibly suggesting (1) that the task was too easy to affect metacognitive judgments in a straightforward way or (2) that the method of recording a confidence reduced actual between-group differences as participants perhaps responded more to end the trial than to indicate their actual confidence. As a result, we use different confidence response measures in latter experiments. That is, group differences were only revealed as part of quite complex 3-way and 2-way interactions in Experiment 1, which was not possible with the reduced design in Experiment 2 and the meta-comparison. Importantly and in agreement with previous findings (e.g., Ciesielski et al., 2007; Henseler et al., 2008) we did not observe general differences in WM capacity between high and low checkers: performance on resolvable and no probe-1 trials was comparable in both experiments as well as in the extreme groups meta-comparison. This suggests that group differences are not a WM capacity issue *per se* - especially with low demands employed here and in other research (e.g., Morein-Zamir et al., 2010; Purcell et al., 1998a; van der Wee et al., 2003).

According to our group hypothesis regarding probe-2 accuracy, we observed differences in performance accuracy in Experiment 1 when irrelevant but misleading probes were maximally concentrated: for misleading trials when these trials where highly frequent (misleading block). This crucial finding was replicated in Experiment 2 and was also revealed in the extreme group comparison across experiments underpinning the clinical relevance of our findings. This provides a convergence of support for our *strong hypothesis* that in misleading trials high checkers' WM performance will be poorer compared to low checkers.

We conclude that our experimental manipulation resonated with personal checking dispositions and affected WM representations. It appears that low checkers have learned more readily to ignore irresolvable probes especially in an experimental context where such probes were highly frequent so their irrelevance became even more obvious (misleading block). In contrast, high checkers might have "checked yet another time" whether the probe letter "really" wasn't anywhere. That is, high scorers appeared to be less able to suppress the misleading probes and the associated intrusive thoughts. In turn, this might have initiated repeated scans through WM to compare the irresolvable probe with each letter-location binding over and over again. We propose that the competition between a strong, visually present letter-stimulus and the fragile letter-location bindings in the EB weakens these multimodal representations. This assertion is supported by the *simplified comparison theory* of Makovski, Sussman and Jiang (2008). In this they suggested that exhaustively comparing every probe item (i.e., misleading P1) with those maintained in memory (encoding set: letters in locations) may come at a cost to WM performance. Therefore, repeated checking due to insufficient suppression of misleading information might have therefore resulted in repeated competition and increasingly weaker bindings. This is in agreement with research showing that: (1) checkers (not washers) have impairments in memory that are associated with dysfunctional inhibitory control (Omori et al., 2007), (2) urges to check are mediated by the degree of experienced intolerance of uncertainty (Rachman & Hodgson, 1980; Tolin et al., 2003) (i.e., which checkers likely experienced when externally challenged by a misleading letter) and (3) doubt/uncertainty regarding the contents of memory will increase the likelihood of covert checking (Alcolado & Radomsky, 2011; McNally & Kohlbeck, 1993), which in turn may interfere with the integrity of internal representations (Makovski, Sussman, & Jiang, 2008; Nedeljkovic & Kyrios, 2007; Radomsky & Alcolado, 2010). This latter point is supported by the established sensitivity of attention-dependent bindings of the EB to interference.

In Figure 1 we provided our original adaptation of Baddeley's (2000) model to predict and interpret our findings of compulsive checking. Thus, while we originally proposed Baddeley's (2000) model of WM in explanation of the manner (i.e., executive-memory interaction) and cognitive location (i.e., EB) underlying memory impairment in OCD-checking, we now further elucidate upon this. According to this framework compulsive checking could involve three components that together make up a vicious circle.

(1) Executive dysfunction could result in a lack of suppression of misleading information, which is in strong agreement with the susceptibility to intrusive thoughts (Bannon, Gonsalvez, & Croft, 2008; Salkovskis, 1999) and the general executive dysfunction (Olley et al., 2007; Omori et al., 2007) reported in clinical OCD samples. The misleading/intrusive information can be internally generated in form of intrusive thoughts ("I think I left a burner on") or can be externally provided in form of challenging questions ("Where was the letter?" or "Are you ABSOLUTELY SURE that you turned all burners off?"). This explains domain-specific WM deficits because WM performance is only disrupted when the WM task requires a component of the central executive that is dysfunctional, e.g., does not suppress intrusive information (Dek et al., 2010; Radomsky & Alcolado, 2010). If there is no external challenge or the stimulus domain does not induce intrusive thoughts then no performance deficit will be observed (Friedman & Miyake, 2004). This point should be considered in all WM research that compares OCD performance to typical populations. In the case of our experiments we provided an external challenge and we observed the effects although the stimuli were not related to individual checking domains. Our considerations might also provide an explanation for the potential progression of subclinical checking towards clinical. Checking might be likely to create conditioned associations over time between external or internal challenges of performance (e.g. "Did you/I turn the stove off?") and intrusive thoughts (e.g. "I think I left a burner on") mediated by anxiety (e.g., MacLeod & Mathews, 1991). This would in turn incrementally increase the likelihood of detrimental checking in this specific domain and could lead to self-reinforcement of intrusive thoughts (e.g., Hartston & Swerdlow, 1999).

(2) The lack of suppression of misleading information in turn might trigger repeated checking of the EB contents. In the case of our findings, the competition between a visually present probe letter and fragile letter-location bindings in the EB of WM weakens these bindings the more often this competition is repeated. With intrusive thoughts the challenge for the bindings is generated within the system itself and the more often the bindings are reactivated and their veridicality challenged the less reliable they will become. Paradoxically, while high-scorers check to improve their performance it actually undermines performance by reducing the accuracy of the WM representation (Radomsky & Alcolado, 2010). In that sense checking critically differs from mere rehearsal. Both processes imply reactivation of memory representations, yet, while rehearsal reactivates "without questioning", checking seems to imply a lack of confidence in the veridicality of the reactivated information that is detrimental without the original sensory information to check against, or even with competing new sensory information present.

(3) The final component is the consolidation of EB representations into episodic LTM. If the EB representations are progressively weakened by checking then the consolidated representations in LTM will be affected as well (Tolin et al., 2001), thus, further increasing the likelihood of subsequent checking in LTM that has been shown to decrease accuracy and confidence in episodic representations (van den Hout & Kindt, 2003a, 2003b). Savage et al. (1999; and Deckersbach et al., 2000; Penades et al., 2005; Savage et al., 2000; Segalas et al., 2008) reported intact copy but impaired immediate and delayed memory performance of OCD patients on the Rey-Complex Figure Task (i.e., a complex visuospatial stimulus requiring the organization and maintenance of multiple feature/object-location bindings). Preserved copy performance and no additional loss of information between the immediate and delayed conditions indicated that memory capacity did not moderate memory performance. Rather, a failure in executive functioning to efficiently encode visuospatial information during the copy phase mediated impaired performance in their immediate and delayed recall. In other words, impairment of the visuospatial organization and reconstruction in the observers' EB had a direct impact on the amount of information encoded and recalled immediately after construction and the longer term. Furthermore, OCD checkers have exhibited poorer memory confidence for threatening stimuli that they had been repeatedly exposed to (akin to repeated checking) one week earlier compared to non-checking OCD patients (Tolin et al., 2001). A self-awareness of repeated loss of accuracy and confidence in memories may finally increase the likelihood and the strength of misleading intrusive thoughts which would then be harder to ignore (e.g. Hartston & Swerdlow, 1999). High checkers might therefore end up in a vicious circle of checking at various stages of memory that does not improve but further deteriorates memory traces (Nedeljkovic et al., 2009b). The notion proposed here slightly shifts the explanatory focus from retrieval to consolidation, which has direct clinical relevance. We suggest that a combined WM and LTM explanation might provide a comprehensive etiological starting point for the qualitatively different experience that individuals with checking disorders appear to exhibit in their pathological desire to check and their dissatisfaction with it after it has been executed.

Therefore, we place executive dysfunction at the heart of high checkers WM impairments as opposed to deficits in WM capacity *per se*.

2.4.1. Limitations and future research

While we argue that our data have added to the checking-memory literature in an important way we have identified the following limitations and future avenues of research that have emerged with respect to Experiments 1 and 2. Firstly, the use of sub-clinical samples of high checkers may appear to limit the conclusions that can be made with respect to clinical populations. However, we argue that the results of our extreme groups meta-comparison (clinically scoring *versus* lowest scoring participants across Exps. 1 and 2) substantiates the clinical implications of our findings. Furthermore, the result that even within the typical population checking tendencies impact on WM performance is of importance. For example, subclinical checkers have shown similar deficits to those observed in clinical OCD, i.e., the Wisconsin Card Sorting Task (Gershuny & Sher, 1995) and the Wechsler Memory Scale (Sher, Mann, & Frost, 1984). Further, this same group have shown memory deficits for everyday activities (Sher, Mann, & Frost, 1984), prospective memory impairments (Cuttler & Graf, 2007, 2008, 2009) and were poorer at distinguishing real from imagined events (Rubenstein et al., 1993). This has lead some researchers to suggest that a subclinical analogue is a valid means of understanding a variety of features relevant to clinical OCD, especially as they are free from confounds such as medication, clinical state, or co-morbidity (Mataix-Cols et al., 1997; 1999a). Indeed, considering this alongside the commonality of checking in OCD (50-80%; (50-80%; Antony, Downie, & Swinson, 1998; Henderson & Pollard, 1988; Rasmussen & Eisen, 1988) and the population generally (15%; Stein et al., 1997), subclinical checkers may provide a 'purer' means for determining the specific impact of executive deficits upon WM functioning. Future research, however, should ensure the validity of our claims by using a larger clinically scoring or a clinically diagnosed sample. Also, if the observed WM performance is specific to pathological checking, then it should differ from performance associated with other obsessive-compulsive sub-types (e.g., hoarding, contamination, cf. Abramowitz, McKay, & Taylor, 2005) and other disorders, i.e., generalized anxiety disorder, social phobia and depression. Secondly, with respect to the probe-1 design we did not explicitly manipulate checking per se but hypothesized that high checkers are likely to check the content of WM more often relative to low checkers if irrelevant but misleading information is provided. It could however be that checkers are simply more distracted by an irrelevant probe, which reduces the attentional processes required for rehearsing the encoded information (see Cuttler & Graf, 2007, for a similar notion). This does not necessarily imply enhanced checking behaviour per se. Nevertheless, the detriment in memory performance of high checkers was observed for misleading trials only, while resolvable and no-probe-1 trials were comparable. This suggests that it was the misleading content and not the mere presence of a distracting probe that attenuated WM performance. Yet this does not fully rule out the possibility of distraction and future research should directly manipulate checking within a WM paradigm, which unfortunately is not trivial without overly affecting the primary WM task. For example, in a delayed-match-to-sample-task Rotge et al. (2008) provided OCD patients with the opportunity to check and recheck the original encoded-set to allow verifications with respect to the accuracy of the memory probe. They reported that while OCD patients WM performance was intact, they did make more verifications and spent longer before subsequent checks compared to healthy controls. Interestingly, these behavioural patterns were more pronounced in checkers. Thus, allowing checkers to physically check may show that they do in fact check but it will likely attenuate WM impairments which are sensitive to verifications occurring purely within WM. Indeed, we test this very hypothesis in Experiment 5, where we measure the eye movements of high and low checkers to examine group differences when presented with misleading compared to resolvable probes. In addition, what participants were experiencing during the WM task could have been recorded in more detail after the experimental procedure. For example, participants could have been asked to: (1) rate the degree of uncertainty they felt when presented with a misleading compared to a resolvable probe, (2) explain how and when they actually check the contents of their memory, and/or (3) what different strategies did they employ (if any) for misleading, resolvable and no-probe-1 trials. This information could have then been independently coded and analysed, with the aim of revealing phenomenological differences between high and low checkers in how they experienced and dealt with a misleading compared to resolvable intermediate probes. Thirdly, the use of letters and locations has limited ecological validity with respect to checkers' idiographic believe systems and anxieties. The use of ecologically valid stimuli within a WM paradigm might reveal even stronger effects than the ones reported here – especially if the high-checking group was drawn from a clinical population. This could also shed light on the implications of anxiety associated with specific checking domains (e.g. MacLeod & Mathews, 1991). This point is addressed specifically in Experiments 7 and 8 by requiring participants to encode and recall electrical kitchen appliances located on a kitchen countertop. Finally, the basic WM task employed here was very easy. Stronger group differences regarding the impact of misleading information could be revealed with a harder task (cf. Van der Wee et al., 2003). A point specifically addressed in Experiments 3 and 4 presented below.

3. Checkers' memory impairments persist in more complex working memory experiments

Experiments 3 and 4 are a logical extension from the findings and methodology of Experiments 1 and 2 (Harkin & Kessler, 2009). Whereby, we set out to determine in more detail the relation between WM, misleading information, and checking disposition. Our experimental extensions were two-fold. Firstly, in Experiment 3 we increased the complexity of the WM task by including a further feature dimension (colour) to test whether a harder task would increase the performance difference between high and low checkers (i.e., van der Wee et al., 2003). Our reasoning being that in normal subjects, inhibitory functioning is impaired when concurrent demands upon the central executive are high (Eysenck et al., 2007). For example, Graydon and Eysenck (1989) reported that the negative effect of distracting stimuli on task performance increased as a function of greater load within WM. Also Lavie et al. (2004) showed that selective attention performance was more negatively affected by distracting stimuli when demands upon WM were high but not low. Therefore, as our present experimental manipulation (Experiment. 3) calls upon extra attentional resources – compared to Experiments 1 and 2 – interference with this attention may further attenuate memory performance, especially in checkers who previously were shown to be poorer at inhibiting a misleading intermediate probe. Secondly, in Experiment 4 we challenged the fragile letter-location bindings via their "weaker link" by asking which letter had been in a specific <u>location</u>, while there either had or had not been a letter. In this case, locations were the weaker link as they have no permanent representations in LTM to aid WM encoding. In contrast, Harkin and Kessler (2009) asked where a specific <u>letter</u> had been, thus, accessing the representations via their stronger letter-identity part (stronger because letters are stored in LTM). The expectation was that this could further undermine the trust that high checkers have in their memory representations resulting in more pronounced group effects.

3.1. Experiment 3

Similar to Harkin and Kessler (Exp. 1 and 2; 2009) the checking manipulation consisted of presenting an intermediate probe (between the encoding set and the actual memory test) that could be misleading in the sense that it was not resolvable. In Experiment 3 (see fig. 8) this probe asked for the colour of a letter that was either *part* of the encoding set, hence resolvable (e.g., What colour was Z), or *not part* of the encoding set, hence irresolvable (e.g. What colour was K). We wanted to investigate whether enhancing the WM task difficulty by adding colour as another feature dimension would result in stronger group effects with high checkers' performance being dramatically worse than low checkers' for misleading/irresolvable trials. Checking induced by the misleading information could have an increasingly negative effect the more difficult the task is. We were also expecting to observe similarly enhanced group effects for confidence, i.e., high checkers revealing less confidence in their WM performance than low.

3.1.1. Method

Participants

40 Participants (mean 19.55 years; 7 male, 33 female) from the University of Glasgow gave written informed consents. British Psychological Society (BPS) ethical requirements were met. We used the checking subscale of the VOCI (Thordarson et al., 2004) and employed a median split of checking scores to obtain two groups: low (mean = 1.74, SD = 1.69) and high (mean = 12.57, SD = 5.32) "checkers".

Stimuli and Procedure

Participants sat 90cm from a 19'' computer screen ran at 800x600 resolution with their head on a chin rest. Stimuli were capital letters in font Arial, size 18 and were presented against a black background within a 2 (columns) by 3 (rows) matrix covering an area of 300x420 pixels. After 1000ms fixation, 4 letters were presented randomly in 4 of the 6 possible locations and participants had 2000 ms to encode the identity and the location of each letter (fig. 8). After 500ms, the probe-1 question asked for the colour of a specific letter. Participants indicated the colour through 6 colour coded keypad responses and were instructed to respond within 4000 ms (to keep the WM delay constant). Whether the probe-1 letter had or had not been part of the encoded set created the resolvable versus misleading (irresolvable) trials. Asking for the colour of a letter added to the difficulty of this task, particularly in the case of misleading trials. In a baseline condition probe-1 was omitted to measure WM performance on the primary task under ideal conditions.

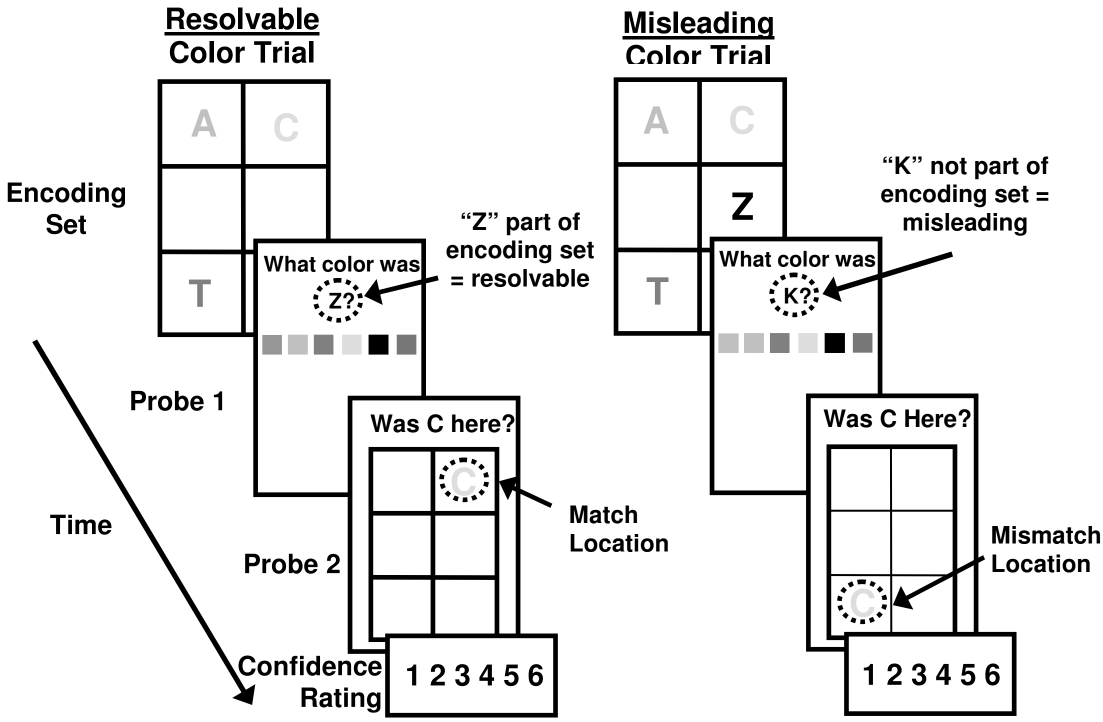

Figure 8. Experiment 3: Schematic procedure of resolvable and misleading trials. A set of 4 letters in 6 possible colors were presented randomly in 4 out of 6 possible locations had to be encoded within 2 s. Encoding was then followed after 500 ms by probe (probe-1) asking for the color of a letter that was present or not, i.e., was resolvable or misleading. Subsequently participants had to indicate if the probe-2 letter match or mismatched it location with respect to the encoded set, which was the actual memory test. Finally confidence in probe-2 response had to be indicated on a scale from 1 (highly certain) to 6 (highly uncertain). Further explanations in the text.

A 1000 ms interstimulus interval (ISI) separated probe-1 and probe-2. Since baseline trials did not include the intermediate probe-1 a black screen was shown for 5500 ms between encoding and probe-2 (equalling the ISI between encoding and probe-2 on the other trial types). Probe-2 was the actual memory test for each trial and required participants to indicate if a letter was a match or a mismatch in terms of location with respect to the originally encoded set. It is important to note that we only analysed probe-2 performance and how it changed depending on the various probe-1 manipulations. In all trials the probe-2 letter had been part of the encoded set in terms of identity while the probe location was a match on 50% of the trials. Finally, a scale was displayed prompting participants to indicate their degree of confidence in their probe-2 response (6 levels: 1=totally certain to 6=totally uncertain). Three self-paced breaks were included and the experiment lasted approximately 90 minutes. The resolvable block comprised 180 trials with 120 resolvable trials, 40 misleading trials, and additional 20 baseline trials (no-probe-1). Correspondingly, the misleading block (180 trials) was made up of 40 resolvable trials, 120 misleading trials, and again 20 baseline trials. The sequence of these blocks was counterbalanced across participants in order to avoid order effects.

Design

We employed a 2 (*group*: low vs. high checkers) by 2 (*block type:* mostly resolvable vs. mostly misleading block) by 3 (*probe-1 trial type*: resolvable, misleading, no probe-1) mixed design with *group* as the between- and *block* and *probe-1* as the within-subjects factors.

3.1.2. Results

MANOVA's were employed due to violations of the sphericity assumption (Mauchly's tests). Statistics for the 2x2x3 design were carried out for reaction times, accuracy and confidence on probe-2 responses. Note we only analysed performance on probe-2 (depending on the different levels of the intermediate probe-1). As our theoretical predictions specifically focused on the effect of checking induced by resolvable vs. misleading probe-1 trials we also conducted a 2 x 2 x 2 ANOVA removing the no-probe-1 trials.

Probe-2 response latencies

The MANOVA (2 x 2 x 3) for probe-2 latencies failed to reveal any significant main effects or interactions.

Probe-2 response accuracy

The MANOVA (2 x 2 x 3) for probe-2 accuracy revealed a main effect of probe-1 trial-type ($F(2,37)=18.38$, $p<0.001$). Further analyses revealed that resolvable and misleading probe-1 significantly differed from no-probe-1 ($F(1,38)$, $p<0.001$, $p<0.001$, respectively). Resolvable versus misleading probe-1 approached but did not reach significance ($F(1,38)=3.0224$, $p=0.09$). This indicates greater probe-2 accuracy for no-probe-1 trials compared to misleading and resolvable trials and that there was a trend toward less accuracy for misleading trials. There was a significant main effect of probe-2. Correctly located probe-2 trials where significantly less accurate than incorrectly located trials (74.4% vs. 88.7%: $F(1,38)=33.33$, $p<0.001$). We argue that an accurate correct probe-2 response requires the precise memory of the probe letter in its original location so that the match between probe and memory exceeds the response threshold. In contrast, incorrectly located probe-2 trials can be accurately performed using incomplete/partial information such as overall letter locations and/or possibly letter shape information that can quickly generate a mismatch (i.e., round 'D' vs. jagged 'X'). Especially with a complex task like the one employed here (in contrast to Harkin & Kessler, 2009) where several features have to be bound together this asymmetry has become obvious in performance.

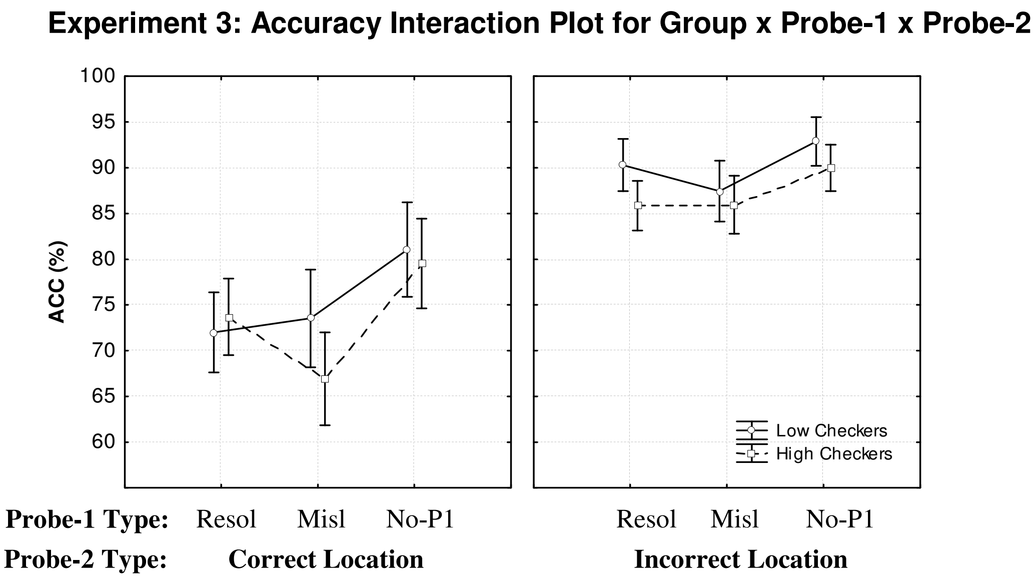

Figure 9. Experiment 3: Group (low vs. high checkers) x probe-2 (correct vs. incorrect) x trial type (resolvable, misleading, no-probe-1) interaction plot for probe-2 accuracy. Vertical bars denote standard errors.

Most importantly, there was also a significant 3-way interaction (fig. 9) for group x probe-2 x probe-1 trial type. This interaction was the result of high and low checkers having different accuracy for different levels of probe-2 and probe-1 trial-type. First, the main effect of probe-2 was apparent in the data pattern in Figure 9: accuracy was poorer overall for correct compared to incorrect probe-2 trials. Second, no group differences (low vs. high checkers) were revealed for mismatch probe-2 across the three probe-1 trial types ($F(1,38)$= resol: p=0.12; misl: p=0.65; no-p1: p=0.27). This indicates that group differences are likely to reside in the match probe-2 condition. Therefore, we conducted an ANOVA by removing the no-probe-1 trials as no significant group differences were revealed, indicating that WM capacity was intact. The ANOVA for this reduced design again revealed the significant 3-way interaction for group x probe-2 x trial type ($F(1,38)=7.54$, $p=0.009$). The simple interaction for group x probe-1 trial type for correct probe-2 trials was significant ($F(1,38)=4.95$, $p=0.032$) (left plot fig. 9). To determine which effects generated this interaction we analysed the simple effects. The only effect that reached significance was the high checkers' performance on resolvable (73.1%) versus misleading (67.1%) trials ($F(1,38)=6.95$, $p=0.012$). The same comparison for low checkers failed to reach significance (71.9% vs. 73.4%; $p=0.58$). This supports our hypothesis that high checkers have an executive deficit in inhibiting information that is misleading and irrelevant. As a result checkers seem to look for the colour of a letter that was actually not presented disrupting the 'true' information retained in memory.

Probe-2 confidence ratings

The MANOVA (2 x 2 x 3) for confidence on probe-2 responses revealed significant main effect of probe-1 ($F(2,37)=28.415$, $p<0.001$). No-probe-1 trials had the most confidence and were statistically different from resolvable ($F(1,38)=41.99$, $p<0.001$) and misleading trials ($F(1,38)=51.99$, $p<0.001$). There was a significant main effect of probe-2 trial type ($F(1,38)=27.256$, $p<0.001$), indicating that there was less confidence for correct probe-2 trials versus incorrect. The MANOVA revealed a 3-way interaction for group x block x probe-2 that approached significance ($F(1,38)=3.88$, $p=0.056$). However, the ANOVA for the reduced design (removed no-probe-1 trials) produced a significant 3-way interaction ($F(1,38)=5.04$, $p=0.031$) (fig. 10a: MANOVA and fig. 10b: ANOVA).

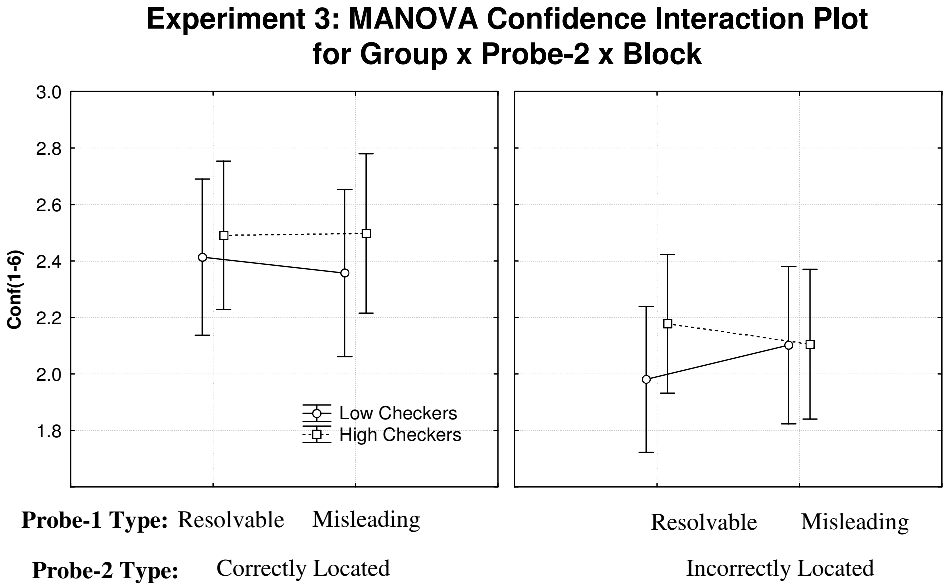

Figure 10a. Experiment 3: MANOVA for Group (low vs. high checkers) x block (resolvable vs. misleading) x probe-2 (correct vs. incorrect) interaction plot for confidence in probe-2 responses. The scale ranged from 1 (highly certain) to 6 (highly uncertain), i.e., the lower value reflect higher confidence. Vertical bars denote standard errors.

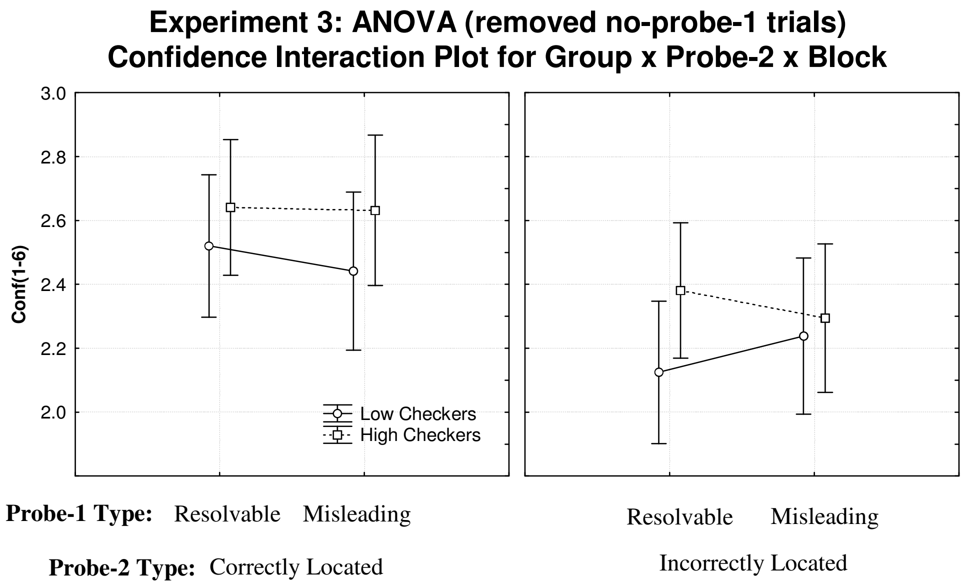

Figure 10b. Experiment 3: ANOVA for Group (low vs. high checkers) x block (resolvable vs. misleading) x probe-2 (match vs. mismatch) interaction plot for confidence in probe-2 responses. The scale ranged from 1 (highly certain) to 6 (highly uncertain), i.e., the lower value reflect higher confidence. Vertical bars denote standard errors.

There was a tendency to have less confidence in correct than incorrect probe-2 responses, a pattern that corresponds with poorer accuracy in correct versus incorrect probe-2 responses. The removal of no-probe-1 trials (ANOVA) increased the magnitude of difference between groups, with high checkers having poorer confidence across conditions (block and probe-2) compared to low. This suggests that for no-probe-1 trials, high checkers have comparable confidence to low checkers.

3.1.3. Discussion of Experiment 3

In conclusion, high checkers' performance was poorest for misleading trials in the correct probe-2 condition. No difference was observed between groups in the easier incorrect probe-2 condition, indicating that irrelevant and misleading stimuli capture the attention of high checkers to a greater extent than low. This also indicates the capacity differences between groups are not responsible for the slight group performance difference in the correctly located probe-2 condition. However, while this pattern was not confined to the misleading block only, as had been the case in our previous findings (Harkin & Kessler, 2009), it was not more pronounced in that the difference between the groups was not dramatically enhanced. It would seem that by increasing the complexity of the WM task performance drops for everyone by a similar amount with high checkers not suffering disproportional losses of performance. Confidence responses, however, revealed that the mere presence of an intermediate probe (resolvable or misleading) resulted in high checkers' poorer confidence overall, which was not observed as clearly in our previous studies. For high checkers, therefore, the presence of an irrelevant intermediate probe in a hard WM task appears to affect confidence more strongly than their actual performance compared to low checkers.

3.2. Experiment 4

Since increased complexity of the WM task resulted in a general drop in performance without a more accentuated group effect we employed an alternative strategy. If checking is really detrimental to bindings by inducing a competition between incoming perceptual information (misleading probe) and the fragile multimodal bindings in the EB then it could be even more detrimental for high checkers if the veridicality of the encoded representations was questioned with respect to their ‘weaker link’. In our case the locations are the weaker link compared to letters as they do not have a LTM trace that could support their retention. The prediction therefore was that we would observe very clear group effects with a misleading probe that specifically challenges the spatial part of the WM representations.

Hence, for the intermediate probe (probe-1) we asked which letter was presented at a particular location while there either *had* (resolvable) or *had not* been a letter (misleading) (see fig. 11). We expected high checkers' memory performance to be generally impaired for resolvable and misleading conditions. This would support a general executive difficulty in suppressing irrelevant information (Wolters and Raffone, 2008). Specifically, the largest impairment was expected for the most difficult (match probe-2) and frustrating conditions (misleading trials/block). Intact basic WM capacity is expected in baseline no-probe-1 trials.

3.2.1. Method

Participants

40 Participants (mean 20.12 years: 10 male, 30 female) from the University of Glasgow gave written informed consents. BPS ethical requirements were met. A median split of VOCI checking scores was used to obtain two groups: low (mean = 0.89, SD = 1.15) and high (mean = 10.48, SD = 5.96) “checkers”.

Stimuli, Procedure, and Design

This experiment used the same encoding (4 letters in 6 locations) and memory test (probe-2 correct or incorrect location) as Experiment 1. However, in this case the 4 letters were randomly selected from D, F, G, H, J, and K. This served the intermediate probe-1 manipulation. After 500 ms, the 2x3 matrix was again presented, but empty, and this time the participant was asked to indicate what letter had been at an indicated location. Participants selected the letter they believed to be at that location by pressing the corresponding letter-key on the keyboard and were instructed to respond within 4000 ms (to keep the WM delay constant). The probe-1 spatial cue either indicated a location where a letter had been present (resolvable) or a location that had been empty (misleading).

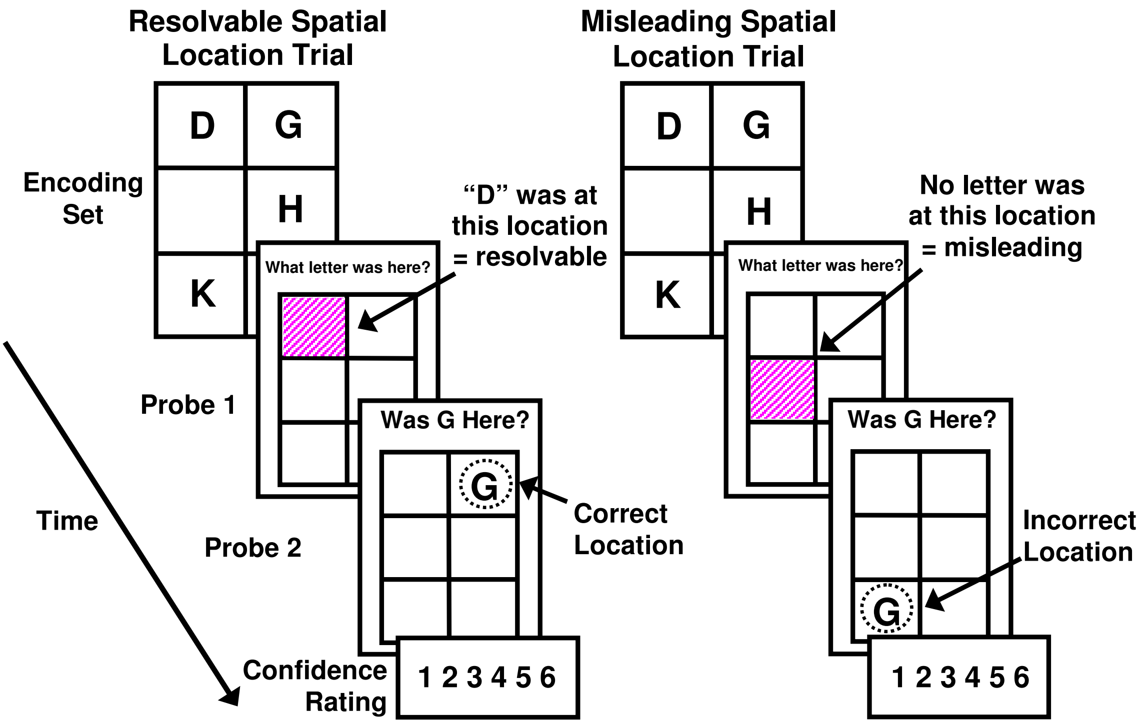

Figure 11. Experiment 4: Schematic procedure of resolvable and misleading trials. A set of 4 letters presented randomly in 4 out of 6 possible locations had to be encoded within 2 s. Encoding was then followed after 500 ms by probe (probe-1) asking what letter was at a cued location, where a letter was present or not, i.e., was resolvable or misleading. Subsequently participants had to indicate if the probe-2 letter either correctly or incorrectly located with respect to the encoded set, which was the actual memory test. Finally confidence in probe-2 response had to be indicated on a scale from 1 (highly certain) to 6 (highly uncertain). Further explanations in the text.

In a baseline condition probe-1 was omitted to measure WM performance on the primary task under ideal conditions. Finally, as in Experiment 3 a scale was displayed prompting participants to indicate their degree of confidence in their probe-2 response (6 levels: 1=totally certain to 6=totally uncertain). Three self-paced breaks were included and the experiment lasted approximately 90 minutes. The same overall design as Experiment 3 was employed.

3.2.2. Results

Again full MANOVA (2 x 2 x 3) and reduced ANOVA (2 x 2 x 2) designs were employed to analyse, reaction times (RT), accuracy (ACC), and confidence (CF).

Probe-2 response latencies

The MANOVA (2 x 2 x 3) for probe-2 latencies revealed a main effect for probe-2 trial type ($F(1,38)=25.18$, $p<0.001$) with correct probe-2 responses (1969ms) faster than those that were incorrect (2120ms). There was a significant 2-way interaction for block x trial-type ($F(2,76)=6.16$, $p<0.004$). Resolvable trials in a resolvable block had a faster RT than misleading trials, a pattern that was reversed for misleading trials in the misleading block. This suggests that when a trial and a block were congruent then RTs were faster than when they were incongruent. In addition, no-probe-1 trials were slower in a predominantly misleading block than a resolvable block. This indicates that a misleading block increases decision-making time and block context was sufficient to influence decision-making time in baseline WM trials.

Probe-2 response accuracy

The MANOVA (2 x 2 x 3) for probe-2 accuracy revealed a main effect for probe-2 ($F(1,38)=17.65$, $p<0.001$), with more accurate responses for incorrect compared to correct, replicating Experiment 3. The main effect of block was significant ($F(1,38)=10.98$, $p<0.003$), with less accuracy overall in the misleading block compared to the resolvable. This matches our expectations that a misleading block is particularly distracting and attenuates WM performance. The main effect of trial-type was significant, and indicated significantly greater accuracy in the no-probe-1 trials compared to resolvable and misleading (both comparisons: $p<0.001$). The absence of this main effect in the reduced ANOVA (removal of no-probe-1 trials) supports the impact of no-probe-1 trials.

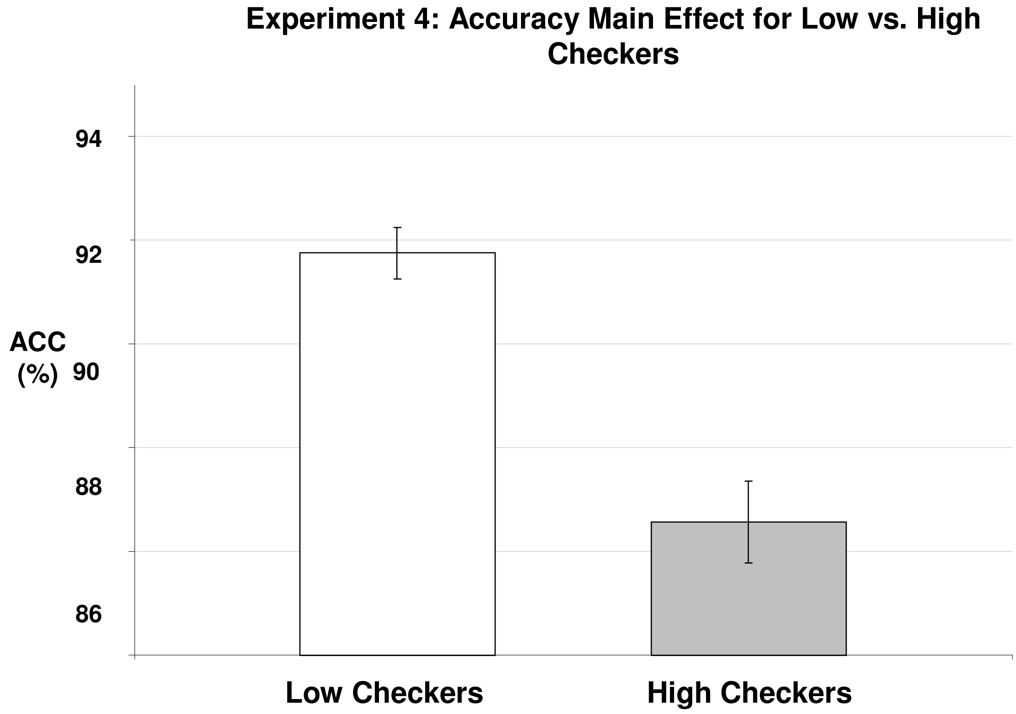

Figure 12. Experiment 4: Main Effect of Group (low vs. high checkers) for probe-2 accuracy. Vertical bars denote standard errors.

Finally and most importantly, the main effect of group reached significance ($F(1,38)=5.83$, $p<0.021$) with high checkers being less accurate than low checkers across all conditions (fig. 12). This group difference was observed for resolvable ($t(1,38)=5.66$, $p<0.023$) as well as for misleading ($t(1,38)=5.53$, $p<0.024$) trials (fig. 13, left). While the group difference did not reach significance for the baseline no-probe-1 trials, there was a statistical trend for lower performance of high checkers ($p=0.092$).

Although we found a main effect of group for the first time, which supports the claim that our strategy of challenging WM via the 'weaker spatial link' was indeed more detrimental for high checkers, we also expected at the same time that basic WM capacity in the baseline trials (no-probe-1) would not differ between high and low checkers. Hence, we conducted a more specific group analysis for the no-probe-1 trials and found that high checkers had significantly less accurate no-probe-1 responses in a misleading compared to a resolvable context ($t(1,38)=14.82$, $p<0.001$), a difference that was not observed for low checkers ($t(1,38)=2.42$, $p=0.127$) (fig. 13, right). Thus, the trend for a group effect on no-probe-1 trials was mainly driven by the misleading context. Comparable performance in the resolvable context indicates that, conform to our expectations, high checkers are not impaired in WM capacity *per se* but negatively influenced by the misleading context. However, we are aware of the limitation of conducting post-hoc contrasts in the absence of a significant interaction, In defence, we propose that as we predicted repetitive checking within a misleading context (strong hypothesis) that this carried over onto no-probe- trials, thus we justify the use of these exploratory contrasts but highlight their limited statistical robustness.

