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Error-related negativity in individuals with obsessive–compulsive symptoms: Toward an understanding of hoarding behaviors

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ABSTRACT

The error-related negativity (ERN), an event-related potential component elicited by error responses in cognitive tasks, has been shown to be abnormal in most, but not all, studies of obsessive–compulsive disorder or obsessive–compulsive symptoms (OCD/S); these inconsistencies may be due to task selection, symptom subtype, or both. We used meta-analysis to further characterize the ERN in OCD/S, and pooled data across studies to examine the ERN in OCD/S with hoarding. We found an enhanced ERN in OCD/S relative to controls, as well as heterogeneity across tasks. When stratified, OCD/S showed a significantly enhanced ERN only in response conflict tasks. However, OCD/S + hoarding showed a marginally larger ERN than OCD/S–hoarding, but only for probabilistic learning tasks. These results suggest that abnormal ERN in OCD/S is task-dependent, and that OCD/S + hoarding show different ERN activity from OCD/S – hoarding perhaps suggesting different pathophysiological mechanisms of error monitoring.

1. Introduction

Obsessive–compulsive disorder (OCD) is a neuropsychiatric disorder that affects approximately 2% of the population world-wide and has strong biological underpinnings and a well-defined neurocircuitry (APA, 2000; Karno et al., 1988). Neuroimaging studies have implicated the ventromedial prefrontal cortex (VMPFC), the anterior cingulate cortex (ACC), the orbitofrontal cortex (OFC), the striatum (particularly the caudate nucleus), and the thalamus, as being involved in the pathophysiology of OCD (Grundler et al., 2009; Harrison et al., 2009; Saxena and Rauch, 2000). These brain regions are interconnected in multiple recurrent loops, making up the cortico-striatal-thalamic-cortical circuit, and are thought to be involved in action selection, performance monitoring, and goal directed behaviors (Gilbert et al., 2008; Harrison et al., 2009; Menzies et al., 2008; Nieuwenhuis et al., 2005). Hyperactivity of this circuit has been demonstrated in individuals with OCD, both at rest and following symptom provocation (Adler et al., 2000; Menzies et al., 2008; van den Heuvel et al., 2005). It has been proposed that, in this disorder, such cortico-striatal hyperactivity leads to a persistently high error signal, ultimately resulting in the psychopathology characteristic of OCD (Grundler et al., 2009; Remijnse et al., 2006). In this model, the brain's error monitoring system compares intended responses (or "expected outcomes") to actual responses (or "actual outcomes" in environmental stimuli, thoughts, feelings, and actions), and generates an error signal when a conflict is detected. It has been suggested that this error signal is amplified in individuals with OCD, leading to the feeling that something is "out of line" (thus, generating irrational fears or obsessions) or that an action was not completed correctly according to a set of internal unattainable rules, triggering repetitive, compensatory behaviors (*i.e.*, compulsions) (Gehring et al., 2000; Pitman, 1987).

The hypothesis of a persistent and enhanced cortico-striatal error signal was first put forward by Pitman (1987), and since then, the enhanced error-signal hypothesis has been tested by multiple investigators using electrophysiological measures associated with performance monitoring and error detection, most specifically the error-related negativity or ERN (Endrass et al., 2008; Gehring et al., 2000; Hajcak et al., 2008; Hajcak and Simons, 2002; Johannes et al., 2001; Pitman, 1987; Santesso et al., 2006). The ERN (Gehring et al., 1993), or error negativity (Ne) (Falkenstein et al., 1991), component of the response-locked event-related potential (ERP) associated with performance errors in speeded choice-response tasks is evident following overt error responses (Gehring et al., 1995) and peaks 50–150 ms after the error is committed. Larger (*i.e.*, more negative) ERNs are associated with instructions emphasizing accuracy over speed, faster errors, lower error rates, attempts to correct errors, greater post-error slowing, and greater error salience (Bernstein et al., 1995; Falkenstein et al., 2000; Gehring et al., 1995; Scheffers and Coles, 2000). Topographic scalp maps show the ERN to have a fronto-central maximum (Falkenstein et al., 1991; Gehring et al., 1995). Converging evidence from dipole modeling of the

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ERN (Dehaene et al., 1994), functional magnetic resonance imaging (fMRI), (Carter et al., 1998; Kiehl et al., 2000; Mathalon et al., 2003), and intracranial recordings from monkeys (Brooks, 1986; Gemba et al., 1986; Niki & Watanabe, 1979), suggests that the ACC is the principal generator of the ERN, which, as noted, has been implicated in OCD. The ERN has been suggested to reflect simple error detection (Falkenstein et al., 1991), high levels of response conflict (Botvinick et al., 2001; Carter et al., 1998; Danielmeier et al., 2009) but see (Carbonnell and Falkenstein, 2006; Masaki et al., 2007), and reward prediction errors in which outcomes are worse than expected (Holroyd and Coles, 2002). Interestingly, the ERN can be evoked by errors committed outside of conscious awareness (Nieuwenhuis et al., 2001; O'Connell et al., 2007).

