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Hoarding disorder and obsessive–compulsive disorder show different patterns of neural activity during response inhibition

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ABSTRACT

Although hoarding disorder (HD) has been historically conceptualized as a subtype or dimension of obsessive–compulsive disorder (OCD), preliminary evidence suggests that these two disorders have distinct neural underpinnings. The aim of the present study was to compare the hemodynamic responses of HD patients, OCD patients, and healthy controls (HC) during response inhibition on a high-conflict Go/NoGo task that has previously proved sensitive to OCD. Participants comprised 24 HD patients, 24 OCD patients, and 24 HCs who completed a Go/NoGo task during functional magnetic resonance imaging (fMRI). Although behavioral data showed no difference among the groups in Go/NoGo task performance, significant differences in hemodynamic activity were noted. During correct rejects (successful response inhibition), HD patients showed greater right precentral gyrus activation, whereas OCD patients exhibited greater right orbitofrontal activation, as assessed using a region of interest approach. During errors of commission (response inhibition failures), OCD patients, but not HD patients, were characterized by excessive activity in left and right orbitofrontal gyrus. The present results lend further support to the biological distinction between HD and OCD, and they are consistent with previous research suggesting frontal hypoactivity in HD patients during hoarding-unrelated tasks.

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1. Introduction

Hoarding disorder (HD), a new diagnosis in DSM-5 (American Psychiatric Association, 2013), is characterized by a pathological inability to discard objects, resulting in debilitating clutter (Frost and Gross, 1993). Historically, hoarding has been conceptualized as a subtype or dimension of obsessive–compulsive disorder (OCD). However, most individuals with HD do not meet other symptom criteria for OCD (Frost et al., 2011), most individuals with OCD do not report significant hoarding behaviors (Samuels et al., 2007), and hoarding demonstrates weak correlations with classic OCD symptoms (Wu and Watson, 2005; Abramowitz et al., 2008). Furthermore, the prevalence of HD may actually be higher than that of OCD (Samuels et al., 2008).

Preliminary evidence suggests that the neural underpinnings of HD and OCD may differ as well. OCD is most robustly characterized by hyperactivity in the orbitofrontal–striatal loop (Whiteside et al., 2004; Rotge et al., 2008), although a dorsolateral prefrontal–striatal loop has also been implicated (Menzies et al., 2008; Rotge et al., 2008). Hyperactivity in these loops may project to other structures such as the hippocampus, anterior cingulate cortex (ACC), and basolateral amygdala (Rauch et al., 1994; Saxena et al., 1998; Adler et al., 2000; Friedlander and Desrocher, 2006; Simon et al., 2010). Neuroimaging studies of HD using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) suggest a different neural dysfunction. At rest, OCD patients with prominent hoarding symptoms show low baseline glucose metabolism in the ACC (Saxena et al., 2004); during symptom provocation (imagined or real discarding of possessions), excessive hemodynamic activity is seen in the ventromedial prefrontal cortex (VMPFC) in OCD patients with hoarding symptoms (An et al., 2009). Finally, excessive orbitofrontal cortex (OFC) activity is noted in primary HD patients (Tolin et al., 2009).

To date, there have been few direct comparisons between OCD patients and HD patients. Most previous studies sampled OCD patients with and without hoarding symptoms (Saxena et al., 2004; An et al., 2009), which might not be representative of the majority of HD patients who do not have OCD. To our knowledge, only one neuroimaging study to date has compared primary HD patients and OCD patients; compared with OCD patients and healthy control participants, HD patients were characterized by a biphasic abnormality in the insula and ACC during a hoarding-relevant decision-making task (Tolin et al., 2012b). Additional comparison studies are needed to understand the neural similarities and differences between HD and OCD. It is noted that the previous comparison study used a hoarding-specific task, and OCD patients showed an overall lack of activation on that task. It would

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be helpful, therefore, to compare the neural function of HD and OCD patients using a task that has been shown to selectively activate neural regions of interest (ROIs) in OCD patients.

Neural mechanisms of executive functions are a promising area for comparison between HD and OCD. Executive functions have been identified as the primary area of neuropsychological deficit in OCD patients (Olley et al., 2007; Kashyap et al., 2013). These executive deficits include impairments in behavioral response inhibition (Morein-Zamir et al., 2010). Studies of executive function in HD patients have yielded mixed results (Grisham et al., 2007, 2010; Tolin et al., 2011), and it is not clear whether that disorder is characterized by the presence of executive deficits, although a problem of motor inhibition seems particularly unlikely. Of note, patterns of neural activity underlying executive functions may differ between OCD patients and healthy controls. During a Go/NoGo task, in which participants must alternatively execute or inhibit a prepotent, planned response (e.g., button press) based on stimulus presentation, on correctly rejected NoGo trials OCD patients ($n=11$) showed excessive activation in several frontal and striatal brain regions compared with healthy control subjects. These included rostral and caudal ACC, lateral prefrontal cortex (LPFC), lateral orbitofrontal cortex (LOFC), caudate, and thalamus, as well as portions of the posterior cingulate cortex (PCC) (Maltby et al., 2005). Furthermore, OCD severity was positively correlated with activity in the PCC on these correctly rejected NoGo trials. During errors of commission (button press following a NoGo stimulus), OCD patients showed excessive activation in rostral ACC, LOFC, LPFC, and PCC compared with healthy controls (Maltby et al., 2005). The authors of that study concluded that because of the high conflict from prepotent response tendency created by the task instructions, OCD patients might have selectively activated error-monitoring regions even in the absence of actual errors, which could help explain the repetitive nature of compulsive behaviors. A higher conflict (lower ratio of NoGo to Go trials) study (Page et al., 2009), in which response inhibition was contrasted with response execution (NoGo > Go), also found excessive activity in unmedicated OCD patients ($n=10$) vs. healthy controls in the PCC, as well as in the right VMPFC and premotor cortex. However, contrary to the results of Maltby et al. (2005), OCD patients showed attenuated activity in the ventromedial OFC, ACC, caudate, and thalamus. The higher response conflict in that study could have elicited "oddball" effects (Stevens et al., 2000). Furthermore, it is likely that the NoGo > Go contrast more selectively examines mechanisms of response inhibition than of error monitoring. It is also noted that 1 of the 10 OCD subjects in that trial had prominent hoarding symptoms. In a substantially lower conflict task (equal ratio of Go to NoGo trials), during NoGo > Go trials), OCD patients ($n=12$) showed diminished activity (compared with healthy controls) in right medial and inferior frontal gyri, precentral and postcentral gyri, superior temporal gyrus, and fusiform gyrus. In addition, OCD severity correlated inversely with NoGo > Go activity in right OFC and ACC, and positively with thalamic and posterior cortical activations. OCD patients showed excessive activity in left insula, lingual gyrus, and head of the caudate (Roth et al., 2007). Neither depressed mood nor medication status appeared to mediate the group differences. As those authors note, due to the equal ratio of NoGo to Go trials, errors were quite infrequent and therefore that study may have shown effects more consistent with response inhibition than with error monitoring.