Experiment 4: Accuracy Plot for (1) Group x Probe-1 (left) and (2) Group x Block (right)

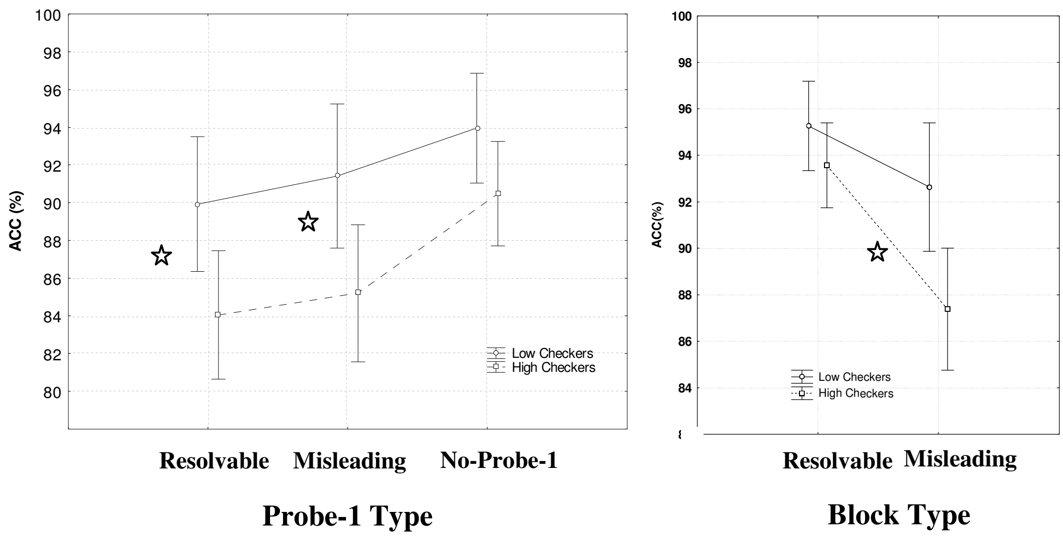

Figure 13. Experiment 4: Left Graph: Group (low vs. high checkers) x trial-type (resolvable, misleading, no-probe-1) interaction plot for probe-2 accuracy. Right Graph: Group (low vs. high checkers) x block-type (resolvable vs. misleading) for no-probe-1. Star denotes significant difference. Vertical bars denote standard errors.

Probe-2 response confidence ratings

The MANOVA (2 x 2 x 3) for confidence in probe-2 responses revealed a significant main effect of group ($F(1,38)=4.25$, $p<0.05$), with high checkers having less confidence overall than low checkers (see fig. 14). High checkers revealed a lack of confidence that operates irrespective of a specific condition. Such a general deficit was not observed previously, suggesting that an intermediate spatial probe was particularly detrimental to checkers' confidence. A significant main effect of probe-1 trial type ($F(1,38)$, 16.10, $p<0.001$) was driven by the significant differences of misleading and resolvable trials compared to no-probe-1 trials. This was further modulated by block as indicated by a significant 2-way interaction between block and trial-type ($F(1,38)=5.45$, $p<0.007$). This interaction directly reflected the block x trial-type accuracy pattern with less confidence for conditions with less accuracy and vice-versa.

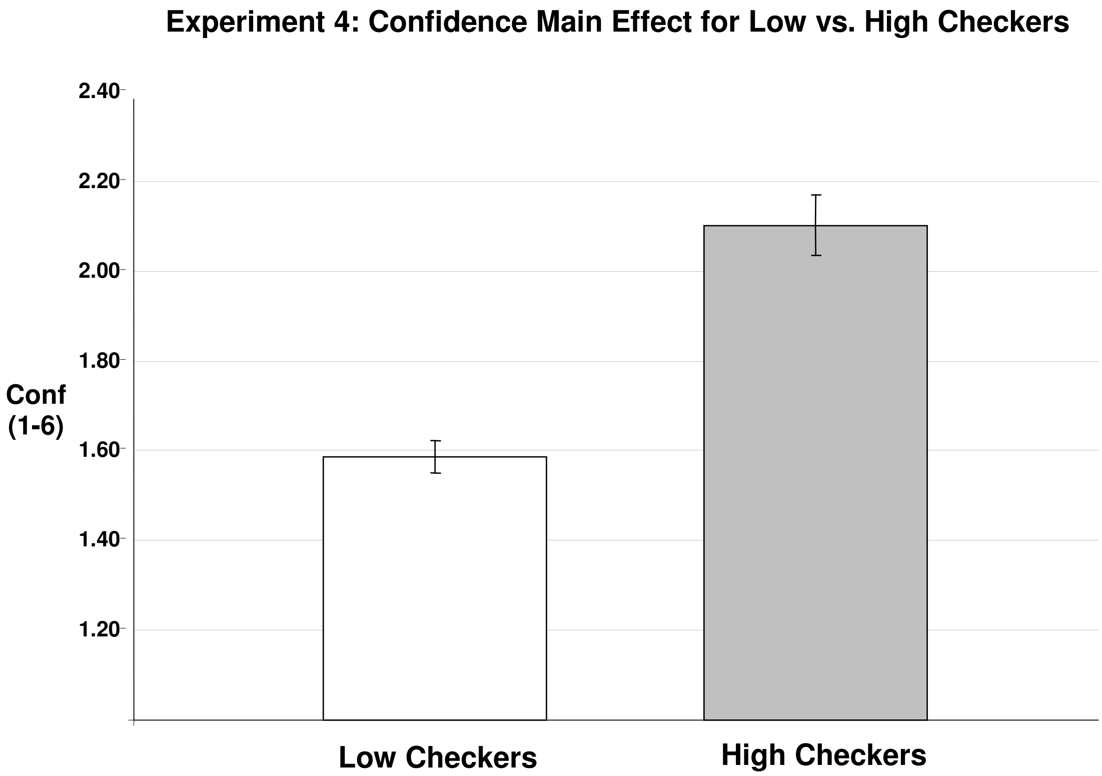

Figure 14. Experiment 4: Main Effect of Group (low vs. high checkers) for confidence. Vertical bars denote standard errors.

3.2.3. Discussion of Experiment 4

Experiment 4 revealed that high checkers had poorer accuracy and less confidence overall. In support of our *weaker* hypothesis (see Section 1.4.2.) high checkers' memory was generally impaired by the presence of distracting spatial information in form of an intermediate probe whether it was resolvable or not. Checkers were also significantly poorer at suppressing the cumulative effect of misleading spatial information (misleading block) which interfered with baseline performance (no-probe-1 trials). Reaction time data supported this, as a misleading block context was sufficient to increase decision making time also in baseline trials. A misleading block was, therefore, generally distracting but especially so for high checkers.

3.3. General Discussion

Overall the results support and extend the previous findings that checking goes hand in hand with a lack of confidence and may lead to attenuated WM performance under certain circumstances. This seems to be the case when distracting and/or misleading information is presented, which, instead of being ignored, seems to induce repeated checking of the encoded memory traces. Paradoxically, while high-scorers check to improve their memories it actually undermines performance by reducing the accuracy of the WM representation. In that sense checking critically differs from mere rehearsal. Both processes imply reactivation of memory representations, yet, while rehearsal reactivates "without questioning", checking seems to imply a lack of confidence in the veridicality of the reactivated information that is detrimental without the original sensory information to check against - especially if competing new sensory information is present.

In Experiment 3 checkers' memory was poorest in a combination of misleading and correct probe-2 trials. Overall performance for both groups was poorer for correct-probe-2 trials, suggesting we may have induced a certain degree of checking in all participants by increasing WM task difficulty. This is in agreement with MacDonald et al. (1997) who in a very difficult task (recall 50 words that were presented for 1sec after 7mins of distractor tasks) reported no difference in recall proportion between checkers (0.179), non-checkers (0.142) and controls (0.188). As indicated by the low memory scores, in such an experiment (and perhaps ours) extant OCD/checkers' executive-memory impairments would need to be extremely acute to impact memory performance and significantly differentiate them from controls. However, for checkers, misleading trials were especially disrupting conform to our hypotheses. High checkers also had less confidence in their responses, indicating a metacognitive deficit that seems to affect WM performance which corroborates the findings in Harkin and Kessler (2009). In line with previous findings (Ciesielski et al., 2007; Henseler et al., 2008; Harkin & Kessler, 2009) there were no group differences on the resolvable and the no-probe-1 trials, suggesting that even in a difficult WM task the observed differences between high and low checkers on misleading trials were not an issue of WM capacity *per se*. However, the group differences on misleading trials were not dramatically enhanced compared to the previously reported effects in an easy WM task (Harkin & Kessler, 2009). This indicates that high checkers did not suffer disproportionally from the enhanced task difficulty, which further underpins our claim that checkers are not impaired at the level of WM capacity *per se*.

In Experiment 4 we challenged participants in a more refined way. In this case, an intermediate probe questioned participants about a specific location which was either resolvable (letter at this location) or misleading (no letter at this location). We argue that this challenges the integration of letter-location representations through their 'weakest link', i.e., spatial location. Considering that memory spans are better for word stimuli compared to nonsense word stimuli, with the only difference being the availability of words in LTM (Hulme, Maughan, & Brown, 1991). We propose that while letters have an existent representation in LTM, contributing to retention in WM, spatial locations do not, which should make the latter more sensitive to interference affecting this dimension across memory, i.e., bindings of locations to letters. Support for this can be drawn from the research which has shown impacted verbal-spatial (Elsley & Parmentier, 2009) *versus* intact object-feature binding (Allen, Baddeley, & Hitch, 2006) with concurrent mental load. From this we can infer that attention is mobilized to a greater extent when binding occurs across the boundaries of the slave systems of WM, i.e., the visuospatial sketchpad (location) and phonological loop (letters) (Elsley & Parmentier, 2009). The stimuli we present are multimodal which refers to fact that different components of our stimuli are processed in different cortical streams, specifically: the ventral ('What') and dorsal ('Where') streams for object and location representations, respectively (Goodale & Milner, 1992). Therefore, if accurate task performance is dependent upon accurate object ('What') and location ('Where') information then this will rely upon the maintenance of accurate object-location conjunctions in what Baddeley had termed the "EB" (see Keizer, Colzato, & Hommel, 2008; Olson et al., 2006). Within the WM model of Baddeley (2000) this suggests that information which requires binding across the 'What' and 'Where' streams is likely to be more sensitive to interference compared to that which is processed primarily within one stream. Thus, due to the lack of location representations in LTM sustained attention is required for their accurate maintenance in WM, which in turn makes letter-location bindings particularly sensitive to interference when challenged at the level of location.

This may explain why Experiment 4 was the first to reveal poorer accuracy overall for high compared to low checkers in an easy WM task, suggesting in line with our *weaker* hypothesis (see Section 1.4.2.) that an intermediate spatial probe was in fact strongly distracting for checkers (fig. 13, left). The observed trend towards poorer performance for high compared to low checkers in the baseline condition (no-probe-1 trials) is noteworthy (fig. 13, left). We attribute this to the cognitive style that checkers adopt in a misleading context which they 'carry-over' to the processing of baseline trials (see fig. 13, right). This also fits the established profile of clinical checking/OCD typified by the inflexibility to shift cognitive processing style in the face of changing demands and despite its detriment to performance (e.g., Fenger et al., 2005; Omori et al., 2007; Veale et al., 1996). In conclusion, the very clear group differences we obtained in Experiment 4 indicates that checkers WM performance are susceptible to challenges by distracting or even misleading information, especially if this challenge is directed towards weakly encoded information like the episodic spatio-temporal context of events that is not supported by LTM concepts.

Together Experiments 3 and 4 provide evidence that the episodic spatio-temporal context is indeed the weaker link in the EB representations. In Experiment 3 high-checkers performance is attenuated in the more difficult correct probe-2 condition which seems to require exact letter-location information relative to incorrect trials that can be accurately performed using partial information only. However, the overall greater difficulty of Experiment 3 reduced the between-group effects (c.f., MacDonald et al., 1997) whereas accessing representations via spatial locations in Experiment 4 enhanced group effects. High-checkers' questioning of the veridicality of letter-location bindings accessed via the weaker location feature resulted in deteriorated overall performance (86.6%) compared to the performance observed by Harkin and Kessler (2009) with an identical encoding and retrieval task, yet, with an identity-cue as probe-1 task (93%). Low-checkers, on the other hand, revealed more comparable overall performance here (91.8%) and in the original Harkin and Kessler (2009) paper (92.8%). An alternative explanation could be that the high checker group in the present Experiment 4 was special. However, this is not the case: in Harkin and Kessler (2009) Experiment 1 revealed a mean score of 9.5, Experiment 2 a score of 8.7 and in the extreme group comparison the high checkers reached a score of 15.8. The present Experiment 3 revealed a mean score of 12.6, so the score of 10.5 in the present Experiment 4 falls well within this range. Together these points support our argument that location – extending to the spatio-temporal context in general - is the weaker link in EB bindings compared to letter identity, and that this weakness becomes most apparent when individual checking disposition is high.

3.3.1. Limitations and future research

Two main limitations of the current experiments should be mentioned. Firstly, we did not manipulate checking *per se* but assume checking is responsible for poorer WM performance as opposed to general distraction caused by an intermediate probe. With respect to Experiment 3, however, if distraction was causal then impairment would be expected for resolvable trials, whereas checkers performance is only attenuated on misleading trials. This allows us to argue that misleading trials are special for checkers whereby they check and compare it to each letter of the encoding set. General distraction is likely to underlie checkers poorer performance in resolvable and misleading trials in Experiment 4. However, for checkers misleading trials were particularly salient and difficult to shift attention from, suggesting that general distraction is not the whole story. Future research should directly manipulate checking within a WM paradigm, which is not easy to implement without confounding impact on the complexity of primary WM task. Secondly, as previously suggested (Experiment 1 and 2) letters and locations have limited ecological validity to the specific symptoms of checkers, a criticism we deal with in subsequent Experiments 5, 6, 7, and 8.

4. Do high checkers' actually check: An eye movement study.

The previous experiments have helped shed some light on the cognitive processes which differentiate the WM performance of high from low checkers (Harkin & Kessler, 2009, 2011a; Harkin, Rutherford, & Kessler, 2011). In our original WM task (Harkin & Kessler, 2009) we employed a simple delayed-match-to-sample paradigm, where participants had to encode 4 letters and their locations and then after a delay recall if one of the letters was correctly or incorrectly located. Our novel manipulation was to present an intermediate probe – between encoding and the memory task – which asked participants to indicate the location of a letter that was either part (resolvable) or not part (misleading) of the encoding set. We found that only high checkers' WM performance (correct/incorrect letter location task) was impaired when preceded by a misleading but not a resolvable trial. Considering that an intermediate probe is irrelevant to the performance of the memory test, we conclude that checkers are more distracted by a misleading probe as it is not part of the encoded set. Checkers either cannot suppress the distractor itself, or cannot suppress the urge to check triggered by the misleading distractor (cf. Harkin & Kessler, 2009). This is a process which we suggest is perhaps driven by impairment in inhibitory functioning specific to the checking but not the washing subtype (Omori et al., 2007). We propose that as misleading trials are special to high checkers they check the contents of WM to verify if a misleading letter was present or not. This is consistent with Lind and Boschen (2009) who reported that intolerance of uncertainty (i.e., raised by a misleading probe) mediated the propensity to check . However, as observed in the research discussed above (Radomsky & Alcolado, 2010), checking only serves to impair the veridicality of the contents of WM which occurs, we suggest, at the level of letter-location bindings maintained in the EB of WM (Baddeley, 2000).

While we provided a more precise characterisation of the relationship between inhibitory dysfunction and episodic short-term memory in checkers, we were aware that our conclusions were somewhat limited. Specifically, we could not determine with certainty whether the presence of a misleading probe indeed differentiated the manner in which high and low checkers scanned the contents of their WM. Therefore, the present study addressed this question by comparing eye movements of high and low checkers specifically during the presentation of the misleading distractors. .While previously we had placed a time-constraint of 4000ms on the responses to the misleading distracters (henceforth called 'Probe-1') (Harkin & Kessler, 2009) we now provided participants with unlimited time to make their Probe-1 response (see fig. 15). We hypothesised that high checker's responses would be slow, which would also allow us to investigate in detail their eye movement patterns in contrast to low checking controls. Rotge et al. (2008) indeed reported that OCD checkers took longer than OCD non-checkers for verifying WM probes. They concluded that increased response time for 'choice making' represented the degree of uncertainty and doubt that checkers had at the moment of choice. Unsurprisingly, in trials where checkers had longer response times this lead to more overt repetitive checking behaviors, i.e., uncertainty motivated checking (Lind & Boschen, 2009; Tolin et al., 2003). Accordingly we expected to find eye movement patterns in our study that would reflect the internal (i.e. mental) checking behaviours of high checkers. This would confirm our conclusion based on our previous research that misleading distractors triggered repeated mental checking of WM contents in high checkers only.

Thus, measuring eye movements in our WM task (fig. 15) allowed us to answer our own outstanding research question and to add substantially to the existing OCD eye movement research which has revealed mixed results at best (for reviews see Gooding & Basso, 2008; Jaafari et al., 2011; Sweeney, Levy, & Harris, 2002). For example, in a recent review of thirty-three eye movement studies Jaafari et al. (2011) reported that OCD patients were characterised only by rather unspecific deficits in form of smooth pursuit impairments and longer response latencies in anti-saccade tasks. The majority of these studies concentrated purely on the functionality of the oculomotor system bearing little information on the cognitive and emotional deficits in compulsive checking. No emphasis has been put so far on eye movements during more complex cognitive or memory tasks, thus, the present study was likely to make a substantial contribution in this respect.

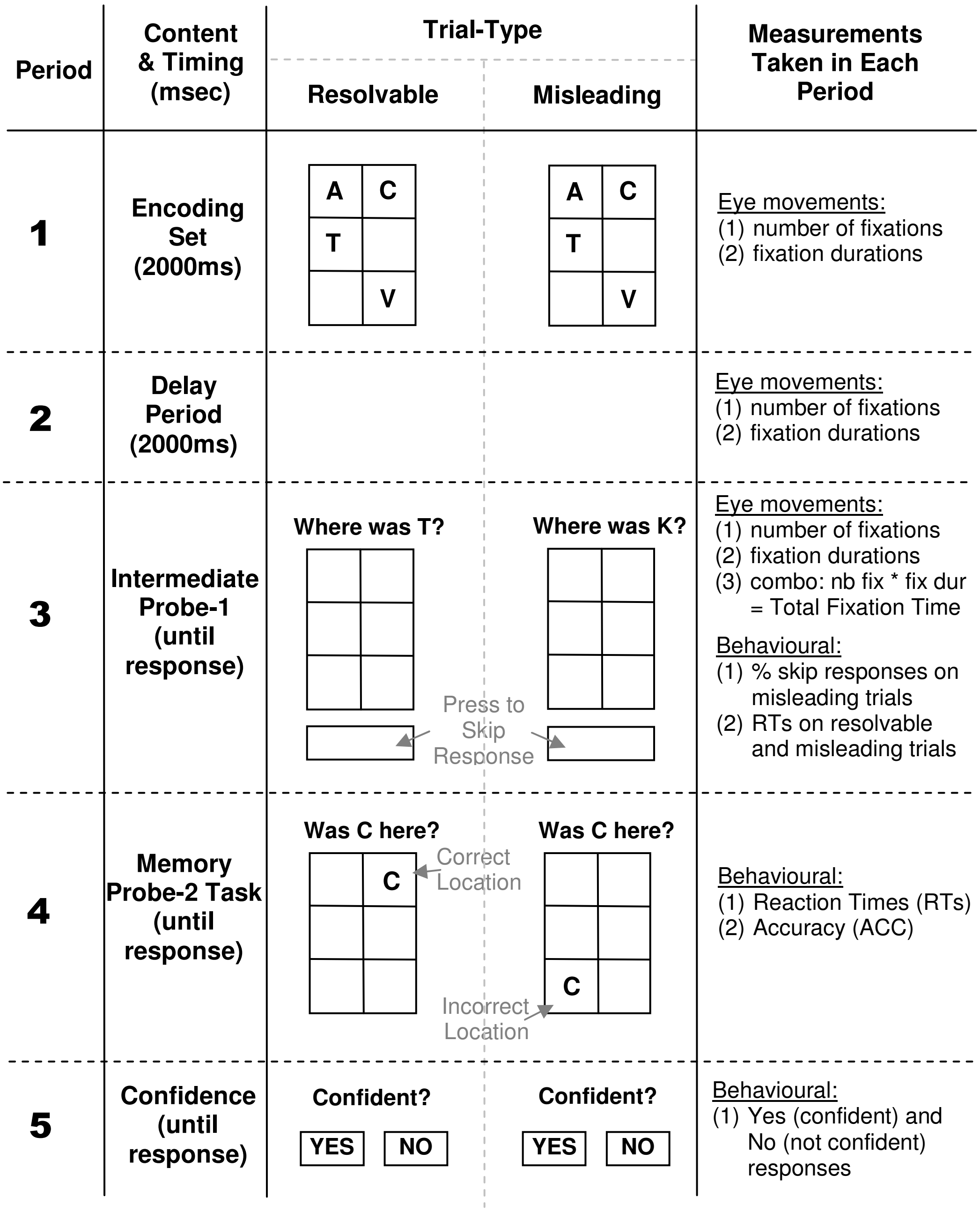

Figure 15. Schematic representation of Periods 1-5 for resolvable and misleading trials: (1) *Period 1*: encoding set of 4 letters presented randomly in 6 possible locations (2000ms), (2) *Period 2*: delay period (2000ms), (3) *Period 3*: probe letter (Probe-1) which was either part (resolvable = T) or not part (misleading = K) of the encoded set, (4) *Period 4*: probe letter (Probe-2) which was wither correctly or incorrectly located with respect to the encoded set, which was actually the memory task, and (5) *Period 5*: confidence in the memory task was then indicated using a confidence and not confident response. The eye and/or behavioural measurements recorded and analysed in each period are also provided. Further explanations in the text.

Specifically, it has been argued that eye movements reflect both attention and rehearsal within WM making it a valid measure of differences in executive function between high and low checkers in the present study (for review see Theeuwes, Belopolsky, & Olivers, 2009). For example, it has been repeatedly observed that participants tend to fixate on the previous location of an encoded item during delay, indicating that the contents of short-term memory guide attention which in turn guides eye movements (Altmann, 2004; Deubel & Schneider, 1996; Olivers, Meijer, & Theeuwes, 2006). An assertion corroborated by Theeuwes, Belopolsky and Olivers (2009) who suggested that attention always precedes an eye movement, and that attention may serve as the vehicle by which information is stored in WM (Dehaene et al., 2006; Dehaene, Sergent, & Changeux, 2003; B. K. Schmidt et al., 2002).

In the present study we divided our WM task (see fig. 15) into three 'periods of interest' during which we recorded eye movements. We concentrated our analysis on number and duration of fixations, which were most likely to reflect internal checking behaviours, i.e., more and longer fixations reflecting internal checking. *Period 1* was the 2000ms encoding period, where 4 letters were presented in 6 possible locations. *Period 2* was the 2000ms delay period after encoding and before the presentation of the intermediate (resolvable or misleading) Probe-1. Accordingly, *Period 3* refers to the presentation of a resolvable or misleading intermediate Probe-1 trial. As shown in Figure 15, the employed WM task included two further Periods, referring to Probe 2 presentation and indication of confidence, respectively. However, eye movements were not recorded during these periods, hence, only behavioural data will be reported for each period (response times, accuracy and response confidence, respectively).

It was an open question whether we would observe group differences in eye movements during *Periods 1* or *2*. Either checking as a cognitive style could already take place during encoding and during the undisturbed delay period, or checkers might not differ from non-checkers unless their executive attention deficit was explicitly triggered by a misleading probe. Conform to previously reported findings, the latter was likely under conditions of low memory load as employed here (Boldrini et al., 2005; Ciesielski et al., 2007; Harkin & Kessler, 2009, , 2011a; Henseler et al., 2008; Morein-Zamir et al., 2010; Moritz et al., 2003; Purcell et al., 1998a, , 1998b; van der Wee et al., 2003; Zielinski, Taylor, & Juzwin, 1991; Zitterl et al., 2001). However, in *Period 3* we expected high checkers to make more and longer fixations in misleading compared to resolvable trials, as misleading trials specifically tap into the inhibitory impairments of high- but not of low checkers (see Harkin & Kessler, 2009) fuelling their urge to overcome uncertainty by means of excessive checking (Veale et al., 1996). We therefore also expected high checkers to have slower response times on Probe-1s in misleading trials compared to low checkers. In contrast, for resolvable trials we did not expect to observe group differences for eye movements or response times if our hypothesis was correct that the executive impairments of high checkers had to be specifically triggered by a misleading Probe-1 (Omori et al., 2007; Harkin & Kessler, 2009, 2011a, 2011b; Harkin, Rutherford, Kessler, 2011).

Taking these arguments to a finer level of analysis we expected to observe that on misleading trials high checkers will spend longer examining the six locations of the encoding set matrix and specifically empty encoded set locations, in comparison to low checkers and resolvable trials. If supported, this will provide an exact indication that checkers' executive impairments result in them accessing the encoded set matrix as a whole and that specifically they spend longer perseverating on empty locations where no letter had been presented at all.

Confirmation of these hypotheses will provide evidence that checkers' inhibitory impairments do in fact lead them to check the contents of WM in a manner which is unnecessary (specifically in misleading trials) and uninformative (empty locations). Checking uninformative locations would require additional time and resources, possibly affecting -or at least delaying behavioural performance.

4.1. Experiment 5

4.1.1. Method

Participants

35 student participants (mean 20.8 years: 18 males, 17 females) from the University of Glasgow gave written informed consents. British Psychological Society ethical requirements were met, including that of participant debriefing. The Vancouver Obsessional Compulsive Inventory (VOCI; Thordarson et al., 2004) was employed to evaluate all participants regarding their checking tendencies. The VOCI is a 55 item, self-report questionnaire for assessing the severity of OCD symptoms. Conform to our previous research (Harkin & Kessler, 2009, 2011a; Harkin, Rutherford & Kessler, 2011), the checking subscale was used in the present study to created obtain two groups: 17 low (mean: 0.71, SD: 0.92) and 18 high (mean: 12.67, SD: 5.78) ‘‘checkers’’.

Eye Tracking

Eye movements were recorded at a sampling rate of 1000 Hz with the SR Research Desktop-Mount EyeLink 2K eyetracker (with a chin/forehead-rest), which has an average gaze position error of about 0.25°, a spatial resolution of 0.01° and a linear output over the range of the monitor used. Only the dominant eye of each participant was tracked although viewing was binocular. The experiment was implemented with E-prime®. Calibrations of eye fixations were conducted at the beginning of the experiment using a nine-point fixation procedure as implemented in the EyeLink API (c.f. EyeLink II User Manual: SR.Research.Ltd, 2002) and using E-prime® software. Calibration was validated with the EyeLink software and repeated when necessary until the optimal calibration criterion was reached. At the beginning of each trial, participants were instructed to fixate a dot at the centre of the screen to perform a drift correction. If the drift correction was more than 1°, a new calibration was launched to insure optimal recording quality.

Procedure

Participants sat 60cm from a 19’’ computer screen ran at 800x600 resolution with their head on a chin rest. Stimuli were capital letters in font Arial, size 18 and were presented against a grey background within a 2 (columns) by 3 (rows) matrix covering an area of 300x420 pixels, subtending 11.5 degrees horizontally of visual angle and 13.3 of visual angle vertically. After 1000 ms fixation, 4 letters were presented randomly in 4 of the 6 possible locations and participants had 2000 ms to encode the identity and the location of each letter (fig. 15). After 2000 ms, the probe-1 question requested the location of a specific letter which had been either part (hence, resolvable) or <u>not</u> (hence, misleading) of the encoded set. Participants indicated the location through a 2x3 spatially mapped keypad and responded in their own time. Participants could ‘skip’ the intermediate probe by pressing the ‘0’ button on the number pad with their most dominant thumb at any time. This provided reaction times and ‘skip’ percentages specific to the termination of resolvable and misleading Probe-1 trials which we could then analyse statistically (see fig. 15, Period 3). This differed from the original Harkin and Kessler (2009) procedure which limited the probe-1 response period to 4000 ms. In a baseline condition probe-1 was omitted to measure WM performance on the primary task under ideal conditions. A 1000 ms interstimulus interval (ISI) separated probe-1 and probe-2. Since baseline trials did not include the intermediate probe-1 a grey screen was shown for 5000 ms between encoding and probe-2. Probe-2 was the actual memory test for each trial and required participants to indicate if a letter was correctly located with respect to the originally encoded set. In all trials the probe-2 letter had been part of the encoded set in terms of identity while the probe location was correct only on 50% of the trials. Finally, through a binary response option participants were then prompted to indicate their degree of confidence in their probe-2 response (1 = confident vs. 2 = not confident). There were 190 trials in total, 10 of which (at the beginning) we practice trials including resolvable and no-probe-1 trials only. The main experiment was then done in two blocks (with 5min rest period between), each comprising of 60 misleading, 20 resolvable, and 10 no-probe-1 trials presented in random order. This asymmetric trial type distribution was adopted from Kessler and Harkin (2009).

4.1.2. Results

Breakdown of individual Periods (1-5)

We present our data analyses (eye movement and/or behavioural responses) in the same sequence in which the participant viewed and/or responded to each aspect of the experiment: Period 1 (encoding), Period 2 (2000ms delay), Period 3 (Probe-1), Period 4 (Probe-2) and Period 5 (confidence). We focused our eye movement recordings on Periods 1, 2 and 3 as these were the intervals of interest specifically related to our group hypotheses.

Period 1: 2000ms encoding set presentation

Independent-samples t-tests revealed that low and high checkers did not statistically differ in terms of fixation durations (t=1.32, df=33, p=0.19) or number of fixations (t=0.87, df=33, p=0.39) they made during the 2000ms presentation period of the encoding set (Period 1). Conform to our expectations high and low checkers do not differ in their allocation of attention during early encoding.

Period 2: 2000ms delay period

Independent-samples t-tests revealed that low and high checkers did not statistically differ in terms of fixations durations (t=1.76, df=33, p=0.088) or number of fixations (t=1.71, df=33, p=0.09) they made during the 2000ms delay (Period 2) between the encoding set and intermediate Probe-1.

Period 2b: 5000ms extended delay in no-probe-1 trials

We conducted separate independent sample t-tests for no-probe-1 trials, due to them having a longer 5000ms delay period. In terms of fixation duration there was no statistical difference between low and high checkers (t=1.46, df=33, p=0.16). However, we did find that high checkers (9.08) made significantly less fixations than low checkers (10.97) (t=2.12, df=33, p=0.04). While this finding is surprising it actually serves to highlight the abnormality of high checkers' making more fixations during misleading trials in our subsequent Period 3 analysis.

Period 3: misleading and resolvable intermediate Probe-1

Response Times (RT)

A two (*Group*: low checkers vs. high checkers) by two (*trial-type*: resolvable vs. misleading) mixed design was used with *group* as the between- and *trial-type* as the within-subjects factors. There was a main effect for Trial-Type ($F(1,33)=51.123$, $p<0.000$), with slower RTs for resolvable (2240.9ms) compared to misleading (1807.7ms) trials. Critically, there was a Group x Trial-Type interaction ($F(1,33)=6.065$, $p<0.02$). Analysis of the simple comparisons revealed that there was no significant group difference in RTs for resolvable trials (LC = 2196.4ms vs. HC = 2285.5ms: $F(1,33)=0.308$, $p=0.58$), compared to a significant group difference for misleading trials (LC = 1613.9ms vs. HC = 2001.4ms: $F(1,33)=4.871$, $p<0.04$) (see fig. 16). This suggests that both low and high checkers match a resolvable probe to its location within the encoded set. In contrast, on misleading trials, only high checkers appear to 'check' if a misleading probe "really" was there, whereas low checkers quickly dismiss it and quickly terminate the presentation of misleading probes. Critically, there was no difference between low and high checkers in their percentage of 'Skip' responses (LC: 97.9% vs. HC: 96.9%; $p=0.28$) on misleading trials. This indicates that despite high checkers taking longer to confirm that a misleading probe is absent they do so at the same ceiling level as low checkers.

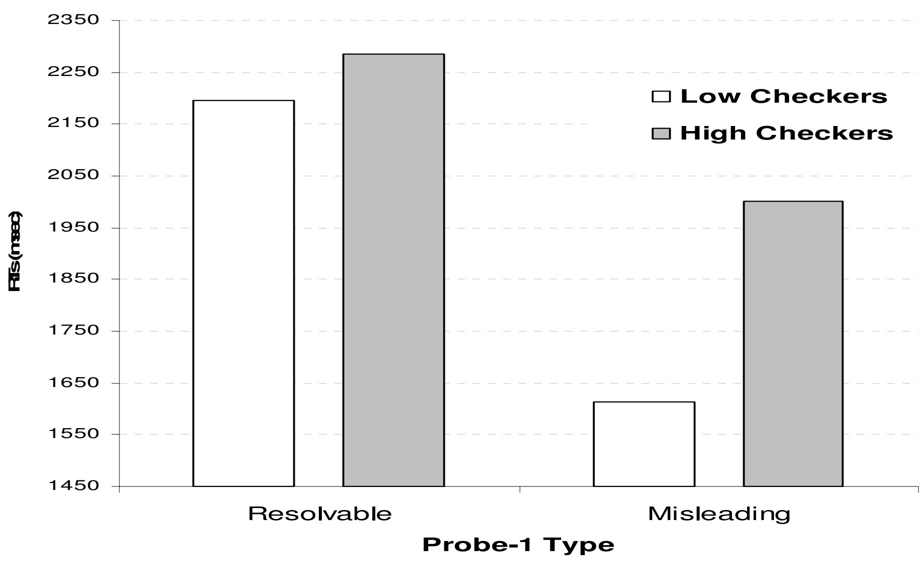

Figure 16. Probe-1 RTs for Group (Low checkers vs. High checkers) for Trial-Type (resolvable and misleading).

Eye Measurements

Period 3 is the most critical of our three analyses, specifically, as high checkers had slower Probe-1 RTs for misleading trials (compared to low checkers; see Fig. 15). We expected that in Period 3 high checkers would also engage in more and longer fixations in misleading trials relative to low checkers. We employed a two (*Group*: low checkers vs. high checkers) by two (*trial-type*: resolvable vs. misleading) mixed design was used with *group* as the between- and *trial-type* as the within-subjects factors. Thus, we conducted a 2 x 2 ANOVA design for fixation duration and number of fixations separately. For fixation duration a main effect of Trial-Type ($F(1,33)=71.98$, $p<0.000$) was observed, reflecting shorter fixation durations on average in misleading (226.5ms) compared to resolvable trials (250.5ms). No effects involving group reached significance (all $p<0.17$).

For the number of fixations a main effect of Trial-Type ($F(1,33)=10.19$, $p<0.004$) was again observed, reflecting fewer fixations in misleading (6) compared to resolvable trials (6.6). However, a significant Group x Trial-Type interaction ($F(1,33)=5.69$, $p<0.023$) was also observed. Most importantly, this was the result of high checkers executing significantly more fixations (6.6) than low checkers (5.4) in misleading trials ($F(1,33)=4.795$, $p<0.04$), a pattern that was not present on resolvable trials (HC: 6.7 vs. LC: 6.5: $F(1,33)=0.305$, $p=0.59$) (see fig. 17). Thus, low checkers mirrored the previous interaction for Trial-Type (less fixations for misleading compared to resolvable trials), whereas high checkers did not. Furthermore, considering that misleading trials are the most common trial-type presented (66%) this did not result in high checkers having carry-over effects (i.e., based on expectations) which inflated eye movements during encoding (Period 1), maintenance (Period 2) or for resolvable Probe-1s (Period 3). This highlights the methodological relevance of measuring eye movements during Periods 1 and 2 and allows us to argue that high checkers do not seem to develop trial expectations (i.e., based upon the majority of trials being misleading) which influence how they either encode (Period 1) or maintain (Period 2) letters and their locations.

Fixations in encoding locations in Period 3

Consistent with our first hypothesis, we had observed that during Period 3 high checkers made more fixations during misleading trials compared to low checkers (see fig. 17). However, as these fixations were calculated from all possible screen locations of a misleading (and resolvable) probe, we cannot determine with certainty that high checkers actually access the encoded set or if they perhaps made more fixations to the Probe-1 prompt (misleading trials: “Where was K?”; fig. 15) relative to low checkers.

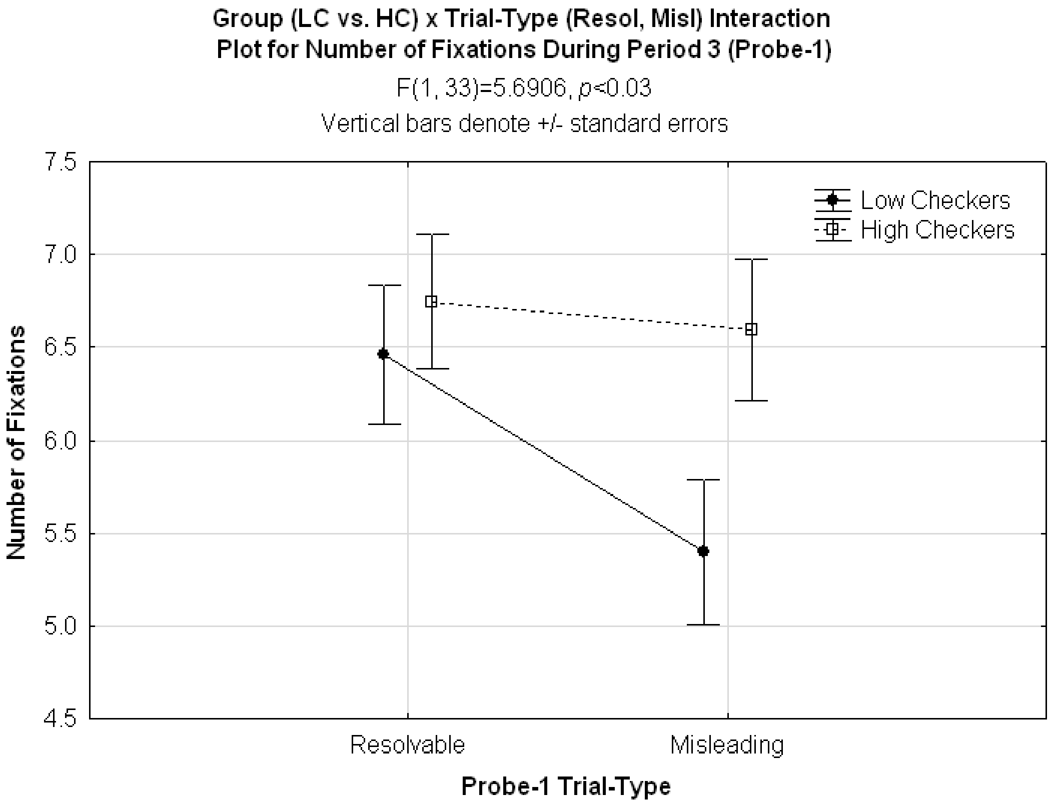

Figure 17. Fixation number for Group x Trial-Type interaction plot.

Based on our finer-grained hypotheses we expected that when presented with a misleading probe high checkers examined the matrix of six locations presented empty during Period 3 (see fig. 15). We further expected that they particularly perseverated on empty locations compared to low checkers and that these checking-related patterns would be observed in misleading but not in resolvable trials. This would provide evidence that, when confronted with a misleading letter probe, checkers experience a particularly high degree of uncertainty regarding the presence or absence of the probe, which they attempt to negate by checking all locations even those where no letter had been presented. To this end, we re-coded the matrix of six locations - presented empty during Period 3 (see fig. 15) - according to their contents during encoding (Period 1). Specifically, we determined whether a particular location had contained 1) the target (resolvable trials only), 2) any letter (resolvable and misleading trials) or 3) whether a location had been empty (see fig. 18). With this information we could then determine where participants specifically looked during Period 3, in terms of the 'correct' contents of WM, despite the 2 x 3 matrix being empty. In concordance with our re-coded locations we multiplied number of fixations by fixation duration to provide a "total fixation time" (TFT) on (1) target locations (resolvable trials only), (2) non-target letter locations, and (3) empty locations.

Results for TFT

For comparing misleading and resolvable trials we focused on total fixation time (TFT) measures for empty and non-target letter locations only (there was no target location in misleading trials). We calculated a 2 (*Group*: high checkers vs. low checkers) x 2 (*Trial-Type*: misleading, resolvable) x 2 (*Encoded Set Content*: empty, letter) ANOVA, with Group as a between- and Trial-Type and Encoded Set Content as the within-subjects factors. The number of fixations and fixation duration values for low (LC) and high checkers (HC) which were combined to create the TFT values are provided in Table 2. It is important to note that these values are smaller than those previously reported in Figure 17 as we now focused our analysis on the six matrix locations as opposed to the whole intermediate probe screen (incl. the probe sentence "Where was K?"; see fig. 15).

A significant group effect ($F(1,33)=5.85$, $p<0.022$) revealed that high checkers (443.8ms) spent longer overall looking at the locations (empty and letter) of the encoded set matrix compared to low checkers (315.2ms). The Group x Trial-Type interaction approached significance ($F(1,33)=3.75$, $p=0.06$). Consistent with our hypothesis, this was driven by high checkers revealing significantly longer TFT measures in misleading trials compared to low checkers (F(1,33)=7.62, p<0.01), whereas no group differences were observed in resolvable trials (F(1,33)=2.29, p=0.14). Critically, this supports our previous Group x Trial-Type interaction presented in Figure 17 and shows that when presented with a misleading probe high checkers access the six encoded set locations to a greater extent (TFT) than low checkers.

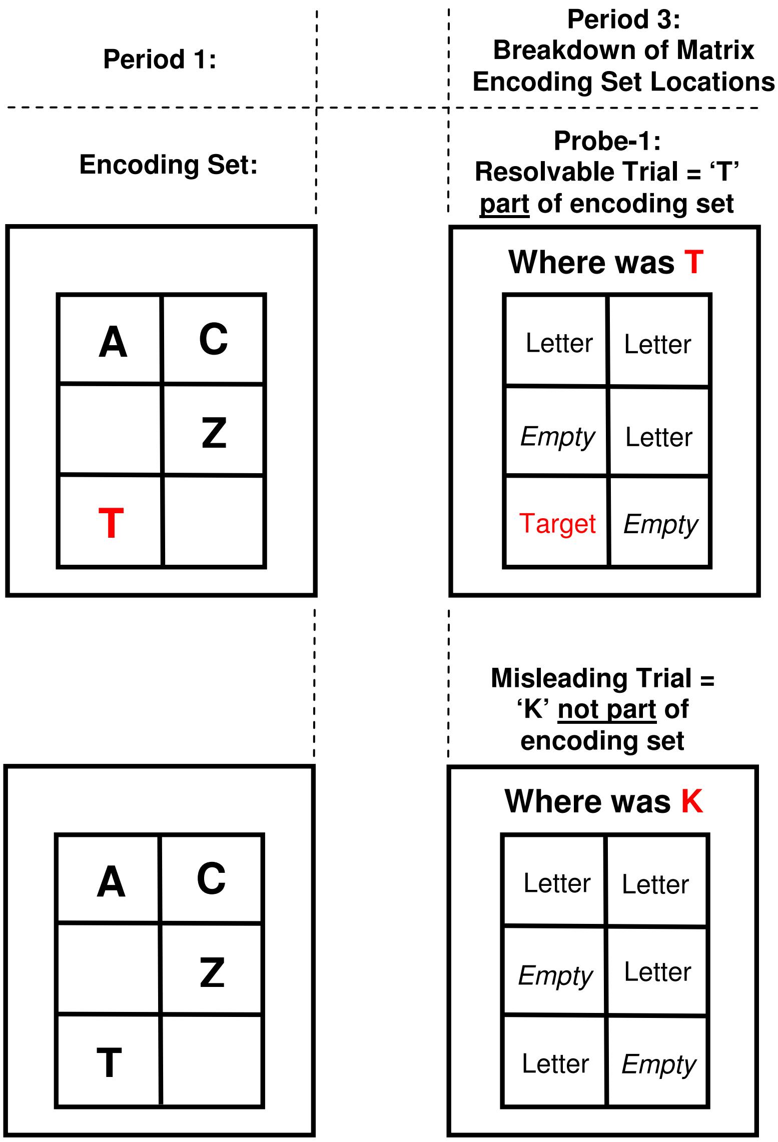

Figure 18. Breakdown of Period 3 analysis for resolvable (top) and misleading (bottom) trials in terms of encoding set contents presented in Period 1.