To date, the published studies examining the ERN among individuals with OCD or high levels of OC symptoms (OCS) have been variable, with many (Endrass et al., 2008, 2010; Gehring et al., 2000; Hajcak et al., 2008; Johannes et al., 2001; Riesel et al., 2011; Ruchsow et al., 2005; Stern et al., 2010), but not all (Hammer et al., 2009; Nieuwenhuis et al., 2005), reporting an increased ERN amplitude in OCD/S subjects compared to controls. A recent hypothesis put forward by Grundler et al. (2009) suggested that some of the observed variability between studies could be due to differences in the task choice—*i.e.*, that the relationship between ERN activation and error processing is task dependent, with *hyperactivity* seen in response conflict tasks, and *hypoactivity* seen in probabilistic or reinforcement learning tasks (Grundler et al., 2009). In response conflict tasks where the correct response is known to the subject, the ERN is generated from quick and impulsive errors known as "slips." Slips are distinguished from "mistakes," which reflect inaccurate intentions based on faulty knowledge (Reason, 1990). Under Grundler's hypothesis, in an individual with OCD/S, protective measures against slips are added, thereby enhancing error signals and perpetuating maladaptive compensatory strategies. In contrast, probabilistic tasks enlist "NoGo learning," (Frank et al., 2005; Holroyd and Coles, 2002) where the subject learns task rules from feedback on a trial-by-trial basis, and learns to inhibit responses that caused the error. If this system is under-activated, individuals with OCD/S may compulsively engage in repetitive behaviors, possibly due to attenuated error signals, leading to suboptimal performance. However, the hypothesis of task divergent ERN activation in OCD/S has not yet been tested (Grundler et al., 2009).

1.1. ERN and OCD symptom subtype

OCD is a heterogeneous disorder, with multiple symptom subtypes, including contamination/cleaning, "taboo" symptoms (religious, sexual, and aggressive obsessions), doubts/fears of causing or incurring harm (often characterized by checking behaviors), rituals and superstitions, and hoarding (Katerberg et al., 2010; Pinto et al., 2008). Of these, the hoarding subtype appears to be the most distinct with regard to treatment response, and perhaps also with regard to neurocognitive and neuroanatomic patterns (An et al., 2008; Gilbert et al., 2008; Pertusa et al., 2008; Rachman et al., 2009; Saxena, 2008). In addition to the hallmark symptoms of compulsive collecting, poor organizational skills, and difficulty discarding, we, and others, have shown that many individuals with compulsive hoarding exhibit cognitive deficits across domains including information processing speed, decision-making, categorization, attention, and memory, and problematic behaviors including procrastination and slowed task completion (Grisham et al., 2007; Hartl et al., 2004; Lawrence et al., 2006; Luchian et al., 2007; Mackin et al., 2010; Mataix-Cols et al., 2004; Tolin et al., 2008).

Based on clinical observations, a recent model of the pathognomonic features of OCD (*i.e.*, obsessions, compulsions, avoidance, and pathological doubt) reframes these symptoms as being secondary or observed manifestations of a more fundamental feature of this disorder, intolerance of uncertainty (Tolin et al., 2003). While the larger, more inclusive construct of intolerance of uncertainty may be a core feature of OCD, more recent theorists suggest that hoarding symptoms are part of a discrete clinical syndrome that also includes indecisiveness and poor organizational ability (Steketee and Frost, 2003). Compared to non-hoarding OCD patients, hoarders have less impulsivity, higher sensitivity to punishment (Fullana et al., 2004), more severe interpersonal disability (Steketee and Frost, 2003) and lower global functioning (Saxena et al., 2002). In addition, they often have less insight into their symptoms than non-hoarding OCD patients, making them less likely to seek treatment (Saxena, 2008). Thus, individuals with hoarding behaviors may have distinct neurocognitive abnormalities that lead to hoarding *per se*. Neuropsychological and neuroimaging examination of hoarding has implicated brain regions including ACC, dorsolateral prefrontal cortex, and orbitofrontal cortex, among others (Gilbert et al., 2008; Mataix-Cols et al., 2004; Saxena, 2008; Tolin et al., 2009) that are involved in response selection, decision-making, conflict monitoring, and error detection, further supporting the hypothesis that abnormalities in error monitoring may be seen in individuals with hoarding behaviors (An et al., 2008; Gilbert et al., 2008; Mataix-Cols et al., 2004). However, to date, no studies of the ERN to assess error monitoring in compulsive hoarding have been conducted.

1.2. Goals of the present study

The goals of this paper are to use currently published/available data to: (1) conduct meta-analyses to verify that the ERN deflection in OC-affected individuals is task-dependent, and to obtain a more precise estimate of the magnitude of the abnormality of the ERN in OCD/S compared to controls, and (2) conduct the first examination into the ERN among OCD/S with hoarding behaviors or symptoms (OCD/S + hoarding) compared to those without hoarding behaviors or symptoms (OCD/S–hoarding). As noted, although the available published studies have, for the most part, shown an association between OCD and an enhanced ERN, the relationship between task type and symptom subtype has not yet been fully examined. To our knowledge, there are no published analyses addressing potential differences in the ERN based on symptom profile. Although an analysis of all symptom subtypes would be of interest, we chose to focus on hoarding in this analysis in part because of the growing body of evidence that hoarding, although overlapping with other OCD symptoms, may have distinct etiologies and distinct neurocognitive profiles (An et al., 2008; Gilbert et al., 2008; Pertusa et al., 2008; Rachman et al., 2009; Saxena, 2008). We predicted that the meta-analysis would uphold the predominant findings in the individual studies, that is, that the ERN is larger (more negative) in OC-affected individuals when compared to healthy controls. We also predicted that this difference would be apparent primarily in those studies that used a response conflict task rather than a probabilistic learning task, as postulated by Grundler et al. (2009). Furthermore, we hypothesized that the phenotypic distinction between individuals with hoarding behaviors relative to those without hoarding behaviors would be reflected by differences in ERN amplitude dependent upon task type.

2. Methods

2.1. Meta analysis

2.1.1. Study selection

Published studies examining the ERN in individuals with OCD or OCS and in controls were initially identified using the MEDLINE-PubMed databases, using the terms OCD, obsessive–compulsive disorder, obsessive–compulsive, action