

Recent Advances in Compulsive Hoarding

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Compulsive hoarding is a common and often disabling neuropsychiatric disorder. This article reviews the conceptualization, phenomenology, diagnosis, etiology, neurobiology, and treatment of compulsive hoarding. Compulsive hoarding is part of a discrete clinical syndrome that includes difficulty discarding, urges to save, excessive acquisition, indecisiveness, perfectionism, procrastination, disorganization, and avoidance. It was thought to be part of obsessive-compulsive disorder or obsessive-compulsive personality disorder, but recent evidence indicates that it should be classified as a separate disorder with its own diagnostic criteria. Compulsive hoarding is a genetically discrete, strongly heritable phenotype. Neuroimaging and neuropsychological studies are elucidating its neurobiology, implicating dysfunction of ventral and medial prefrontal cortical areas that mediate decision-making, attention, and emotional regulation. Effective treatments include pharmacotherapy and cognitive-behavioral therapy. More research will be required to determine the prevalence, etiology, and pathophysiology of compulsive hoarding and to develop better treatments.

Introduction

Hoarding is defined as the acquisition of and inability to discard items even though they appear (to others) to have no value [1]. Hoarding behavior has been observed in several neuropsychiatric disorders, including schizophrenia, dementia, autism, and mental retardation, as well as in nonclinical populations [2], but it is most commonly associated with obsessive-compulsive disorder (OCD). Approximately 18% to 42% of patients with OCD have hoarding and saving symptoms [2,3,4•].

Although standard diagnostic classifications consider OCD to be a single diagnostic entity, factor analyses of OCD symptoms have consistently identified at least four principal symptom factors: 1) aggressive, harm-related, sexual, and religious obsessions with checking compulsions; 2) symmetry and order obsessions with arranging, repeating, and counting compulsions; 3) contamination obsessions with washing and cleaning compulsions; and 4) compulsive hoarding and saving symptoms [5,6]. These symptom factors appear to be relatively stable over time and show different patterns of genetic inheritance, comorbidity, and treatment response [6]. Cluster analyses, which identify mutually exclusive, categorical subgroups, indicate that some of these symptom factors, including hoarding, may constitute discrete subtypes of OCD [7].

The hoarding symptom factor has been identified in phenomenologic and epidemiologic studies of OCD in the United States, Brazil, Canada, Costa Rica, France, Germany, Italy, Japan, the Netherlands, Poland, Turkey, Egypt, Singapore, and South Africa, where it is found in both blacks and whites [6,8]. Thus, compulsive hoarding is not a culture-bound syndrome.

The Compulsive Hoarding Syndrome

Frost and Hartl [9] developed the first systematic definition and diagnostic criteria for clinically significant compulsive hoarding: 1) the acquisition of and failure to discard a large number of possessions that appear (to others) to be useless or of limited value, 2) living or work spaces sufficiently cluttered so as to preclude activities for which those spaces were designed, and 3) significant distress or impairment in functioning caused by the hoarding behavior or clutter. They found that hoarding and saving symptoms are part of a discrete clinical syndrome that includes the core symptoms of urges to save, difficulty discarding, and excessive acquisition but also indecisiveness, perfectionism, procrastination, disorganization, and avoidance [2]. In addition, many compulsive hoarders are slow to complete tasks, are frequently late for appointments, and display circumstantial and overinclusive language. Patients with prominent hoarding and saving symptoms who display these other associated symptoms thus are considered to have the compulsive hoarding syndrome [2,10].

Compulsive hoarding is most commonly driven by obsessional fears of losing important items that the patient believes will be needed later or making the “wrong” decision about what to keep and what to discard. These fears cause substantial distress and lead to compulsions to acquire and save items to prepare for every imaginable contingency. Hoarders also frequently have excessive emotional attachments to possessions and distorted beliefs about possessions’ importance [1], leading to excessive saving. The consequent clutter can cause significant social and occupational impairment [2,10]. In severe cases, it can produce health risks from infestations, falls, fires, and inability to cook or eat in the home [2]. Avoidance is prominent and includes behavioral avoidance of discarding items, putting things away, or cleaning, as well as cognitive avoidance of making decisions or even thinking about the clutter or its consequences.

The mean age at onset of compulsive hoarding symptoms is 12 to 13 years [11•,12,13]. Difficulty discarding and clutter usually precede excessive acquiring [11•]. Symptoms generally worsen from mild during the teenage years to moderate when patients are in their 20s to severe when they are in their 30s. Recognition of symptoms develops last, generally not until patients reach their 30s and 40s [11•].