As we were interested in TFT at empty locations we conducted a 2 (*Group*: high checkers vs. low checkers) x 2 (*Trial-Type*: misleading, resolvable) ANOVA. There was a marginal Group x Trial-Type interaction (F(1,33)=3.75, p=0.063) (see fig. 19; left plot). Analysis of the simple group comparisons revealed that, in comparison to low checkers, high checkers had a significantly longer TFT in misleading (LC: 493.3ms vs. HC: 732.7ms; $F(1,33)=6.09$, $p<0.019$) but not resolvable trials ($F(1,33)=0.77$, $p<0.39$). Thus, high checkers spent 239.4ms longer looking at empty locations relative to low checkers. This suggests that checkers' inhibitory impairments for misleading trials result in them checking locations where no task-relevant information is present and may reflect an attempt to negate uncertainty, i.e., "Was that (misleading) letter there, I will check every possible location to be sure." Also within group effects revealed that high checkers had a significantly larger TFT ($F(1,33)=14.27$, $p<0.0007$) on misleading compared to resolvable trials, a pattern not present on low checkers ($F(1,33)=0.97$, $p<0.34$). Importantly, there were no group effects for letter locations (see fig. 19; right plot) suggesting that the Group x Trial-Type interaction in the 3 way ANOVA was driven by high checkers perseverating on empty locations.

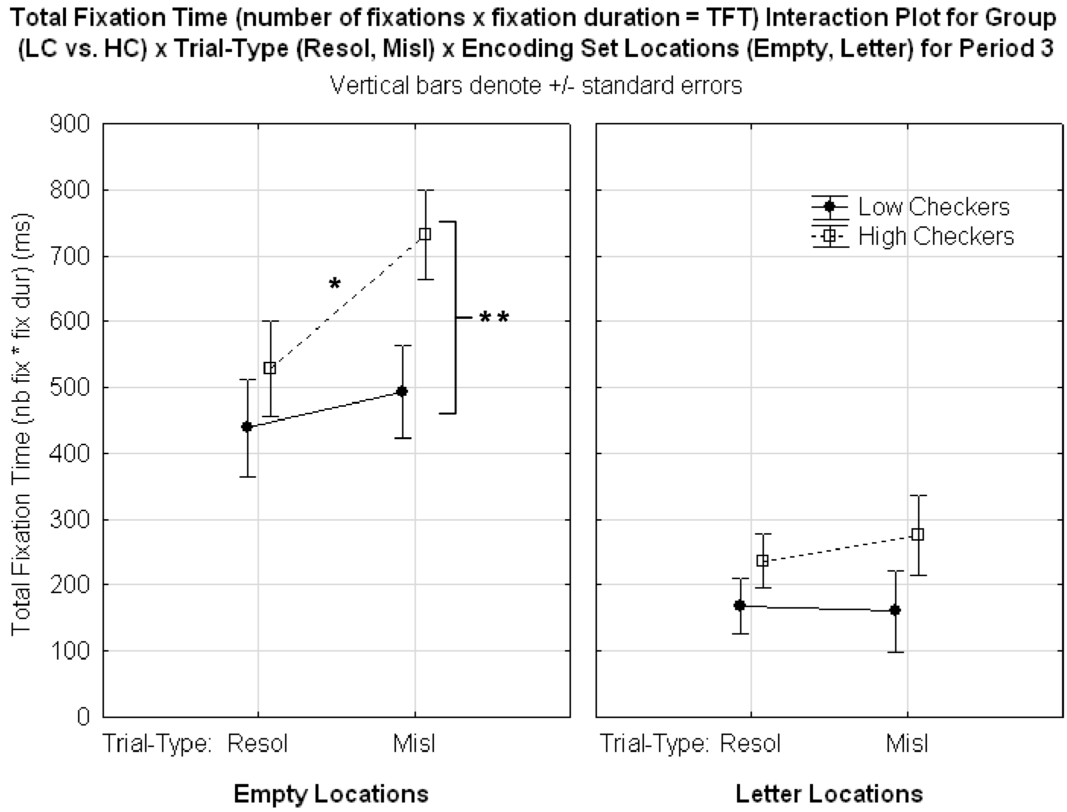

Figure 19. Group (Low Checker vs. High Checker) x Trial-Type (Resolvable, Misleading) x Encoding Set Content (Empty; left plot, Letter; right plot) Total Fixation Time (number of fixations x fixation duration = TFT) interaction plot for Period 3. Please note that * denotes $p<0.0001$ significance level and ** $p<0.019$. There were no other significant effects between or within the high and low checking groups.

Table 2. Number of Fixations and Fixation Durations (mean/stdev) for low (LC) and high checkers (HC) which were combined to create the Total Fixation Time (TFT) values at specific encoding content locations (see interaction plot in fig. 19). For resolvable and misleading trials alike this included empty (**E**) and letter (**L**) locations, and specifically for resolvable trials a Probe-1 target letter location (**T**).

Measure		**Number of Fixations**						**Fixation Durations**				
Trial-Type		**Resolvable**			**Misleading**			**Resolvable**			**Misleading**	
Encoding Content		**E**	**L**	**T**	**E**	**L**		**E**	**L**	**T**	**E**	**L**
LC	Mean	1.36	0.86	2.15	1.49	0.85		292.54	186.32	584.14	313.54	176.96
	Std	0.50	0.20	0.52	0.41	0.20		97.66	56.70	207.68	79.33	66.55
HC	Mean	1.45	0.95	1.98	1.74	1.03		344.90	221.24	656.04	403.34	223.78
	Std	0.41	0.33	0.79	0.43	0.45		91.78	97.45	236.69	94.54	115.40

Finally, high and low checkers did not significantly differ (*p*=0.64) in TFT to correct Probe-1 target-letter locations (resolvable trials only). This highlights that high checkers are not impaired in their ability to accurately locate an actual target letter based on their WM representations. Overall, on misleading trials high checkers focus significantly more on the six encoding set locations as a whole, and specifically longer at empty locations in comparison to low checkers and resolvable trials.

Periods 4: (Probe-2 response times and accuracy

A two (*Group*: low checkers vs. high checkers) by three (*trial-type*: resolvable, misleading, no-probe1) by two (*probe-2 location*: correct, incorrect) mixed design was used with *group* as the between- and *trial-type* and *probe-2 location* as the within-subjects factors. Thus, ANOVAs for a 2 x 3 x 2 design were carried out on Probe-2 reaction times and accuracy.

Response Times (RT)

A main effect of Trial-Type (F(2,66)=11.20, *p*<0.000), reflected faster RTs for misleading (1896.8ms) compared to resolvable (2130.8ms) and no-probe-1 trials (2153.9ms). Critically, the reaction time pattern for misleading and resolvable trials was reversed to that which we observed in our original experiment (Misl: 1896.7ms vs. Resol: 1782ms; Harkin & Kessler, 2009). Therefore, the between-experiment difference exists for resolvable trials. We suggest that the self- *versus* automatic-termination of the preceding intermediate Probe-1 in our present and original experiment (respectively) likely explains this. In the present experiment, participants had to provide the actual location (i.e., corresponding button response within 2x3 matrix) of a resolvable Probe-1 letter. This deliberate response process was observed in significantly slower reaction times for resolvable compared to misleading Probe-1 responses, a pattern which appears to have carried-over into slower Probe-2 reaction times in the resolvable condition. In contrast, in the previous experiment, participants did not have to exogenously or endogenously locate a resolvable Probe-1 letter (i.e., it was up to them but the task did not demand it) which was then reflected in their faster (relative to current experiment) Probe-2 responding in the resolvable condition. A main effect for Probe-2 Location ($F(1,33)=70.39$, $p<0.000$) revealed that RTs were overall faster for a correctly located (1919.5ms) compared to an incorrectly located (2183.5ms) letter. There was a significant Group x Trial-Type x Probe-2 Location interaction, which was driven by different between-group response patterns in the correct and incorrect Probe-2 conditions. Specifically, the only between-group (LC vs. HC) comparison to statistically differ in the correct probe-2 condition was for no-probe-1 trials ($F(1,33)=4.77$, $p<0.004$), whereas in the incorrect probe-2 condition the group difference was only present for misleading trials ($F(1,33)=4.96$, $p<0.03$).

Accuracy (ACC)

No main effects or interactions with or without group reached significance, the absence of group effects support our expectations. We suggest that allowing participants to self-terminate the presentation of the intermediate Probe-1 allowed high checkers to compensate for existing executive impairments. This we argued possibly removed high checkers' WM impairments specific to misleading trials which we previously observed when Probe-1 terminated automatically (see Harkin & Kessler, 2009).

Period 5: Confidence Responses (CR)

Confidence (CR) was calculated as the individual percentage of responses (per trial type) indicating that the participant was not confident that their response with respect to Probe-2 had been correct. CR data were subject to the same design and 2 x 3 x 2 ANOVA that were carried out in Period 5. A main effect for Trial-Type ($F(2,62)=34.6$, $p<0.000$) reflected less confidence overall for misleading trials compared to resolvable ($F(1,31) = 22.77$, $p<0.000$) and no-probe-1 trials ($F(1,31) = 43.2$, $p<0.000$). Further, resolvable trials resulted in less confidence than no-probe-1 trials ($F(1,31) = 38.3$, $p<0.000$). This suggests that a misleading intermediate probe resulted in less confidence for all participants. No effects involving group reached significance. Two participants were removed from the confidence analysis due to an error in the data sampling.

4.2. General Discussion

Conform to our hypotheses checkers' eye movements revealed that they were less able to ignore a misleading probe than non-checkers. Firstly, checkers made more fixations during the presentation of a misleading probe compared to low checkers, a group difference that was not observed for resolvable trials. This group by trial-type interaction was mirrored in response times, where checkers took significantly longer to 'skip' a misleading trial relative to low checkers; again a pattern not present for resolvable trials. Secondly, we used the contents of the encoding set (Period 1) to determine what was driving participants' fixations, i.e. what types of information they preferably checked during the Probe-1 period (Period 3). This revealed that in misleading trials high checkers' Total Fixations Times (TFT) were greater to the six locations of the encoding set matrix and specifically its empty locations, in comparison to low checkers and resolvable trials. No group effects were observed for letter locations suggesting that high checkers greater TFTs to the encoding set matrix as a whole were driven by group differences at empty locations. The specificity of this pattern argues against the idea that checkers simply made more fixations as the result of their longer manual Probe-1 RTs. If this was the case then checkers would not show such a specific preference for empty locations in misleading trials. No similar group differences in eye movements were observed during Period 1 or 2, which indicates that checkers were not affected in their default mechanisms for how they either encode or maintain letters in locations within the EB of WM (Baddeley, 2000). Further, we observed that on the extended 5000ms delay period for no-probe-1 trials high checkers actually made less fixations than low checkers, which serves to highlight the specificity and importance of the relationship between misleading probes and the greater eye movements of high checkers. In addition, the TFT to a resolvable target letter indicated that checkers were not impaired in their ability to correctly locate a simple letter representation within WM. Rather, checkers' inhibitory impairments only impacted on behaviour (eye movements) when they were challenged by a misleading probe. Therefore, conform to our current expectations and previous papers (Harkin & Kessler, 2009, 2011a), misleading trials tap into checkers' established impairments in inhibition (Olley, Malhi, & Sachdev, 2007; Omori et al., 2007) which results in them engaging in excessive checking of their representations in WM, comparing these even against empty, uninformative locations.

The abnormal 'searching' eye movements of high checkers during misleading trials are consistent with OCD patients having impairments in performance monitoring. Performance monitoring in OCD has been examined with event related potentials (ERP), specifically with respect to the so-called 'error related negativity' (ERN; Gehring et al., 1993) produced by the anterior cingulate cortex (ACC). While the literature on the ERN is extensive, it reflects a number of cognitive functions potentially associated with obsessive-compulsive symptoms, such as error checking, detection of conflicting responses/stimuli, monitoring of performance/conflict, "worse than expected outcomes", strategy implementation, and uncertainty (Botvinick et al., 2001; Braver et al., 2001; Gehring et al., 1993; Holroyd & Coles, 2002; Ridderinkhof et al., 2004; van Veen et al., 2001). It is therefore unsurprising that enhanced ERN amplitudes have been observed in OCD and that these correlated with symptom severity (Ciesielski et al., 2011; Gehring, Himle, & Nisenson, 2000; Ursu et al., 2003). Also van der Wee et al. (2003) observed that in an n-back WM task OCD patients had greater ACC activity at all levels of task difficulty relative to controls. This was not interpreted as a deficit in WM capacity but rather as one of abnormal performance monitoring and/or compensatory executive processes. This is highly consistent with our current findings, where WM performance (Probe-2) was not affected in checkers, but where we observed atypical eye movement patterns during misleading distractions, reflecting inhibitory deficits and compensatory mechanisms for coping with enhanced uncertainty as a result (e.g. checking empty locations just “to make sure”).

Enhanced ERNs have also been observed in subclinical high scoring obsessive-compulsive participants (Hajcak & Simons, 2002), which highlights the possible quantitative nature of inhibitory/performance monitoring impairments across subclinical and clinical participants. This is consistent with the perspective that a subclinical analogue is a valid means of understanding a variety of features relevant to clinical OCD, especially as they are free from confounds such as medication, clinical state, or co-morbidity (Mataix-Cols et al., 1997; 1999a). Subclinical checkers may therefore provide a ‘purer’ indication of inhibitory impairments in our WM task. Specifically, checkers’ inhibitory impairments reduced their ability to inhibit a misleading probe, which likely induced uncertainty and resulted in them checking the contents of WM at empty, uninformative locations.

In a manner similar to Ciesielski et al. (2007) and Henseler et al. (2008) our findings reveal latent inhibitory impairments despite WM performance being intact. It is therefore important for us to explain why checkers did not show the same WM impairment (Period 4) when preceded by a misleading intermediate probe (Period 3) as we had previously reported (see Harkin & Kessler, 2009). In our previous experiments, the intermediate probe was terminated automatically after 4000ms. It can therefore be assumed that, in misleading trials, high checkers were unnecessarily searching the contents of WM when this process was terminated ‘mid-flow’. This, in turn, may have interfered with attention to bindings maintained in the EB, thus impairing memory. By contrast, in our present experiment, participants could terminate an intermediate probe in their own time; this provided high checkers with sufficient time to achieve their elevated threshold of satisfaction (i.e., overcome uncertainty) before terminating a misleading trial. This is consistent with the observation that checkers take longer before making a choice in a situation of uncertainty (see Rotge et al., 2008), and that uncertainty *per se* motivates checking (Lind & Boschen, 2009; Rotge et al., 2008; Tolin et al., 2003). In the current case self-pacing most likely allowed checkers to engage and optimise their compensatory mechanism and search the contents of WM in a manner which did not interfere with episodic bindings, preserving their memory accuracy in this low load task. Indeed, the fact that on misleading trials there were no significant group differences on ‘Skip’ responses – and that both groups performed at an optimal level (both >96.9%) – is evidence that high checkers used the extra time to attain certainty (i.e., correctly skip misleading P1 in their own time) and preserve WM performance.

4.3. Conclusion

Using eye movement measures we show for the first time that high checkers’ inhibitory impairments for misleading information results in them unnecessarily searching the contents of WM (four letters and two empty locations). Behaviourally, this was expressed with checkers taking significantly longer to terminate a misleading intermediate probe in comparison to non-checkers who quickly dismissed it as misleading and irresolvable. Furthermore, the fact that both groups were similarly excellent at correctly skipping a misleading probe suggests that while high checkers took longer to achieve certainty (i.e. that it was not there) self-termination allowed them to preserve the integrity of the bindings maintained in the EB. We concentrated our eye movement measures on number and duration of fixations which were the best candidates for reflecting internal checking behaviours. Specifically, during the presentation of misleading probes, not only did checkers execute more fixations, but they fixated longer on the six encoding set locations and specifically at locations that had been empty during encoding. Thus, not only do misleading trials trigger internal checking behaviours in checkers, but for these trials they are also more likely to search locations where no actual task relevant information had been presented. It would appear that misleading trials specifically tap into the inhibitory impairments of checkers, inducing uncertainty which they try to overcome by means of excessive checking, searching even uninformative, empty locations.

5. Using ecologically valid stimuli to address previous experimental concerns

Tasks (6 and 7) and experiments (8 and 9) address two central points with respect to our previous experiments (1-5). First, while we previously concluded that checkers suffer from impairments in inhibitory functioning, we do not provide explicit evidence of this impairment. Second, that while we reported robust and replicable effects using letters in locations (Experiments 1 to 4; Harkin & Kessler, 2009, 2011a), it is apparent that such stimuli do not directly relate to checking compulsions in clinical obsessive-compulsive disorder (OCD). We therefore provide a necessary methodological step forward by employing electrical kitchen appliances that are more concordant to checkers primary concerns (Rachman, 2002; Thordarson et al., 2004). As such we address our methodological concerns in two different experimental paradigms using the same electrical kitchen appliance stimuli. Task 6 and 7 make a direct attempt to determine if checkers *do in fact* have executive impairments regarding Inhibition of Return (Posner & Cohen, 1984) effects. Then, Experiments 8 and 9 use our classic WM task with an intermediate spatial probe (i.e., Exp. 4; Harkin & Kessler, 2011a). As we use the same stimuli in both types of experiments, inhibitory (attenuated IOR) and memory impairment for the same stimulus features will provide a strong indication that executive dysfunction leads to memory impairment (Greisberg & McKay, 2003).

5.1. Deficient Inhibition-of-Return in checkers only when attention is directed to the threatening aspects of a stimulus

In our original experiments (Exp. 1 and 2; Harkin & Kessler, 2009) considering that an intermediate probe is irrelevant to the performance of the memory test, we conclude that checkers are more distracted by a misleading probe as it is not part of the encoded set. Checkers either cannot suppress the distractor itself, and/or cannot suppress the urge to check triggered by the misleading distractor (strong hypothesis). A process which we suggested is driven by impairment in inhibitory functioning specific to the checking but not the washing subtype (Omori et al., 2007). However, in alignment with our weak hypothesis our second series of experiments revealed that checkers suffered similar memory impairments for resolvable and misleading spatial probes (see Exp. 4; Harkin & Kessler, 2011a). Thus, while there is a delicate balance between resolvability (strong hypothesis) and general distraction (weak hypothesis), in either case it appears that checkers' poorer memory is due to an executive deficit of inhibitory functioning which impairs attention-dependent bindings within the EB. However, while we concluded that checkers suffer from impairments in inhibitory functioning, we did not provide explicit evidence of this impairment. Therefore, Tasks 5 and 6 are a direct attempt to determine if checkers *do in fact* have executive impairments regarding Inhibition of Return (IOR; Posner & Cohen, 1984) effects. Thus, if these experiments show that checkers do in fact suffer inhibitory impairments for checking specific stimuli then this will inform the inconsistent OCD literature on the need for symptom and stimulus specificity.

5.2. Inhibition impairments in OCD highlight a need for experimental-symptom specificity

Generally, it is argued in the literature that OCD has a common underlying trait: a reduced ability to selectively inhibit irrelevant external stimuli or internal thoughts (e.g., "Did I leave the iron ON?"), which, in turn may trigger subsequent neutralizing compulsions (e.g., repeatedly checking that a switch is turned OFF) and memory deficits (Bannon, Gonsalvez, & Croft, 2008; Harkin & Kessler, 2009, 2011a, 2011b). The centrality of impaired inhibitory control is further underlined by it being proposed as a possible candidate endophenotypic marker of OCD (Chamberlain et al., 2005, 2007; Chamberlain & Menzies, 2009; Penades et al., 2007).

However, not all paradigms of selective attention involving stimulus inhibition have consistently revealed deficits in OCD (for review see Muller & Roberts, 2005). For example, word Stroop tasks have revealed both interference and non-interference (Kampman et al., 2002; Kyrios & Iob, 1998; McNally et al., 1994; Moritz et al., 2008) effects for emotional words in OCD. These inconsistencies may in part be due the fact that words are not particularly relevant to the symptoms of those with OCD and as such fail to adequately and consistently interfere with attention (Moritz et al., 2008). Another measure of inhibitory functioning that has revealed inconsistent findings in OCD is the Inhibition of Return task. This paradigm presents an irrelevant cue to the left or right of fixation before a subsequent target appears in either the cued (valid) or in the uncued (invalid) location. It was found that response latencies to targets were longer to a previously attended location (valid), than an unattended location (invalid). Thus, attending to a target in a previously cued, yet irrelevant location was slower as inhibition impeded attention from returning to that location, i.e., IOR. Furthermore, it is now known that attention and inhibition are not purely space-based (Behrmann, Zemel, & Mozer, 1998; Grison et al., 2005; Jordan & Tipper, 1998; Kessler & Tipper, 2004; Tipper, Grison, & Kessler, 2003; Tipper, Jordan, & Weaver, 1999), but also occur in relation to objects as research revealed greater IOR after the cuing of an object compared to that of a 'pure' location (Jordan & Tipper, 1998; Tipper, Jordan, & Weaver, 1999). IOR is thought to be of adaptive value by biasing attention away from previously attended locations and objects to those that are novel and unsearched (Klein & MacInnes, 1999). Impaired IOR could therefore result in perseverations on previously searched locations or objects, and by failing to attend to new stimuli the individual is more likely to repeatedly revisit the same items/locations again and again (Tipper, Grison, & Kessler, 2003). This bears a striking similarity to the core symptoms of perseveration we observe during compulsive checking in OCD.

Despite this theoretical overlap, however, IOR effects in OCD have revealed a mixed pattern of results. In one instance, OCD patients were generally slower for targets following cue images (for valid and invalid) that were relevant to OCD obsessions, whereas in other studies no group differences in inhibitory functioning were reported at all. Thus, in these studies it appears that while OCD patients were distracted by OCD relevant images IOR remained intact. Furthermore, in OCD patients inconsistent findings have been reported regarding visual fields, i.e., reduced IOR in the left visual field (LVF; Rankins et al., 2004) or in the right visual field (RVF: E. Nelson, Early, & Haller, 1993). Specifically, in the latter case IOR was even reversed into positive priming

(PP) in the RVF, while IOR was preserved (but decreased) in the LVF. These discrepancies may be in part due to differences in IOR methodology, with some of the studies using the classic abstract IOR paradigm (Moritz & von Muhlenen, 2005; E. Nelson, Early, & Haller, 1993; Rankins et al., 2004) while others employed word- (Moritz & von Muehlenen, 2008) or image cues (Moritz et al., 2009) that are relevant to OCD symptomatology.

In order to resolve these discrepancies the present studies extend the previous work on emotional IOR tasks (Moritz & von Muehlenen, 2008; Moritz et al., 2009). We believe that a combination of stimulus and symptom specificity within the domain of checking may be required to reveal robust impairments of executive control and inhibitory functioning (Enright, Beech, & Claridge, 1995; Harkin & Kessler, 2011b; Omori et al., 2007). An assertion we justify with the following observations. First, in an extensive review of memory and attention in OCD, Muller and Roberts (2005) highlighted that OCD is a heterogeneous disorder comprised of multiple subtypes each with their own unique psychological markers (i.e., checkers vs. washers; see Omori et al., 2007). Thus, Muller and Roberts recommended that attention and memory tasks may benefit from using stimuli that are specific to an individuals primary OCD concerns. For example, Amir, Najmi and Morrison (2009) highlighted that high scoring OCD participants had an attentional bias to ideographically selected – therein threatening – word stimuli (*versus* neutral words) and that this bias correlated with symptom severity. This highlights that the tighter the symptom-stimuli concordance then this increases the likelihood of observing an attentional bias specific to the symptomatic but not the asymptomatic group. Second, in our previous work on WM deficits in high checkers we revealed that fragile multimodal integration of stimulus-identity and – location was most susceptible to distraction (Harkin & Kessler, 2009, , 2011a). Suggesting that in the correct experimental circumstances high checkers' attention can be distracted from the primary memory task.

Specifically, in two IOR tasks we employed electrical kitchen appliances as stimuli that could be switched 'ON' or 'OFF'. The two tasks were administered to the same participants within the same session, so in fact were two experimental blocks, which we balanced in sequence. The two tasks differed in the way the unpredictive cues were administered. In Task 6 the cues were yellow coloured outlines around one of the two objects that were left and right of fixation (see fig. 20, left). Always, one appliance was 'ON' and the other one 'OFF'. In Task 7 (see fig. 20, right) the irrelevant cue was administered by switching one of the two appliances 'ON' and then 'OFF' again. The two tasks/blocks were counterbalanced across two groups of participants in order to control for sequence effects. Thus, our tasks differ from previous OCD IOR studies in two critical ways. First, in both tasks ecologically valid stimuli are presented throughout the task as opposed to the brief presentation of unpredictive abstract cues (Moritz & von Muhlenen, 2005; E. Nelson, Early, & Haller, 1993; Rankins et al., 2004), OCD relevant words (Moritz & von Muehlenen, 2008) or ecologically valid images (Moritz et al., 2009) before the target. As a result, we suggest that our tasks bear greater similarity to the prolonged nature of checking, where they repeatedly check the content (i.e., ON/OFF switches) of ecologically valid stimuli (i.e., iron, kettle, stove) for the presence and/or absence of threat. Second, Task 7 provides a novel addition to the literature by explicitly manipulating the content of ecologically valid stimuli (OFF to ON) to act as the unpredictive cue.

Therefore, due to the differences between the tasks we arrived at two probable hypotheses. First, a general hypothesis applicable to high checkers performance in both tasks, where we predicted that 'ON' appliances in Task 6 and 'ON' cues in Task 7 would grab the attention of high checkers and attenuate their IOR effect in both tasks. Second, a task-specific hypothesis where we predicted that focusing high checkers' attention directly onto the electrical state of the appliances (OFF to ON) in Task 7 would attenuate IOR, while with an abstract cue (yellow outline in Task 6) the state of the appliance might go unnoticed, thus not affecting IOR. An outcome conform to this second, task-specific prediction would help explain the rather fragile and inconsistent IOR findings in the literature and highlight the need for ecologically valid stimuli in this research.

5.3. Tasks 6 and 7

5.3.1. Methods general to Tasks 6 and 7

Participants

102 participants (mean age: 23.44 years; 32 males, 70 females) from the University of Glasgow gave written informed consents. British Psychological Society ethical requirements were met, including that of participant debriefing.

We counterbalanced the two task-related blocks across two groups of participants and used their checking scores on the VOCI to create a low and high checking group within each sequence. As we wanted to measure inhibitory functioning in relation to checking related stimuli we used a stringent cut-off criterion using the checking subscale of the VOCI to create two distinct groups: (1) <=1 for low checkers and (2) >=7 for high checkers. For sequence 1 this resulted in mean checking scores for low (n = 24) and high checkers (n = 25) of 0.33 (SD: 0.48) and 14.6 (SD: 6.23), respectively. For sequence 2 this resulted in mean checking scores for low (n = 26) and high checkers (n = 27) of 0.29 (SD: 0.47) and 12.96 (SD: 4.86), respectively. Thus, both low checking groups (Sequence 1 and 2) scored within the range of healthy community adults and so likely had little or no issues with checking. In comparison, both high checking groups (Sequence 1 and 2) scored in the clinical checking range for OCD patients (see Thordarson et al., 2004). This underlines that they do in fact have a problem with repeated checking and that this checking is for appliances, ON/OFF switches, etc (cf. VOCI-items on the checking subscale), justifying the use of such stimuli/manipulations in our tasks. It is important to note that we observed no statistical differences in the age (p=0.81) or gender distribution (p=0.2) between low and high checkers.

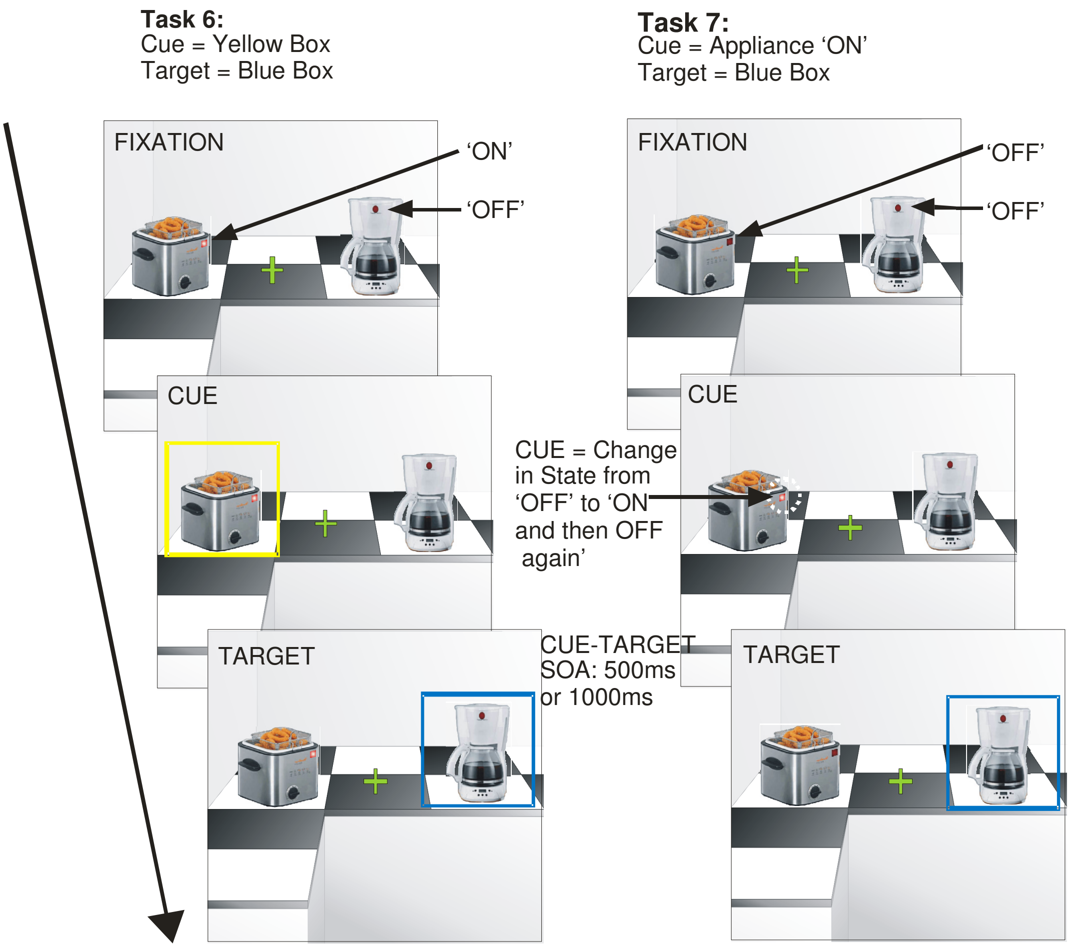

Figure 20. Schematic representation of the procedures used in Task 6 (left) and Task 7 (right). Further explanations in the text.

Stimuli

We employed ecologically valid stimuli that were concordant with checking/OCD symptomatology. For example, the Vancouver Obsessional-Compulsive Inventory (VOCI; Thordarson et al., 2004), and the checking subscale specifically, ask respondents to indicate if they repeatedly check and recheck things like “switches, faucets, appliances, and doors” and “that the stove is turned off” (Thordarson et al., 2004). Additionally, Rachman (2002) highlighted the specific nature of perseverations: “Yes, I remember that I did check the stove but I cannot remember if I checked it satisfactorily. Was the switch fully turned off? I cannot remember if it is safe” (p. 631). In concordance with this symptomatology, we used images of electric kitchen appliances (fryer, iron, kettle, toaster, coffee machine, hob, microwave, sandwich maker) as stimuli and manipulated their “ON” and “OFF” states in two object-based variations of the IOR task in Tasks 6 and 7, respectively. Due to the different cueing procedures in the two tasks the designs were not directly comparable; hence, we conducted separate ANOVA analyses. We will therefore report the two tasks separately.

5.4. Task 6

5.4.1. Method

Procedure

Participants sat in front of a computer screen set to 1680x1050 resolution with their head on a chin rest at 60 cm viewing distance. An experimental trial was initiated by the participant pressing the space bar; this revealed a kitchen countertop with two kitchen appliances for 2000ms. One of these appliances was always ‘ON’ and the other ‘OFF’ (see fig. 20, left schematic). A fixation cross was then presented between the two appliances and the presentation time of this was varied (600ms, 800ms, 1000ms) to prevent the build up of temporal cue expectancies, which are known to influence the orientation of attention (Posner & Snyder, 1975). A yellow cue square was then flashed for 100ms around one of the appliances; participants were instructed not to respond to this. Then, after a delay (SOA) of either 500ms or 1000ms, a blue target square was flashed for 100ms around one of the appliances as the target. Participants indicated if it had been presented around the left (left index finger, ‘X’ key) or right appliance (right index finger, ‘M’ key). In addition to these experimental cue-target trials we also added target-only filler trials, where the blue target was presented right away during the time interval where usually the yellow cue would appear. This manipulation was intended to maintain participants’ attention during the cueing interval of the trial.

Appliance state and visual field (‘ON’ left or ‘ON’ right), side of cue (left or right), target validity (valid/cued or invalid/uncued) and SOA (500, 1000ms) were all counterbalanced. There were 166 trials in total, including 6 practice, 80 valid and, 80 invalid cue trials. RTs were the main dependent variable. RTs were discarded when less than 150ms (anticipations) and greater than 1500ms (misses).

Design

RT data were submitted to a 6-way mixed ANOVA with *Group* (low vs. high) and *Sequence* (Task 5 first vs. Task 5 second) as between-subjects factors and *SOA* (500ms vs. 1000ms), *Target State* (ON vs. OFF), *Validity* (valid/cued vs. invalid/uncued) and *Visual Field* (left vs. right) as within-subjects factors. We employed median RTs as individual statistics to reduce the influence of variance inherent in using sub-clinical sample.

5.4.2. Results

Importantly Validity reached significance ($F(1, 98)$ 89.94, $p< 0.001$): valid (347.1ms) were slower than invalid trials (312.9ms), indicating a typical IOR effect. Sequence revealed a main effect ($F(1, 98)$ 6.09, $p< 0.02$): Task 6 first (349.7ms) was slower than Task 5 second (310.4ms), indicating a possible practice effect for Task 5 when it was performed second in sequence. There was a main effect for Stimulus Onset Asynchrony (SOA) ($F(1, 98)$ 32.6, $p< 0.000$) which reflected faster responding for 1000 SOA (324.8ms) compared to 500 SOA (335.3ms). The SOA x Sequence interaction was significant ($F(1, 98)$ 5.06, $p< 0.03$). This appeared to reflect a greater difference between an SOA of 500 and 1000 when Task 6 was performed second (317.7ms – 303.1ms = 14.6ms) compared to first (352.8ms – 346.5ms = 6.3ms).

Two higher order interactions involving Group reached significance. Group x Validity x Target x Visual Field reached significance ($F(1, 98)$ 5.33, $p< 0.03$). This appeared to reflect two main data patterns: (1) greater IOR for high checkers in the left visual field for ON compared to OFF targets and (2) greater IOR for high checkers in the left compared to the right VF. Group x Validity x SOA x VF x Sequence reached significance ($F(1, 98)$ 4.46, $p< 0.04$). This appeared to reflect two main data patterns: (1) greater IOR for high checkers in the left visual field at 500 SOA in Sequence 2 (Task 1 performed second) compared to Sequence 1 (Task 6 performed first) and (2) greater IOR for checkers in the right visual field at 1000 SOA in Sequence 2 compared to Sequence 1. While these two higher order interactions are complex and difficult to interpret, we suggest that the primary finding is that IOR functioning is intact (if slightly enhanced in certain conditions) for high checkers in Task 6, possibly reflecting the complex IOR pattern in relation to visual fields observed in previous studies. This is important when considered alongside the abnormal IOR response pattern of this group in Task 7.

Table 3. Task 6: Mean and StDev RTs for Group (low LC vs. high HC checkers) x SOA x Target State x Validity.

	SOA	**500**	**500**	**500**	**500**	**1000**	**1000**	**1000**	**1000**
	Target State	OFF	OFF	ON	ON	OFF	OFF	ON	ON
	Validity	Invalid	Valid	Invalid	Valid	Invalid	Valid	Invalid	Valid
LC	*Mean*	312.54	347.83	311.34	347.88	304.09	339.95	301.57	338.88
	Stdev	90.11	92.90	91.68	91.08	92.43	98.72	87.79	100.70
HC	*Mean*	326.28	350.78	321.88	358.00	311.82	346.67	307.23	341.11
	Stdev	84.61	85.97	85.85	87.74	80.71	92.79	86.99	92.66

5.5. Task 7

5.5.1. Method

As before, pressing the spacebar revealed a kitchen countertop with two kitchen appliances, however in this task both appliances were 'OFF' (see fig. 20, right schematic). After a variable delay (1600ms, 1800ms, 2000ms) one of the appliances flashed 'ON' (for 300ms) then 'OFF' again. Then, after a delay (SOA) of 500ms or 1000ms, a blue target square was flashed for 100ms around one of the appliances.

The side of cue ('ON' left vs. 'ON' right), target validity (valid/cued vs. invalid/uncued) and SOA (500 vs. 1000ms) were all counterbalanced. There were 160 trials in total, including 80 valid and 80 invalid cue trials. RTs were the main dependent variable. RTs were subject to outlier rejection based upon being greater than 150ms (anticipations) and less than 1500ms (misses).

Design

RT data were submitted to a 5-way mixed MANOVA with *Group* (low vs. high) and *Sequence* (Task 7 first vs. Task 7 second) as between-subjects factors and *SOA* (500ms vs. 1000ms), *Validity* (valid/cued vs. invalid/uncued) and *Visual Field* (left vs. right) as within-subjects factors.

5.5.2. Results

SOA reached significance (F(1, 98) 94.2, $p< 0.000$): RTs were slower at 500 ms SOA (327.7ms) compared to 1000 ms SOA (309.9ms). Validity reached significance (F(1, 98) 19.81, $p< 0.001$): valid (322.9ms) were slower than invalid trials (314.7ms), indicating a typical IOR effect. The Group x SOA interaction was significant (F(1, 98) 10.4, $p< 0.002$). This appeared to reflect marginally significantly different RTs for high (323.7ms) compared to low checkers (323.7ms – 296.2ms = 27.5ms) at 1000 SOA (p=0.07) compared to more similar RTs (335.5ms – 319.9ms = 15.6ms) at 500 SOA (p=0.3).

Importantly the Group x Validity interaction was significant (F(1, 98) 10.09, $p<$ 0.002): Low checkers had faster RTs for invalid (301.1 ms) compared to valid trials (314.9 ms) an effect which was small but highly consistent reaching significance (F(1, 98) 38.53, $p< 0.000$). In comparison, there was little numerical difference for high checkers' RTs between invalid (328.4ms) and valid trials (330.8ms) which was reflected in it failing to reach significance (F(1, 98) 0.83, $p= 0.37$). Thus, the low scoring group showed a typical IOR pattern (invalid – valid = -13.8 ms), whereas the high scoring group showed an abnormally attenuated IOR pattern -2.4 ms) (see fig. 21). The robustness of this Group x Validity interaction was further reflected in its significance when Task 6 was performed by different groups of low and high checkers either first (F(1, 51) 4.63, $p< 0.037$: LC: -14.8 vs. HC: -4.6) or second (F(1, 51) 5.9, $p< 0.02$: LC: -13.2 vs. HC: -1.9) in sequence. Analysis of the validity effects for each sequence separately revealed that in either sequence low checkers had typical IOR effects (both $p<0.000$), while IOR effects were attenuated for high checkers in either sequence (both $p>0.4$). The fact that different groups of low and high checkers performed Task 7 in Sequence 1 and 2 shows that this effect is specific to high checkers and to the experimental cue manipulation (i.e., switch ON and OFF) as opposed to an effect related to a specific task sequence and/or sub-group of individuals.

No other higher order interactions with or without group reached significance in Task 7. This is important as it may suggest that the complex interactions involving group observed in Task 6 may have been due to the more complex design (i.e., cued appliance ON/OFF as well as target appliance ON/OFF) and the resulting smaller trial numbers per cell.

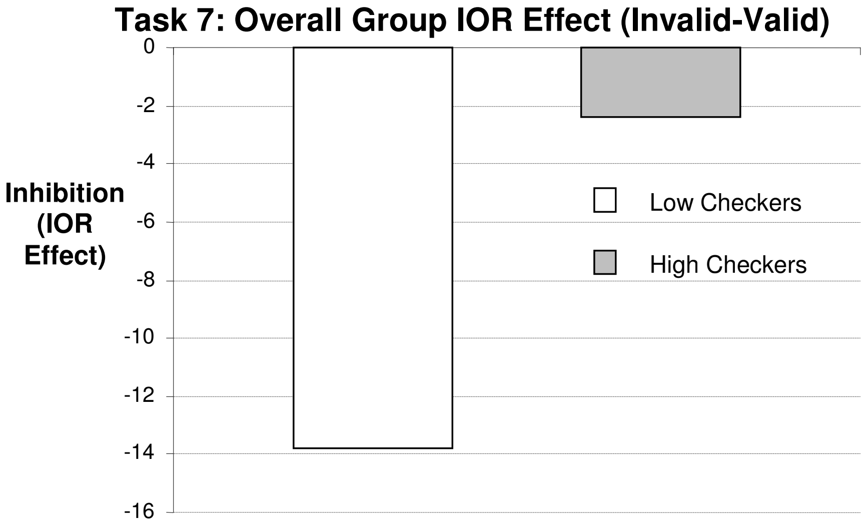

Figure 21. Group IOR Effects in Task 6. The high checking group showed attenuated and almost absent IOR compared to the typical IOR effect of the low checking group.

Table 4. Task 7: Mean and StDev RTs for Group (low checkers: LC vs. high checerks: HC) x SOA x Validity

	SOA	**500**	**500**	**1000**	**1000**
	Validity	Invalid	Valid	Invalid	Valid
LC	*Mean*	310.57	328.91	291.34	300.85
	Stdev	71.03	74.93	71.67	74.23
HC	*Mean*	335.49	336.58	322.18	325.76
	Stdev	88.76	84.30	87.63	90.01

5.6. General Discussion

Conform to our second, task-specific hypothesis, we attenuated IOR for high checkers when the cue involved an explicit manipulation of the state of a kitchen appliance, i.e., ‘off’-‘ON’-‘off’ (Task 7). In comparison, typical IOR was observed for high checkers in the more classic IOR task, where the cue was an irrelevant yellow box flashing around an appliance (Task 6). This suggests that high checkers did not have a global impairment of attentional disengagement and subsequent inhibition (i.e., IOR intact in Task 6) but rather that their attentional functioning was impaired when the task directly engaged existing attentional biases for threatening stimuli (Task 7). This reveals the rather fragile nature of atypical IOR patterns in high scoring checkers, hence, shedding some light on the variability of findings reported in the literature so far.