Thus, across studies, OCD patients exhibit abnormal neural activity during NoGo trials, although methodological differences among the studies preclude many direct comparisons. Results could be related to hyperactive error monitoring (Maltby et al., 2005), exaggerated oddball effects (Page et al., 2009), or under-activation of response–inhibition mechanisms (Roth et al., 2007). The aim of the present study is to compare, using a larger number of participants than in previous trials, the hemodynamic responses of HD patients, OCD patients, and healthy controls during NoGo trials, using a high-conflict Go/NoGo task that has previously proved sensitive to OCD (Maltby et al., 2005). It was predicted that during correct reject trials (successful response inhibition), OCD patients would show excessive activity in ACC, LOFC, caudate, and thalamus compared with the other two groups. During errors of commission (failed response inhibition), OCD patients were expected to show excessive activity in ACC, LOFC, LPFC, and PCC compared with the other two groups. HD participants were expected not to show the same pattern of hyper-activation abnormalities. Rather, based on previous studies not involving hoarding-related decisions (Saxena et al., 2004; Tolin et al., 2012b), it was expected that HD patients would show decreased activity, compared with the other two groups, in ACC, PCC, and insula.

2. Methods

2.1. Participants

Participants comprised 24 patients with HD, 24 patients with OCD, and 24 healthy controls. All provided written, informed consent in accordance with Hartford Hospital IRB procedures. Patients with HD were recruited using advertisements for people with "clutter problems" or "hoarding," as well as from an existing patient group at a clinic specializing in HD treatment. The patients with OCD were recruited using advertisements seeking people with OCD, and the healthy controls were recruited using advertisements for a brain-imaging study. All assessments were conducted by well-trained postdoctoral fellows or postgraduate research assistants. Participants were classified as having HD if they met diagnostic criteria (Frost and Hartl, 1996; American Psychiatric Association, 2013), hoarding was their primary diagnosis as defined by clinical severity ratings on the Anxiety Disorders Interview Schedule for DSM-IV (Brown et al., 1994), the Clinician's Global Impression (Guy, 1976) rating was "moderately ill" or above, and symptom duration was at least 1 year. One potential participant with comorbid HD and OCD was excluded from the study, given that the primary study aim was to compare HD and OCD. Where there were questions about the severity of hoarding, symptom severity was confirmed via home visit or analysis of current photographs of living space. The patients with OCD met diagnostic criteria for a primary diagnosis of (nonhoarding) OCD, had at least moderate symptom severity as evidenced by a clinician's global impression rating of moderately ill or above, and had a symptom duration of 1 year or more. HD or OCD patients were excluded if they had a history of psychotic disorder, neurological disorder, substance abuse, or serious suicidal ideation. Healthy controls were excluded if they met criteria for a current or past Axis I or Axis II disorder, had a history of neurological disorders, or were taking psychiatric medications. Participants, regardless of diagnostic group, who were unsuitable for MRI scanning (e.g., those with severe claustrophobia, pregnancy, or metal implants) were also excluded.

2.2. Measures

Demographic information, including self-reported race and ethnicity, was collected via a questionnaire. The HD diagnoses were made using the Hoarding Rating Scale-Interview (Tolin et al., 2010b), a semistructured interview that assesses the severity of clutter, acquisition, difficulty discarding, distress, and impairment, each on a 0- to 8-point scale. Other psychiatric diagnoses were ascertained using the Anxiety Disorders Interview Schedule for DSM-IV (Brown et al., 1994). Severity of HD was assessed using the Saving Inventory-Revised (Frost et al., 2004), a 23-item questionnaire. Nonhoarding OCD severity was assessed using the Obsessive Compulsive Inventory-Revised (OCI-R) (Foa et al., 2002), an 18-item self-report measure. For the present purposes, given the intent to contrast OCD and HD, a total OCI-R score was calculated with omission of the three hoarding items. Depression severity was assessed using the Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1960), a 17-item semistructured interview. The structured interview guide for the HRSD (Williams, 1988) was used for administration. Global impressions of illness severity were recorded using the Clinician's Global Impression (Guy, 1976) scale.

2.3. Apparatus

Images were acquired on a Siemens Allegra 3T head-only scanner. A single-shot echo-planar gradient recalled pulse sequence (repetition time/echo time = 1500/28 ms; flip angle = 65°; field of view = 24 cm; matrix = 64; in plane resolution = 3.4 mm; slice

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