Poor memory confidence mediates the association between inattention symptoms and hoarding severity and impairment

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1. Introduction

Hoarding disorder (HD) is a costly and prevalent disorder that is defined primarily by difficulty discarding possessions, resulting in severe clutter that precludes use of living spaces (Frost & Gross, 1993; Frost & Hartl, 1996). Previously considered a subtype of obsessive-compulsive disorder (OCD), HD was classified as a distinct diagnostic entity in DSM-5. One reason for this reclassification was the observation that hoarding symptoms do not reliably co-occur with other OCD symptoms (e.g., excessive checking or cleaning) at elevated levels, as would be expected if hoarding was a subtype of OCD (Frost, Steketee, & Tolin, 2011; Pertusa et al., 2008). However, hoarding is characterized by elevated symptoms of depression and attention deficit-hyperactivity disorder (ADHD), particularly the inattentive subtype of ADHD (ADHD-I; Frost, Steketee, & Tolin, 2011; Hall, Tolin, Frost, & Steketee, 2013; Sheppard et al., 2010).

The elevated comorbidity between HD and ADHD-I has generated increased attention in recent years. Converging evidence from several studies suggests that between 20 and 33% of individuals with HD meet criteria for ADHD-I (Frost et al., 2011; Sheppard et al., 2010). Among individuals with clinically significant hoarding, the presence of inattention symptoms results in a more severe clinical presentation, including more problems with impulsivity, cognition, activities of daily living, and squalor, compared to HD without ADHD-I (Hall et al., 2013). Among individuals with HD, inattention (but not hyperactivity) is also associated with greater severity of core symptoms of hoarding disorder, including more severe clutter, acquiring, and excessive saving of possessions (Tolin & Villavicencio, 2011). ADHD symptoms also appear to differentiate individuals with and without clinically significant hoarding (Hacker et al., 2012).

Despite the clear association between HD and ADHD-I, there have been relatively few attempts to explain this elevated pattern of comorbidity, and to date no studies have attempted to examine mechanisms by which inattention symptoms may increase HD severity. A recent account points to shared neuropsychological and neurobiological impairments between HD and ADHD (Lynch, McGillivray, Molding, & Byrne, 2015). Neuropsychological findings in HD have been mixed, but several studies have linked HD to impairments in visuospatial memory (Blom et al., 2011; Hartl et al., 2004; Testa, Pantellis, & Fontenelle, 2011) and sustained attention (Grisham, Brown, Savage, Steketee, & Barlow, 2007; Tolin, Villavicencio, Umbach, & Kurtz, 2011; see Woody, Kellman-McFarlane, & Welsted, 2014 for a review). Similarly, adult ADHD is characterized by impairments in memory and sustained attention, among other
deficits (Hervey, Epstein, & Curry, 2004). HD and ADHD also show similar patterns of neurobiological dysfunction, including abnormal glucose metabolism in prefrontal cortex, anterior cingulate cortex, and the amygdala, regions associated with executive functioning and emotion regulation (see Lynch, McGillivray, Molding, & Byrne, 2015 for a brief review). Lynch et al. (2015) suggest that these neurobiological and neuropsychological deficits may represent a shared vulnerability to both HD and ADHD, perhaps reflecting a shared genetic vulnerability.

Although the above account highlights a plausible explanation for elevated rates of ADHD-I among individuals with HD, it does not describe potential mechanisms by which inattention symptoms are associated with increased severity and impairment in HD. There are many potential pathways by which this association could take place. We propose one such pathway, a causal model that is illustrated in Fig. 1. We first suggest that ADHD-I symptoms result in poor memory confidence and overreliance on visuospatial memory strategies. For the present paper, we operationalize poor memory confidence as high scores on the memory subscale of the Savings Cognitions Inventory (Frost et al., 2004), which includes items such as “my memory is so bad I have to leave this [object] in sight or I’ll forget about it,” “saving this [object] means I don’t have to rely on my memory,” and “if I put this [object] into a filing system, I will forget about it.”