For example, Moritz et al. (2009) found that OCD patients were generally slower in responding to a target (dot) when the cue was an image relevant to OCD symptomatology (e.g., for checking: fire; for washing: dirty toilet). As this effect failed to interact with cue validity or SOA they concluded that OCD patients were generally more distracted by OCD-related visual images. In line with their previous research (Moritz & von Muehlenen, 2008; Moritz & von Muhlenen, 2005) they proposed that OCD patients did not exhibit generally impaired inhibitory functioning. Tapping into such impairments may require stimuli that elicit negative and more acute responses in those with OCD (Harkin & Kessler, 2009; Moritz et al., 2009), which may explain the divergent results for our high checkers across the two tasks (IOR in Task 6, attenuated IOR in Task 7) and generally within the research so far. Specifically, in Task 1, the mere presence of an 'ON' appliance failed to interfere with the disengagement of attention after an irrelevant yellow box cue. As a result, we found a typical IOR effect in both groups. On the other hand, the explicit use of an appliance's state as a cue (Task 7: switching between 'OFF' - 'ON' - 'OFF') was sufficient to interfere with the normal functioning of attention in the high but not the low checking group. In other words, switching an appliance 'ON' as a cue was sufficiently salient to override normal disengagement and re-orienting of attention to the centre followed by inhibition of the just attended irrelevant object/location (Grison et al., 2005; Jordan & Tipper, 1998). In contrast, for the low checking group switching 'ON' an appliance merely acted as an irrelevant cue, again producing typical IOR.

As attentional biases to threat play a central role in the etiology and maintenance of anxiety disorders (Mathews & MacLeod, 2002; Mogg & Bradley, 1998; Williams et al., 1997), our research may be particularly informative to potential interventions which target attentional processes in checking/OCD (Wells, 1990; 2000) conform to interventions proposed for other anxiety disorders. For instance, the so-called attentional modification training (AMT) proposed by MacLeod et al. (2002) attempts to improve attentional efficiency by targeting and directing attention away from threat/anxiety inducing information to less threatening information not associated with anxiety by using a dot-probe discrimination task. MacLeod et al. (2002) simultaneously presented a threatening and a neutral word followed by a visual (dot) probe. The primary task was to indicate as quickly as possible the location of this dot-probe. The key manipulation was to randomly assign participants to one of two conditions where there was either a strong contingency between the location of the probe and a threat-related word or a neutral word. Participants in the attend-threat condition had faster response latencies to threat-words and had higher levels of negative mood when they performed a stressful task compared to participants in the attend-neutral condition. Despite their results being limited to non-clinical mildly anxious students they proposed that AMT may provide an appropriate means of treating clinical anxiety. A suggestion corroborated by Amir et al. (2009) who trained (8 sessions over 4-weeks) individuals with generalized anxiety disorder (GAD) to selectively attend to the location of a non-threat word in one group (AMT) but not in another group (i.e., no contingency between probe location and word-type). GAD participants in the AMT condition reported a decrease in their attentional bias to threat words and a decrease in their anxiety. The authors concluded that attention is central to the etiology and maintenance of GAD symptoms as retraining attention reduced anxiety. The robustness of AMT in the dot-probe is further substantiated by two studies which reported a similar attenuation in symptoms and anxiety levels using face stimuli (i.e., disgust *versus* neutral) for groups with high anxiety (Eldar & Bar-Haim, 2010) and generalized social phobia disorder (Schmidt et al., 2009). Therefore, not only has AMT shown that attentional biases are malleable to intervention but also that systematically directing attention away from threat reduces anxiety, i.e., attention moderates symptoms.

Thus, while little (if any) research has been conducted into AMT and checking/OCD so far, the similarities between the dot-probe and the present IOR task point towards attention training as a possible means for attenuating the attentional bias and symptoms of high checkers. Firstly, could the dot-probe be systematically applied (i.e., similar to Amir et al.) to high checkers' attentional bias to ‘ON’ states which would then restore normal inhibitory functioning for ‘ON’ cues in the IOR task? Secondly, if AMT could reverse high checkers’ attentional bias for ‘ON’ states and generally to threatening stimuli/features, would this then translate to improved memory performance for these stimuli/features?

Critically, we subsequently report (Exp. 8) that high checkers have a robust impairment in their ability to recall if a kitchen appliance was either ‘ON’ or ‘OFF’ (Harkin, Rutherford, & Kessler, 2011). Based on our theory (Harkin & Kessler, 2009, 2011b) we take this as evidence for a strong interaction between executive functioning (selective attention) and WM processes (binding within the ‘EB’, cf. Baddeley, (Baddeley, 2000)). We proposed that attention to the threatening feature of an appliance (i.e., its ON/OFF state) hampered the binding of the state to the actual appliance and its location (see Harkin, Rutherford & Kessler, 2011) by either directly interfering in form of an exogenous distractor and/or by initiating repetitive detrimental checks of WM contents in form of an endogenous distraction. This argument is explored in more detail in Experiment 8. This is important as Salkovskis, Forrester and Richards (1998) pointed out that OCD is characterised by endogenous distractions in form of “intrusive thoughts”. Accordingly, it has been found that high checkers’ memory performance is improved when attentional focus is shifted away from the actual memory task (Radomsky, Ashbaugh, & Gelfand, 2007). This suggests that contrary to the checkers’ intuition, a relaxing, non-checking attentional focus actually improves memory performance particularly when combined with reduced attention to intrusive thoughts. Therefore, training selective attention with respect to exogenous distractors only (e.g. AMT) might not be most effective intervention for OCD in the long run. Wells (Wells, 2000) proposed an attention-based intervention specific to the intrusive thinking of OCD. This attentional training (ATT) method aims to enhance executive control over attention and cognitive processes through selective attention, attention switching and divided attention exercises. ATT treats spontaneously occurring intrusive thoughts as “noise” that does not require attention but suppression. However, in one of the few studies of ATT in relation to OCD, Watson and Purdon (2008) failed to show that it improved symptoms beyond those of thought replacement, distraction or no intervention control conditions. In this case, it may have been that the single ATT session was insufficient to improve symptoms beyond a placebo effect. Alternatively, considering the specificity of our IOR group-effects in Task 7, it is possible that the effectiveness of ATT could have been enhanced by tailoring the attentional aspects of the intervention to the symptoms of specific subgroups (i.e., 'ON' states for checkers) or individual patients (i.e., ideographic selection). Taking all these consideration into account, we propose that training of exogenous as well as endogenous selective attention within the wider context of WM processing, aiming to specifically attenuate repetitive checking of WM contents (Harkin & Kessler, 2009, 2011a, 2011b; Harkin, Rutherford, & Kessler, 2011) may be most effective for improving a wider range of OCD symptoms in the long term.

5.7. Conclusion

In conclusion, we have confirmed our second, task-specific hypothesis regarding IOR being affected in high checkers. For high checkers, IOR mechanisms were basically intact (Task 6) and only affected when attention was drawn to a threatening aspect of ecologically valid stimuli (Task 7: switching ON an electric appliance). This is an essential piece of evidence that potentially explains why IOR effects in OCD are rather fragile and somewhat inconsistent in the literature. Not only does this highlight the necessity of symptom- and stimulus-specificity but it directs future research to measuring IOR effects for other OCD subtypes using stimuli specific to their symptoms. A limitation of this study is that participants were not asked to subjectively appraise the threat of our stimuli and so we cannot determine how threatening our stimuli were for high compared to low checkers. However, as our group effects were specific to Task 7 this indicates that the presence of an unpredictive ON cue was sufficiently threatening to grab the attention of high checkers at the cost of normal IOR functioning. Indeed, these effects occurred by using stimuli that were general (i.e., present on checking subscale of VOCI) to the symptoms of checking despite them not being idiographically selected or appraised (in contrast to Amir et al., 2009).

Suggesting that if we had allowed individual high checkers to select visual stimuli relevant to their unique symptoms we may have observed a greater attenuation of IOR effects than presently observed. Alternatively, we would have expected larger effects if we had used checkers whose concerns were only for electrical appliances.

Regardless, our findings corroborate the notion that distractions that are salient to OCD symptomatology cannot be easily ignored by those with clinical and subclinical expressions of checking/OCD. As we have argued in our recent research on WM impairments in subclinical checkers, deficits in attentional selection and suppression could be the essential factors for episodic memories being affected in the short- and long-term. Episodic representations are inherently multimodal, hence, fragile and susceptible to persistent interference by irrelevant external stimuli (an iron left 'ON') or internal thoughts ('Did I leave the iron ON?') that cannot be efficiently suppressed (Harkin & Kessler, 2009, , 2011a, , 2011b; Harkin, Rutherford, & Kessler, 2011).

Another possible limitation of the present study was that in using a subclinical group this raises the issue of their relevance as an analogue to a clinical group. We agree, however, with Mataix-Cols et al. (1997,1999a) that subclinical OCD groups are a valid means of determining which cognitive factors play a role in clinically defined OCD, particularly considering their reduced medication and potential for co-morbidities. We therefore expect that the pattern observed here with subclinical checkers could be more pronounced using clinical OCD patients, yet, also more variable. We conclude that drawing attention to the threatening aspect of an ecologically valid stimulus is the most promising candidate to reveal deficient disengagement of attention, yielding attenuated IOR.

5.8. Impaired executive functioning in checkers with ecologically valid stimuli reveals novel and classic working memory impairments

Experiments 8 and 9 use the same ecologically valid stimuli that were used in the previous IOR tasks where high checkers showed attenuation in normal inhibitory functioning for electrical appliances when they were cued with an 'ON' state. Therefore, Experiments 8 and 9 in using such stimuli addressed two central issues to our research: (1) Our previous WM experiments (1-4) used letters in locations which have little validity with respect to the primary concerns of high checkers, and (2) if high checkers showed attenuated IOR for 'ON' cues will they show a related memory impairment for the same and/or associated features? Thus, we presented 4 electrical kitchen appliances located in 6 possible locations, of which two were 'ON' (electrical light was bright red) and two were 'OFF' (electrical light was dark red). The primary memory task (probe-2) required the participants to recall if an appliance had been 'ON' or 'OFF' (Exp. 8) or if an appliance was correctly located (Exp. 9) as shown in Figure 22. In both experiments, we used an intermediate spatial-location probe similar to Experiment 4 of Harkin and Kessler (2011a), where it had produced stable group effects (i.e., low standard deviations) and substantial memory impairments in high compared to low checkers. This intermediate probe was presented at a location where an appliance had either been present (resolvable) or at a location that had been completely empty (misleading), participants had to indicate if the appliance at that location had been 'ON' or 'OFF'. An additional yet critical development of our methodology related to trial-type ratio. In our previous experiments we presented two blocks, one with predominantly misleading trials (66%) and a counterbalanced block of resolvable trials as a result we could not exclude the influence that this had upon checkers' WM performance. Therefore, we currently used an equal trial-ratio (33% resolvable, 33% misleading, 33% no-probe-1) which allowed us to develop a clearer understanding of the specific effect(s) of trial-type and/or group on memory performance (probe-2). We predict that using such stimuli and probing the spatial location of threatening aspects of them may potentially enhance executive dysfunction, impair attention-dependent bindings (i.e., Exp. 7: state to appliance or Exp. 8: appliance to location) and perhaps produce novel memory and metacognitive impairments compared to our previous work.

5.9. Experiment 8

5.9.1. Method

Participants

40 Participants (mean 20.8 years: 12 males, 28 females) from the University of Glasgow gave written informed consents. British Psychological Society ethical requirements were met, including that of participant debriefing. We used the checking subscale of the VOCI and used a median split of checking scores to obtain two groups: 20 low (mean: 0.5, SD: 0.61) and high (mean: 13.85, SD: 4.12) ‘‘checkers’’. Further, no statistical differences between the low and high groups were revealed in gender distribution (p=0.72) or age (p=0.27).

Procedure

Participants sat 60cm from a computer screen with their head on a chin rest. At the beginning of each trial a fixation cross was presented for 2000ms. A kitchen countertop was then presented for 6000ms with 4 electrical kitchen appliances presented randomly in 6 possible locations as shown in Figure 22. Two of these appliances were ‘ON’ as indicated by a red light and two were shown to be ‘OFF’ with no accompanying light. After this a mask was presented for 1000ms, this was to reduce the influence that possible image retention may have played in subsequent retrieval (i.e., distinct appliances and/or their ‘ON’ states), thus isolating disturbances in later memory-probe performance to those of WM. After this a probe-1 question asked if a device at a specific location was either ‘ON’ or ‘OFF.’ As in our previous research (Exp. 2; Harkin & Kessler, 2011a) this probe was presented (3000ms) at a location where there <u>had been</u> (resolvable) or <u>had not been</u> (misleading) a device in the original encoding set. Participants were asked to indicate if the device at this location (resolvable or misleading) was either ‘ON’ (left index finger of right hand) of ‘OFF’ (middle index finger of right hand). This probe previously produced stable group effects (i.e., low standard deviations) and substantial memory impairments in high compared to low checkers. Additionally, using such an intermediate probe was motivated by our recent findings that an explicit, yet task irrelevant ‘ON’ cue interfered with normal inhibitory functioning (i.e., Inhibition of Return; Posner & Cohen, 1984) of high but not low checkers (Task 2; Harkin & Kessler, in press). Thus, for high checkers drawing attention to the functional and threatening aspects of electrical appliances and probing empty locations may resonate with the established executive impairments of high checkers in inhibiting irrelevant thoughts and/or stimuli (Olley, Malhi, & Sachdev, 2007; Omori et al., 2007; Savage et al., 2000). Baseline trials were also included; these presented an empty kitchen countertop (i.e., no probe-1) designed to measure WM under ideal conditions. A mask was again presented (1000ms) before the actual memory task. In Experiment 8, probe-2 simply presented a single electrical appliance at the centre of the screen, the participant had to indicate if they recalled it as being ‘ON’ (right index finger) or ‘OFF’ (right middle index finger) with respect to the original encoded set. Finally, participants were asked to indicate their confidence in their probe-2 decision as indicated simply by a ‘Confident’ (right index finger) or ‘Not Confident’ (right middle index finger) response.

There were 156 trials in total, 12 of which (at the beginning) were practice including resolvable and no-probe-1 trials only. The main experiment was then done in two blocks (with 5min rest period between), each comprising 24 resolvable, 24 misleading and 24 no-probe-1 trials presented in random order. Importantly, we employed an equal ratio of trial type in the current experiments: 33% resolvable, 33% misleading, 33% no-probe-1, while in our previous studies we had employed at least one block with 66% misleading trials (- and a counterbalanced block of predominantly resolvable probe-1 trials, cf. Harkin & Kessler, 2009, 2011). We did this to remove the influence of trial-type ratio which had to be counter-balanced across 2 blocks in our previous experimental designs. This allowed us to develop a clearer understanding of the specific effect(s) of trial-type and/or group on memory performance (probe-2). For example, in our original experiment (Harkin & Kessler, 2009) it is possible that high checkers’ poor performance on misleading trials was driven by the novelty/surprise caused by an unfamiliar trial type.

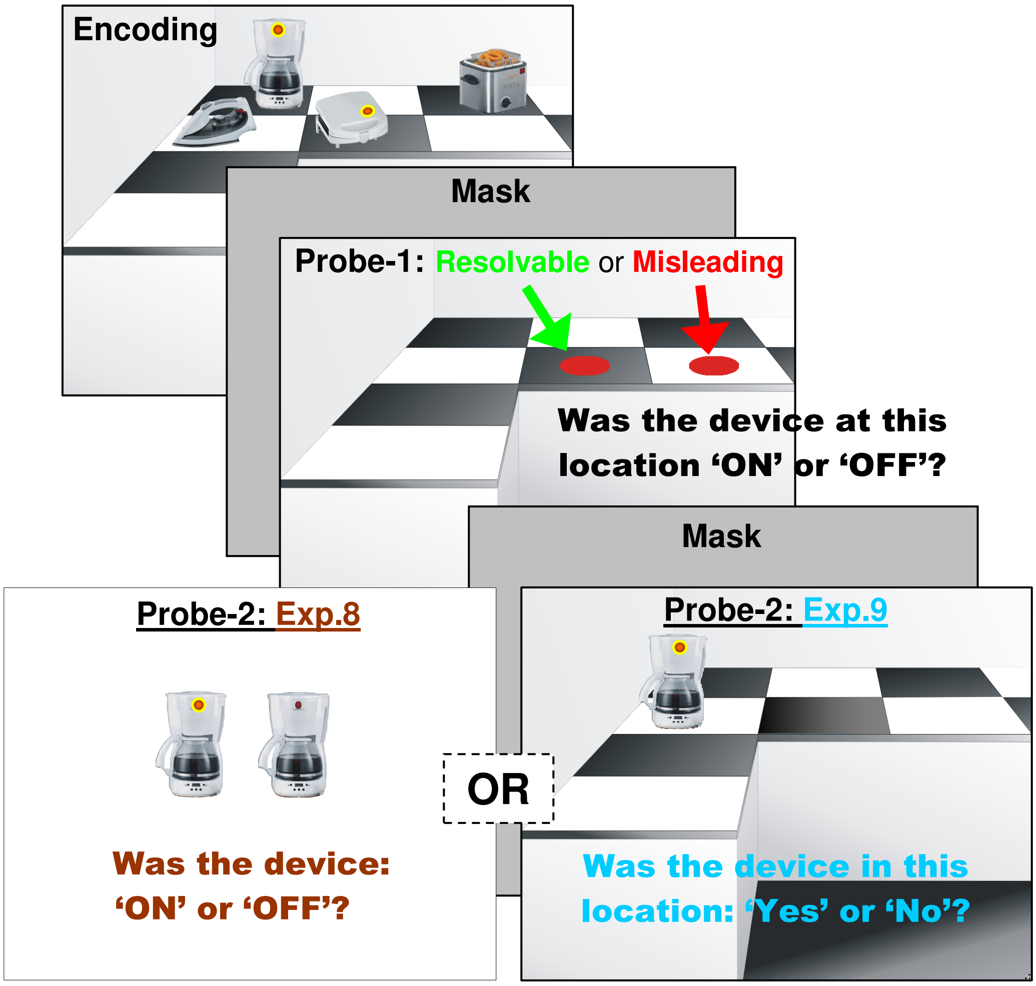

Figure 22: Procedure and stimuli in Experiments 8 and 9. The Experiments differed only on Probe-2. Please note that appliances were only ever presented in the top 2 (vertical) x 3 (horizontal) countertop locations.

Design

A two (*Group*: low vs. high checkers) by three (*Probe-1*: resolvable, misleading, no-probe-1) by two (*Probe-2 State*: ON, OFF) mixed design was employed with *group* as the between- and *probe-1* and *probe-2 state* as the within-subjects factors.

5.9.2. Results and Discussion of Experiment 8

MANOVAs for a 2 x 3 x 2 design were carried out for reaction times, accuracy and confidence on probe-2 responses due to violations of the sphericity assumption (Mauchley's tests).

Probe-2 Response Latencies

The MANOVA (2 x 3 x 2) for probe-2 latencies revealed a main effect of group, high checkers (1898.4ms) were significantly slower in responding than the low group (1573.4ms) ($F(1,38) = 10.65$, $p<0.05$) (see fig. 23). A main effect for trial type ($F(2,76) = 5.59$, $p<0.006$), reflected slower RTs overall for misleading trials compared to resolvable ($F(1,38) = 9.32$, $p<0.005$) and no-probe-1 trials ($F(1,38) = 9.20$, $p<0.005$). This suggests that for all participants making a probe-2 location decision is particularly sensitive to a misleading intermediate probe: encouraging participants to examine the state of an appliance at a location where there is none slows subsequent location based responding.

A significant main effect for probe-2 state ($F(1,38) = 24.7$, $p<0.001$) revealed that all participants were slower in responding to an appliance that was 'OFF' (1847.6ms) compared to 'ON' (1624.2ms) in the encoded set.

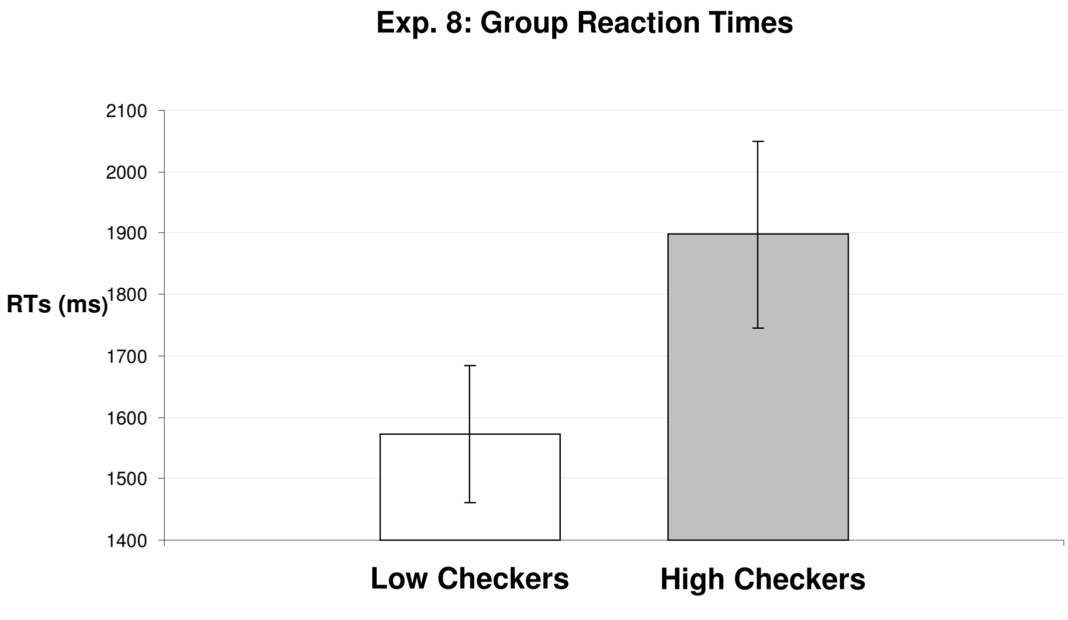

Figure 23. Probe-2 response latencies: High checkers (1898.4ms) were significantly slower overall than low checkers (1573.4ms) in making their probe-2 responses ($p<0.05$). Vertical bars denote standard errors.

Probe-2 Accuracy

The MANOVA (2 x 3 x 2) for probe-2 accuracy revealed a main effect of group ($F(1,38) = 4.27$, $p<0.05$), with high checkers (87.3%) significantly less accurate than the low group (94.1%) (see fig. 24). Importantly, as high checkers were significantly slower in making their responses, we can rule out a speed-accuracy trade-off as an explanation for their poorer accuracy. A main effect for trial type ($F(2,76) = 4.08$, $p<0.05$), reflected no-probe-1 trials were more accurate than resolvable ($F(1,38) = 5.93$, $p<0.02$) or misleading trials ($F(1,38) = 6.70$, $p<0.05$). Therefore, for all participants an intermediate probe resulted in poorer probe-2 state accuracy compared to trials with no intermediate probe.

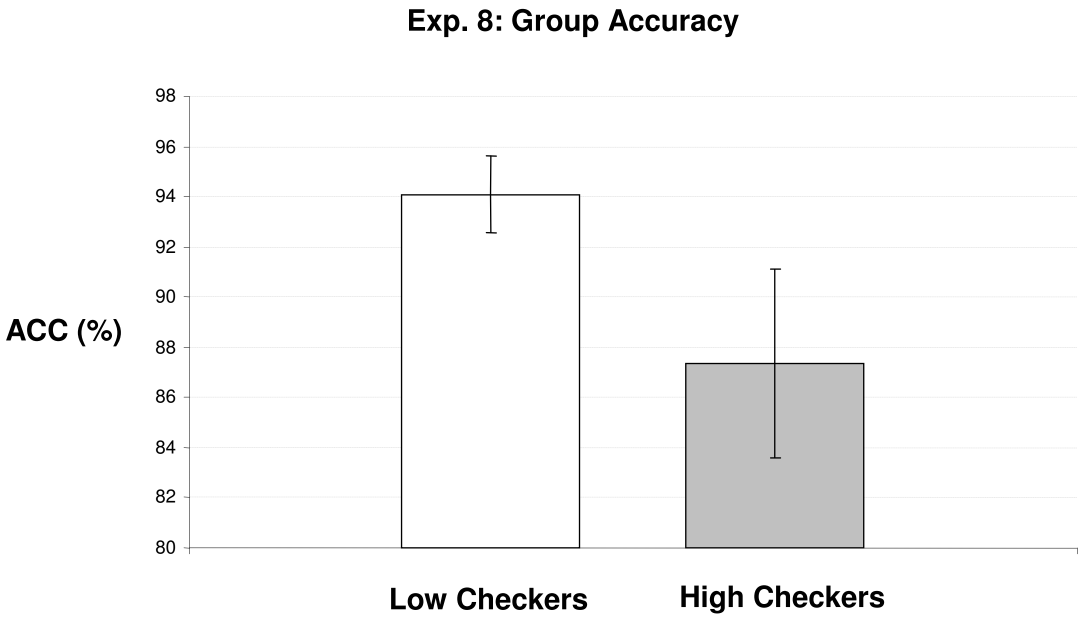

Figure 24. Probe-2 accuracy (ACC%) for group: High checkers (87.3%) were significantly less accurate overall in making their probe-2 responses than low checkers (94.1%) ($p<0.05$). Vertical bars denote standard errors.

Confidence Responses

The MANOVA (2 x 3 x 2) for confidence responses concentrated upon the total 'not-confident' responses of each participant in each condition. A main effect for trial type ($F(2,76) = 7.99$, $p<0.003$) reflected lower confidence for all participants for misleading trials compared to resolvable ($F(1,38) = 4.60$, $p<0.04$) and no-probe-1 trials ($F(1,38) = 10.27$, $p<0.003$). Also a main effect of probe-2 state ($F(1,38) = 26.68$, $p<0.001$) indicated that all participants had less confidence for an electrical appliance that had been 'OFF' than 'ON'. No effects involving group reached significance.

To sum up, we found a general accuracy deficit for high checkers that could reflect general capacity issues. However, based on our previous research (Harkin & Kessler, 2009; 2011 and research reported by others (Ciesielski et al., 2007; Henseler et al., 2008), we did not believe this to be the case. In contrast, we hypothesised that the employed probe-2 may have focused checkers' attention too strongly on the threatening aspect of the stimuli (electric on/off status), hence introducing a generally higher level of interference during encoding, maintenance, and/or retrieval fuelled by anxiety.

Hence, we devised a second Experiment that differed from Experiment 8 regarding the feature dimension of the memory test (probe-2). Instead of probing the state of an appliance (on vs. off) we probed its location (correct vs. incorrect). We expected a more differentiated pattern across conditions with a special role for misleading trials.

5.10. Experiment 9

5.10.1. Method

Participants

40 Participants (mean 21.85 years: 13 males, 27 females) from the University of Glasgow gave written informed consents. British Psychological Society ethical requirements were met, including that of participant debriefing. As before, the checking subscale was used to obtain two groups: 20 low (mean: 0.0, SD: 0.0) and high (mean: 13.75, SD: 6.16) ‘‘checkers’’. Further, no statistical differences between the low and high groups were revealed in gender distribution (p=0.31) or age (p=0.58).

Procedure

Experiment 8 was identical to Experiment 9 with two exceptions. (1) *Probe-2*: We presented an electrical appliance either at the correct (50%) or incorrect (50%) location with respect to the encoding set and asked participants to indicate if it was correctly or incorrectly located (see fig. 22). (2) *Confidence*: We asked participants to indicate their confidence on a sliding scale from 0 (no confidence at all) to 100 (complete confidence). We expected this scale to be more sensitive in detecting between-group differences in meta-cognition than the binary response option employed in Experiment 8.

There were 156 practice trials in total, 12 of which were practice trials including resolvable and no-probe-1 trials only. The main experiment was then done in two blocks (with 5min rest period between), each comprising of 24 resolvable, 24 misleading and 24 no-probe-1 trials presented in random order. As in Experiment 7 an equal ratio of misleading, resolvable and no-probe-1 trials were used.

Design

A two (*Group*: low vs. high checkers) by three (*Probe-1*: resolvable, misleading, no-probe-1) by two (*Probe-2 Location*: Correct, Incorrect) mixed design was employed with *group* as the between- and *probe-1* and *probe-2 location* as the within-subjects factors.

5.11. Results and Discussion of Experiment 9

MANOVAs for a 2 x 3 x 2 design were carried out for reaction times, accuracy and confidence on probe-2 responses due to violations of the sphericity assumption (Mauchley's tests).

Probe-2 Response Latencies

A main effect of trial type ($F(2,76) = 4.01$, $p<0.023$) reflected the linear increase in RTs across resolvable (1847.4ms), misleading (1943.9ms) and no-probe-1 trials (2019.9ms). We suggest that the presence of an intermediate probe (resolvable or misleading) may focus the attention of checkers to responding which primes them to subsequent responding, leading to faster responding in these conditions compared to when no intermediate probe (i.e., no response priming) is presented. This pattern was previously observed in our original experiments, which when considered in relation to the different probe-1 RTs of Experiment 1 (Misleading > Resolvable = No-Probe-1) indicates that the relationship between probe-1 and the specificity of probe-2 is sufficient to influence RTs. A main effect of probe-2 location ($F(1,38) =$

39.31, $p<0.001$) showed that participants responded slower to an appliance that was correctly located (2067.8ms) with respect to the encoded set compared to one that was incorrectly located (1806.4ms).

Probe-2 Accuracy

The main effect of probe-2 location reached significance ($F(1,38) = 42.86$, $p<0.001$), which reflected poorer accuracy for correctly (79.4%) compared to incorrectly located appliances (94.5%). When considered alongside the RT main effect for probe-2 this suggests that correctly located appliances are more difficult to resolve which is reflected in slower RTs. In explanation, an incorrect location can be disproved by at least two partial representations such as remembering which object actually had been in the probe location or by remembering the correct location of the probe object. This is not the case for correct probes where this particular object-location binding has to be received veridically. The group x trial type interaction was significant ($F(1,38) = 3.42$, $p<0.04$). Analysis of the simple main effects for group at each level of trial-type revealed a significant group difference (low=90.5% vs. high=82.3%) for misleading trials ($F(1,38) = 7.52$, $p=0.009$) (see fig. 25), whereas, for resolvable and no-probe-1 trials no statistically significant group difference was observed ($p=0.084$ and $p=0.366$, respectively). However, we are aware that the lack of significant differences does not necessarily equate to a demonstration of similarity but rather could be explained by a lack of sensitivity. We further analysed the simple main effects within each group to determine the locus of between-condition performance differences. For the low group, no differences were reported between resolvable, misleading or no-probe-1 trials (i.e., all $p>0.3$). On the other hand, for high checkers, responses were less accurate for misleading trials than no-probe-1 trials ($F(1,38) = 5.99$, $p<0.02$), but responses for resolvable and no-probe-1 trials were similarly accurate ($p=0.361$). Thus, despite the visually attenuated performance of high checkers across trials, the significant group x trial interaction is due to the special role of misleading trials which is then reflected with a significant group difference specific to this condition.

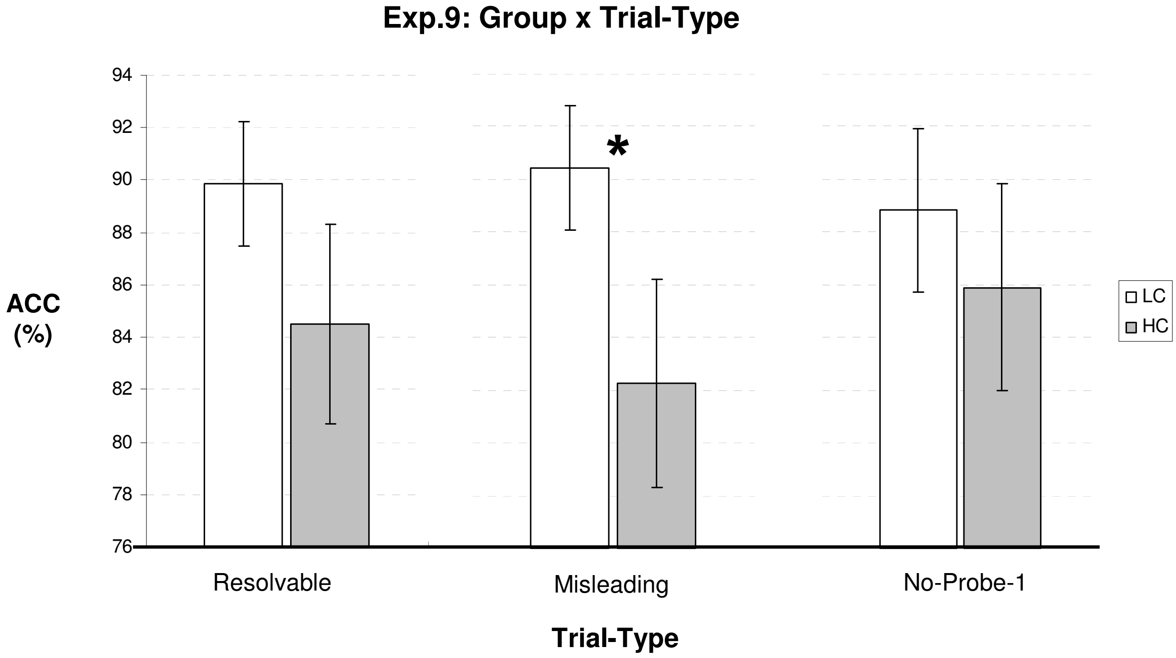

Figure 25. Probe-2 accuracy (ACC%) for group (low vs. high) at each level of trial type (resolvable, misleading, no-probe-1). * Denotes significance at *p*<0.01 level. Vertical bars denote standard errors.

Confidence Ratings

A main effect of group revealed ($F(1,38) = 5.30$, $p<0.028$) that high checkers (70.67) had poorer confidence overall compared to low checkers (80.71) (see fig. 26). Trial-type reached significance ($F(2,76) = 5.87$, $p<0.005$), which was driven by poorer confidence on misleading trials compared to resolvable ($F(1,38) = 4.67$, $p=0.037$) and no-probe-1 trials ($F(1,38) = 8.15$, $p<0.008$). This suggests that a misleading intermediate probe was sufficient to reduce confidence in all participants. Probe-2 location reached significance ($F(1,38) = 20.51$, $p<0.001$) and reflected less confidence for correctly compared to incorrectly located appliances. Poorer confidence for a correctly located appliance reflected the poorer accuracy that all participants had in this condition. The group x probe-2 location interaction approached significance ($F(1,38) = 3.65$, $p=0.064$), with group differences observed for incorrectly ($F(1,38) = 8.31$, $p=0.006$) but not correctly located appliances ($F(1,38) = 2.23$, $p=0.144$). Thus, the low checkers mirrored the general trend of the probe-2 location main effect (i.e., poorer performance for correct than incorrect), whereas the high group had poorer confidence across both conditions.

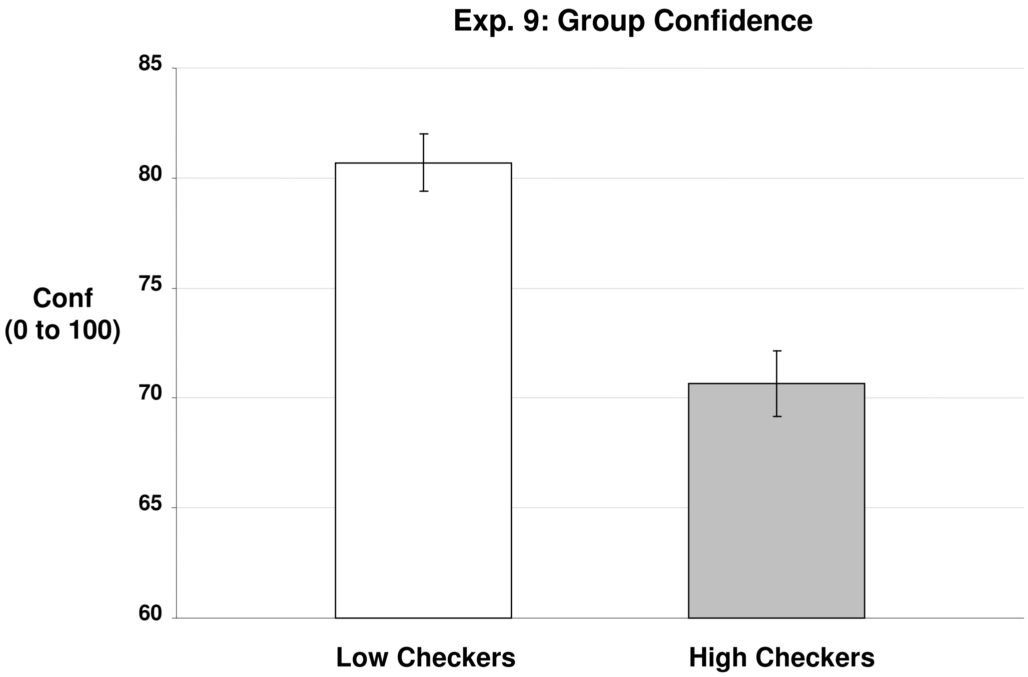

Figure 26. Confidence (0-100) for group: High checkers (70.7) had significantly less confidence overall than low checkers (80.7) (p<0.05). Vertical bars denote standard errors.

Correlations between accuracy and confidence were conducted for each group and both groups showed significant relationships (low group: r=0.56, n=20, p=0.01; high group: r=0.71, n=20, p=0.000) indicating that for all participants confidence mirrors accuracy. In a further analysis we subtracted confidence scores from accuracy scores for each participant in each condition, which produced what we termed a discrepancy score. A discrepancy score of zero indicates that accuracy and confidence mirror each other, whereas an increasing discrepancy score indicates that confidence is numerically less than preceding accuracy. We were primarily interested in group differences in discrepancy scores across trial-types, as this could indicate conditions, where confidence and accuracy might only diverge in high checkers, revealing a metacognitive deficit.

In a MANOVA analysis of the discrepancy scores the interaction between group x trial-type reached significance ($F(1,38) = 3.14$, p=0.049, η^2_p=.076). Analysis of the simple main effects for group at each level of trial-type revealed a significant group difference (LC=6.42 vs. HC=14.18) for no-probe-1 trials ($F(1,38) = 5.42$, p=0.025) but not for resolvable ($F(1,38) = 0.60$,

p=0.442) or misleading trials (F(1,38) = 0.76, p=0.389). This indicates that low and high checkers confidence-accuracy discrepancy is similarly inflated in trials when there is an intermediate probe: accuracy is greater than confidence. However, in no-probe-1 trials low checkers accuracy-confidence is more concordant (6.42) compared to high checkers whose discrepancy score (14.18) is similar to that observed in resolvable (12.47) and misleading trials (14.04). We interpret that high checkers suffer a task independent impairment in their metacognitive functioning which is expressed here as less confidence in their accuracy on no-probe-1 trials.

5.12. General Discussion

The present experiments used electrical kitchen appliances that were concordant with the symptomatology of those afflicted with obsessive-compulsive checking (Rachman, 2002; Thordarson et al., 2004). We did this in an attempt to address a primary criticism of our previous research (Harkin & Kessler, 2009, 2011a) that letters in locations do not resonate with the primary concerns of checkers. We predicted that for high checkers using episodically rich stimuli and questioning a threatening aspect of them (i.e., 'ON/OFF' state of probe-1) would provide a greater challenge to the attention-dependent bindings required for accurate memory recall.

We observed that group effects differed between experiments, a finding we attribute to employing ecologically valid stimuli and probing different features of the memory in Experiment 8 (electric state on/off) compared to Experiment 8 (location). Experiment 8 supported our claim that our stimuli were compatible with OCD/checking symptomatology by revealing a main group effect in reaction times and accuracy. However, reaction times and accuracy data also indicated that the particular manipulations in Experiment 8 may have resulted in a degree of interference in all participants. Specifically, probe-2 reaction times were slower after a misleading intermediate probe, suggesting that this experiment encouraged all participants to access the 'ON/OFF' states of the appliances which then slowed subsequent responding to a state-based probe-2 question. Memory decisions regarding appliances'

'ON' or 'OFF' states (probe-2) were significantly slower for all participants in misleading compared to resolvable or no-probe-1 trials. Memory accuracy was significantly poorer after resolvable and misleading trials compared to no-probe-1 trials. So for all participants continually focusing on ON/OFF states appears to have come at the cost to their performance. Together, the strengths of these general effects could have been sufficient to obscure group effects but this proved not to be the case: High checkers were generally slower and poorer at recalling the state of an electric appliance compared to low checkers (weak hypothesis).

As we did not include an independent cognitive index of WM functioning, high checkers' poorer accuracy overall (compared to low scoring checkers) could be interpreted as impaired WM capacity. However, we argue against this for a number of reasons (for a review see Harkin & Kessler, 2011b). Firstly, if checkers have a general WM capacity impairment then this would have influenced our previous results (Harkin & Kessler, 2009, 2011a). A general impairment would negatively affect WM performance irrespective of the content of the encoded set, i.e., similar no-probe-1 impairment for letters and electrical appliances. Secondly, if checkers suffered from basic capacity impairment, then memory would *not* be influenced by the specificity of the probe-2 question, whereby they would necessarily have impaired appliance-location (Exp. 9) memory in the no-probe-1 condition. Thirdly, there is a convergence of evidence showing that basic WM capacity is intact (Ciesielski et al., 2007; Henseler et al., 2008) with impairment only observed at high load levels when tasks stress dysfunctional components of executive control in OCD patients (Boldrini et al., 2005; Morein-Zamir et al., 2010; Moritz et al., 2003; Purcell et al., 1998a, , 1998b; van der Wee et al., 2003; Zielinski, Taylor, & Juzwin, 1991; Zitterl et al., 2001). Finally, considering that in simple memory tasks subclinical checkers have outperformed OCD patients (Tuna, Tekcan, & Topcuoglu, 2005) and controls (Irak & Flament, 2009), it is unlikely that our group of subclinical checkers had anomalous capacity issues. Rather, it is likely that they have executive impairments analogous to those observed in clinical OCD (Mataix-Cols et al., 1999a; Mataix-Cols et al., 1997; Omori et al., 2007), which interferes with efficient state-appliance-location bindings during encoding and/or maintenance. This is in agreement with the perspective that memory impairments in OCD are secondary to executive dysfunction (Greisberg & McKay, 2003) and it is further in agreement with the metacognitive deficit revealed in Experiment 9.

The differences between low and high checkers were somewhat more subtle, yet even more revealing in Experiment 9: (1) Performance of high checkers was significantly affected on misleading trials compared to baseline (no-probe1 trials). (2) The misleading condition revealed the strongest group difference with the best performance for low- and the worst performance for high checkers across all trial conditions. (3) In contrast to Experiment 8, high checkers' performance on no-probe-1 trials did not significantly differ from the performance of low checkers. Finally, there was a statistical trend for a group difference on the resolvable trials that was reminiscent of the significant differences we had observed before with a spatial probe and abstract stimuli (letters in locations, Expt. 4 in Harkin and Kessler, 2011a). There, a spatial probe had been generally distracting for high checkers. Here however, when the stimuli were relevant to checkers' symptoms (electric appliances with switches) a misleading probe provides additional impairment to that caused by an intermediate spatial probe resulting in the main, statistically reliable difference. This is corroborated by the significant interaction between group and trial type and further detailed analysis which revealed that high checkers performed significantly worse on misleading compared to baseline trials while performance on resolvable compared to baseline trials did not significantly differ (supporting the strong hypothesis). In contrast, the performance of low checkers did not significantly differ for any trial-type comparison.