Diagnostic Classification of Compulsive Hoarding

Relationship of compulsive hoarding to OCD

Although compulsive hoarding long has been considered a subtype or symptom factor within OCD, recent evidence suggests otherwise. Whereas the harm/checking, contamination/cleaning, and symmetry/rituals symptom factors are strongly intercorrelated, hoarding/saving symptoms do not correlate strongly with the other major factors in clinical and nonclinical samples [6,14]. Patients with OCD do not report more hoarding symptoms than healthy controls or patients with other disorders [14,15]. Moreover, many compulsive hoarders have no other OCD symptoms [1,2,14]. A recent taxometric analysis of OCD symptoms found that hoarding showed significant evidence of taxonicity, indicating that it constituted a categorical latent subclass; the other OCD symptoms were found to be dimensional, varying by degrees along a continuum [16••]. The taxonic latent structure of compulsive hoarding indicates that it is a discrete categorical entity that may have an etiologic mechanism distinct from that of other OCD symptoms [16••]. Taken together, these findings call into question the idea that compulsive hoarding is simply part of OCD and suggest that it may be a separate but related OCD spectrum disorder that is frequently comorbid with OCD, similar to the way body dysmorphic disorder and trichotillomania are now conceptualized [17].

The phenomenology of compulsive hoarding is consistent with its conceptualization as an OCD spectrum disorder, as its core features include obsessions, compulsions, and avoidance [9]. However, compulsive hoarding also has similarities to impulse control disorders. Many hoarders are prone to

excessive buying, excessive acquisition of free items, and even shoplifting [2]. These behaviors are ego-syntonic in some patients, who derive pleasure from acquisition.

Hoarding versus nonhoarding OCD

OCD patients with hoarding symptoms differ from nonhoarding OCD patients in many important ways. They have earlier age at onset but older age when presenting for treatment [2,10]; greater prevalence of symmetry, ordering, counting compulsions, and indecisiveness [4•,12]; more anxiety, depression, schizotypal, and dependent personality disorder symptoms [2,4•,8,12]; and less insight than nonhoarding OCD patients [4•]. Child and adolescent OCD patients with significant hoarding symptoms were found to have more magical thinking obsessions, ordering/arranging compulsions, anxiety, aggression, somatic complaints, overall externalizing and internalizing symptoms, and worse insight than nonhoarding children with OCD [18]. Compared with nonhoarding OCD patients, compulsive hoarders have more severe family and social disability and lower global functioning [2,10,13]. Hoarding OCD patients are less likely to be married [12,13], have significantly lower incomes [13], and report significantly more traumatic life events than nonhoarding OCD patients [19]. They also show a different pattern of comorbidity than nonhoarding OCD patients, with significantly greater prevalence of social phobia, generalized anxiety disorder, specific phobias, bipolar disorder, dysthymia, brief depression, personality disorders, and pathologic grooming disorders [4•,9,12,13]. Child and adolescent OCD patients with significant hoarding symptoms have higher rates of panic disorder than nonhoarding children with OCD [18].

Compulsive hoarding versus obsessive-compulsive personality disorder

Currently, the inability to discard worthless items is listed in the *DSM-IV* as a symptom of obsessive-compulsive personality disorder (OCPD). However, the available evidence argues strongly against classifying hoarding as part of OCPD. Compulsive hoarders have been found to have no more OCPD traits than controls [2], and only a small percentage of them have comorbid OCPD [4•]. Of the current diagnostic criteria for OCPD, inability to discard worthless items has the lowest specificity, positive predictive value, and total predictive value [20]. Conversely, it is the best at discriminating among OCD, panic disorder, and major depressive disorder diagnoses [21]. In a student sample, hoarding severity correlated with scores on an OCD inventory but not with scores on an OCPD scale [1]. Taken together, these findings indicate that hoarding should be removed from the diagnostic criteria for OCPD [17].

Etiology

Family and genetic studies

Compulsive hoarding clearly runs in families. About 84% to 85% of hoarders report having a first-degree relative

who was a “pack rat,” whereas only 37% to 54% report having a family member with a different OCD [1,22]. Relatives of hoarding OCD patients have significantly higher prevalence of hoarding symptoms, dysthymia, and indecisiveness than relatives of nonhoarding OCD patients [4•,13]. Hoarding symptoms in relatives are related to indecisiveness in probands, suggesting that indecisiveness may be a risk factor for compulsive hoarding. The hoarding symptom factor is strongly familial, with robust correlations among sibling pairs [23], and shows an autosomal recessive inheritance pattern [4•].

Only a few genetic studies have examined compulsive hoarding. A genome-wide scan in sibling pairs with Tourette’s syndrome found that the hoarding phenotype was significantly associated with genetic markers on chromosomes 4, 5, and 17 [24]. The met/met (L/L) genotype of the COMT Val158Met polymorphism on chromosome 22q11 was found to be significantly more prevalent in Afrikaner OCD patients with hoarding symptoms than in Afrikaner nonhoarding OCD patients or controls [8]. The OCD Collaborative Genetics Study found “suggestive” linkage of compulsive hoarding to a marker on chromosome 14 in families with early-onset OCD [25••]. These findings indicate that compulsive hoarding is a genetically discrete phenotype.