To our knowledge, no study has examined memory confidence or maladaptive memory strategies such as saving in individuals with ADHD. However, ADHD symptoms are present since childhood by definition, and have been independently linked to global (Hervey et al., 2004) and visuospatial (Barnett, Maruff, & Vance, 2005) memory impairments. It therefore seems likely that inattention symptoms would lead to poor confidence in one’s ability to remember important information, by virtue of their association with genuine memory deficits. The present model proposes that poor memory confidence leads to increased saving of possessions to facilitate remembering, which in turn contributes to clutter and functional impairment. As an illustration of the proposed model, an individual with a longstanding history of inattention symptoms might have little confidence in her ability to remember a discussion with a colleague without keeping a tangible reminder of the conversation (e.g., notes taken during the meeting). Excessive reliance on this memory strategy, perhaps in the absence of a clear organizational system and in the presence of other HD risk factors, may lead to excessive clutter and corresponding functional impairment.

This suggestion – that poor memory confidence leads to excessive saving of possessions – is in keeping with the leading cognitive-behavioral model of HD, which proposes that HD symptoms arise in part from perceived or actual memory disturbances (Frost & Hartl, 1996). Poor memory confidence and exaggerated beliefs about the negative consequences of forgetting are prominent in HD (Frost & Hartl, 1996; Hartl et al., 2004). These negative memory beliefs are proposed to lead to increased saving of possessions and keeping possessions within sight to facilitate remembering, which in turn contributes to clutter. We extend this model by proposing that poor memory confidence is the result of inattention symptoms, which are present on a continuum in the population (Marcus & Barry, 2011), independently associated with poor memory (Hervey et al., 2004; Barnett et al., 2005), and elevated in HD even in the absence of a DSM-5 diagnosis of ADHD-I (Hartl, Duffany, Allen, Steketee, & Frost, 2005).

It is important to emphasize that this model represents one of many potential mechanisms of hoarding pathology; it is necessarily simplified in order to allow an empirical test of one potential causal pathway. We do not include the multitude of other risk factors that may play a role in HD, including genetic vulnerabilities, deficits in planning and organization, and maladaptive beliefs that are unrelated to memory (e.g., “I must not be wasteful”). Nor do we attempt to distinguish between perceived and actual memory deficits, both of which have been identified in HD (Hartl et al., 2004). Instead, this model represents a first attempt to explain the mechanisms linking inattention symptoms to increased HD severity. To our knowledge, this is also the first study to use mediation analyses to test claims made by the leading cognitive-behavioral model of HD (Frost & Hartl, 1996).

2. Method

2.1. Participants and procedure

Participants were treatment-seeking adults (age 20–60) with HD (n=32) and age-matched healthy controls with no current or past psychiatric disorders (n=26) who were recruited as part of a larger neuroimaging and treatment study. Both HD and healthy participants were included in the present study to ensure a full range of scores on the predictors and outcomes of interest, and because taxometric research indicates that hoarding is best conceptualized as a dimensional construct (Timpano et al., 2013). Inclusion criteria for the HD sample included a primary (most severe) hoarding diagnosis of at least moderate severity as assessed by a semi-structured diagnostic interview administered by a licensed psychologist. Exclusion criteria included a history of traumatic brain injury or loss of consciousness > 5 min, current (past 12 months) substance use disorder, a previous adequate course (> 10 sessions) of cognitive-behavioral therapy for HD, and MRI-related rule-outs (e.g., metal in the body). Certain medications (primarily selective serotonin reuptake inhibitors) were permitted provided the dose was stable for 8 weeks prior to participation.

2.2. Measures

2.2.1. Diagnostic status

HD and healthy control status were determined using a semi-structured diagnostic interview (the Diagnostic Interview for Anxiety, Mood, Obsessive-compulsive, and Related Neuropsychiatric Disorders [DIAMOND]; Gilliam et al., 2014; Tolin et al., in review), which was administered by a licensed clinical psychologist with considerable experience in diagnosing and treating hoarding. The DIAMOND, which assesses DSM-5 criteria for HD and other disorders, was administered by a licensed clinical psychologist. The DIAMOND shows good psychometric properties, including
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