In explanation, based on the findings from Task 6 we argue that checkers' attention is generally biased toward the threatening aspects of the appliances. In Experiment 9 this is moderated by the emphasis on spatial locations of probe-2, but may still provide high checkers with a slight advantage in accessing the state of an appliance at a resolvable compared to a misleading location during probe-1. This may explain why the group difference for resolvable trials did not reach significance while it did for misleading trials. We argue that our explanation in terms of attention biased by the threatening aspects of the stimuli may be particularly true when locations are being challenged during probe 2 (cf. Exp. 9) rather than if stimuli identities are challenged. This proposal is supported by our previous findings (Exp. 1-4) which showed that high checkers exhibited memory impairments when questioned about the location of a certain stimulus, but not when questioned about the identity of a stimulus at a certain location. That is, maintaining the correct location of an appliance in WM depends more strongly on sustained attention than maintaining the identity of the appliance. Indeed, identity representations may be harder to disrupt than location representations because the identity of a stimulus is based on concepts stored in long-term memory (LTM), whereas the location of a stimulus is arbitrary and specific to the experimental context. Also as we proposed in relation to the findings of Experiment 4 (Harkin & Kesssler, 2011a), cross-modal stimuli (i.e., binding across the ventral and dorsal streams) require greater attentional resources than those processed within one stream (i.e., contrast: impaired object-location binding; Elsley & Parmentier, 2009 *versus* intact object-feature binding; Allen, Baddeley, & Hitch, 2006). As a result, location-identity bindings are particularly vulnerable to interference as sustained attention is necessary to their veridicality in WM. In contrast to our previous studies, however, we employed an equal ratio of misleading, resolvable and no-probe-1 trials throughout our two experiments (in contrast to counter-balanced ratios across two blocks in Harkin & Kessler, 2009, 2011a) which further underpins the robustness of our findings with ecologically valid scenarios.

Finally, we suggest that high checkers' intact no-probe-1 performance in Experiment 9, in contrast to generally impaired performance in Experiment 8, is due to task differences regarding the memory probe (probe 2). Specifically, Experiment 8 required the accurate recall of the appliances' 'ON/OFF' status while Experiment 9 probed the correct location of an appliance. As this no-probe-1 impairment was neither previously reported (Exp 1 to 4; Harkin & Kessler, 2009, 2011a) nor was it observed in Experiment 9, the locus of the difference must be specific to the probe-2 task in Experiment 8 where attention was again focused on the threatening aspects of the stimuli (electric on/off status). We propose that this may have in turn affected the encoding, maintenance and/or retrieval of multimodal bindings in Experiment 8 in form of interference fuelled by anxiety. In fact we regard the group main effect in Experiment 8 as confirmation of the ecological validity of our stimuli.

Specifically, our findings from Experiment 8 are not only supported by checkers specific attenuations of normal IOR functioning (Task 6) for threatening aspects (Task 7) of stimuli but are also in agreement with Attentional Control Theory of Eysenck et al. (2007; Eysenck & Derakshan, 2011). This theory proposed that stimuli which evoke anxiety (i.e., those relevant to checkers symptoms) divert attention from the goal-directed (i.e., maintenance of bindings in EB) to the stimulus-specific (i.e., ‘ON/OFF’ states) attentional system. This theory explains why anxiety is particularly interfering to memory that is reliant upon bindings of features to locations. For example, Lavric, Rippon, and Gray (2003) observed that threat-evoked anxiety impaired performance in a spatial but not a verbal *n*-back WM task. The authors proposed that anxiety interfered with executive functioning, which impaired spatial WM as it more reliant upon sustained attention than verbal WM (for review see Harkin & Kessler, 2011b). This agrees with attenuated IOR effects we saw for high checkers when their attention was drawn to threatening ‘ON’ cues (Task 7). In short, this means that high scorers’ attention perseveres on a threatening stimulus once it was drawn to it, underpinning the ecological validity of our stimuli. Thus, it is possible that as ‘ON/OFF’ states are salient to the symptoms of checkers they attend to them at the cost of their binding to the appliance. Thus, we provide tentative evidence which concurs with Omori et al. (2007) that checkers likely have executive impairments of inhibition which interferes with the veridicality of bindings maintained within the EB.

While group differences in confidence were not observed in Experiment 8, Experiment 9 revealed a group main effect for a lack of confidence in high scorers. This highlights that a continuous confidence scale (Exp. 9) is not only more sensitive for detecting group effects but it also lends itself to a wider range of statistical analyses compared to the binary forced-choice (Exp. 8). The main effect in Experiment 9 indicates that high checkers have a global (trial-type independent) impairment in confidence compared to low checkers. That is, although correlations where high between accuracy and confidence for both groups, only high checkers showed a significant discrepancy for the no-probe1 trials. This dissociation between performance and confidence in the baseline condition in particular, suggests a metacognitive deficit in form of impaired performance monitoring that is present in high- but absent in low checkers.

5.12.1. Conclusions

The current findings confirm that checkers' memory impairments are secondary to executive dysfunction, especially when ecologically valid stimuli are employed. The different accuracy patterns of high compared to low checkers between Experiment 8 and 9 allow us to make the following conclusions. In Experiment 8, we observed a novel finding with high checkers showing a robust impairment in their ability to accurately recall the state ('ON' or 'OFF') of an electrical appliance (weak hypothesis). A group effect which was surprisingly not influenced by trial type (resolvable, misleading, no-probe). While superficially this appears to indicate a general impairment in WM capacity, we have highlighted a number of reasons why this is an unsatisfactory explanation. We conclude that this novel, general impairment is rather specific to the memory task (probe-2) in Experiment 8 that biased subclinical checkers towards the threatening electric on/off status of the appliances (Harkin & Kessler, in press), which in turn generally interfered with multimodal bindings in the EB. In contrast, Experiment 9 revealed the expected, more differentiated pattern with a special status for misleading trials: Performance of high checkers was significantly affected on misleading trials compared to baseline trials and the strongest group difference was observed in the misleading condition (strong hypothesis).

In Experiment 9 we successfully employed a continuous confidence scale that allowed us to calculate discrepancy scores between accuracy and confidence for each participant in each condition. The main result was that while there overall strong correlations between accuracy and confidence in both groups, only the high checkers revealed a significant discrepancy in the baseline condition. Although they reached their highest performance levels in this condition, their confidence did not improve, which we interpret as supporting a metacognitive deficit that is absent in low checkers. The importance of memory and metacognitive impairments in OCD is corroborated by reports that poor memory and checking influences the severity of obsessional thinking (Park et al., 2006; Purcell et al., 1998a).

5.12.2. Limitations

The following limitations of our study have to be considered. Firstly, using a subclinical group always raises the issue of their relevance as an analogue to a clinical group. We agree, however, with Mataix-Cols et al. (1997; 1999a) that subclinical OCD groups are a valid means of determining which cognitive factors play a role in clinically defined OCD, particularly considering their reduced medication and potential for co-morbidities. We therefore expect that the pattern observed here with subclinical checkers could be more pronounced using clinical OCD patients, yet, also more variable. Secondly, despite the claim that a subclinical group provides a ‘purer’ indication of the cognitive impairments specific to this subtype; we did not control for anxiety or depression nor did we provide an independent cognitive index of WM functioning and so cannot exclude possible group differences. Thirdly, subjects were not explicitly matched for education; however, they were selected from an undergraduate population, thus, ensuring a homogenous educational background for all participants, which is yet another advantage of a subclinical sample. However, future research with clinical patients could examine in more detail the relationship between severity of symptoms and completion of formative school which may then influence general intelligence. Fourthly, we did not counterbalance the keys for the forced-choice confidence responses in Experiment 7 and so cannot determine if a lateralization bias influenced participants’ responding and possibly masking existing group differences.

6. The role of working memory in compulsive checking and OCD: A systematic classification of 58 experimental findings

The importance of memory in checking/OCD is not only evident in the present thesis but is also reflected in a number of reviews covering the topic, for example, Coles and Heimberg (2002), Woods et al. (2002), Kuelz et al. (2004), Muller and Roberts (2005) and Cuttler and Graf (2009). However, despite this large body of research the evidence for memory impairments in OCD is described as mixed at best (Hermans et al., 2008). For example, there are inconsistent findings regarding a general mnestic deficit (e.g. Tallis, 1997 vs. MacDonald et al., 1997; McNally & Kohlbeck, 1993), verbal memory (e.g., intact: Henseler et al., 2008 *versus* deficit: Tuna, Tekcan, & Topcuoglu, 2005) and generally affected visuospatial memory (Hermans et al., 2008; Mataix-Cols et al., 1999a; Muller & Roberts, 2005).

We attribute this to the traditional pursuit of OCD memory impairment as one of the general capacity and/or domain specific deficits (visuospatial vs. verbal). In contrast, a body of research indicates a more subtle relationship, with memory impairments secondary to executive dysfunction (Greisberg & McKay, 2003). If a memory task taps into a dysfunctional component of executive functioning (see Table 5), attenuated memory impairment will follow. In this understanding, it is executive deficits in conjunction with task requirements that differentiate memory functioning in OCD from controls (Olley, Malhi, & Sachdev, 2007). This review provides a more precise level of explanation: EB functionality (binding) is vulnerable to interference through executive dysfunction. In other words, interference from executive dysfunction reduces the veridicality of multimodal bindings within the EB, attenuating OCD memory performance. Irrespective of the exact mechanism for binding multimodal features together into a representation, researchers tend to agree that attentional effort is required for their generation and maintenance (i.e., Hyun, Woodman, & Luck, 2009). Therefore, memory impairments occur if distraction is sufficient to interfere with attention-dependent bindings. For example, Fougnie and Marois (2009) hypothesized that attention iteratively refreshes multimodal representations in WM: it is only when a distractor sufficiently interferes with attention that there is a failure to maintain features in a bound manner (e.g., Harkin & Kessler, 2009, 2011a; Harkin, Rutherford, & Kessler, 2011; Kessler & Kiefer, 2005). This dove-tails nicely with findings showing that the more emotionally engaging a given distractor is to an individual (or group) the more it interferes with attention-dependent bindings (Mather et al., 2006).

The present review therefore concentrates on identifying the mechanisms and parameters underlying executive-memory impairment in OCD, i.e., disrupted attention-dependent bindings in the EB. To this end, we provide a synthesis of our research which has concentrated on checking in WM performance (Harkin & Kessler, 2009, 2011b; Harkin, Rutherford, & Kessler, 2011). Basically, our novel paradigm aimed at the 'EB' component of WM (Baddeley, 2000). During the delay period of a WM task (i.e., between *memory-set*: letters in locations & *memory task*: "Where was letter?") participants were presented with misleading information which was detrimental to high checkers' memory more than for low. Checkers, it appears, are poorer at inhibiting irrelevant and misleading information (i.e., executive dysfunction) which interfered with the maintenance of bindings (letter in locations) in the EB. From this, we identify three common factors (EBL: **E**xecutive-functioning efficiency, **B**inding complexity, and memory **L**oad) that we generalise to 58 experimental findings from 46 OCD memory studies and explain otherwise inconsistent research, e.g., intact *versus* deficient verbal memory.

Table 5. Studies reporting executive deficits in OCD.

Study	Test/Task	Executive Function	OCD Impairment
Head et al. (1989) Roh et al . (2005) Sanz et al. (2001)	Wisconsin Card Sorting Test (WCST)	Set-Shifting	More perseverative errors
Bohne et al. (2005)	WCST	--	Poorer at learning from feedback
Goodwin & Sher (1992) Harvey(1987) Head et al. (1989) Hymas et al. (1991) Sanz et al. (2001)	WCST	--	Completed less categories
Chamberlain et al. (2006) Purcell et al. (1998a) Veale et al. (1996) Watkins et al. (2005)	Intra-Dimensional/ Extra-Dimensional (ID/ED) Task	Selective attention and set-shifting	Stuck in previous attentional set/impaired in switching cognitive set

Elliot et al. (1995) Fenger et al. (2005) Veale et al. (1996)			
Enright & Beech (1993) Enright et al. (1995) Hoenig et al. (2002)	Negative Priming	Inhibition	Preattentive deficit in cognitive inhibition
Moritz et al. (2009)	Inhibition of Return (IOR)	Inhibition	Slower RTs when targets preceded with threat stimuli
Harkin & Kessler (in press)	IOR	--	Normal inhibition overcome by attention to threat
Omori et al. (2007)	Stroop Test, Trail Making Test, Go/No Go Task Category Fluency	Inhibition, cognitive flexibility, and multi-tasking	Only checkers (not washers) was inhibition impairments correlated with poor episodic memory
Bannon et al. (2006) Bannon et al. (2008)	Go/No Go Task Stroop	Selective attention	Deficit (while OCD symptoms were reduced)
Kim et al. (2002) Penades et al. (2005) Roh et al. (2005)	Trail-Making Task (TMT)	Organization	Consistently slower on Part A(organization impairment) and B (set-shifting)
Fenger et al. (2005)	Figural Fluency Task (FFT)	--	Impaired in organizing spatial information

6.1. A working-memory explanation

Baddeley's original model (1986) included a central executive, phonological loop and visuospatial sketchpad and was deemed separate from long-term memory (LTM). While this simple model explained a range of data (e.g., phonological similarity, word-length effect), it could not account for all experimental phenomena. For example, the visuospatial sketchpad, a capacity limit of 4 units was observed for the maintenance of individual features (colors *or* orientations) as well as for integrated objects with colors *and* orientations (Luck & Vogel, 1997). The so-called "binding problem" (e.g., Treisman, 1996) refers to the fact that information presented in visual scenes rarely consists of isolated features. Rather, features pertain to objects, objects to locations, and objects are further embedded into episodes together with a plethora of contextual information. A parallel processing architecture like the human brain needs mechanisms for tracking "what goes with what" in order to generate and maintain bindings between multiple features (Hinton, McClelland, & Rumelhart, 1986). Therefore, accurate memory (WM and LTM) requires the encoding, maintenance and retrieval of bindings between various aspects of a multimodal episode (Allen, Baddeley, & Hitch, 2006). Baddeley (2000), therefore, extended his classic 1986 WM model to include an "EB" that allowed for multimodal, temporarily integrated representations and served as an interface with episodic LTM. Based on this development, we proposed (Harkin & Kessler, 2009) that an executive dysfunction (e.g., unsuppressed intrusive thoughts/stimuli) interferes with fragile multimodal bindings in the EB, resulting in the consolidation of affected episodes into WM and LTM.

6.2. Empirical evidence from high checkers' memory performance

With these points in mind, our recent experiments (Harkin & Kessler, 2009, 2011b; Harkin, Rutherford & Kessler, 2011) set out to: (1) *engage* the EB using stimuli that required multimodal conjunctions between various object features and spatial locations and to (2) *hamper* EB functionality by confronting high and low checkers with misleading/irresolvable information during the WM retention interval. In Harkin and Kessler (2009), we employed this novel paradigm for the first time. We presented 4 letters (see fig. 2) randomly in 6 possible locations and asked participants to indicate 4 seconds later if a test letter was in the correct (50%) or incorrect (50%) location. The novel manipulation that was meant to induce checking was presented as an additional probe between the encoding-set and the actual test letter. This intermediate probe (probe-1) was either resolvable (e.g., "Where was T") or misleading (e.g., "Where was K") referring to its presence or absence in the encoding-set, respectively (see fig. 2). Misleading trials were hypothesized to induce frustrating and unnecessary checking in those with such a predisposition as no correct answer was possible but in order to proceed, suppression of the misleading information and of the urge to check was required.

Conforming to our expectations, high scoring checkers' memory performance was attenuated compared to low checkers when interfered with by misleading information, yet, performance was not statistically different when the distracting intermediate probe was resolvable or absent. Importantly and in agreement with previous findings (e.g., Ciesielski et al., 2007; Henseler et al., 2008), this further underpins that there is no general difference in WM capacity *per se* between high and low checkers.

We extended these experiments in Harkin and Kessler (Harkin & Kessler, 2011a) to include the same 4 letters in 6 locations but with an additional feature dimension (color) in one experiment and a different distractor probe (spatial) in another. Adding color enhanced the memory load in the EB and resulted in overall reduced performance but not in a specifically enhanced deficit for checkers. Thus, we may have induced a greater degree of checking/uncertainty in all participants. This further emphasises how careful one must consider the requirements of a task in order to obtain a checker-specific performance deficit. Employing a spatial probe as the intermediate distractor, however, had the desired effect regarding a checker-specific deficit, although WM load per se was not increased. We asked which letter had been presented at a particular location where there either *had* (resolvable) or *had not* been a letter (misleading). This spatial distractor manipulation boosted group differences, as it tapped into more specific executive deficits of high checkers (i.e., suppression of distraction) while low checkers were not challenged by this modification. Furthermore, the use of eye tracking measures in our WM task revealed that high checkers made more fixations during misleading trials to primarily empty encoded set locations (Exp. 5). Indicating that impairments in their ability to inhibit misleading stimuli induced a degree of uncertainty which motivated checkers to search the contents of WM at empty locations where no additional task-relevant information was present (Harkin, Miellet, & Kessler, subm).

While we reported robust and replicable effects in the aforementioned studies we were aware of the limitations of using letters in locations, as it is unlikely that they evoke a strong emotional response in checkers (see Moritz et al., 2008). Our third series of experiments, therefore, used ecologically valid stimuli in the form of electrical kitchen appliances (Harkin, Rutherford & Kessler, 2011). We presented 4 kitchen appliances in 6 possible locations on a kitchen countertop: two appliances were ‘ON’ and two were ‘OFF.’ Again, we used an intermediate spatial probe asking if the appliance at a cued location had been ‘ON’ or ‘OFF’ (an appliance had either been there = resolvable, or not = misleading). When the primary WM task required remembering the correct location of an appliance we found a very similar pattern of group differences as we previously had with letters, yet, statistically and experimentally more robust (stronger effect sizes with fewer trials) and, most importantly, accompanied by a metacognitive deficit in high checkers, reflected in reduced confidence even when performance was at ceiling and did not differ statistically from the low checking group, i.e., in the baseline condition without a distracting probe.

6.3. The EBL (Executive-Functioning, Binding Complexity, Memory Load) classification system

Our synthesis so far leads us to conclude that checkers’ memory impairment results from a complex interaction between (1) *executive dysfunction* in encoding organization, multimodal integration, selective attention (inhibition), maintenance control, and set-shifting and (2) the *task components* of load (e.g., high load, requiring chunking), multimodality (e.g., location+identity+color), distraction (e.g., dual task paradigm), retrieval dimension (e.g., location), and stimulus salience (e.g. electric switches). We proposed that the likely locus where these deficits interact and potentially augment each other is the EB and we have reviewed supporting findings and arguments. In conclusion, we further propose that there are etiological and explanatory factors common to OCD, which can be summarised along the following three dimensions that serve as our basis for predicting and classifying WM deficits in compulsive checking and OCD:

(1) ***Executive Function Efficiency (E)***: Checking (Cha et al., 2008), rumination (Exner, Martin, & Rief, 2009), and disinhibition (Omori et al., 2007) are all associated with poorer memory in OCD, implying that if these impairments of executive function are present or induced by a task then OCD patients will experience a detriment in memory functioning relative to controls. We follow Wolters and Raffone’s (2008) tri-partite definition of executive

functioning consisting of (1) *Attentional Control*: top-down selective activation of task-relevant representations and suppression of task-irrelevant stimuli and responses, (2) *Maintenance*: holding task-relevant information in an active state, and (3) *Integration*: flexibly bind and manipulate information from multimodal sources, in the service of controlling task execution. Efficient executive functioning can improve performance by reducing outside interference and by selecting mnemonic strategies such as chunking of information based on long-term-memory knowledge (Miller, 1956). In this understanding, OCD memory impairment occurs when: (1) Experimental manipulations aggravate existing impairments in executive functioning which interfere with attention-dependent bindings. For example, when the encoding-set is concordant with OCD symptomatology it may divide attention between threat and encoding (Coles & Heimberg, 2002), which reduces quality of attention to bindings, impairing memory performance. (2) Inappropriate use of executive strategies decreases binding efficiency and/or the overall load of a given memory representation. We will discuss that an inability to appropriately structure and organize stimulus input is typical of OCD (Kuelz, Hohagen, & Voderholzer, 2004).

(2) ***Binding Complexity (B)***: Binding different (multimodal) features together and maintaining these representations over time impose a challenge that increases with the number of features and their multimodality. We propose that the executive function deficit ‘allows’ distracting information to affect the fragile complex bindings in OCD. The inherently greater binding complexities of visuospatial tasks (e.g., multiple objects-to-location bindings) are more likely to reveal OCD impairments than verbal tasks. Complex bindings are susceptible to interference and place greater strain upon correct executive control – especially when multimodal bindings are involved (Harkin & Kessler, 2009, 2011b; Harkin, Rutherford & Kessler, 2011; Olley, Malhi, & Sachdev, 2007). Verbal deficits, however, will occur if the task relies to a similar extent upon the maintenance of complex bindings (e.g. position of letters in space or sequence). This places memory impairment primarily as an outcome of disrupted multimodal bindings and secondarily as one of memory domain. It just so happens that linguistic/verbal material is usually more

strongly subserved by LTM concepts (if not artificially scrambled, e.g. non-words), thus, providing semantic/lexical knowledge that facilitates complex bindings. We expect Binding Complexity to play a predominant role during maintenance, when attention is required to ensure veridicality of WM representations over time.

(3) ***Memory Load (L)***: Assuming that there is no basic capacity issue involved in OCD (e.g., Ciesielski et al., 2007; Henseler et al., 2008; Harkin & Kessler, 2009, 2011a; Harkin, Rutherford & Kessler, 2011), performance deficits under high load would crucially depend on executive strategies (van der Wee et al., 2003): An increase in load (i.e., number of chunks to retain) places greater stress upon the correct implementation of organization strategies (chunking), updating, and overall task-management (Smith & Jonides, 1999). Efficient executive control reduces the overall complexity and/or load of a representation that is subsequently maintained in WM. For example, when recalling a sequence of unrelated words, performance drops when the number of words exceeds five or six as it is beyond the functional capacity of the phonological loop. But, if the words create a sentence, then span can reach as high as sixteen, far exceeding loop capacity (Baddeley, 1987). Hence, chunking improves efficiency as items are not individually maintained (Miller, 1956). Therefore, verbal tasks that benefit from semantic clustering could reveal OCD impairments as they fail to efficiently chunk and reduce the load of the encoding-set. Memory impairment in OCD is not an issue of basic WM capacity (e.g., Harkin & Kessler, 2009) but rather of creating appropriate mnemonic associations and hierarchical groupings using existing knowledge that alleviates the burden on WM (see Ericsson, Chase, & Faloon, 1980). So, while poorer performance is expected for ‘everyone’ at high loads, we provide an explanation for when and how people with OCD are particularly affected (e.g., van der Wee et al., 2003).

6.3.1. The role of anxiety in executive function efficiency (E)

In our model (Harkin & Kessler, 2009) as well as in our *EBL* classification system we focus on the cognitive mechanisms that mediate specific forms of information processing that have been found to be deficient in OCD. We would like to emphasise that the emotional state associated with specific stimuli and situations may *boost* these deficiencies: Anxiety and lack of confidence in their ability to control a given situation (Rachman, 2002) may further attenuate existent cognitive deficiencies in OCD. In other words, we are careful to state that anxiety/lack of confidence is sufficient but not necessary for executive-memory impairment to occur. For example, we report findings from two studies (Roh et al., 2005; Rao et al., 2008) where resolution of OCD symptoms (and anxiety; Rao et al., 2008) was not associated with improvements in WM functioning. While it could be that some executive deficiencies are part of the OCD endophenotype it is likely that cognitive functions may either become deficient as a consequence of a futile attempt to counteract anxiety by 'over-using' specific executive functions - e.g., memory retrieval may turn into compulsive memory checking (Harkin & Kessler, 2009, 2011a) – or cognitive functions may become progressively impeded due to constant insecurity fueled by anxiety, manifesting itself as hampered executive selection between stimuli, goals, and actions (Harkin, Rutherford, & Kessler, 2011). Thus, anxiety is likely to act in a manner similar to a dual-task paradigm (Baddeley, 1986) by reducing the amount of attention on the primary memory task (see Tasks 6 & 7; Harkin & Kessler, in press). In the following, we implicitly assume a 4^{th} dimension as the level of induced anxiety/insecurity and we propose that this implicit dimension predominantly affects executive functioning and is therefore inherent to the E-dimension of the *EBL* system. Specifically, we assume that the more threatening the employed stimuli (e.g. switches, electric appliances) or procedures (e.g., pressure, distraction, misleading information) are in a given study, the more likely executive functioning will be modulated, with knock-on effects for memory performance. Paradoxically, memorized threatening items might even improve performance by biasing attention toward these items during encoding.

6.4. Applying the EBL classification system to 58 experimental findings

Figure 27 explains where we *do* and *do not* expect to observe OCD memory impairments relative to controls; this we suggest is influenced by the degree of executive function efficiency (E), binding complexity (B) and memory load (L) within any given neuropsychological task. First, we do not expect memory performance to differ between OCD patients and controls for tasks that are low in executive demand, binding and load (see: white region in top-left quadrant of fig. 27). Second, likelihood for OCD-specific deficits increases as a combination of high load, binding complexity, and executive function requirements (increasingly black area in the bottom-right quadrant). But finally, as we move toward the extreme end of the *EBL* continuum, memory impairment reduces in magnitude and eventually disappears because due to a simple floor effect operating for OCD patients and controls alike. We suggest that task requirements must be sufficient to tap into executive dysfunction but at the same time not be so extreme to reduce all participants' performance (i.e., controls and OCD) thus obscuring OCD impairments.

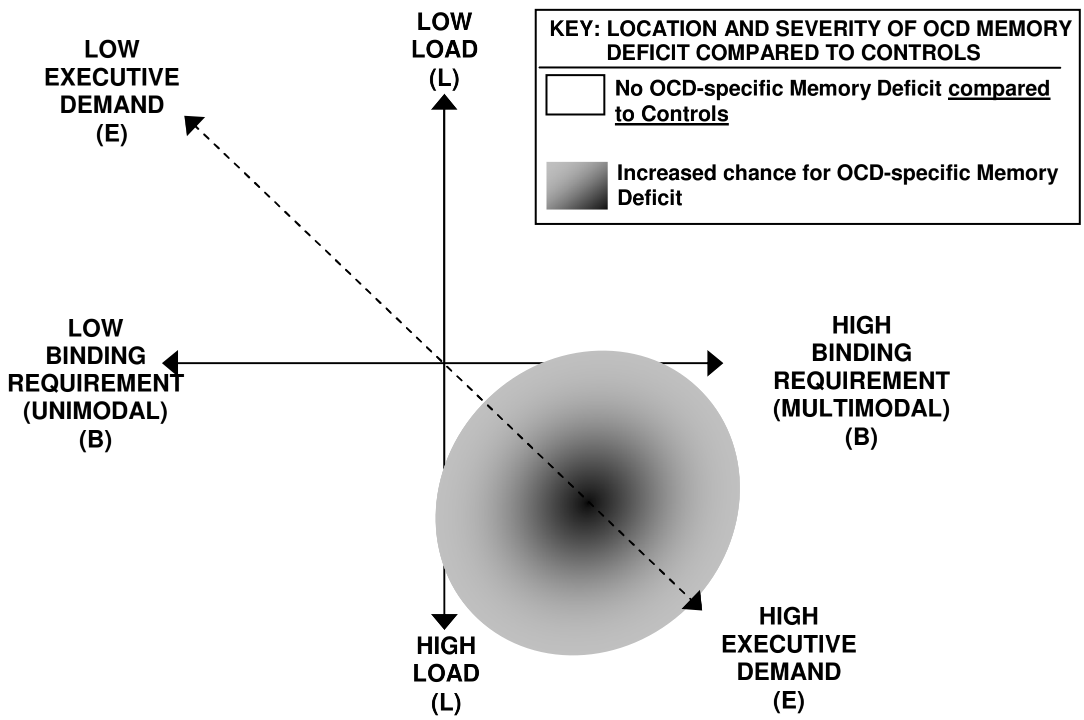

Figure 27. The *EBL* Classification System.

In light of this, we suggest that differences in the *EBL* scores of verbal and visuospatial tasks make OCD memory impairments more likely in the latter, especially if spatial locations are relevant to the task. We shall see that verbal tasks, generally, present verbal information in a format (stories, word lists) that is high in load but low in binding complexity. In this case, performance is benefited by efficient executive processes that utilize existing representations in LTM, i.e. chunking according to categories, that reduces load (see fig. 28A).

Thus, verbal impairments in OCD are due to poor executive functioning failing to reduce the load of verbal stimuli and so they operate primarily within the dimensions of E and L. In contrast, visuospatial tasks inherently have a greater binding demand, where successful performance depends on the veridical binding of multimodal features (spatial + visual). Generally, if visuospatial tasks employ multimodal stimuli that cannot be directly linked to a LTM concept (letters or words can) that could support the chunking of WM representations, then memory performance in OCD depends on the bidirectional relationship between executive organization strategies (*E*) and multimodal binding complexity (*B*) which strongly influences the actual load (*L*) of all representations in the EB. In certain instances, tasks that steadily increase load within a visuospatial domain (e.g., *n*-back, corsi-block) will see a detriment in OCD memory performance at higher levels, as it is at this point their executive inefficiencies fail to match task demands, impairing memory relative to controls. In sum, we expect that OCD visuospatial memory impairments will be more evenly distributed between the three *EBL* dimensions as depicted in Figure 28B. We propose that it is the *EBL* requirement (high scores – but not too high – on all three dimensions) of a task that determines if verbal or visuospatial memory impairments in OCD are observed rather than the domain *per se*.

In the following we will examine studies that investigated OCD memory performance and locate each study's methodology within the *EBL* classification system. It is important to stress that it is impossible to exactly quantify the 'scores' we allocate for a particular study on each dimension. We will explain to the best of our knowledge why there are good reasons to believe that a given study scores highly or lowly on the three *EBL* dimensions based on its task requirements and by comparing it to other studies. We believe that these virtual scores will help the research community to gain a clear overview of the major findings in the field and allow explaining and predicting under which circumstances memory deficits in OCD do occur and under which they do not. Our analysis will break down the literature into the classic distinction between verbal and visuospatial memory and will discuss for each domain separately why memory functioning remained intact in some studies and then why and which studies did reveal deficits.

6.5. Verbal Memory

The literature paints an inconsistent picture with respect to OCD verbal memory performance. We argue that this is due to the manner in which tests of verbal memory differ in their executive-functioning, binding complexity and memory load scores.

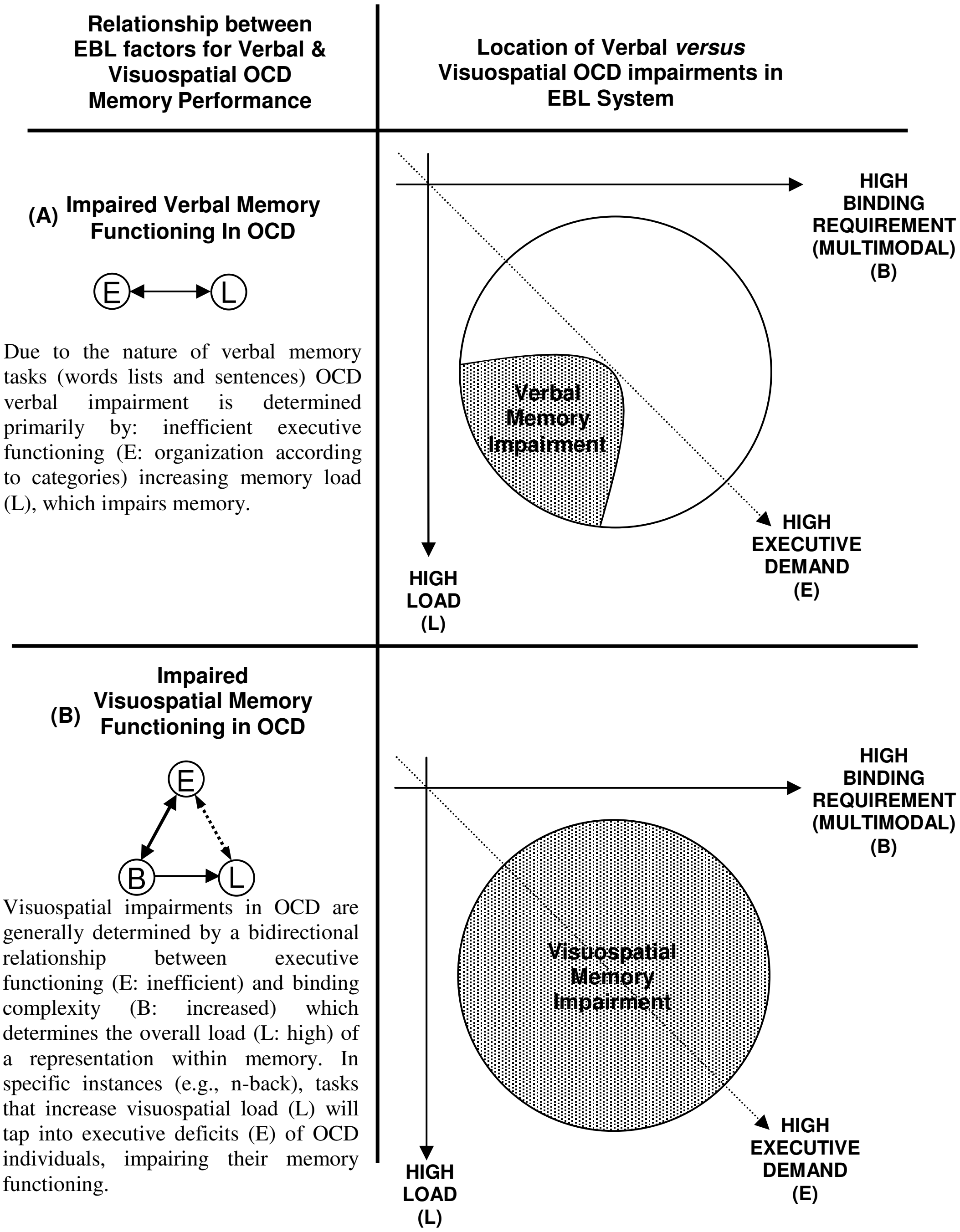

Figure 28. The contribution of *EBL* factors for verbal (A) and visuospatial (B) OCD memory performance and their respective locations of impairment within *EBL* dimensional space.

6.5.1. Intact verbal memory in OCD

Studies (see Table 6) showing intact verbal memory invariably share the same characteristics: (1) low executive demands (minimal strategy and/or attention allocation necessary), (2) low binding complexity and (3) low memory load, i.e., within phonological loop capacity (6 items). On the other hand, an extremely difficult task that impairs all participants to the same extent is likely to mask any OCD-specific memory impairment.

Table 6. Studies reporting no verbal memory deficits in OCD.

Authors	Method	Task Requirements	Groups Compared	Behavioural Findings
Henseler et al. (2008)	Delayed Match to Sample (WM task)	Encode 4 letters, identify if probe letter was in 4	11 OCD patients (YBOCS; 21.0) vs. 11 controls	No differences
Foa et al. (1997)	Sentence Recognition	Contamination vs. neutral sentences presented in 3 levels of noise	15 OCD patients (YBOC: 24.7) vs. 15 controls (2.8)	No differences
Martin et al. (1995)	Self-paced word selection task	Always select a different word	18 OCD patients (DSM-III-R criteria) vs. 18 controls	No differences
MacDonald et al. (1997)	Verbal recall & recognition	Memorize 48 words presented for 1 sec each	10 OCD checkers (≥ 4 on checking MOCI) & 10 OCD non-checkers (<4 on checking MOCI) vs. 10 controls	No differences
Rubenstein et al. (1993; Exp. 2)	Verbal recall	Memorize 50 words presented for 4 seconds each	20 subclinical checkers (≥ 4 checking MOCI) vs. controls (≤2)	No differences

In a simple (encode: 4 letters and memory task: same/different single letter) delayed-match-to-sample task (DMTS), Henseler et al. (2008) failed to report any significant group differences as OCD patients (92.6%) performed at a similar ceiling level to controls (93.5%). This task called minimally upon the *EBL* factors: there were no distractors to suppress, the stimuli were non-threatening, and binding requirement was minimal as successful performance required the remembrance of 4 individual letters (within loop limits) not letter-to-location bindings. On a self-paced test (recall and recognition) of verbal WM, Martin et al. (1995) presented participants with 16 words on a page, in a book of 16 pages. The only measure that revealed a significant group differences was total time taken, with OCD patients taking longer than controls to make 16 successive choices. As this task is predominantly visuospatial in nature (locate different words in spatial locations), we argue (based on the findings reported in Chapter 3) that this is evidence of organizational impairments (i.e., 'E': executive functioning efficiency) slowing OCD patients' processing of each page. If this is the case, we predict that if individuals with OCD require longer to process a piece of information to their satisfaction relative to controls, interrupting this mid-flow will interfere with their ability to efficiently encode words, thus, highlighting that an executive impairment must be sufficiently operant to impair memory. As another example, Foa et al. (1997) reported that checkers' memory for contamination and neutral words was intact despite showing a concurrent perceptual distractibility (i.e., rated background noise as louder than controls). According to the *EBL* system we would not expect OCD memory impairments in this case as the disruption is not task-related and the task itself does not impose high *EBL* requirements.

In a classic study, often cited as evidence for lack of verbal deficits in OCD, MacDonald et al. (1997) investigated verbal recall and recognition. The experiment consisted of the following phases: (i) Study Phase 1, (ii) Distraction Phase 1, (iii) Recall Test, (iv) Study Phase 2, (v) Distraction Phase 2, and (vi) Recognition Memory Test. Specifically, (i) forty-eight words were presented, each for 1 second with 750 ms between each word, (ii) then a 7 minute distractor task was administered between the 48th word and the (iii) beginning of the free recall period. Then, after (iv) study phase 2 (identical in format to the first but with different words), there was a (v) 10 minute distractor task followed by a (vi) recognition task which presented single words requiring participants to indicate if they had (old judgment) or had not (new judgment) been presented in study phase 2 (iv). Considering this methodology in the *EBL* system presentation of a word for 1 second calls upon WM resources (i.e., executive-attention, phonological rehearsal) and LTM word representations (Cowan, 1999). Successful recall requires quick consolidation into verbal LTM, before presentation of the next word in 750 ms. An encoded word will experience primacy and recency interference from previous and subsequent words, respectively (Murdock, 1962), in addition to the substantial interference from the distractor tasks. This threatens veridicality of a word within early encoding, which likely impairs subsequent recall and recognition. In sum, this very difficult task obscures group differences by inducing a floor effect in all participants, an assertion supported by the very low recall proportion for checkers, non-checkers and controls of 0.179, 0.142, and 0.188, respectively. Furthermore, in a task of similar difficulty (Exp. 2; 50 words – 4secs each – from 5 categories), Rubenstein et al. (1993) failed to report any differences in memory of checkers (47%) compared to controls (49.6%). In these experiments, extant OCD/checkers' executive-memory impairments would need to be extremely acute to impact memory performance and significantly differentiate them from controls.

Summary: Intact verbal memory in OCD

For the aforementioned studies, ceiling (e.g., Henseler et al., 2007) or floor effects (MacDonald et al., 1997; Exp. 2: Rubenstein et al., 1997) may underlie lack of verbal deficits. However, we are aware that the low group numbers of 11, 15, and 10 of Henseler et al. (2008), Foa et al. (1997), and MacDonald et al. (1997), respectively, may have resulted in these studies being underpowered. However, we see below that studies with similar group sizes (e.g., van der Wee et al., 2003, 2007; Tallis et al., 1999; Simpson et al., 2006) reported significant group effects suggesting that OCD performance is better explained by scores on the *EBL* dimensions as opposed to group size (see fig 27).

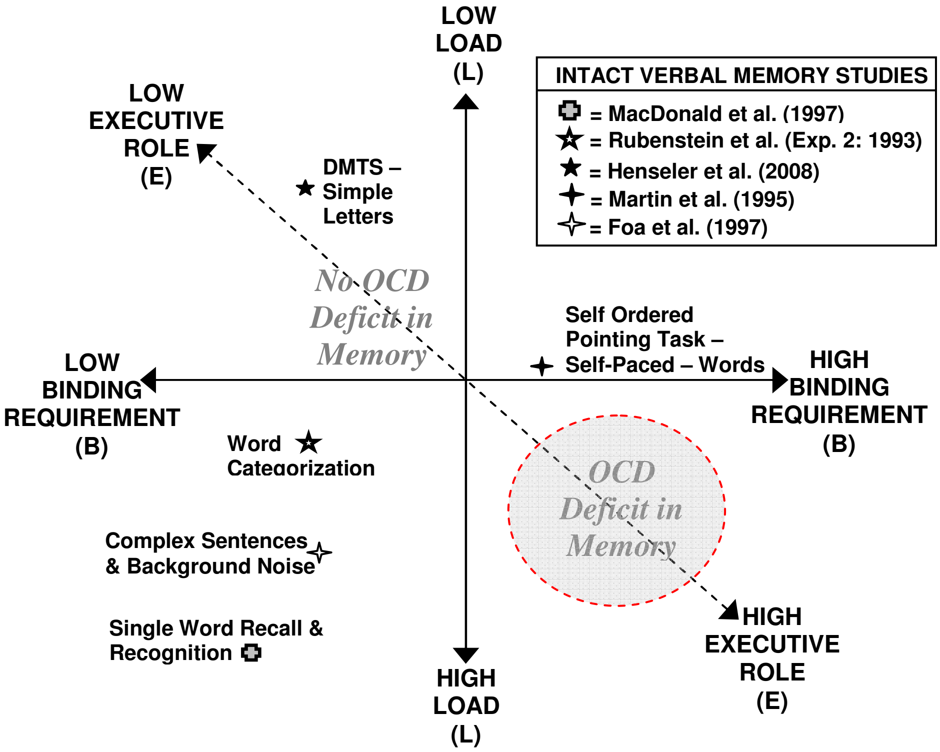

Figure 29. Location of intact verbal memory studies within the *EBL* Classification of OCD Memory Deficits.

6.5.2. Deficient verbal memory in OCD

Verbal memory impairment in OCD is invariably seen in studies that use words/sentences that benefit from organization according to implicit categories (see Table 7). Due to inefficiencies in their executive functioning, OCD patients fail to use mnemonic strategies (e.g., chunking according to categories) which reduces their memory performance relative to controls.

Table 7. Studies reporting verbal memory deficits in OCD.

Authors	Method	Task Requirements	Groups Compared	Behavioural Findings
Sher et al. (1984)	Logical Method subtest of WMS	Listen to short story, recall & recognition requires semantic linking	Frequent vs. occasional vs. infrequent checkers vs. controls (MOCI)	Checkers deficit in recalling meaningfully linked sequences
Tuna, Tekcan, and Topcuoglu (2005)	Cued word recall and recognition	Memorize 48 word pairs presented for 3 secs: 24 neutral-neutral & 24 neutral-threat	17 OCD patients (YBOCS: 22.3) vs. 16 subclinical checkers & 15 controls (MOCI)	OCD patients had poorer recall & recognition for all word pair types = general memory deficit
Irak & Flament (2009)	Focused, Divided & Passive Attention	Attend to words (threat vs. neutral) in a range of conditions. Various recall & Recognition tasks at end.	24 subclinical checkers (>4 checking MOCI) vs. 22 controls (0-1)	Subclinical-checkers had attentional bias and better recall & recognition for threat stimuli compared to controls.

Rubenstein et al. (Exp. 3: 1993)	Cued word recognition	Memorize 60 word pairs presented for 5 secs. Identify study words among 60 lures	20 subclinical checkers vs. 20 controls	Advantage for checkers
De Geus et al. (2007)	California Verbal Learning Task (CVLT)	Recall (short & long term), recognition, semantic clustering, attention.	39 Chronic therapy resistant OCD patients (YBOCS: 27.3) vs. 26 controls	OCD patients had poorer 1st trial recall & learned less words over 5 trials
Savage et al. (2000)	CVLT	Recall (short & long term), recognition, semantic clustering, attention.	33 OCD patients (YBOCS: 19.5) vs. 30 controls	OCD patients poorer recall, recognition, and semantic clustering
Deckersbach et al. (2004)	CVLT	Recall (short & long term), recognition, semantic clustering, attention.	30 OCD patients (YBOCS: 19.3) vs. 30 Bipolar Disorder vs. 30 controls	OCD patients were poorer organizing word lists. OCD's long-delayed free recall mediated by semantic clustering during encoding
Deckersbach et al. (2005)	CVLT	Recall (short & long term), recognition, semantic clustering, attention.	20 OCD patients (YBOCS: 22.5) vs. 20 Bipolar Disorder vs. 20 controls	Improved semantic clustering when directed to group words to category
Zielinski et al. (1991)	CVLT	Recall (short & long term), recognition, semantic clustering, attention.	OCD patients (DSM-III-R/MOCI) vs. controls	OCD patients poorer only on intrusions measure
Segalas et al. (2008)	Spain-Compluten ase Verbal Learning Task (modified CVLT)	Recall (short & long term), recognition, semantic clustering, attention.	50 OCD patients (YBOCS: 20.2) vs. 50 controls	OCD patients poorer recall, recognition not moderated by org strategies
Cabrera et al. (2001)	Complex sentences	Content extraction and recognition	21 OCD patients (DSM-IV) vs. 21 controls	OCD patients poor semantic integration no difference in recognition
Sawamura et al. (2005)	Modified version of Iddon et al's (1998) verbal strategy task	Recall of 20 words presented for 1 min. Recognize these 20 words among 20 distractors. Semantic categorization.	16 OCD patients (YBOCS: 14.6) vs. 16 controls (MOCI-J)	OCD patients had poorer recall & recognition. Slower to semantically categorise words.

Sher, Mann, and Frost (1984) examined a range of verbal (and visuospatial) memory tests but only found verbal deficits for checkers in the Logical Memory subtest of the Wechsler Memory Scale (WMS; Wechsler & Stone, 1945). A short story is read to the participant with recall occurring immediately and then after 30 min. This is one of the earliest studies to highlight the importance of encoding impairments (i.e., in organizing meaningful episodic information) which we propose would occur in the EB (failure of *E* to reduce *B* and *L*) and so explain checker's poorer memory.

Tuna, Tekcan, and Topcuoglu (2005) tested recall and recognition for neutral-

neutral word pairs (e.g., "shirt"-"book") and neutral-threat word pairs (e.g., "music"-"fire"). OCD patients had poorer recall and recognition than subclinical checkers and controls for both neutral and threat-relevant stimuli, which was taken as evidence of a general mnestic deficit not influenced by memory task (recall vs. recognition) or emotional valence (neutral vs. contamination vs. threat). The performance advantage of subclinical checkers for threatening words over neutral was also observed in a study that used three attentional tasks (focused, divided, and passive) that measured recall and recognition memory (Irak & Flament, 2009). The stability of this effect was further substantiated by Rubenstein et al. (Exp.3: 1993) who reported a similar advantage for checkers in word-pair recall and recognition. Revealingly, in the same study, checkers had impaired memory for actions (Exp. 1A; discussed below in deficient visuospatial memory section 6.6.3.), leading the authors to conclude that differences in schematic organization may have differentiated their memory performance from controls. We argue that word-pair and action tasks likely stressed different cognitive resources: simple rehearsal within the phonological loop vs. visuospatial maintenance involving executive organization, complex binding, and high load. Therefore, in these experiments, checkers' perseveration/attentional biases may provide a memory advantage (vs. OCD patients; Tuna et al., 2005 or controls; Irak & Flament, 2009; Rubenstein et al., 1993) for stimuli that have a low classification score across the *EBL* dimensions, i.e., over-rehearsal increases the strength of words maintained and subsequently retrieved from memory. This is in agreement with the evolutionary basis of OCD, where OCD can be imagined on the extreme end of a continuum of fitness-promoting and/or avoidance strategies (Bruene, 2006). However, as observed in OCD generally and this EBL system this cognitive style may cause more harm than intended good.

A frequent measure of verbal memory and learning in OCD is the California Verbal Learning Test (CVLT; Delis et al., 1988). The CVLT is usually administered in the following manner. First, 16 words are presented orally for 5 trials with free recall occurring after each trial. An interference list is presented after the 5^{th} trial. Second, a test of short- and long-delayed (20/30 min) free recall is administered. Third, a delayed recognition test requiring participants to identify previously presented words among distractors. As a result the CVLT measures: (1) attention and WM (recall after first trial), (2) short and long term free recall, (3) semantic clustering (ability to categorize words over trials 1-5), and (4) recognition. De Geus et al. (2007) reported reduced trial-1 recall accuracy for therapy resistant OCD patients relative to controls, no differences were observed for trials 2 through 5 indicating intact verbal memory capacity. Trial 1 is more a measure of attention (immediate span) than memory *per se* and as such, group differences are attributable to an inability to correctly attend to each word. The consistency of this impairment across studies (e.g., Deckersbach et al., 2004; Savage et al., 2000; Segalas et al., 2008) indicates that poor initial attention is a stable deficit in OCD CVLT performance. Savage et al. (2000) reported that OCD patients: (1) memorized less information during encoding (trial 1), (2) used less efficient organizational strategies, and (3) had no deficit in capacity for verbal information over short and long delays. Indeed, when given category cues, OCD patients showed a disproportionate improvement in long-delayed recall where performance was now normal, a pattern also observed by Deckerbach et al. (2005). However, it is important to note that several CVLT studies (Deckersbach et al., 2004; Segalas et al., 2008; Zielinski, Taylor, & Juzwin, 1991) and two using complex verbal material (Cabrera, McNally, & Savage, 2001; Sawamura et al., 2005) have reported similar, additional, and different performance profiles for OCD (see Table 7 for more details).

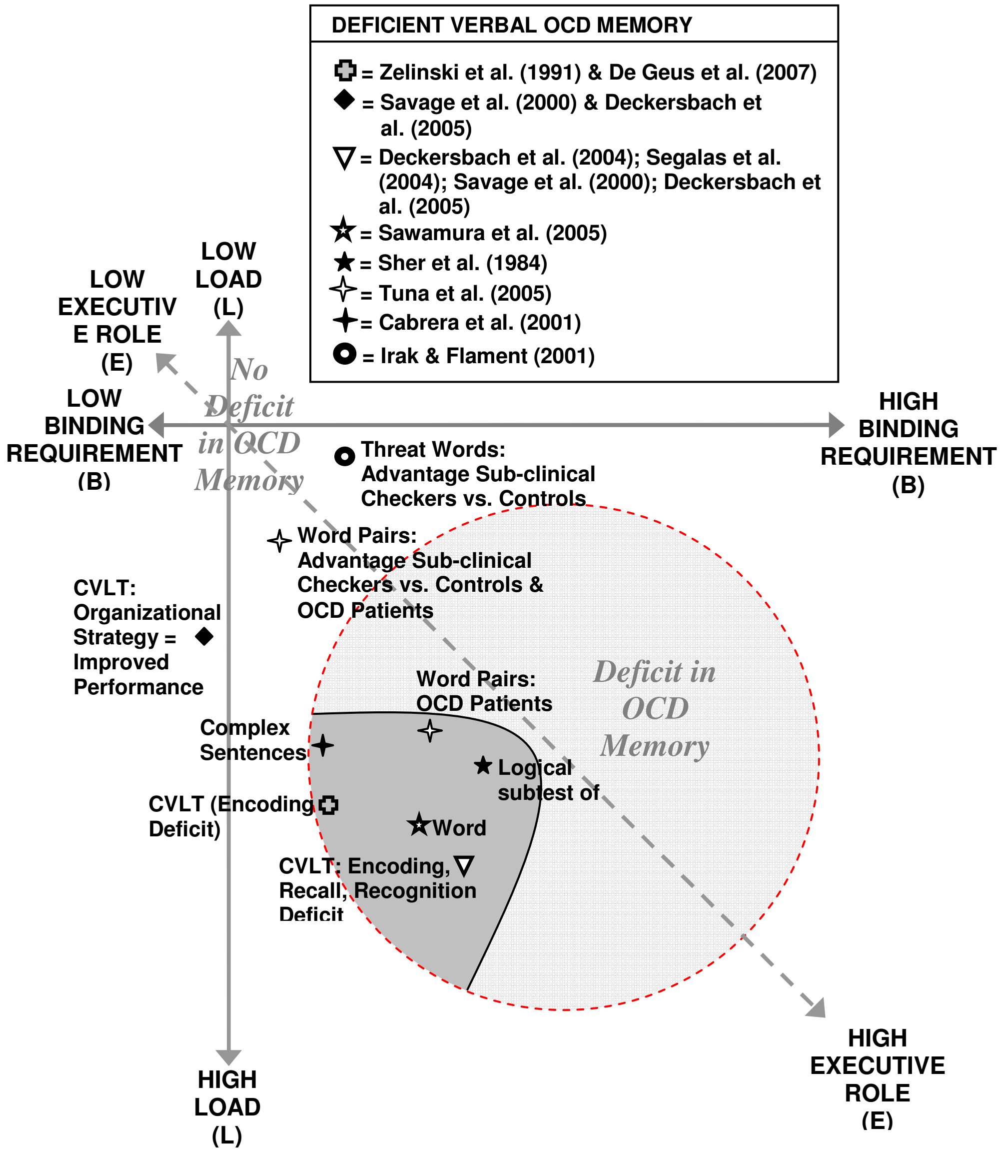

Figure 30. Positioning of deficient verbal memory studies within the *EBL* Classification. The scale has been adjusted to allow clearer representation of verbal memory studies. Observe that verbal memory impairments cluster around inefficient executive functioning (*E*) and memory load (*L*) as proposed in the distinction we draw between verbal and visuospatial memory impairments in OCD (see fig. 28A vs. 28B).

Summary: Deficient verbal memory in OCD

Generally, OCD deficits in verbal memory occur when the task benefits from some form of input organization, which was evident in story recall (Sher et al., 1984), word list categorisation (Sawamura et al., 2005), and CVLT performance (e.g., Savage et al., 2000). We saw that in the CVLT task impairment was influenced by the specific cognitive profile of each OCD group: Efficient or inefficient executive functioning (*E*) will increase or decrease memory load (*L*), respectively (see fig. 28A), which influences the magnitude and type (e.g., trial-1 vs. semantic clustering) of memory impairment observed. OCD patients compared to sub-clinical checkers showed impaired and enhanced word-pair memory performance, respectively (Tuna, Tekcan, & Topcuoglu, 2005; Irak & Flament, 2009), which leads us to propose that executive functioning differs between these two groups. For example, sub-clinical checkers may over-rehearse (e.g., Tuna, Tekcan, & Topcuoglu, 2005) and/or have attentional biases (e.g., Irak & Flament, 2009) which strengthen the representation of simple stimuli in memory (see fig. 30). In addition, co-morbidities in patients (e.g. depression) might amplify their executive deficits compared to subclinical checkers (cf. Moritz et al., 2003; Rampacher et al., 2010).

6.6. Visuospatial Memory

Visuospatial memory impairments are most commonly observed in OCD, however, when visuospatial tasks are low on all *EBL* dimensions then no impairments in memory should occur. In addition, we expect studies that varied load to report intact and deficient OCD memory for lower and higher load levels, respectively, which we attribute to executive functioning failing to meet increasing task demands.

6.6.1. Intact visuospatial memory in OCD

Studies that score low on the *EBL* dimensions invariably report intact visuospatial memory as they are: (1) within visuospatial sketchpad capacity (i.e., low memory load), (2) low executive requirements (successful maintenance requires low attention and/or organization if undisturbed (e.g., Kessler & Kiefer, 2005) and (3) low binding requirement (see Table 8).

Table 8. Studies reporting intact non-verbal memory in OCD.

Authors	Method	Task Requirements	Groups Compared	Behavioural Findings
Henseler et al. (2008)	Delayed Match to Sample (WM task)	Encode 5 x 5 matrix with 4 squares filled. Indicate if a probe is correctly located	11 OCD patients (YBOCS: 21.0) vs. 11 controls	No differences
Ciesielski et al. (2007)	Delayed Match to Sample (WM task)	Encode 3 x 3 matrix with 2 squares filled. Choose correct probe from 2 choices	8 OCD patients (YBOCS: 25.6) vs. 8 controls	No difference for simple DMTS or distractor DMTS
Roth et al.	Self-Ordered	Self-paced abstract	30 OCD patients	No differences

(2004)	Pointing Task	selection task	(YBOCS: 28.1) vs. 24 controls	
Martin et al. (1995)	Self-paced selection of drawings of animals and nonsense objects	Always select a different animal/object/word	18 OCD patients (DCM-III-R) vs. 18 controls	No differences

Two simple delayed-match-to-sample (DMTS) tasks failed to report any difference in OCD memory performance relative to controls (Henseler et al., 2008; Ciesielski et al., 2007) due to low scores on all *EBL* dimensions. Roth et al. (2004) mainly used the Self-Ordered Pointing Task (SOPT; Petrides & Milner, 1982) as a measure for executive WM requiring the ability to generate and monitor a sequence of responses. On each page of a booklet with 12 pages several abstract designs were presented. On page 1, participants were asked to select a design by pointing at it, then to turn to page 2 and point to a different design until they completed the full 12 page booklet. Participants were instructed not to choose the same design more than once and not to choose designs in the same spatial location on two consecutive pages (designs and locations were randmized across pages). There were no differences between OCD patients and controls in terms of errors, time taken, likelihood of using an organizational strategy, and specific organizational strategy used. One potential explanation for these null findings is the observation that on average all participants took approximately 20 seconds per page which may have been sufficient to allow OCD patients to compensate for extant executive dysfunction (see also Martin et al., 1995).

Summary of intact visuospatial memory

Low load tasks (e.g., Henseler et al., 2008; Ciesielski et al., 2007; Rotge et al., 2008) with minimal executive, binding and load requirements are unlikely to produce OCD memory deficits. In addition, self-pacing appears to prevent performance deficits in OCD patients (e.g., Martin et al., 1995; Roth et al., 2004) by allowing individuals to attain higher threshold of certainty or to satisfy their obsessions and/or compulsions to some degree (see fig. 31). Following this logic, limiting decision-making time curtails some or all of these strategies which may put OCD patients' central executive sufficiently under pressure to impair their memory.

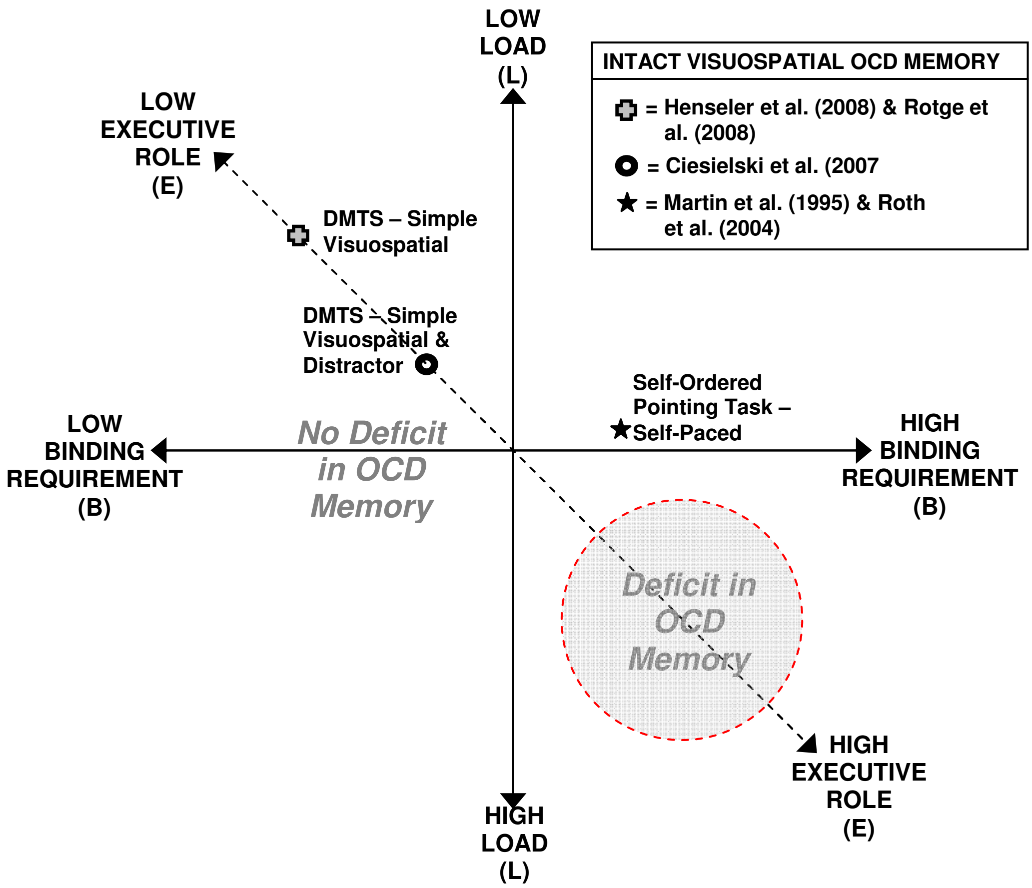

Figure 31. Positioning of intact verbal memory studies within the *EBL* Classification.

6.6.2. Intact and deficient visuospatial memory within the same study

The following are examples of intact and deficient visuospatial memory within the same study (see Table 9) they highlight the delicate manner in which executive-functioning, binding complexity and load interact to negate or produce visuospatial memory deficits.

Table 9. Studies reporting intact and deficient non-verbal memory in OCD. Please observe that we include a study by Morein-Zamir et al., (2010) * in this section which failed to show OCD spatial memory impairment in the SWM task (i.e., as used by Purcell et al. (1998a, 1998b), as they did report memory impairment in another spatial task (Paired Association Learning).

Authors	Method	Task Requirements	Groups Compared	Behavioural Findings
Van der Wee et al. (2003)	*n*-back (0,1,2,3 load levels)	Continual monitoring and updating of information in WM	11 OCD patients (YBOCS: 25.8) vs. 11 matched controls	No diff at 0, 1, 2 *n*-back. Diff for 3 *n*-back task
Van der Wee et al. (2007)	*n*-back (0,1,2,3 load levels). Before &	Continual monitoring and updating of information in WM	14 psychotropic free OCD patients. 7 Responders (YBOCS: 24.4) vs.	Improvement at 3-back level only for responders

	after pharmacological intervention		7 Non-Responders (24.7)	
Purcell et al. (1998a)	Spatial WM	Spatial search task in spatial locations.	23 OCD patients (YBOCS: 22.4) vs. 23 matched controls	No diff for 2, 3, 4 (low difficulty). Diff for 6 & 8 (high difficulty)
Purcell et al. (1998b)	Spatial WM	Spatial search task in spatial locations.	30 OCD patients (YBOCS: 24.1) vs. 30 matched controls	No diff for 2, 3, 4 (low difficulty). Diff for 6 & 8 (high difficulty)
Morein-Zamir et al. (2010) *	Spatial WM	Spatial search task in spatial locations.	18 OCD patients (DSM-IV-TR) vs. 18 matched controls	No diff on between-search errors or strategy scores
Morein-Zamir et al. (2010) *	Paired Association learning	Learning associations between geometric patterns and spatial locations	18 OCD patients (DSM-IV-TR) vs. 18 matched controls	No diff level 3 (low difficulty) Diff for 6 & 8 (high difficulty)
Zielinski et al. (1991)	Corsi Block-Tapping Test	Spatial span & number of correct repeated sequences	OCD patients (DSM-III-R/MOCI) vs. controls	OCD patients poorer span & correct sequences
Zitterl et al. (2001)	intermediate (Lern- und Gedachtnistest; LGT-3) and immediate (Corsi Block-Tapping Test)	Spatial span & number of correct repeated sequences	27 non-depressed OCD patients (YBOCS: >16) vs. 27 controls	OCD patients poorer on intermediate and immediate measures
Moritz et al. (2003)	Corsi Block-Tapping Test	Spatial span & number of correct repeated sequences	32 OCD patients (YBOCS: 23.52) vs. 20 controls.	OCD patients poorer at corsi block tapping
Boldrini et al. (2005)	Corsi Block-Tapping Test	Span and Supraspan	25 OCD patients (YBOCS: 22.7) vs. 15 Panic vs. 15 Controls	OCD patients impaired on Span and Supra span

Van der Wee et al. (2003) used a spatial variant of the *n*-back WM task with four levels of load. OCD individuals and controls had equivalent performance for 0-, 1-, and 2-back indicating that OCD spatial WM capacity was intact. It was only at the 3-back load level that patients with OCD significantly differed from controls with errors of 48% *versus* 25%, respectively. Further, van der Wee et al. (2007) reported that OCD patients which responded favourably to pharmacological treatment showed improvement only in their 3-back performance. Thus, poor OCD 3-back performance is attributable to dysfunctional executive control (*E*) failing to provide efficient strategies in the face of attention-dependent multimodal bindings (*B*) and increased memory load (*L*) (see fig. 28B), with improvements in memory likely attributable to improvements in executive functioning at the level of organization and/or suppression.

Stability of OCD impairment at higher load levels is supported across a range of tasks. For example, the Paired Association Learning task (PAL; Sahakian et al., 1988) which required the binding and maintenance of shapes to spatial locations in memory across increasing levels of load and so scored highly in the *EBL* classification system. Morein-Zamir et al. (2010) attributed the impairment of the OCD group (at more demanding load levels 6 and 8) to a dysfunction in nonspatial associative learning. However, they did report intact performance in a test of spatial WM (SWM) at low and high load levels, which was interesting as another group reported impaired OCD performance at higher load levels (see Purcell et al., 1998a, 1998b). Purcell and colleagues observed that OCD patients were more likely to return to a previously searched box at higher load levels (i.e., 6 and 8 boxes), which was indicative of impairment in adopting a systematic search strategy (*E*: organization) and inability to correctly manipulate internal WM representations. Critically we suggest that absence (Morein-Zamir et al., 2010) and presence (Purcell et al., 1998a, 1998b) of OCD memory impairment in this SWM task suggest that the specificity of executive dysfunction (*E*) between OCD-groups may differ between studies. Further evidence for OCD memory impairment at higher (not lower) load levels is supported by their performance on the Corsi block-tapping test (see Table 5: Boldrini et al., 2005; Moritz et al., 2003; Zielinski, Taylor, & Juzwin, 1991; Zitterl et al., 2001).

Summary of intact and deficient visuospatial memory within the same study

In all these tasks (n-back, SWM, PAL, Corsi-block) we saw that increasing load in the SWM domain differentiates OCD patients from controls; it is only when executive functioning is stressed at high loads that the contents of memory become unmanageable, i.e., inefficient executive functioning (*E*) fails to reduce memory load (*L*) (see fig. 32). Van der Wee et al. (2007) proposed that OCD performance on the *n*-back was state dependent, as treatment responders showed significantly less errors in 3-back performance compared to non-responders.

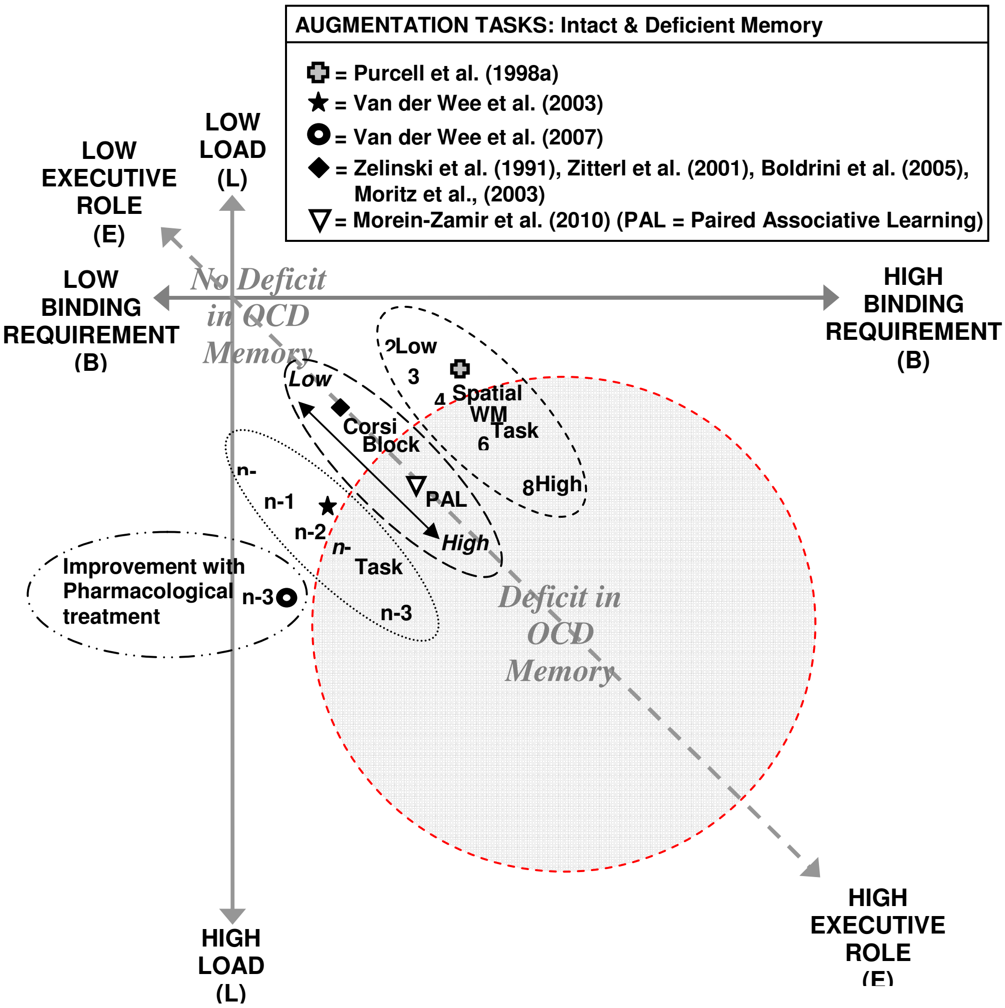

Figure 32. Positioning of intact and deficient visuospatial memory studies within the *EBL* Classification.

6.6.3. Deficient visuospatial memory

Studies that show deficits in visuospatial memory invariably share the following characteristics: (1) they exceed visuospatial sketchpad capacity (>6 items), (2) have high executive requirements, and (3) are high in binding complexity (see Table 10). In essence these are the same characteristics as for the high load conditions in the studies reviewed in the previous section (see fig. 32).

Table 10. Studies reporting deficient non-verbal memory in OCD

Authors	Method	Task Requirements	Groups Compared	Behavioural Findings
OCD MEMORY IMPAIRMENTS IN SINGLE METHODOLOGIES				
Rubenstein et al. (Exp. 1A: 1993)	Write, observe, or perform 90 actions	After completing 90 actions write down all actions they could remember	20 subclinical checkers (MOCI-checking: unknown) vs. 20 controls (MOCI-checking: ≤ 2)	Checkers remembered fewer actions and greater errors vs. controls
Purcell et al. (1998a)	DMTS	Maintain complex visuospatial stimulus and select it from 3 close alternatives	23 OCD patients (YBOCS: 22.39) vs. 23 matched controls	OCD patients poorer DMTS selection vs. controls
Tallis et al. (1999)	Recurring Figures Task	Maintain previously copied abstract figure and recall immediately & after 30 mins	12 OCD patients (primarily checkers: Pauda: 72.6) vs. 12 matched controls	OCD patients poorer than controls on RFT
Zielinski et al. (1991)	Recurring Figures Task	Maintain previously copied abstract figure and recall immediately & after 30 mins	OCD patients (DSM-III-R/MOCI) vs. controls	OCD patients impaired on immediate and delayed components of RFT
Simpson et al. (2006)	Benton Visuospatial Retention Test	View abstract design then recall from memory	15 Comorbid OCD (YBOCS: 26) vs. Current OCD (19.5) vs. History-of-OCD (9.8) vs. Controls (0.34)	OCD patients less correct responses
OCD RCFT PERFORMANCE IMPAIRMENT STUDIES				
Martionot et al. (1990)	Rey Complex Figure Task (RCFT)	Overall memory score and completion time	16 nondepressed OCD patients (MOCI:16.9) vs. 8 controls	OCD patients impaired in memory score and slower
Savage et al. (1999)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	20 OCD patients (YBOCS: 20.9) vs. 20 Controls (0.4)	OCD patients impaired immediate and delayed recall. Immediate recall mediated by org strat during copy.
Savage et al. (2000)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	33 OCD patients (YBOCS: 19.5) vs. 30 Controls	OCD patients impaired immediate recall, copy to immediate recall and copy organization
Deckersbach et al. (2000)	RCFT (Reliability and Validity of Scoring)	Copying of abstract figure, immediate, delayed recall, recognition, and organization	71 OCD Patients (YBOCS: 21.2) vs. 55 Controls	OCD patients impaired in organization, copy accuracy, copy organization.
Segalas et al. (2008)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	50 OCD patients (YBOCS: 20.2) vs. 50 Controls	OCD patients impaired on immediate, delayed recall and recognition
Boldrini et al. (2005)	RCFT	Copying of abstract figure and recall	25 OCD patients (YBOCS: 22.7) vs. 15 Panic vs. 15 Controls	OCD patients impaired on copy and overall recall
Penades et al. (2005)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and	35 OCD patients (YBOCS: 29.3) vs. 33 Controls	OCD patients impaired immediate recall and copy

		organization		organization
Shin et al. (2004)	RCFT	Qualitative analysis of copy, immediate, delayed, recognition and organization	30 OCD patients (MOCI: 14.5) vs. 30 Controls (3.5)	OCD patients impaired immediate recall and copy organization. Qualitative analysis: copy = poorer planning & fragmentation
Rampacher et al. (2010)	RCFT	Copying of abstract figure, delayed recall, and organization	40 OCD patients (YBOCS: 20.9; BDI: 15) vs. 20 Major Depressives (YBOCS: 0; BDI; 16.3) vs. 40 Controls	OCD patients impaired on copy but not organization compared to MDD patients. Only OCD severity correlated with visuospatial organization.
Jang et al. (2010)	RCFT	Copying of abstract figure, delayed recall, and organization	144 OCD patients (YBOCS: 23.1; BDI 17.95; BAI: 19.67) vs. 144 Controls	OCD patients impaired in recall and organization which correlated with obsession/checking and symmetry/ordering dimensions
SPLIT OCD GROUP BY AGE OF ONSET OR PRIMARY SYMPTOM				
Hwang et al. (2007)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	24 early-onset (≤ 17 years: YBOCS: 22.2) OCD vs. 24 late-onset (≥ 21: YBOCS: 23.4) vs. 24 controls	Late-onset impaired on immediate and delayed recall
Roth et al. (2005)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	21 early-onset (≤ 12 years: YBOCS: 23.4) OCD vs. 13 late-onset (≥ 24.8: YBOCS: 23.4) vs. 24 controls	Late-onset impaired on delayed recall
Cha et al. (2008)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	24 checking-OCD (25.4) vs. 23 cleaning-OCD (24.7) vs. 20 controls	Checkers significantly impaired in immediate and delayed recall vs. cleaners and controls. No difference in copy accuracy.
NO OCD RCFT IMPAIRMENTS FOUND				
Simpson et al. (2006)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	15 Comorbid OCD (YBOCS: 26) vs. Current OCD (19.5) vs. History-of-OCD (9.8) vs. Controls (0.34)	OCD patients did not differ from controls on any RCFT measure
Bohne et al. (2005)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	21 OCD patients (YBOCS: 16.9) vs. 23 trichotillomania vs. 26 controls	OCD and TTM did not differ from controls
Moritz et al. (2003)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization – Controlling for depression	32 OCD patients (YBOCS: 23.52) vs. 20 controls.	OCD patients did not differ from controls on any RCFT measure

RECOVERED OCD PATIENTS – RCFT IMPAIRMENTS REMAIN				
Rao et al. (2008)	RCFT	Copying of abstract figure, immediate, delayed recall, recognition, and organization	30 Recovered OCD patients (YBOCS: 2.57) vs. 30 controls (2)	Recovered OCD patients remained impaired on Immediate and Delayed Recall
OCD RCFT THERAPY STUDIES				
Kim et al. (2002)	RCFT-Pharmacological Intervention	Baseline vs. 4 month comparison. Pharmacological intervention.	39 OCD patients (YBOCS at baseline: 25.4) vs. 31 Controls (0.2)	OCD patients immediate and delayed impairments still after 4 months
Roh et al. (2005)	RCFT-Pharmacological Intervention	Baseline vs. 4 month vs. 1 year follow up. Pharmacological intervention.	21 OCD patients (YBOCS at baseline: 26.9) vs. 20 Controls	OCD patients immediate and delayed impairments still after 1 year
Kuelz et al. (2006)	RCFT-Cognitive Behavioral Therapy	Baseline vs. 3 month follow-up. Cognitive-Behavioral Treatment	30 OCD patients (YBOCS at baseline: 24.2) vs. 39 Controls (0.5)	OCD patients immediate and delayed improvements, specific to major responders
Buhlmann et al. (2006)	RCFT-Cognitive Training	Organization training vs. no training	35 OCD patients (YBOCS at baseline: 20.1) vs. 36 Controls	Training improved organization during encoding. Immediate and delayed recall still impaired
Park et al. (2006)	RCFT-Cognitive Training	Before vs. After: Cognitive Training for 5 weeks	Baseline: 15 Treatment OCD patients (YBOCS:21.1) vs. 15 No-treatment OCD (18.7)	Treatment group improved: copy, immediate, delayed, organization and symptoms

Rubenstein et al. (Exp. 1a: 1993) examined sub-clinical checkers' ability to recall if they had written, observed, or performed an action they had heard. They had unlimited time to complete the memory tasks. Subclinical checkers remembered fewer actions (56.2 vs. 66.1), were more likely to confuse whether they had written, observed or performed a given action (1.2 vs. 0.4) and made more errors of commission compared to controls (0.5 vs. 0.1). This shows that checkers are poorer at recalling their own actions in general and deficient in recalling details of their actions specifically. No group differences in a control condition (memory for cartoons) suggests that impairments are a property of actions not memory capacity *per se*. Remembering actions in their situational context taps into the EB deficits in terms of attention-demanding multimodal bindings described in Section 6.4.

In a DMTS task, Purcell et al. (1998b) presented a complex target stimulus (rectangle with different internal arrangements of color and shape) for 4 seconds. The participant then had to select the correct target from three distractors. OCD patients were significantly less accurate than controls (85.11% vs. 90.43%), which is interesting as the DMTS tasks of Henseler et al. (2007) and Ciesielski et al. (2007) failed to report group differences. Overall, accuracy was high for all three studies suggesting low overall load (all>85%). However there are two features of the particular methodology employed by Purcell et al (1998b) that may explain the memory impairment in OCD patients. First, binding requirements were much higher as an arbitrary shape, color and location had to be integrated requiring more executive control during encoding and maintenance than the other two studies. Second, the employed recall probe was more complex with 4 options being presented and where two of these were partially correct (in shape or colour). Thus, the 4 options at recall may have been particularly distracting for OCD patients' already challenged executive control, hence, interfering with correct retrieval. Taken together, executive control was much more challenged during encoding, maintenance and retrieval in the Purcell et al. task, leading to the observed group differences.

Figures Recall, Recurring Figures Task and Benton Visual Retention Task

Tallis, Pratt, and Jamani (1999) reported impaired performance of OCD (primary symptom was checking) patients on two tests of visuospatial memory. First, in the Figures Recall task (Coughlan & Hollows, 1985), where the participant has to copy an abstract line drawing and then recall it immediately and after a delay. Second, in the Recurring Figures Task (RFT; Kimura, 1963), where 20 geometric or irregular nonsense figures are presented for 3 seconds each. After this the participant must identify those 20 cards from 140 in total by classifying each card as 'old' or 'new.' In this latter task performance for OCD patients was poorer overall and they were more likely to identify new stimuli as old (i.e., false positives; see also Zielinski et al., 1991). Increasing symptom severity was associated with poorer overall score and more false positives. In the task similar to the RFT, Simpson et al. (2006) reported attenuated OCD performance on the Benton Visual Retention Task (BVRT; Benton, 1974). We suggest that executive impairments of organization as observed in the Figural Fluency (e.g., Fenger et al., 2005) and Trail Making Tasks (Penades et al., 2005; Roh et al., 2005, Kim et al., 2002) (see Table 5) explain OCD RFT and BVRT performance: poor executive organization (*E*) during encoding reduces the veridicality of memory traces that are maintained in WM and passed into LTM which in turn play a role in symptom severity.

Summary of deficient visuospatial memory

All the aforementioned tasks require extensive executive control within the visuospatial domain which manifested itself in a number of OCD memory impairments. First, checkers were poorer at remembering actions, which by their nature are episodically rich requiring the integration of information from a number of domains, such as, temporal order and spatial location of actions (e.g., Rubenstein et al., Exp 1a: 1993). Second, Purcell and colleagues highlighted that OCD patients were poorer at remembering abstract shapes, their colours and their locations, a task requiring focused attention of (1) shape-colour-location bindings and (2) suppression of distractors that shared features with the target during recall. Third, OCD performance on the FR and RF tasks (Tallis et al., 1999; Zielinski et al., 1991) and BVR (Simpson et al., 2006) tasks indicates that OCD patients have consistent executive deficits which impair their ability to efficiently attend, organize, and actively retain visuospatial information (see fig. 33 and Table 5: Executive Impairments).

Rey Complex Figure Task

The most common measure of visuospatial memory performance in OCD is the Rey Complex Figure Test (RCFT: Osterrieth, 1944). First, participants are presented with the Rey Complex Figure (RCF) that they draw immediately without distraction revealing their ability to copy/encode. Then, distractor tasks are completed and after 3 min they recall the RCF, which provides a measure of immediate recall. Next, more distractor tasks are completed and after 30 min they again re-draw the RCF as a measure of delayed recall. Finally, twenty-four figures are presented and the participant has to identify twelve that belong to the RCF from twelve that do not, serving as a measure of recognition (Segalas et al., 2008). Chiulli et al. (1995) highlighted the functional distinctions of the RCFT: (1) *Copy*: perceptual, visuospatial, and organizational, (2) *Immediate recall*: amount and quality of information encoding, and (3) *Delayed recall*: amount and quality of information stored and retrieved from episodic memory.

Savage et al. (1999; and Deckersbach et al., 2000; Penades et al., 2005; Savage et al., 2000; Segalas et al., 2008; see also Martinot et al., 1990) reported intact copy but impaired immediate and delayed performance in OCD patients. Preserved copy performance and no additional loss of information between the immediate and delayed conditions indicated that memory capacity (see also Penades et al., 2005 who reported intact memory for faces) did not moderate memory performance. Rather, Savage et al. (1999; and Savage et al., 2000; Penades et al., 2005) suggested that poor use of organizational strategies during the copy condition mediated performance in the immediate recall condition. A point supported by Savage et al. (1999) who observed that OCD patients are more likely to attend to details and less likely to shift their attention to larger RCFT components compared to controls (see also Shin et al., 2004). Furthermore, Penades et al. (2005) highlighted that obsessional severity was associated with greater impairments in organizational strategies and immediate recall. This suggests that unnecessary attention to detail (*E*: organization, set-shifting = longer copy times on RCFT; focusing on details over whole) interferes with early encoding (i.e., fragmentation in EB) which impairs memory (*B* and *L*) and possibly plays a role in obsessional symptoms.

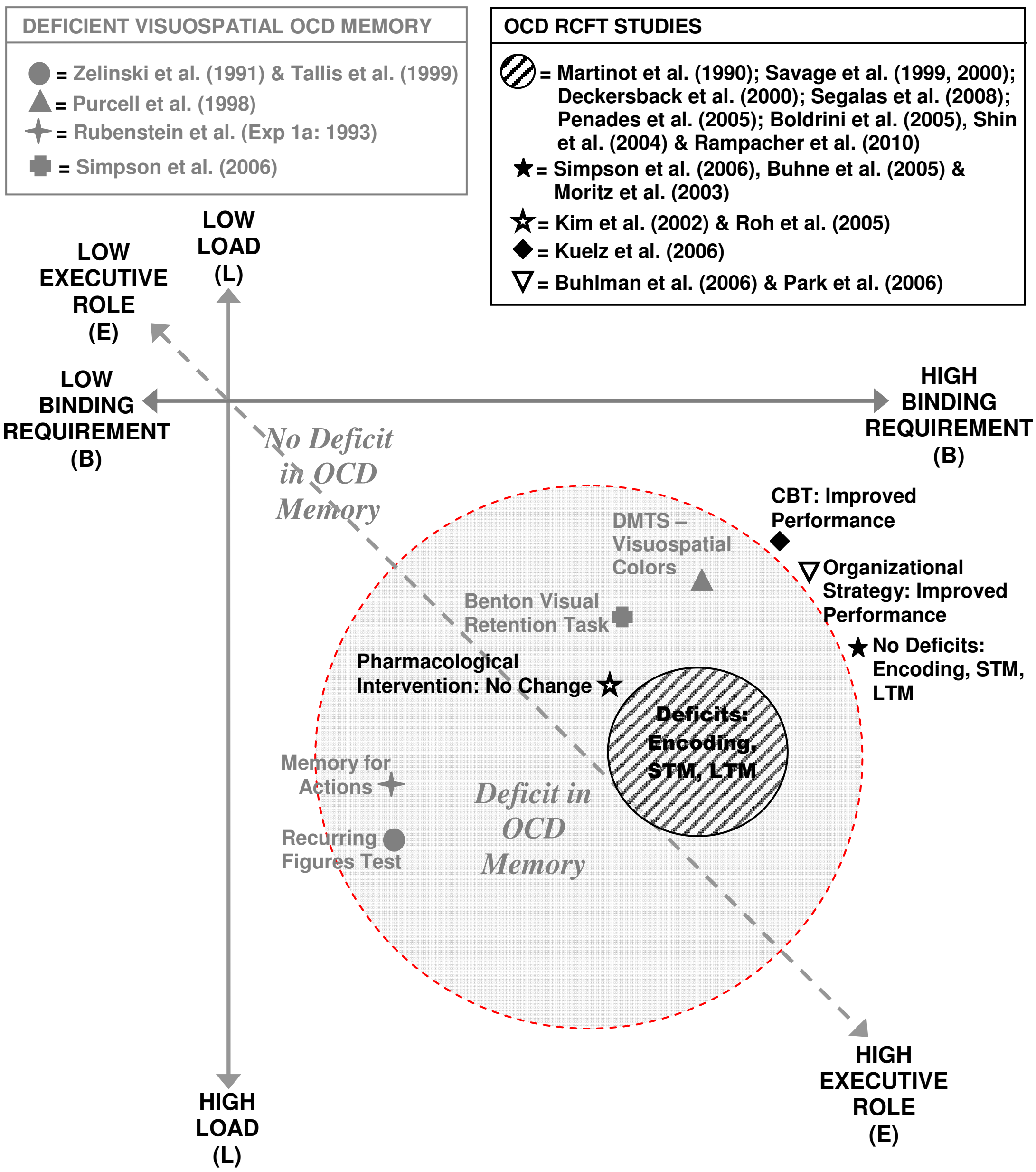

Figure 34. Positioning of deficient visuospatial (light gray text) RCFT (black text) studies within the *EBL* Classification. The scale has been adjusted to allow clearer representation of visuospatial memory studies. To minimise cluttering we have used a shaded area to indicate the dimensional location of the RCFT studies that reported OCD impairments at the level of encoding and/or recall and/or recognition.

No Group Differences in RCFT Performance

Simpson et al. (2006) proposed that depression and/or between study ratio differences in executive dysfunction may explain a failure to report OCD RCFT memory impairments. Both of these fit the current *EBL* explanation in that performance differences between studies are attributable to the respective executive deficits of the OCD group tested: (1) *Depression*: Moritz et al. (2003) reported that OCD patients with higher comorbid depression forgot more RCFT information between copying and delayed recall compared to those with lower depression scores. They concluded that memory dysfunctions in OCD are moderated by comorbid depression a finding also supported by Segalas et al. (2008). However, Rampacher et al. (2010) proposed that organizational impairments were specific to OCD and not to major depressive disorders but did concede that depression may aggravate existing deficits in OCD. (2) *Sub-group Ratios*: Cha et al. (2008) found that a predominantly checking OCD subgroup had poorer immediate and delayed recall compared to cleaners and controls (also observed by Jang et al., 2010), which conforms to our notion of checking compulsions as the primary source of executive deficits. In sum, a specific type of executive dysfunction is required to observe a memory impairment, one that is predominant in one OCD sub-group (checkers) but generally absent in another (cleaners), which may be aggravated by comorbid depression, and possibly influenced by age of onset (Hwang et al., 2007; Roth et al., 2005).

RCFT and Pharmacological and Psychological Interventions

Kim et al. (2002) examined OCD patients on the RCFT (among other tests) before and after a 4-month period of pharmacological treatment. At baseline OCD patients had similar copy- but impaired immediate and delayed recall compared to controls. Despite a significant improvement of immediate recall from baseline to follow-up, they remained significantly impaired compared to controls (see also Rao et al., 2008; Roh et al., 2005). These studies indicate that certain executive and non-verbal deficits are stable and possibly candidate endophenotype markers for OCD (see Bannon et al., 2006; Chamberlain et al., 2005; Rao et al., 2008) resisting pharmacological treatment. Psychological interventions which either implicitly (i.e., cognitive-behavior therapy; Kuelz et al., 2006) or explicitly (i.e., cognitive retraining; Buhlmann et al., 2006; Park et al., 2006) targeted organizational strategies have been associated with improvements on RCFT memory performance and obsessional severity in OCD (i.e., Park et al., 2006). This highlights that not only is executive efficiency (E) malleable to intervention by improving how patients encode (integrated B = low L) information in memory (see fig. 28B) (see also Buhlmann et al., 2006) but it can also attenuate symptom severity.

Summary of RCFT OCD performance

The RCFT is a task with the following *EBL* requirements that make OCD deficits very likely: (1) ***Executive-Functioning***: For the RCFT, OCD patients show consistent executive impairments (*E*) in: (1) organization during early encoding, (2) attention to details over the whole and (3) shifting cognitive set from details to the whole. A failure to reveal OCD impairments on the RCFT is likely due to the tested OCD group not having a sufficient number of executive impaired patients, e.g., more cleaners than checkers (see Cha et al., 2008). (2) ***Binding Complexity***: successful memory of multiple geometric shapes relies on binding. This occurs at the level of within-object binding (i.e., sides of triangle in bottom left corner) and between-object binding, where veridicality depends on the correct binding of parts in space relative to other parts (i.e., position of circle with 3 dots within triangle). Thus, poor *executive functioning* interferes with the veridicality of multiple RCF bindings (*B*) in encoding, WM maintenance and LTM. (3) ***Load***: load in the RCFT depends on the *executive efficiency* and *binding complexity*, in other words, the ability to chunk the complex figure into manageable sub-parts. For OCD patients, executive impairments (*E*) increase the load (*L*) and the binding complexity (*B*) of the RCF in memory (see fig. 28B).

6.7. Comparing EBL system to other models in the OCD literature

The *EBL* classification system allowed us to explain, in a unified manner, how executive impairments observed in OCD/checking tend to impair memory when the EB is extensively relied upon. However, we are aware that our *EBL* classification system is primarily cognitive in nature, which poses the question: How does it relate to alternative and more phenomenogical explanations of OCD symptoms in general and of memory impairments in particular?

Salkovskis (1999) provided one of the most influential models of OCD suggesting an integrated relationship between a number of variables. In the most general sense, this model saw early experiences and critical incidents as primers for the development of faulty assumptions and general beliefs. In turn, this motivates intrusive thoughts, images, urges and doubt which induce a misinterpretation of the personal significance of these intrusions. This misinterpretation is then maintained by an array of factors such as attention and reasoning biases, mood changes, counterproductive safety strategies, and neutralising actions. These then feed back into the maintenance and shaping of existent and future intrusive thoughts. Within this phenomenological model of OCD the cognitive *EBL* factors we propose fall into the category of 'attention and reasoning biases', while our account exactly specifies the executive mechanisms that have distractibility/biases as origin and memory impairment as effect. Compared to Salkovskis' model, we argue for a more direct relationship between executive-memory impairments (as understood in the *EBL* system) and the content of obsessional thinking. The findings that executive functioning (i.e., *'E'*: organization) was associated with memory performance (for visuospatial stimuli high in *'B'*, see: Penades et al., 2005; and *'L'*, see: van der Wee et al., 2007) and severity of symptoms in OCD supports this assertion (see Tallis et al., 1999; Park et al., 2006). We suggest that critical incidents/early experiences/personal dispositions likely prime executive/attentional impairments to become operant when faced with an internal and/or external stimulus/intrusion associated with the original incident. For example, a childhood incident of burning oneself with an iron may manifest subsequently as an attentional bias to irons and/or checking that they are 'OFF.'

The role of inflated personal responsibility (i.e., preventing harms to others) has been identified as important in models of checking and impaired memory (Rachman, 2002; Rachman et al., 1995). In the simplest interpretation, Rachman (2002) proposed that responsibility influences perceptions of harm, increasing anxiety and neutralising checking attempts. However, checking only serves to increase responsibility and impair memory, which leads checkers to believe that their behaviours are out of control. A likely consequence would be increased attention to aspects of a memory representation which are deemed relevant or possibly neutralising to the perceived responsibility/threat. However, as we saw in our work (Exp. 1 of Harkin, Rutherford, & Kessler, 2011) and others' (e.g., Savage et al., 1999), this could result in a narrow focus on specific stimulus details or deficient suppression of distracting thoughts/stimuli, which in any case comes at a cost for memory accuracy.

Van den Hout and Kindt (2003a) validated their OCD-memory model using the remember/know distinction. They showed that repetitively checking the same stimulus resulted in a shift in the nature of their memory recollections from being detailed and vivid ('remember' judgment) to being hazy, indefinite and unclear ('know' judgment). While the authors reported the outcome of checking, the exact mechanism of memory changes was not stated. A more specific indication of the mechanism underlying checking-related memory impairment was revealed by Radomsky and Alcolado (2010). They asked participants to mentally check ("…imagine your hand manipulating the knobs"; p.347) and then recall "Which three knobs did you check on the last trial?" (p.347). Those who engaged in mental checking were significantly less accurate than those who did not mentally check. The unnecessary mental manipulation and increased complexity (i.e., imagining your hand when it is not needed) caused by mental checking (*E*) likely interferes with the veridicality of knob-to-stove bindings (high in '*B*') maintained in the EB.

More specifically, Ferreri, Lapp, and Peretti (2011) proposed that cognitive dysfunction in OCD (and in anxiety disorders in general) could be classified into four domains: (1) executive functioning (primarily attention), (2) memory (WM, episodic, autobiographical), (3) maladaptive cognitions (thoughts and beliefs), and (4) metacognitions (thoughts and beliefs about thoughts and beliefs). We suggest that our *EBL* system helps integrate the first two domains: primary executive dysfunction results in secondary memory impairment. In turn, we have previously proposed (Harkin & Kessler, 2009) that self-awareness (metacognition) of repeated loss of accuracy may decrease confidence in memory and increase the likelihood and strength of misleading intrusive thoughts (maladaptive cognitions) which would then be harder to ignore. This was supported by a recent study (Harkin, Rutherford, &

Kessler, 2011), where we found a metacognitive deficit specific to high checkers (i.e., a dissociation between accuracy and confidence in a baseline condition). We do accept that the direction of causality between memory and metacognition is intricate and likely differs from patient to patients, i.e., poor memory results in reduced confidence in those memories or alternatively poor confidence motivates checking, which we have seen impairs memory. Thus, we argue that our *EBL* system not only complements the models of Salkoskis (1999), Rachman (2002; Rachman et al., 1995) and van den Hout and Kindt (2003a) and the classification proposed by Ferreri et al. (2011) but also provides a more specific and stringent cognitive framework for explaining and predicting executive-memory impairments in OCD.

6.8. Limitations of the EBL classification system

We highlight the following limitations to the *EBL* classification system. First, it is a good fit for OCD patients with prominent checking cognitions/behaviors, but appears not to describe symptoms such as cleaning or hoarding. We propose that if the *EBL* factors are sufficiently stressed (as discussed above) then memory impairment could be observed in symptoms other than checking. However, we do concur that due to the specific impairments (i.e., inhibition; Omori et al., 2007) and cognitive habits (i.e., iteratively checking the contents of memory, perseveration) associated with checking, this symptom is the most likely to affect executive functions that lie at the core of the *EBL* system. Second, we do not make many solid conclusions regarding the relationship between the *EBL* and confidence in memory. Whereby, poor confidence may be a general factor – tightly linked to anxiety – which increases the likelihood that executive dysfunction will impair memory for tasks which load high on *B* and/or *L* dimensions. Alternatively or in addition, executive-memory impairment may result in poorer memory confidence which then motivates detrimental checking and/or obsessional thinking. Third, we make no comment on the reviewed studies with respect to general cognitive abilities like intelligence. However, we agree with the extensive OCD literature review of Kuelz et al. (2004) – which covered many of the papers we examined – who stated that: “It is well established today that general intelligence is not affected in OCD" (p. 223). Finally, these limitations highlight the necessity for future experimental research to see if the *EBL* system does accurately predict where memory impairment will and will not occur.

6.9. Conclusions

This review reconciles inconsistent findings as to memory deficits in OCD by suggesting that the classic view in terms of modality-specific (verbal vs. visuospatial) deficits and/or general capacity issues might not be the optimal way of conceiving of the problem, while we propose to follow and extend the more recent argument that OCD memory impairments are secondary to executive dysfunction. Using our research as a basis, we argue that memory impairments occur when: (1) a task taps into executive deficits of OCD/checkers, and (2) accurate memory performance requires attention-dependent maintenance of bindings and/or the task has high encoding load. Thus, executive dysfunction interferes with the accurate maintenance of complex bindings and/or fails to reduce load, impairing memory. From this we propose the *EBL* classification system, which comprises executive functioning (E), binding complexity (B) and memory load (L) as central dimensions for understanding and predicting OCD memory impairments. This challenges the importance of the modality-specific view, i.e., the visuospatial- vs. verbal-memory distinction, in two important ways. First, impairments are thought to be determined primarily by poor executive functioning (E) and then by the content of the task. Second, visuospatial- compared to verbal stimulus content inherently possesses different resource requirements that are best conceived of as binding- and load-requirements.

In support of this challenge, we reviewed 58 findings across 46 studies. First, we observed that for visuospatial as well as for verbal tasks with low *EBL* scores, no OCD memory impairments were observed compared to controls. Second, tasks that steadily increased load (visuospatial: n-back task) or employed a high inherent load (verbal: CVLT) revealed OCD memory impairment, as the patients' executive deficits failed to match the task demands at higher load levels. Hence, across verbal and visuospatial tasks it is poor executive functioning that cannot cope with increasing cognitive demands that differentiates OCD memory performance from controls. However, we did suggest that default differences in *EBL* scores of verbal compared to visuospatial tasks make OCD memory impairments more likely in the latter (see fig. 28A vs. 28B). Verbal tasks, generally, present verbal information in a format (stories, word lists that benefit from semantic clustering) that are high in load but low in binding complexity. In this case, performance is benefited by efficient executive processes that utilize existing representations in LTM, i.e., chunking according to categories reduces memory load. In contrast, basic visuospatial tasks, especially when random locations are employed, are usually less supported by LTM knowledge, so strategic executive organizing must cope with binding complexity and/or load even at low demands. This increases the number of dimensions (3 in visuospatial, i.e. *EBL*; *vs*. 2 in verbal, i.e. *EL*) where OCD memory impairments can occur, making visuospatial impairments more likely than verbal.

For tasks that are high in binding complexity (memory for actions, Trail-Making Task, Benton Visual Retention Task, Figural Fluency, Recurring Figures Test, Rey Complex Figure Task) consistent OCD impairments were observed across a range of measures. This can be simply surmised as an inability to organize complex visuospatial information in a manner to benefit early encoding, immediate and delayed recall and recognition. For example, in the case of RCFT performance in OCD, poor executive functioning (*E*) fails to reduce the load (*L*) by means of strategic organization, which in turn reduces the veridicality of multiple bindings (*B*) of the RCF representation in memory. Such a representation based on loosely interconnected feature assemblies is not only more difficult to accurately copy and recall than a tightly structured one, but it also places additional strain upon executive processes during maintenance, which are already operating sub-optimally. Further extrapolating these arguments to future studies, tasks that require complex binding of multiple and multimodal features (as in our recent studies) are also likely to tap into OCD-specific deficits due to sub-optimal executive organization of input and deficient 'protection' during maintenance.

The central role of executive dysfunction was further supported by the finding that targeting executive processes in OCD patients with therapeutic intervention not only reduces obsessional symptoms but also improves memory performance. We take this as evidence of a link between executive and memory impairments, anxiety, and the development of obsessions (e.g., doubt and uncertainty; “Did I turn the stove off?”) and neutralizing compulsions (e.g., checking to compensate for poor memory and high anxiety). Finally, we propose that our explanation complements existing OCD models by specifying essential cognitive mechanisms, which will hopefully help guiding future research.

7. Conclusion: Overview, clinical implications, limitations and future research, and contribution to OCD memory research

A summary of the main findings for Experiments 1-9 is provided in Table 11. It highlights the primary experimental manipulations, how high checkers' WM performance faired with respect to the *strong* (high checkers' memory impaired in misleading trials) or *weak* hypothesis (high checkers' memory impaired after misleading and resolvable trials), and the main confidence findings. We review the main findings from our experiments, followed by the possible role of executive-memory impairments in the development and the maintenance of obsessions and compulsions (see fig. 33). We then highlight the limitations of the research and the solutions to these through future research.

7.1. Working memory and inhibitory performance of checkers

The present WM tasks placed an emphasis on the veridical binding of letters-to-locations (Exp. 1-5), 'ON/OFF' states-to-appliances (Exp. 8) and appliances-to-locations (Exp. 9). Accurate memory performance required that attention be allocated to such bindings in a cognitive resource which Baddeley (2000) referred to as the EB. This buffer provided a pragmatic solution to manner in which the cognitive system bound information from different modalities (i.e., a visual letter presented a location in space) into a coherent representation. However, it does come with a cost: Attention (automatic or controlled) is required for the veridical maintenance of bindings in the EB, which implies that interfering with this attention (i.e., away from bindings) will reduce the veridicality of those bindings and impair memory (see fig. 1). Due to differences in the content of the encoding set stimuli (Exp. 1-5: non-threatening letters *versus* Exp. 8 & 9: electrical kitchen appliances) between experiments, we present the results separately for those which employed letters in locations (Exp. 1-5) and ecologically valid stimuli (Exp. 6-9).

7.1.1. Letters in locations: Experiments 1-5

In our original experiments 1, 2 and the extreme meta-comparison we provided support to the *strong hypothesis* as high checkers' WM impairments

Table 11. Overview of the main results for experiments 1 to 9 as divided by the stimuli used (letters vs. ecologically valid stimuli), type of experiment (WM vs. IOR), and key task elements. The results concentrate on WM/IOR performance and confidence of high checkers compared to low checkers. N/A Data = No data was taken with respect to this variable.

Task Stimuli:	**Letters in Locations**						**Ecologically Valid Stimuli**			
Task Type:	**Working Memory**						**Inhibition of Return**		**Working Memory**	
Key Task Elements:	**Simple**			**Colour Added**	**Visuospatial Distractor**	**Eye-Tracker (Similar method to Exp. 1 & 2)**	**Classic IOR Cue**	**Novel 'ON' Cue**	**ON/OFF P2**	**Location P2**
Experiment Number/Type:	**Exp. 1**	**Exp. 2**	**Extreme Comparison**	**Exp. 3**	**Exp. 4**	**Exp. 5**	**Task 6**	**Task 7**	**Exp. 8**	**Exp. 9**
LC check score (mean/SD)	1.11(1.10) [*n*=20]	1.05(1.18) [*n*=20]	0.5(0.71) [*n*=10]	1.74(1.69) [*n*=20]	0.89(1.15) [*n*=20]	0.7(0.9) [*n*=17]	0.31(0.48) [*n*=50]		0.5(0.61) [*n*=20]	0.0(0.0) [*n*=20]
HC check score (mean/SD):	9.53(5.49) [*n*=20]	8.65(3.7) [*n*=20]	15.8(5.32) [*n*=10]	12.57(5.96) [*n*=20]	10.48(5.96) [*n*=20]	12.7(5.8) [*n*=18]	13.78(5.55) [*n*=52]		13.85(4.12) [*n*=20]	13.75(6.16) [*n*=20]
LC vs. HC: Primary Attention/Memory Finding:	HC poorer memory in misleading trials vs. LC	HC poorer memory in misleading trials vs. LC	HC poorer memory in misleading trials vs. LC	HC poorer memory in misleading for correct P2 vs. LC	HC poorer memory for misleading & resolvable trials vs. LC	HC make more fixations during misleading trials at empty locations	HC (and LC) had normal IOR effects	HC attenuated IOR for ON cues vs. LC	HC poorer memory for ON/OFF states vs. LC	HC poorer memory in misleading trials vs. LC
Hypothesis Supported:	Strong	Strong	Strong	Strong	Weak	Strong	Data N/A	Data N/A	Weak	Strong
LC vs. HC: Primary Confidence Finding:	HC meta-cognitive impairment vs. LC. Enhanced in misleading trials	No group difference	No group difference	HC meta-cognitive impairment vs. LC. Moderated by Inter P1	Strong HC meta-cognitive impairment vs. LC	No group difference	Data N/A	Data N/A	HC meta-cognitive impairment vs. LC	Strong HC meta-cognitive impairment vs. LC

were specific to a misleading but not a resolvable context (Harkin & Kessler, 2009). The replication of our findings in Experiment 2 and the extreme group meta-comparison speak to the robustness of our findings. Critically, the only difference between misleading and resolvable trials was the absence or presence of the Probe-1 letter in the encoding set, respectively. When confronted by a misleading mismatch high checkers appear to be unable to ignore it and so unnecessarily and fruitlessly search those letters presently maintained within WM. This assertion is supported by the observation that OCD checkers are poorer at tolerating uncertainty (i.e., misleading probe-1 letter) than OCD non-checkers and controls (Tolin, Woods, & Abramowitz, 2003), and that an inability to tolerate uncertainty is associated with subsequent checking and repeating rituals (Lind & Boschen, 2009; Tolin, Woods, & Abramowitz, 2003). The resultant search and/or competition between a strong, visually present misleading letter to fragile letter-location representations in the EB likely impairs attention directed to those bindings, impairing memory. The extreme group meta-comparison provides evidence that the same misleading specific WM impairment was present in subclinical checkers who scored in the clinical range and that this impairment was numerically larger. This indicates that there may be a relationship between severity of checking symptoms and extent of WM impairment (see Omori et al., 2007). High checkers' performance was intact on no-probe-1 trials proving that basic WM capacity was in this case intact.

Experiments 3 and 4 were a direct attempt to further target and interfere with the fragile bindings maintained within the EB especially in high checkers (Harkin & Kessler, 2011a). Experiment 3 increased the load of the encoding set by adding the additional binding of colour to letters. The intermediate Probe-1 then asked for the colour of a letter which was either misleading or resolvable. The results indicated that we may have induced a degree of checking in all participants which possibly obscured the clear-cut WM impairments observed in the previous experiments. Despite this and in support of the *strong hypothesis*: High checkers had WM impairments in the most difficult memory condition (correctly located Probe-2 letters) in misleading trials. This indicates that the inability of high checkers to ignore misleading trials was still sufficient to result in WM impairment specific to this condition relative to low checkers. Again, no group differences on no-probe-1 trials were observed. Experiment 4 presented a strong visuospatial distractor at a misleading and resolvable location. Therefore, in accord with the *weaker hypothesis* high checkers' WM was impaired in both conditions indicating that a visuospatial distractor generally impaired high but not low checkers' attention to bindings. WM performance in baseline conditions again proved to be intact.

Experiment 5 measured eye movements in a slightly modified version of our original WM task (Exp. 1 and 2). This was a direct attempt to show that checkers' inhibitory impairments for misleading information result in them searching the contents of WM. In line with our *strong hypothesis*, high checkers made more fixations during the presentation of a misleading intermediate Probe-1 compared to low checkers. Further analysis revealed that in misleading trials high checkers fixated longer on empty encoded set locations, in comparison to resolvable trials and low checkers (Harkin, Miellet, & Kessler, subm). This provides evidence that checkers' inhibitory impairments do in fact lead them to check the contents of WM in a manner which is unnecessary (misleading trials) and uninformative (empty locations). Importantly, allowing high checkers to self-terminate the intermediate Probe-1 appears to have reversed their WM impairments which were previously observed when the intermediate probe terminated automatically (Exp. 1-4). These findings are comparable to research which has reported intact WM performance despite abnormal brain functioning in OCD participants (Ciesielski et al., 2007; Henseler et al., 2008). Further, high checkers' intact WM performance across resolvable, misleading and no-probe-1 trials again indicates that basic WM capacity is preserved.

7.1.2. Ecologically valid stimuli: Tasks 6-7 and Experiments 8-9

We then employed ecologically valid stimuli (electrical kitchen appliances) in a novel inhibition of return (IOR; Posner & Snyder, 1975) and WM tasks to address two central limitations identified in the previous experiments. First, while we inferred that high checkers' WM (Exp. 1-4) and eye movements

(Exp. 5) was attributable to their inhibitory impairments, we were aware that this conclusion would be strengthened by explicit evidence of this. Second, despite letters in locations producing robust and replicable results, as stimuli they have limited relevance to the actual symptoms of checkers. Indeed, in our IOR task we showed that while high checkers' inhibitory functioning was intact (Task 6) it was impaired when attention was drawn to a threatening 'ON' cue (Task 7). Thus, an inability to disengage attention from a threatening feature was sufficient to impair otherwise normal inhibitory control (i.e., IOR). In Experiment 8, high checkers were impaired in their ability to correctly recall if an appliance had been 'ON' of 'OFF'. This is consistent with the previous findings from Task 7: Focused attention to threatening states may interfere with the binding of that state to that actual appliance. In contrast, Experiment 9 produced the more classic WM pattern, where high checkers' memory impairment was more focused to the misleading context. The intact no-probe-1 performance of high checkers in Experiment 9 (and Exp. 1-5 generally) provides important evidence against the argument that a basic capacity impairment underlies their general (across resolvable, misleading, no-probe-1 trials) WM impairment for appliance states observed in Experiment 8. Further, a basic impairment in capacity would not have influenced WM capacity in an isolated manner (i.e., Exp. 8 only) but would have impaired WM performance across all conditions and experiments (Exp. 1-7).

7.2. Confidence

Confidence responses revealed a mixed pattern across the present WM experiments (see Table 11). In a manner consistent with a large body of literature (for review see Woods et al., 2002), high checkers have a general task independent impairment in their confidence (Exp. 4 and 9). However, a misleading context appears to further attenuate their already inferior confidence (Exp. 1), perhaps as the result of the uncertainty and checking which arises in this condition (Exp. 5). The complex nature of high checkers confidence is further reflected in Experiment 3, where the removal of no-probe-1 trials from the statistical analysis increased the magnitude of difference between groups, with high checkers having poorer confidence across conditions (resolvable and misleading) compared to low checkers. This also indicates that in this experiment, for no-probe-1 trials, high checkers have comparable confidence to that of low checkers. These divergent findings may be explained by the different type of confidence responses used between experiments. For example, confidence responses were provided in the following manners: (1) Experiments 1-4: on a scale ranging from 1 (totally confident) to 6 (totally not-confident), (2) Experiments 5 and 8: used a binary response option for confident versus not-confident and (3) Experiment 9 used a quantitative scale ranging from 1 (totally not-confident) to 100 (totally confident). Furthermore, Experiments 1-5 and 8 used keyboard responses while Experiment 9 required the participant to make their response by shifting their hand from the keyboard to a mouse. Therefore, the absence and presence of general confidence impairment is more likely due to the use of the binary/keyboard response option in Experiment 8 as compared to the quantitative/mouse option in Experiment 9. In addition, using a 0-100 scale in Experiment 9 provided the option of calculating sensitive confidence-accuracy correlations which were not available with the confidence response options used in the other experiments (1-5 & 8). Therefore, in our future research we will continue to use the 1-100 scale as it is flexible to different statistical designs and more sensitive to between group differences than the other measure employed.

7.2.1. An intricate relationship between working memory performance and confidence

The relationship between WM performance and confidence in high checkers is both delicate and complex. High checkers suffer from inhibitory deficits which in the correct experimental/environmental circumstances impair their memory. They also appear to 'carry-around' a task-independent metacognitive impairment, which potentially primes them to question their memories, actions, and thoughts in relation to stimuli/activities that are concordant with their symptoms (Exp. 5 & 9). An absence of a general impairment in WM capacity (i.e., intact no-probe-1 trials), argues against the idea that poor memory explicitly mediates confidence. Rather, in specific circumstances, high checkers' dysfunctional inhibitory control attenuates a general metacognitive impairment which was reflected in their poorer confidence when preceded by a misleading (Exp. 1) or intermediate probe (i.e., Exp. 3). However, the direction and strength of causality between inhibitory dysfunction and confidence is unclear and highlights an interesting avenue for future research

7.3. Clinical implications of checkers' executive and working memory impairments

From the executive and WM impairments identified in Experiments 1-9 and the EBL (**E**xecutive Functioning, **B**inding Complexity, Memory **L**oad) classification system (see Section 6), we propose a model (see fig. 27) where primary executive dysfunction and secondary memory impairment potentially plays a role in the development and maintenance of obsessive-compulsive symptoms. For example, in Task 7, high checkers were less able to disengage their attention from a threatening 'ON' cue to the detriment of normal inhibitory functioning (Task 6). In a related manner, Experiment 8 showed that high checkers were generally impaired in their ability to accurately recall the state ('ON' or 'OFF) of an electrical kitchen appliance. Combined, the results of Task 7 and Experiment 8 indicate that high checkers suffer from a primary executive dysfunction in disengaging their attention from threatening states which results in secondary memory impairment by impairing state-appliance bindings within the EB. In turn, this poor memory will likely evoke a degree of anxiety and doubt regarding its original status which will increase the likelihood of intrusive obsessions ("Did I turn it OFF?") and neutralizing checking compulsions (cognitive: checking the contents of WM; behavioural: physically checking if it was 'ON' or 'OFF'). This is supported by the finding that mental and physical checking of electrical stoves impaired memory of the actual knobs checked on the last trial (Radomsky & Alcolado, 2010). This suggests that if executive-memory impairment plays a key role in the development of obsessions and compulsions then targeting executive-memory dysfunction should necessarily reduce the frequency and severity of obsessions and compulsions. Indeed, a body of evidence from the anxiety literature indicates that targeting and reducing attentional biases (i.e., executive functioning efficiency) to threat also attenuated anxiety levels (Amir, Najmi, & Morrison, 2009; Eldar & Bar-Haim, 2010; Schmidt et al., 2009).

Furthermore, within this model we highlight three important points regarding executive impairments. Firstly, while inhibition, set-shifting, organization and attention is the primary executive impairments observed in OCD (see Table 4 in EBL) this is by no means an exhaustive list. This is supported by Miyake et al. (2000) who reported that while inhibition, set-shifting and updating were relatively independent constructs, they were interconnected in terms of their unified reliance upon the attentional resources of the central executive. The identification of these executive constructs supports the executive-memory link proposed here, whereby an inability to ignore irrelevant stimuli will potentially reduce the attention allocated to the concurrent updating of information presently maintained in the EB of WM. Secondly, executive impairments do not operate in isolation, for example, dysfunctional organization (whole-object *vs.* parts) will influence attentional focus (broad *vs.* narrow, respectively). Finally, we argue that executive impairments are state-like and situationally dependent compared to obsessions and compulsions which once established become increasingly trait-like and stable in nature. Thus, while executive dysfunction is consistently observed in OCD (e.g., Bannon, Gonsalvez, & Croft, 2008), they only impair memory in a specific combination of EBL scores (see fig. 27, 28A, 28B) when confronted with stimuli/situations which are concordant with their primary symptoms.

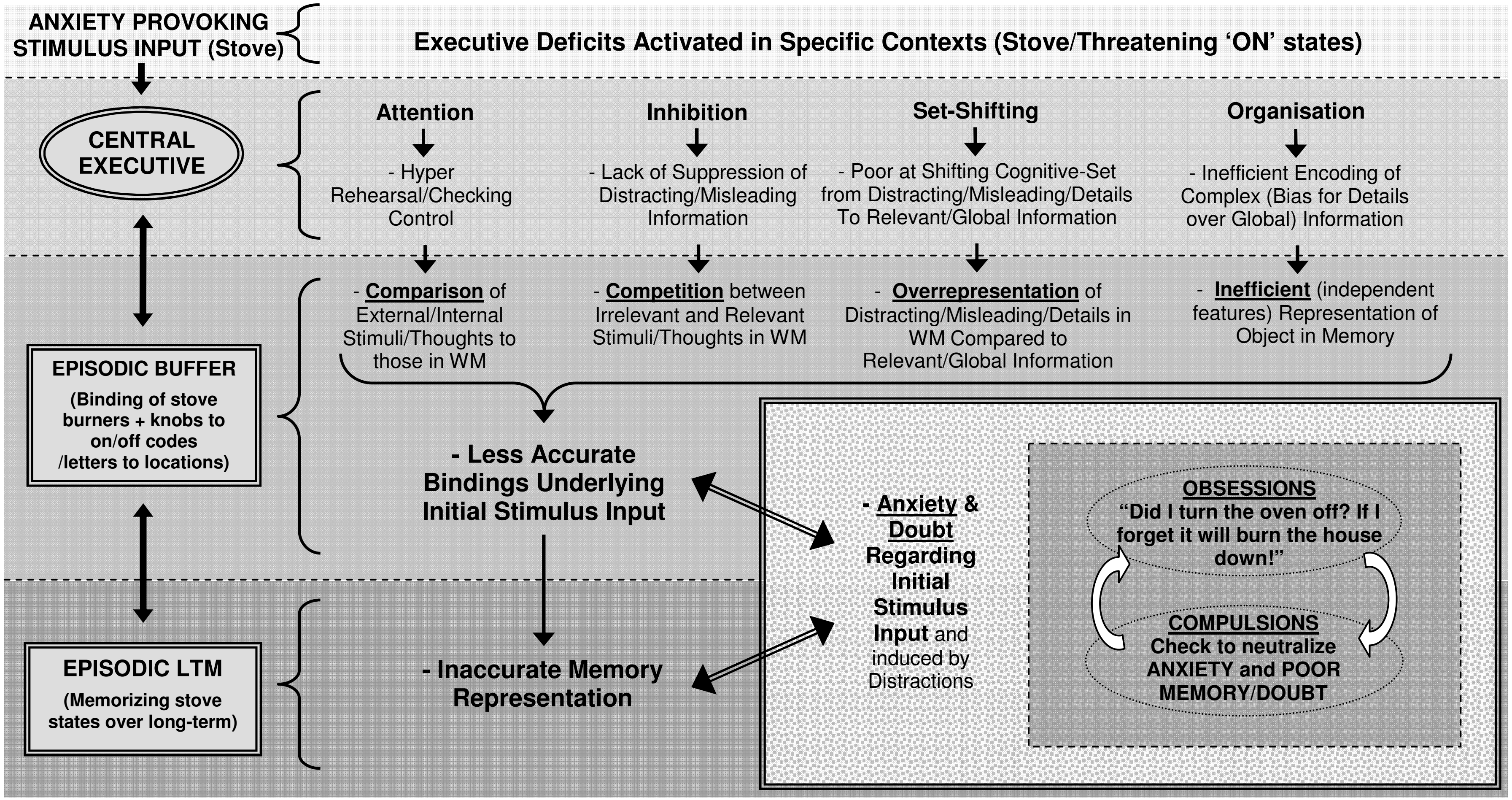

Figure 33. A proposed perspective, based on the original Harkin and Kessler model (2009), on how executive impairments of organization, attention, inhibition and set-shifting interfere with episodic buffer functionality (i.e., binding) impairing memory. From this anxiety and doubt develop with respect to the original memory which increases the likelihood of obsessions (e.g., "Did I turn the iron off?") and subsequent futile compulsions to neutralize anxiety and to overcome poor memory. Further explanations provided in the text.

7.4. Limitations and future research

We now identify the limitations to the present research and, when appropriate, propose solutions to these problems in future research. Firstly, high checkers were selected from a subclinical sample which possibly limits the extent of the conclusions that can be drawn with respect to clinical OCD checkers. However, the extreme group meta-comparison employed high checkers which scored in the clinical range (15.8) on the checking subscale of the VOCI (see Thordarson et al., 2004). In this case, the magnitude of high checkers misleading specific WM impairment was increased, suggesting that the WM performance of subclinical and clinical scoring checkers was quantitatively analogous. Further, in Experiments 3, and 5-9 high checkers had a mean checking score which was in the range of the checking score for OCD patients. The high checking groups in this case were comparable to clinical checking and OCD which further substantiates any conclusions we draw with respect to clinical checking patients. Further, using subclinical checkers from an undergraduate sample likely removes confounding factors such as medication and comorbidity that is likely present in clinical groups (Mataix-Cols et al., 1997; 1999a). Future research can easily address this by employing the present WM and IOR tasks in clinical patient groups. Secondly, across the experiments there was no measurement of anxiety, depression or an independent cognitive index of WM functioning and so we cannot exclude the role of group differences in these areas to the current findings. The first two points are presently addressed in the latest version of our WM task series where we explicitly measure anxiety and depression with the state-trait anxiety inventory (Spielberger et al., 1983) and Beck Depression Inventory (Beck, Steer, & Brown, 1996), respectively. This latter criticism will be addressed in upcoming research where OCD patients will complete the Raven's Progressive Matrices (Raven, Raven, & Court, 2004) as an independent test of WM functioning. Thirdly, a criticism of the first 5 experiments was that letters in locations were not concordant with the primary symptoms of checkers. This was addressed by using ecologically valid electrical kitchen appliances in the subsequent IOR (Task 6 & 7) and WM (Exp. 8 & 9) experiments. A subsequent limitation was the electrical kitchen appliances were not appraised by individual participants and therefore differences in perceived threat of low and high checkers is unknown. However, as high checkers impaired inhibitory functioning was specific to Task 7, this suggests that an unpredictive ‘ON’ cue was sufficiently threatening for high checkers to interfere with otherwise normal inhibitory functioning (Task 6). Indeed, these effects occurred by using stimuli that were general (i.e., present on checking subscale of VOCI) to the symptoms of checking despite them not being idiographically selected or appraised. This suggests allowing for the idiographic selection of visual stimuli relevant to the symptoms of each individual high checker would possibly produce greater IOR and WM impairments than presently observed. Alternatively, we would have expected larger effects if we had used checkers whose concerns were only for electrical appliance. Finally, related to the second and third limitation, the focus on checking limits the conclusions to this subgroup. An interesting avenue of future research would be to see if the WM performance of OCD washers is in the same or opposite direction to that observed for checkers. The body of evidence shows that domain specific checking impairs memory in that domain. Whereas, in contrast, as washers do not have the same cognitive impairments as checkers they may actually show a memory advantage for stimuli (i.e., dirty hands, washing paraphernalia) relevant to their symptoms in a group general or idiographic fashion. If so, this would provide a possible indication of the manner in which the divergent WM performance of checkers compared to washers contributes to the maintenance and development of their unique symptoms. Furthermore, this would highlight the need to separately define checkers and washers in memory experiments which would otherwise define OCD participants in a homogenous manner. This may control for the possibility of producing null findings where checkers’ memory impairments and washers’ enhanced memory cancel each other out.

7.5. Contribution of present work to OCD memory research

Contrary to previous research which has concentrated primarily on the *outcomes* of checking, the present research has proposed and supported the *actual mechanism* of high checkers' memory impairments. Specifically, that bindings maintained within the EB of WM (see Baddeley, 2000) are sensitive to interference, thus when attention from bindings is withdrawn, memory is impaired. Thus, the present work shows for the first time that checkers memory is impaired when the distractor presented is concordant with their inhibitory dysfunctions (Exp. 1-4; Harkin & Kessler, 2009, 2011a). Our eye movement study further revealed that high checkers only make more fixations during misleading trials to empty locations, in comparison to resolvable trials and low checkers (Exp. 5; Harkin, Miellet, & Kessler, subm). This is explicit evidence that checkers' inhibitory impairments result in them checking the contents of WM at locations where no additional task-relevant information is present. Furthermore, checkers appear to suffer from inhibitory (Tasks 7; Harkin & Kessler, in press)) and WM impairments (Exp. 8; Harkin, Rutherford & Kessler, 2011) for the same threatening feature of an electrical kitchen appliance. This indicates that inhibitory impairments for threatening features (ON/OFF states) may interfere with their bindings to appliances in the EB of WM, thus impairing memory. Thus, we show explicitly that checking impairs memory for the very thing (i.e., "Did I turn the iron off?") that theywant to be 100% certain of, which likely motivates further checking and memory impairment. Furthermore, we also observed our classic WM impairment pattern, when high checkers were impaired in recalling the location of an electrical kitchen appliance in a misleading context only compared to low checkers (Exp. 9). Critically, high checkers' intact performance on baseline no-probe-1 trials allows us to exclude a capacity-based explanation of their actual memory impairments. Then using these findings we created a systematic classification system based upon Executive Functioning (**E**), Binding Complexity (**B**) and Memory Load (**L**) (Harkin & Kessler, 2011b). We used this EBL system to clear up an otherwise messy area in OCD memory research, which up until this point has erroneously concentrated upon the visual-verbal distinction and capacity domain as a means of explaining memory performance in OCD. In sum, we use this to highlight the potential role that executive-memory impairments play in the development and maintenance of obsessive-compulsive symptoms and thus provide an explicit target for cognitive interventions to focus upon (see section 7.3. and fig. 33).

Thus, not only does this research indicate the actual mechanism (i.e., bindings within episodic buffer of WM) of memory impairment in checking/OCD but it also provides a research platform (i.e., EBL factors) on which base where we *will* and *will* not observe memory impairments in OCD participants.

References

Abramowitz, J., McKay, D., & Taylor, S. (2005). Special series subtypes of obsessive-compulsive disorder. *Behavior Therapy, 36*(4), 367-369.

Alcolado, G. M., & Radomsky, A. S. (2011). Believe in yourself: Manipulating beliefs about memory causes checking. *Behaviour Research and Therapy, 49*(1), 42-49.

Allen, R. J., Baddeley, A. D., & Hitch, G. J. (2006). Is the binding of visual features in working memory resource-demanding? *Journal of Experimental Psychology-General, 135*(2), 298-313.

Altmann, G. T. M. (2004). Language-mediated eye movements in the absence of a visual world: the 'blank screen paradigm'. *Cognition, 93*(2), B79-B87.

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive-compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157.

Antony, M. M., Downie, F., & Swinson, R. P. (1998). Diagnostic issues and epidemiology in obsessive compulsive disorder. In Richard P. Swinson, Martin M. Antony, S. Rachman & M. A. Richter (Eds.), *Obsessive-compulsive disorder: Theory, research, and treatment.* (pp. pp3-32.). New York: Guilford Press. .

Awh, E., & Jonides, J. (2001). Overlapping mechanisms of attention and spatial working memory. *Trends in Cognitive Sciences, 5*(3), 119-126.

Baars, B. J. (2002). The conscious access hypothesis: origins and recent evidence. *Trends Cogn Sci, 6*(1), 47-52.

Baddeley, A. (1966). Short-term memory for word sequences as a function of acoustic, semantic and formal similarity. *Quarterly Journal of Experimental Psychology, 18*, 362-365.

Baddeley, A. (1986). *Working Memory.* Oxford, England: Clarendon Press.

Baddeley, A. (2000). The episodic buffer: a new component of working memory? *Trends Cogn Sci, 4*(11), 417-423.

Baddeley, A. (2003). Working memory: looking back and looking forward. *Nat Rev Neurosci, 4*(10), 829-839.

Baddeley, A., & Hitch, G. J. (1974). Working Memory. In G. A. Bower (Ed.), *The Psychology of Learning and Motivation* (pp. pp47-89).

Baddeley, A., Papagno, C., & Vallar, G. (1988). When Long-Term Learning Depends on Short-Term Storage. *Journal of Memory and Language, 27*(5), 586-595.

Baddeley, A., Vallar, G., & Wilson, B. (1987). Sentence Comprehension and Phonological Memory - Some Neuropsychological Evidence. *Attention and Performance*(12), 509-529.

Bannon, S., Gonsalvez, C. J., & Croft, R. J. (2008). Processing impairments in OCD: it is more than inhibition! *Behaviour Research and Therapy, 46*(6), 689-700.

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive-compulsive disorder: state or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11-12), 1031-1038.

Beck, A., Steer, R., & Brown, G. (1996). *Manual for Beck Depression Inventory II (BDI-II).* San Antonio, TX: Psychology Corporation.

Behrmann, M., Zemel, R. S., & Mozer, M. C. (1998). Object-based attention and occlusion: Evidence from normal participants and a computational model. *Journal of Experimental Psychology-Human Perception and Performance, 24*(4), 1011-1036.

Benton, A. L. (1974). *The Revised Visual Retention Test Psychological Corporation.* New York

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., Jenike, M. A., Tuschen-Caffier, B., et al. (2005). Visuospatial abilities, memory, and executive functioning in trichotillomania and obsessive-compulsive disorder. *Journal of Clinical and Experimental Neuropsychology, 27*(4), 385-399.

Boldrini, M., Del Pace, L., Placidi, G. P., Keilp, J., Ellis, S. P., Signori, S., et al. (2005). Selective cognitive deficits in obsessive-compulsive disorder compared to panic disorder with agoraphobia. *Acta Psychiatrica Scandinavica, 111*(2), 150-158.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652.

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex, 11*(9), 825-836.

Bruene, M. (2006). The evolutionary psychology of obsessive-compulsive disorder - the role of cognitive metarepresentation. *Perspectives in Biology and Medicine, 49*(3), 317-329.

Buhlmann, U., Deckersbach, T., Engelhard, I., Cook, L. M., Rauch, S. L., Kathmann, N., et al. (2006). Cognitive retraining for organizational impairment in obsessive-compulsive disorder. *Psychiatry Research, 144*(2-3), 109-116.

Cabrera, A. R., McNally, R. J., & Savage, C. R. (2001). Missing the forest for the trees? Deficient memory for linguistic gist in obsessive-compulsive disorder. *Psychological Medicine, 31*(6), 1089-1094.

Cha, K. R., Koo, M.-S., Kim, C.-H., Kim, J. W., Oh, W.-J., Suh, H. S., et al. (2008). Nonverbal memory dysfunction in obsessive-compulsive disorder patients with checking compulsions. *Depression and Anxiety, 25*(11), E115-120.

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2006). Strategy implementation in obsessive-compulsive disorder and trichotillomania. *Psychological Medicine, 36*(1), 91-97.

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, J. (2005). The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience and Biobehavioral Reviews, 29*(3), 399-419.

Chamberlain, S. R., Fineberg, N. A., Menzies, L. A., Blackwell, A. D., Bullmore, E. T., Robbins, T. W., et al. (2007). Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *American Journal of Psychiatry, 164*(2), 335-338.

Chamberlain, S. R., & Menzies, L. (2009). Endophenotypes of obsessive-compulsive disorder: rationale, evidence and future potential. *Expert Rev Neurother, 9*(8), 1133-1146.

Chiulli, S. J., Haaland, K. Y., Larue, A., & Garry, P. J. (1995). Impact of Age on Drawing the Rey-Osterrieth Figure. *Clinical Neuropsychologist, 9*(3), 219-224.

Ciesielski, K. T., Hamalainen, M. S., Geller, D. A., Wilhelm, S., Goldsmith, T. E., & Ahlfors, S. P. (2007). Dissociation between MEG alpha modulation and performance accuracy on visual working memory task in obsessive compulsive disorder. *Human Brain Mapping, 28*(12), 1401-1414.

Ciesielski, K. T., Rowland, L. M., Harris, R. J., Kerwin, A. A., Reeve, A., & Knight, J. E. (2011). Increased anterior brain activation to correct responses on high-conflict Stroop task in obsessive-compulsive disorder. *Clinical Neurophysiology, 122*(1), 107-113.

Coles, M. E., Frost, R. O., Heimberg, R. G., & Rhéaume, J. (2003). "Not just right experiences": perfectionism, obsessive-compulsive features and general psychopathology. *Behaviour Research and Therapy, 41*(6), 681-700.

Coles, M. E., & Heimberg, R. G. (2002). Memory biases in the anxiety disorders: Current status. *Clinical Psychology Review, 22*(4), 587-627.

Coles, M. E., Radomsky, A. S., & Horng, B. (2006). Exploring the boundaries of memory distrust from repeated checking: increasing external validity and examining thresholds. *Behaviour Research and Therapy, 44*(7), 995-1006.

Coughlan, A. K., & Hollows, S. E. (1985). *The adult memory and information procesing batery*. Leeds: St. James's University Hopital.

Cougle, J. R., Salkovskis, P. M., & Thorpe, S. J. (2008). "Perhaps you only imagined doing it": Reality-monitoring in obsessive-compulsive checkers using semi-idiographic stimuli. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(3), 305-320.

Cougle, J. R., Salkovskis, P. M., & Wahl, K. (2007). Perception of memory ability and confidence in recollections in obsessive-compulsive checking. *Journal of Anxiety Disorders, 21*(1), 118-130.

Cowan, A. (1999). An embedded-process model of working memory. In A. a. S. Miyake, P. (Ed.), *Models of Working Memory: Mechanisms of Active Maintenance and Executive Control.* (pp. 62-101.). Cambridge.: Cambridge University Press.

Cuttler, C., & Graf, P. (2007). Sub-clinical compulsive checkers' prospective memory is impaired. *Journal of Anxiety Disorders, 21*(3), 338-352.

Cuttler, C., & Graf, P. (2008). Sub-clinical checking compulsions are related to impaired prospective memory independently of depression, anxiety and distractibility. *Journal of Anxiety Disorders, 22*(4), 642-654.

Cuttler, C., & Graf, P. (2009). Checking-in on the memory deficit and meta-memory deficit theories of compulsive checking. *Clinical Psychology Review, 29*(5), 393-409.

de Geus, F., Denys, D. A., Sitskoorn, M. M., & Westenberg, H. G. (2007). Attention and cognition in patients with obsessive-compulsive disorder. *Psychiatry and Clinical Neurosciences, 61*(1), 45-53.

Deckersbach, T., McMurrich, S., Ogutha, J., Savage, C. R., Sachs, G., & Rauch, S. L. (2004). Characteristics of non-verbal memory impairment in bipolar disorder: the role of encoding strategies. *Psychological Medicine, 34*(5), 823-832.

Deckersbach, T., Otto, M. W., Savage, C. R., Baer, L., & Jenike, M. A. (2000). The relationship between semantic organization and memory in obsessive-compulsive disorder. *Psychotherapy and Psychosomatics, 69*(2), 101-107.

Deckersbach, T., Savage, C. R., Dougherty, D. D., Bohne, A., Loh, R., Nierenberg, A., et al. (2005). Spontaneous and directed application of verbal learning strategies in bipolar disorder and obsessive-compulsive disorder. *Bipolar Disord, 7*(2), 166-175.

Dehaene, S., Changeux, J. P., Naccache, L., Sackur, J., & Sergent, C. (2006). Conscious, preconscious, and subliminal processing: a testable taxonomy. *Trends in Cognitive Sciences, 10*(5), 204-211.

Dehaene, S., & Naccache, L. (2001). Towards a cognitive neuroscience of consciousness: basic evidence and a workspace framework. *Cognition, 79*(1-2), 1-37.
Dehaene, S., Sergent, C., & Changeux, J. P. (2003). A neuronal network model linking subjective reports and objective physiological data during conscious perception. *Proceedings of the National Academy of Sciences of the United States of America, 100*(14), 8520-8525.
Dek, E. C. P., van den Hout, M. A., Giele, C. L., & Engelhard, I. M. (2010). Repeated checking causes distrust in memory but not in attention and perception. *Behaviour Research and Therapy, 48*(7), 580-587.
Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1988). *The California Verbal Learning Test.* San Antonio, Texas.: The Psychological Corporation.
Delorme, R., Gousse, V., Roy, I., Trandafir, A., Mathieu, F., Mouren-Simeoni, M. C., et al. (2007). Shared executive dysfunctions in unaffected relatives of patients with autism and obsessive-compulsive disorder. *European Psychiatry, 22*(1), 32-38.
Delvenne, J. F., & Bruyer, R. (2006). A configural effect in visual short-term memory for features from different parts of an object. *Quarterly Journal of Experimental Psychology, 59*(9), 1567-1580.
Deubel, H., & Schneider, W. X. (1996). Saccade target selection and object recognition: Evidence for a common attentional mechanism. *Vision Research, 36*(12), 1827-1837.
Dolcos, F., & McCarthy, G. (2006). Brain systems mediating cognitive interference by emotional distraction. *Journal of Neuroscience, 26*(7), 2072-2079.
Eldar, S., & Bar-Haim, Y. (2010). Neural plasticity in response to attention training in anxiety. *Psychological Medicine, 40*(4), 667-677.
Elliott, R., McKenna, P. J., Robbins, T. W., & Sahakian, B. J. (1995). Neuropsychological Evidence for Frontostriatal Dysfunction in Schizophrenia. *Psychological Medicine, 25*(3), 619-630.
Elsley, J. V., & Parmentier, F. B. R. (2009). Is verbal-spatial binding in working memory impaired by a concurrent memory load? *Quarterly Journal of Experimental Psychology, 62*(9), 1696-1705.
Enright, S. J., & Beech, A. R. (1993). Reduced cognitive inhibition in obsessive-compulsive disorder. *British Journal of Clinical Psychology, 32 (Pt 1)*, 67-74.
Enright, S. J., Beech, A. R., & Claridge, G. S. (1995). A Further Investigation of Cognitive Inhibition in Obsessive-Compulsive Disorder and Other Anxiety Disorders. *Personality and Individual Differences, 19*(4), 535-542.
Ericsson, K. A., Chase, W. G., & Faloon, S. (1980). Acquisition of a Memory Skill. *Science, 208*(4448), 1181-1182.
Ericsson, K. A., & Kintsch, W. (1995). Long-term working memory. *Psychological Review, 102*(2), 211-245.
Exner, C., Martin, V., & Rief, W. (2009). Self-Focused Ruminations and Memory Deficits in Obsessive-Compulsive Disorder. *Cognitive Therapy and Research, 33*(2), 163-174.
Eysenck, M. W., & Derakshan, N. (2011). New perspectives in attentional control theory. *Personality and Individual Differences, 50*(7), 955-960.
Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353.

Fenger, M. M., Gade, A., Adams, K. H., Hansen, E. S., Bolwig, T. G., & Knudsen, G. M. (2005). Cognitive deficits in obsessive-compulsive disorder on tests of frontal lobe functions. *Nord J Psychiatry, 59*(1), 39-44.

Ferreri, F., Lapp, L. K., & Peretti, C. S. (2011). Current research on cognitive aspects of anxiety disorders. *Current Opinion in Psychiatry, 24*(1), 49-54.

Foa, E. B., Amir, N., Gershuny, B., Molnar, C., & Kozak, M. J. (1997). Implicit and explicit memory in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 11*(2), 119-129.

Foa, E. B., Ilai, D., McCarthy, P. R., Shoyer, B., & Murdock, T. (1993). Information-Processing in Obsessive-Compulsive Disorder. *Cognitive Therapy and Research, 17*(2), 173-189.

Fougnie, D., & Marois, R. (2009). Attentive tracking disrupts feature binding in visual working memory. *Visual Cognition, 17*(1-2), 48-66.

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology-General, 133*(1), 101-135.

Gajewski, D. A., & Brockmole, J. R. (2006). Feature bindings endure without attention: evidence from an explicit recall task. *Psychon Bull Rev, 13*(4), 581-587.

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A Neural System for Error-Detection and Compensation. *Psychological Science, 4*(6), 385-390.

Gehring, W. J., Himle, J., & Nisenson, L. G. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science, 11*(1), 1-6.

Gershuny, B. S., & Sher, K. J. (1995). Compulsive Checking and Anxiety in a Nonclinical Sample - Differences in Cognition, Behavior, Personality, and Affect. *Journal of Psychopathology and Behavioral Assessment, 17*(1), 19-38.

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences, 15*(1), 20-25.

Gooding, D. C., & Basso, M. A. (2008). The tell-tale tasks: A review of saccadic research in psychiatric patient populations. *Brain and Cognition, 68*(3), 371-390.

Goodwin, A. H., & Sher, K. J. (1992). Deficits in Set-Shifting Ability in Nonclinical Compulsive Checkers. *Journal of Psychopathology and Behavioral Assessment, 14*(1), 81-92.

Graydon, J., & Eysenck, M. W. (1989). Distraction and cognitive performance. *European Journal of Cognitive Psychology, 1*(2), 161-169.

Greisberg, S., & McKay, D. (2003). Neuropsychology of obsessive-compulsive disorder: a review and treatment implications. *Clinical Psychology Review, 23*(1), 95-117.

Grison, S., Paul, M. A., Kessler, K., & Tipper, S. P. (2005). Inhibition of object identity in inhibition of return: Implications for encoding and retrieving inhibitory processes. *Psychonomic Bulletin & Review, 12*(3), 553-558.

Hajcak, G., & Simons, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research, 110*(1), 63-72.

Harkin, B., & Kessler, K. (2009). How checking breeds doubt: Reduced performance in a simple working memory task. *Behaviour Research and Therapy, 47*(6), 504-512.

Harkin, B., & Kessler, K. (2011a). How Checking as a Cognitive Style Influences Working Memory Performance. *Applied Cognitive Psychology, 25*(2), 219-228.

Harkin, B., & Kessler, K. (2011b). The role of Working Memory in compulsive checking and OCD: A systematic classification of 58 experimental findings. *Clinical Psychology Review, 31*(6), 1004-1021.

Harkin, B., & Kessler, K. (in press). Deficient Inhibition-Of-Return in subclinical OCD when attention is directed to the threatening aspects of a stimulus. *Depression and Anxiety*.

Harkin, B., Miellet, S., & Kessler, K. (subm). What checkers actually check: An eye tracking study of working memory and executive control.

Harkin, B., Rutherford, H., & Kessler, K. (2011). Impaired executive functioning in subclinical compulsive checking with ecologically valid stimuli in a Working Memory task. *Frontiers in Psychology, 2*.

Hartston, H. J., & Swerdlow, N. R. (1999). Visuospatial priming and stroop performance in patients with obsessive compulsive disorder. *Neuropsychology, 13*(3), 447-457.

Harvey, N. S. (1987). Neurological Factors in Obsessive-Compulsive Disorder. *British Journal of Psychiatry, 150*, 567-568.

Head, D., Bolton, D., & Hymas, N. (1989). Deficit in cognitive shifting ability in patients with obsessive-compulsive disorder. *Biological Psychiatry, 25*(7), 929-937.

Henderson, J. G., & Pollard, C. A. (1988). Types of Obsessive Compulsive Disorder in a Community Sample. *Journal of Clinical Psychology, 44*(5), 747-752.

Henseler, I., Gruber, O., Kraft, S., Krick, C., Reith, W., & Falkai, P. (2008). Compensatory hyperactivations as markers of latent working memory dysfunctions in patients with obsessive-compulsive disorder: an fMRI study. *Journal of Psychiatry and Neuroscience, 33*(3), 209-215.

Hermans, D., Engelen, U., Grouwels, L., Joos, E., Lemmens, J., & Pieters, G. (2008). Cognitive confidence in obsessive-compulsive disorder: Distrusting perception, attention and memory. *Behaviour Research and Therapy, 46*(1), 98-113.

Hinton, G. E., McClelland, J. L., & Rumelhart, D. E. (1986). Distributed representations. In D. E. Rumelhart & J. L. McClelland (Eds.), *Parallel distributed processing: Explorations in the microstructure of cognition* (Vol. 1, pp. 77–109). Cambridge: MIT Press.

Hoenig, K., Hochrein, A., Muller, D. J., & Wagner, M. (2002). Different negative priming impairements in schizophrenia and subgroups of obsessive-compulsive disorder. *Psychological Medicine, 32*, 459-468.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis. of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709.

Hulme, C., Maughan, S., & Brown, G. D. A. (1991). Memory for Familiar and Unfamiliar Words - Evidence for a Long-Term-Memory Contribution to Short-Term-Memory Span. *Journal of Memory and Language, 30*(6), 685-701.

Hwang, S. H., Kwon, J. S., Shin, Y. W., Lee, K. J., Kim, Y. Y., & Kim, M. S. (2007). Neuropsychological profiles of patients with obsessive-compulsive disorder: early onset versus late onset. *Journal of the International Neuropsychological Society, 13*(1), 30-37.

Hymas, N., Lees, A., Bolton, D., Epps, K., & Head, D. (1991). The Neurology of Obsessional Slowness. *Brain, 114*, 2203-2233.

Hyun, J. S., Woodman, G. F., & Luck, S. J. (2009). The role of attention in the binding of surface features to locations. *Visual Cognition, 17*(1-2), 10-24.

Irak, M., & Flament, M. F. (2009). Attention in sub-clinical obsessive-compulsive checkers. *Journal of Anxiety Disorders, 23*(3), 320-326.

Jaafari, N., Rigalleau, F., Rachid, F., Delamillieure, P., Millet, B., Olie, J. P., et al. (2011). A critical review of the contribution of eye movement recordings to the neuropsychology of obsessive compulsive disorder. *Acta Psychiatrica Scandinavica, 124*(2), 87-101.

Jang, J. H., Kim, H. S., Ha, T. H., Shin, N. Y., Kang, D.-H., Choi, J.-S., et al. (2010). Nonverbal memory and organizational dysfunctions are related with distinct symptom dimensions in obsessive-compulsive disorder. *Psychiatry Research, 180*(2-3), 93-98.

Jelinek, L., Moritz, S., Heeren, D., & Naber, D. (2006). Everyday memory functioning in obsessive-compulsive disorder. *Journal of the International Neuropsychological Society, 12*(5), 746-749.

Johnson, M. K., Raye, C. L., Mitchell, K. J., Greene, E. J., Cunningham, W. A., & Sanislow, C. A. (2005). Using fMRI to investigate a component process of reflection: Prefrontal correlates of refreshing a just-activated representation. *Cognitive Affective & Behavioral Neuroscience, 5*(3), 339-361.

Jordan, H., & Tipper, S. P. (1998). Object-based inhibition of return in static displays. *Psychonomic Bulletin & Review, 5*(3), 504-509.

Kampman, M., Keijsers, G. P. J., Verbraak, M., Naring, G., & Hoogduin, C. A. L. (2002). The emotional Stroop: a comparison of panic disorder patients, obsessive-compulsive patients, and normal controls, in two experiments. *Journal of Anxiety Disorders, 16*(4), 425-441.

Keizer, A. W., Colzato, L. S., & Hommel, B. (2008). Integrating faces, houses, motion, and action: Spontaneous binding across ventral and dorsal processing streams. *Acta Psychologica, 127*(1), 177-185.

Kessler, K., & Kiefer, M. (2005). Disturbing visual working memory: electrophysiological evidence for a role of the prefrontal cortex in recovery from interference. *Cerebral Cortex, 15*(7), 1075-1087.

Kessler, K., & Tipper, S. P. (2004). Retrieval of implicit inhibitory processes: The impact of visual field, object-identity, and memory dynamics. *Visual Cognition, 11*(8), 965-995.

Kikul, J., Vetter, J., Lincoln, T. M., & Exner, C. (2011). Effects of cognitive self-consciousness on visual memory in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 25*(4), 490-497.

Kim, M. S., Park, S. J., Shin, M. S., & Kwon, J. S. (2002). Neuropsychological profile in patients with obsessive-compulsive disorder over a period of 4-month treatment. *Journal of Psychiatric Research, 36*(4), 257-265.

Kimura, D. (1963). Right temporal lobe damage. *Archives of Neurology., 8*, 264-271.

Klein, R. M., & MacInnes, W. J. (1999). Inhibition of return is a foraging facilitator in visual search. *Psychological Science, 10*(4), 346-352.

Kuelz, A. K., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: a critical review. *Biological Psychology, 65*(3), 185-236.

Kuelz, A. K., Riemann, D., Halsband, U., Vielhaber, K., Unterrainer, J., Kordon, A., et al. (2006). Neuropsychological impairment in obsessive-compulsive disorder - Improvement over the course of cognitive behavioral treatment. *Journal of Clinical and Experimental Neuropsychology, 28*(8), 1273-1287.

Kyrios, M., & Iob, M. A. (1998). Automatic and strategic processing in obsessive-compulsive disorder: Attentional bias, cognitive avoidance or more complex phenomena? *Journal of Anxiety Disorders, 12*(4), 271-292.

Lavie, N., Hirst, A., de Fockert, J. W., & Viding, E. (2004). Load theory of selective attention and cognitive control. *Journal of Experimental Psychology-General, 133*(3), 339-354.

Lavric, A., Rippon, G., & Gray, J. R. (2003). Threat-evoked anxiety disrupts spatial working memory performance: An attentional account. *Cognitive Therapy and Research, 27*(5), 489-504.

Lavy, E., van Oppen, P., & van den Hout, M. (1994). Selective Processing of Emotional Information in Obsessive-Compulsive Disorder. *Behaviour Research and Therapy, 32*(2), 243-246.

Lind, C., & Boschen, M. J. (2009). Intolerance of uncertainty mediates the relationship between responsibility beliefs and compulsive checking. *Journal of Anxiety Disorders, 23*(8), 1047-1052.

Logie, R. H., Brockmole, J. R., & Vandenbroucke, A. R. E. (2009). Bound feature combinations in visual short-term memory are fragile but influence long-term learning. *Visual Cognition, 17*(1-2), 160-179.

Luck, S. J., & Vogel, E. K. (1997). The capacity of visual working memory for features and conjunctions. *Nature, 390*(6657), 279-281.

MacDonald, P. A., Antony, M. M., Macleod, C. M., & Richter, M. A. (1997). Memory and confidence in memory judgements among individuals with obsessive compulsive disorder and non-clinical controls. *Behaviour Research and Therapy, 35*(6), 497-505.

MacLeod, C., & Mathews, A. (1991). Biased cognitive operations in anxiety: accessibility of information or assignment of processing priorities? *Behaviour Research and Therapy, 29*(6), 599-610.

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology, 111*(1), 107-123.

Makovski, T., Sussman, R., & Jiang, Y. V. (2008). Orienting attention in visual working memory reduces interference from memory probes. *Journal of Experimental Psychology. Learning, Memory, and Cognition, 34*(2), 369-380.

Martin, A., Wiggs, C. L., Altemus, M., Rubenstein, C., & Murphy, D. L. (1995). Working-Memory as Assessed by Subject-Ordered Tasks in Patients with Obsessive-Compulsive Disorder. *Journal of Clinical and Experimental Neuropsychology, 17*(5), 786-792.

Martinot, J. L., Allilaire, J. F., Mazoyer, B. M., Hantouche, E., Huret, J. D., Legaut-Demare, F., et al. (1990). Obsessive-compulsive disorder: a clinical, neuropsychological and positron emission tomography study. *Acta Psychiatrica Scandinavica, 82*(3), 233-242.

Mataix-Cols, D., Junque, C., Sanchez-Turet, M., Vallejo, J., Verger, K., & Barrios, M. (1999a). Neuropsychological functioning in a subclinical obsessive-compulsive sample. *Biological Psychiatry, 45*(7), 898-904.

Mataix-Cols, D., Junqué, C., Vallejo, J., Sànchez-Turet, M., Verger, K., & Barrios, M. (1997). Hemispheric functional imbalance in a sub-clinical obsessive-

compulsive sample assessed by the Continuous Performance Test, Identical Pairs version. *Psychiatry Research, 72*(2), 115-126.
Mataix-Cols, D., Rauch, S. L., Manzo, P. A., Jenike, M. A., & Baer, L. (1999b). Use of factor-analyzed symptom of dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry, 156*(9), 1409-1416.
Mather, M. (2007). Emotional Arousal and Memory Binding An Object-Based Framework. *Perspectives on Psychological Science, 2*(1), 33-52.
Mather, M., Mitchell, K. J., Raye, C. L., Novak, D. L., Greene, E. J., & Johnson, M. K. (2006). Emotional arousal can impair feature binding in working memory. *Journal of Cognitive Neuroscience, 18*(4), 614-625.
Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. *Cognition & Emotion, 16*(3), 331-354.
McNally, R. J., Amir, N., Louro, C. E., Lukach, B. M., Riemann, B. C., & Calamari, J. E. (1994). Cognitive Processing of Idiographic Emotional Information in Panic Disorder. *Behaviour Research and Therapy, 32*(1), 119-122.
McNally, R. J., & Kohlbeck, P. A. (1993). Reality monitoring in obsessive-compulsive disorder. *Behaviour Research and Therapy, 31*(3), 249-253.
Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C. H., Del Campo, N., et al. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*, 3223-3236.
Miller, G. A. (1956). The magical number seven, plus or minus two: some limits on our capacity for processing information. *Psychological Review,, 63*(81-97).
Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49-100.
Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*(9), 809-848.
Morein-Zamir, S., Craig, K. J., Ersche, K. D., Abbott, S., Muller, U., Fineberg, N. A., et al. (2010). Impaired visuospatial associative memory and attention in obsessive compulsive disorder but no evidence for differential dopaminergic modulation. *Psychopharmacology, 212*(3), 357-367.
Moritz, S., Fischer, B. K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., et al. (2008). Words may not be enough! No increased emotional Stroop effect in obsessive-compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104.
Moritz, S., Kloss, M., Jahn, H., Schick, M., & Hand, I. (2003). Impact of comorbid depressive symptoms on nonverbal memory and visuospatial performance in obsessive-compulsive disorder. *Cogn Neuropsychiatry, 8*(4), 261-272.
Moritz, S., & von Muehlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229.
Moritz, S., & von Muhlenen, A. (2005). Inhibition of return in patients with obsessive-compulsive disorder. *Journal of Anxiety Disorders, 19*(1), 117-126.
Moritz, S., Von Muhlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive-compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371.

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive-Compulsive Disorder: a review. *Journal of Anxiety Disorders, 19*(1), 1-28.
Murdock, B. B. (1962). Serial Position Effect of Free-Recall. *Journal of Experimental Psychology, 64*(5), 482-&.
Nedeljkovic, M., & Kyrios, M. (2007). Confidence in memory and other cognitive processes in obsessive-compulsive disorder. *Behaviour Research and Therapy, 45*(12), 2899-2914.
Nedeljkovic, M., Kyrios, M., Moulding, R., Doron, G., Wainwright, K., Pantelis, C., et al. (2009b). Differences in neuropsychological performance between subtypes of obsessive-compulsive disorder. *Australian and New Zealand Journal of Psychiatry, 43*(3), 216-226.
Nedeljkovic, M., Moulding, R., Kyrios, M., & Doron, G. (2009a). The relationship of cognitive confidence to OCD symptoms. *Journal of Anxiety Disorders, 23*(4), 463-468.
Nelson, E., Early, T. S., & Haller, J. W. (1993). Visual-Attention in Obsessive-Compulsive Disorder. *Psychiatry Research, 49*(2), 183-196.
Nelson, T. O., & Narens, L. (1994). *Metamemory: a theoretical framework and new findings.* (Vol. 26). New York: Academic Press.
Olivers, C. N. L., Meijer, F., & Theeuwes, J. (2006). Feature-based memory-driven attentional capture: Visual working memory content affects visual attention. *Journal of Experimental Psychology-Human Perception and Performance, 32*(5), 1243-1265.
Olley, A., Malhi, G., & Sachdev, P. (2007). Memory and executive functioning in obsessive-compulsive disorder: a selective review. *Journal of Affective Disorders, 104*(1-3), 15-23.
Olson, I. R., Page, K., Moore, K. S., Chatterjee, A., & Verfaellie, M. (2006). Working memory for conjunctions relies on the medial temporal lobe. *Journal of Neuroscience, 26*(17), 4596-4601.
Omori, I. M., Murata, Y., Yamanishi, T., Nakaaki, S., Akechi, T., Mikuni, M., et al. (2007). The differential impact of executive attention dysfunction on episodic memory in obsessive-compulsive disorder patients with checking symptoms vs. those with washing symptoms. *Journal of Psychiatric Research, 41*(9), 776-784.
Osterrieth, P. A. (1944). Filetest de copie d'une figure complex: Contribution a l'etude de la perception et de la memoire [The test of copying a complex figure: A contribution to the study of perception and memory]. *Archives de Psychologie, 30*(286-356).
Park, H. S., Shin, Y. W., Ha, T. H., Shin, M. S., Kim, Y. Y., Lee, Y. H., et al. (2006). Effect of cognitive training focusing on organizational strategies in patients with obsessive-compulsive disorder. *Psychiatry and Clinical Neurosciences, 60*(6), 718-726.
Penades, R., Catalan, R., Andres, S., Salamero, M., & Gasto, C. (2005). Executive function and nonverbal memory in obsessive-compulsive disorder. *Psychiatry Research, 133*(1), 81-90.
Petrides, M., & Milner, B. (1982). Deficits on Subject-Ordered Tasks after Frontal-Lobe and Temporal-Lobe Lesions in Man. *Neuropsychologia, 20*(3), 249-262.
Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. In A. a. performance (Ed.), (pp. 531-556). Hillsdale NJ: Erlbaum.

Posner, M. I., Cohen, Y., & Rafal, R. D. (1982). Neural Systems Control of Spatial Orienting. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences, 298*(1089), 187-198.

Posner, M. I., & Snyder, C. R. (1975). Facilitation and Inhibition. In P. Rabbitt & S. Dornick (Eds.), *Attention and Performance V* (pp. 669-682). New York: Academic Press.

Purcell, R., Maruff, P., Kyrios, M., & Pantelis, C. (1998a). Cognitive deficits in obsessive-compulsive disorder on tests of frontal-striatal function. *Biological Psychiatry, 43*(5), 348-357.

Purcell, R., Maruff, P., Kyrios, M., & Pantelis, C. (1998b). Neuropsychological deficits in obsessive-compulsive disorder: a comparison with unipolar depression, panic disorder, and normal controls. *Archives of General Psychiatry, 55*(5), 415-423.

Rachman, S. (2002). A cognitive theory of compulsive checking. *Behaviour Research Therapy, 40*(6), 625-639.

Rachman, S., & Hodgson, R. J. (1980). *Obsessions and compulsions.* Englewood Cliffs, New Jersey: Prentice-Hall.

Rachman, S., & Shafran, R. (1998). Cognitive behavioural features of obsessive–compulsive disorder. In R. Swinson, M. Antony, S. Rachman & M. Richter (Eds.), *Obsessive–compulsive disorder: theory, research and treatment.* (pp. 51-78). New York: Guilford Publications.

Rachman, S., Thordarson, D. S., Shafran, R., & Woody, S. R. (1995). Perceived responsibility: structure and significance. *Behaviour Research and Therapy, 33*(7), 779-784.

Radomsky, A. S., & Alcolado, G. M. (2010). Don't even think about checking: Mental checking causes memory distrust. *Journal of Behavior Therapy and Experimental Psychiatry, 41*(4), 345-351.

Radomsky, A. S., Ashbaugh, A. R., & Gelfand, L. A. (2007). Relationships between anger, symptoms, and cognitive factors in OCD checkers. *Behaviour Research and Therapy, 45*(11), 2712-2725.

Radomsky, A. S., Gilchrist, P. T., & Dussault, D. (2006). Repeated checking really does cause memory distrust. *Behaviour Research and Therapy, 44*(2), 305-316.

Rampacher, F., Lennertz, L., Vogeley, A., Schulze-Rauschenbach, S., Kathmann, N., Falkai, P., et al. (2010). Evidence for specific cognitive deficits in visual information processing in patients with OCD compared to patients with unipolar depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 34*(6), 984-991.

Rankins, D., Bradshaw, J., Moss, S., & Georgiou-Karistianis, N. (2004). Inhibition of return in obsessive-compulsive disorder. *Journal of the International Neuropsychological Society, 10*(1), 54-59.

Rao, N. P., Reddy, Y. C. J., Kumar, K. J., Kandavel, T., & Chandrashekar, C. R. (2008). Are neuropsychological deficits trait markers in OCD? *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 32*(6), 1574-1579.

Rasmussen, S. A., & Eisen, J. L. (1988). Clinical and epidemiologic findings of significance to neuropharmacologic trials in OCD. *Psychopharmacology Bulletin, 24*(3), 466-470.

Raven, J., Raven, J. C., & Court, J. H. (2004). *Manual for Raven's Progressive Matrices and Vocabulary Scales.* San Antonio, TX: Harcourt Assessment.

Reed, G. F. (1985). *Obsessional experience and compulsive behavior. A cognitive-structural approach.* London: Academic Press.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuiss, S. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443-447.

Riesel, A., Endrass, T., Kaufmann, C., & Kathmann, N. (2011). Overactive Error-Related Brain Activity as a Candidate Endophenotype for Obsessive-Compulsive Disorder: Evidence From Unaffected First-Degree Relatives. *American Journal of Psychiatry, 168*(3).

Roh, K. S., Shin, M. S., Kim, M. S., Ha, T. H., Shin, Y. W., Lee, K. J., et al. (2005). Persistent cognitive dysfunction in patients with obsessive-compulsive disorder: A naturalistic study. *Psychiatry and Clinical Neurosciences, 59*(5), 539-545.

Rotge, J. Y., Clair, A. H., Jaafari, N., Hantouche, E. G., Pelissolo, A., Guehl, D., et al. (2008). A challenging task for assessment of checking behaviors in obsessive-compulsive disorder. *European Neuropsychopharmacology, 17*, S300-S301.

Roth, R. M., Milovan, D., Baribeau, J., & O'Connor, K. (2005). Neuropsychological functioning in early- and late-onset obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences, 17*(2), 208-213.

Roth, R. M., Milovan, D. L., Baribeau, J., O'Connor, K., & Todorov, C. (2004). Organizational strategy use in obsessive-compulsive disorder. *Psychiatry Research, 128*(3), 267-272.

Rubenstein, C. S., Peynircioglu, Z. F., Chambless, D. L., & Pigott, T. A. (1993). Memory in sub-clinical obsessive-compulsive checkers. *Behaviour Research and Therapy, 31*(8), 759-765.

Rudner, M., & Ronnberg, J. (2008). The role of the episodic buffer in working memory for language processing. *Cogn Process, 9*(1), 19-28.

Sahakian, B. J., Morris, R. G., Evenden, J. L., Heald, A., Levy, R., Philpot, M., et al. (1988). A Comparative-Study of Visuospatial Memory and Learning in Alzheimer-Type Dementia and Parkinsons-Disease. *Brain, 111*, 695-718.

Salkovskis, P. M. (1999). Understanding and treating obsessive-compulsive disorder. *Behaviour Research and Therapy, 37 Suppl 1*, S29-52.

Salkovskis, P. M., Forrester, E., & Richards, C. (1998). Cognitive-behavioural approach to understanding obsessional thinking. *British Journal of Psychiatry. Supplement*(35), 53-63.

Sanz, M., Molina, V., Calcedo, A., Martin-Loeches, M., & Rubia, F. J. (2001). The Wisconsin Card Sorting Test and the assessment of frontal function in obsessive-compulsive patients: An event-related potential study. *Cognitive Neuropsychiatry, 6*(2), 109-129.

Savage, C. R., Baer, L., Keuthen, N. J., Brown, H. D., Rauch, S. L., & Jenike, M. A. (1999). Organizational strategies mediate nonverbal memory impairment in obsessive-compulsive disorder. *Biological Psychiatry, 45*(7), 905-916.

Savage, C. R., Deckersbach, T., Wilhelm, S., Rauch, S. L., Baer, L., Reid, T., et al. (2000). Strategic processing and episodic memory impairment in obsessive compulsive disorder. *Neuropsychology, 14*(1), 141-151.

Sawamura, K., Nakashima, Y., Inoue, M., & Kurita, H. (2005). Short-term verbal memory deficits in patients with obsessive-compulsive disorder. *Psychiatry and Clinical Neurosciences, 59*(5), 527-532.

Schmidt, B. K., Vogel, E. K., Woodman, G. F., & Luck, S. J. (2002). Voluntary and automatic attentional control of visual working memory. *Perception and Psychophysics, 64*(5), 754-763.

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention Training for Generalized Social Anxiety Disorder. *Journal of Abnormal Psychology, 118*(1), 5-14.

Segalas, C., Alonso, P., Labad, J., Jaurrieta, N., Real, E., Jimenez, S., et al. (2008). Verbal and nonverbal memory processing in patients with obsessive-compulsive disorder: its relationship to clinical variables. *Neuropsychology, 22*(2), 262-272.

Sher, K. J., Frost, R., & Otto, R. (1983). Cognitive deficits in compulsive checkers: an exploratory study. *Behaviour Research and Therapy, 21*, 357-363.

Sher, K. J., Mann, B., & Frost, R. O. (1984). Cognitive Dysfunction in Compulsive Checkers - Further Explorations. *Behaviour Research and Therapy, 22*(5), 493-502.

Shimamura, A. P. (2000). Toward a cognitive neuroscience of metacognition. *Consciousness and Cognition, 9*(2 Pt 1), 313-323; discussion 324-316.

Shin, M. S., Park, S. J., Kim, M. S., Lee, Y. H., Ha, T. H., & Kwon, J. S. (2004). Deficits of organizational strategy and visual memory in obsessive-compulsive disorder. *Neuropsychology, 18*(4), 665-672.

Simpson, H. B., Rosen, W., Huppert, J. D., Lin, S. H., Foa, E. B., & Liebowitz, M. R. (2006). Are there reliable neuropsychological deficits in obsessive-compulsive disorder? *Journal of Psychiatric Research, 40*(3), 247-257.

Smith, E. E., & Jonides, J. (1999). Neuroscience - Storage and executive processes in the frontal lobes. *Science, 283*(5408), 1657-1661.

Spielberger, C. D., Gorssuch, R. L., Lushene, P. R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*.

SR.Research.Ltd. (2002). *Eye Link II: User Manual*. Mississauga, Canada.

Stein, M. B., Forde, D. R., Anderson, G., & Walker, J. R. (1997). Obsessive-compulsive disorder in the community: an epidemiologic survey with clinical reappraisal. *American Journal of Psychiatry, 154*(8), 1120-1126.

Sweeney, J. A., Levy, D., & Harris, M. S. H. (2002). Commentary: Eye movement research with clinical populations. In *Brain's Eye: Neurobiological and Clinical Aspects of Oculomotor Research* (Vol. 140, pp. 507-522).

Tallis, F. (1997). The neuropsychology of obsessive-compulsive disorder: a review and consideration of clinical implications. *British Journal of Clinical Psychology, 36 (Pt 1)*, 3-20.

Tallis, F., Pratt, P., & Jamani, N. (1999). Obsessive compulsive disorder, checking, and non-verbal memory: a neuropsychological investigation. *Behaviour Research and Therapy, 37*(2), 161-166.

Theeuwes, J., Belopolsky, A., & Olivers, C. N. L. (2009). Interactions between working memory, attention and eye movements. *Acta Psychologica, 132*(2), 106-114.

Thordarson, D. S., Radomsky, A. S., Rachman, S., Shafran, R., Sawchuk, C. N., & Ralph Hakstian, A. (2004). The Vancouver Obsessional Compulsive Inventory (VOCI). *Behaviour Research and Therapy, 42*(11), 1289-1314.

Tipper, S. P., Grison, S., & Kessler, K. (2003). Long-term inhibition of return of attention. *Psychological Science, 14*(1), 19-25.

Tipper, S. P., Jordan, H., & Weaver, B. (1999). Scene-based and object-centered inhibition of return: Evidence for dual orienting mechanisms. *Perception & Psychophysics, 61*(1), 50-60.

Tolin, D. F., Abramowitz, J. S., Brigidi, B. D., Amir, N., Street, G. P., & Foa, E. B. (2001). Memory and memory confidence in obsessive-compulsive disorder. *Behaviour Research and Therapy, 39*(8), 913-927.

Tolin, D. F., Abramowitz, J. S., Brigidi, B. D., & Foa, E. B. (2003). Intolerance of uncertainty in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 17*(2), 233-242.

Tolin, D. F., Woods, C. M., & Abramowitz, J. S. (2003). Relationship between obsessive beliefs and obsessive-compulsive symptoms. *Cognitive Therapy and Research, 27*(6), 657-669.

Treisman, A. (1996). The binding problem. *Current Opinion in Neurobiology, 6*(2), 171-178.

Tulving, E. (1985). Memory and Consciousness. *Canadian Psychology-Psychologie Canadienne, 26*(1), 1-12.

Tuna, S., Tekcan, A. I., & Topcuoglu, V. (2005). Memory and metamemory in obsessive-compulsive disorder. *Behaviour Research and Therapy, 43*(1), 15-27.

Unoki, K., Kasuga, T., Matsushima, E., & Ohta, K. (1999). Attentional processing of emotional information in obsessive-compulsive disorder. *Psychiatry and Clinical Neurosciences, 53*(6), 635-642.

Ursu, S., Stenger, V. A., Shear, M. K., Jones, M. R., & Carter, C. S. (2003). Overactive action monitoring in obsessive-compulsive disorder: Evidence from functional magnetic resonance imaging. *Psychological Science, 14*(4), 347-353.

van den Hout, M., Engelhard, I. M., de Boer, C., du Bois, A., & Dek, E. (2008). Perseverative and compulsive-like staring causes uncertainty about perception. *Behaviour Research and Therapy, 46*(12), 1300-1304.

van den Hout, M., Engelhard, I. M., Smeets, M., Dek, E. C. P., Turksma, K., & Saric, R. (2009). Uncertainty about perception and dissociation after compulsive-like staring: Time course of effects. *Behaviour Research and Therapy, 47*(6), 535-539.

van den Hout, M., & Kindt, M. (2003a). Phenomenological validity of an OCD-memory model and the remember/know distinction. *Behaviour Research and Therapy, 41*(3), 369-378.

van den Hout, M., & Kindt, M. (2003b). Repeated checking causes memory distrust. *Behaviour Research and Therapy, 41*(3), 301-316.

van den Hout, M., & Kindt, M. (2004). Obsessive-compulsive disorder and the paradoxical effects of perseverative behaviour on experienced uncertainty. *Journal of Behavior Therapy and Experimental Psychiatry, 35*(2), 165-181.

van der Wee, N. J., Ramsey, N. F., Jansma, J. M., Denys, D. A., van Megen, H. J., Westenberg, H. M., et al. (2003). Spatial working memory deficits in obsessive compulsive disorder are associated with excessive engagement of the medial frontal cortex. *Neuroimage, 20*(4), 2271-2280.

van der Wee, N. J., Ramsey, N. F., van Megen, H. J., Denys, D., Westenberg, H. G., & Kahn, R. S. (2007). Spatial working memory in obsessive-compulsive disorder improves with clinical response: A functional MRI study. *European Neuropsychopharmacology, 17*(1), 16-23.

van Veen, V., Cohen, J. D., Botvinick, M. M., Stenger, V. A., & Carter, C. S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *Neuroimage, 14*(6), 1302-1308.

Veale, D. M., Sahakian, B. J., Owen, A. M., & Marks, I. M. (1996). Specific cognitive deficits in tests sensitive to frontal lobe dysfunction in obsessive-compulsive disorder. *Psychological Medicine, 26*(6), 1261-1269.

Watkins, L. H., Sahakian, B. J., Robertson, M. M., Veale, D. M., Rogers, R. D., Pickard, K. M., et al. (2005). Executive function in Tourette's syndrome and obsessive-compulsive disorder. *Psychological Medicine, 35*(4), 571-582.

Watson, C., & Purdon, C. (2008). Attention Training in the Reduction and Reappraisal of Intrusive Thoughts. *Behavioural and Cognitive Psychotherapy, 36*(1), 61-70.

Wechsler, D., & Stone, C. P. (1945). *Manual for the Wechsler Memory Scale.* New York: Psychological Corporation.

Wells, A. (1990). Panic Disorder in Association with Relaxation Induced Anxiety - an Attentional Training Approach to Treatment. *Behavior Therapy, 21*(3), 273-280.

Wells, A. (2000). *Emotional disorders and metacognition: Inovative cognitive therapy.* Chichester: Wiley.

Wheeler, M. E., & Treisman, A. M. (2002). Binding in short-term visual memory. *Journal of Experimental Psychology-General, 131*(1), 48-64.

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (Vol. (2nd ed.)). Chichester: Wiley.

Wolters, G., & Raffone, A. (2008). Coherence and recurrency: maintenance, control and integration in working memory. *Cognitive Processing, 9*(1), 1-17.

Woods, C. M., Vevea, J. L., Chambless, D. L., & Bayen, U. J. (2002). Are compulsive checkers impaired in memory? A meta-analytic review. *Clinical Psychology-Science and Practice, 9*(4), 353-366.

Zielinski, C. M., Taylor, M. A., & Juzwin, K. R. (1991). Neuropsychological deficits in obsessive-compulsive disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 4*(2), 110-126.

Zitterl, W., Urban, C., Linzmayer, L., Aigner, M., Demal, U., Semler, B., et al. (2001). Memory deficits in patients with DSM-IV obsessive-compulsive disorder. *Psychopathology, 34*(3), 113-117.

Appendix 1: 5 published and 1 submitted paper from this thesis in peer reviewed journals

1. Harkin, B. & Kessler, K. (2009). How Checking Breeds Doubt: Reduced Performance in a Simple Working Memory Task. *Behaviour Research and Therapy*, (47), 504-512.

2. Harkin, B. & Kessler, K. (2011a). How Checking as a Cognitive Style Influences Working Memory Performance. *Applied Cognitive Psychology* (24), 1-16.

3. Harkin, B., & Kessler, K. (2011b). The role of Working Memory in compulsive checking and OCD: A systematic classification of 58 experimental findings. *Clinical Psychology Review,* 31(6),1004-1021

4. Harkin, B., Rutherford, H., & Kessler, K. (2011) Impaired executive functioning in subclinical compulsive checking with ecologically valid stimuli in a Working Memory task. *Frontiers in Psychopathology,* 2 (78).

5. Harkin, B., & Kessler, K. (2012). Deficient Inhibition-Of-Return in subclinical OCD only when attention is directed to the threatening aspects of a stimulus *Depression and Anxiety*.

6. Harkin, B., Miellet, S., & Kessler, K. (accepted pending revision). What checkers actually check: An eye tracking study of working memory and executive control.