Hoarding secondary to brain lesions

There have been several case reports of compulsive hoarding caused by brain damage. Patients have developed compulsive hoarding and collecting behaviors after damage to the orbitofrontal cortex (OFC) and medial prefrontal cortex (mPFC) caused by cerebral hemorrhage from ruptured anterior communicating artery aneurysms [26,27], resection of olfactory meningioma [28], and frontotemporal dementia [29]. Anderson et al. [30•] compared nine patients with compulsive hoarding that began after brain damage with 54 nonhoarding brain-damaged patients. All hoarding patients had damage to the prefrontal cortex, mostly in the medial and inferior areas. The greatest lesion overlap in hoarders was in the right mPFC, orbitofrontal pole, anterior cingulate cortex (ACC), and adjacent white matter. These reports consistently demonstrate that compulsive hoarding can result from localized damage to the OFC, mPFC, ACC, and frontal poles. These brain regions mediate performance on decision-making tests, on which compulsive hoarders perform worse than nonhoarding OCD patients [31•], underscoring the fundamental relationship between indecisiveness and compulsive hoarding.

Functional Neuroanatomy of Compulsive Hoarding

Cerebral glucose metabolism in compulsive hoarding

Our group conducted the first brain imaging study of compulsive hoarders and found that they had a differ-

ent pattern of baseline cerebral glucose metabolism than normal controls and nonhoarding OCD patients [32]. Compulsive hoarders did not have the characteristic hypermetabolism in the OFC, caudate, and thalamus seen in nonhoarding OCD patients [33]. Instead, they showed significantly lower metabolism in the posterior cingulate cortex compared with controls. Across all patients studied, greater hoarding severity was correlated with lower activity in the dorsal ACC and anterior medial thalamus. These results suggested that compulsive hoarding was neurobiologically distinct from OCD [32]. However, this study had several limitations that affected its interpretability. Hoarding and nonhoarding OCD patients were divided retrospectively and were originally recruited and enrolled based on having OCD, not hoarding symptoms. Hoarders were significantly older than controls and nonhoarding OCD patients and had a much higher proportion of women than nonhoarding OCD patients. Therefore, we sought to replicate our findings in a new, larger sample of compulsive hoarders and matched controls, free of the confounds present in the initial study.

We measured cerebral glucose metabolism with [¹⁸F]-fluorodeoxyglucose positron emission tomography in medication-free adults with compulsive hoarding syndrome, compared it with that of age- and gender-matched healthy controls, and found that compulsive hoarders had significantly lower normalized glucose metabolism in the bilateral dorsal and ventral ACC than controls (Saxena et al., unpublished data). Greater hoarding severity was significantly correlated with lower relative activity in the right dorsal ACC, right posterior cingulate cortex, and bilateral putamen. As in our previous study, no differences were found in brain regions usually associated with OCD or major depression. Thus, compulsive hoarding appears to be a neurobiologically distinct disorder with a unique pattern of abnormal brain function that does not overlap with that of nonhoarding OCD (Saxena et al., unpublished data).

These findings have important implications not only for classifying compulsive hoarding but also its neurobiology and treatment. The dorsal ACC is involved in decision-making, attention, reappraising aversive stimuli, and suppressing negative affect. Thus, dysfunction of the dorsal ACC may mediate the difficulty in making decisions and attentional problems seen in compulsive hoarders [31•,34] and also could account for hoarders’ inability to control their fears and distress about losing possessions to which they have sentimental attachments or that they consider potentially useful or valuable. Treatments that increase ACC activity, such as cholinesterase inhibitors, stimulants, or modafinil, may be effective for the compulsive hoarding syndrome (Saxena et al., unpublished data).

Neural correlates of hoarding symptom provocation

Symptom provocation neuroimaging studies reveal patterns of brain activation occurring as patients actively experience

symptoms. Symptom provocation studies of OCD have consistently found activation of the OFC, caudate, and thalamus during the provoked state—usually greater in patients with OCD than in controls—with less consistent activation of the ACC and other regions [33]. However, only two studies to date have investigated brain activation during provocation of compulsive hoarding symptoms.

Mataix-Cols and colleagues [35] provoked hoarding/saving, contamination/cleaning, and aggressive/checking symptoms in patients with OCD using photographs intended to provoke specific obsessional concerns and compulsive urges. During the hoarding-related provocation, patients with OCD showed significantly greater activation of the left dorsal motor/premotor cortex, right OFC, and left fusiform gyrus than controls. The degree of provoked hoarding-related anxiety correlated with the magnitude of activation of the left dorsal motor/premotor cortex. In contrast, provoked contamination-related anxiety correlated with activation of different brain regions—the fusiform and lingual gyri, right superior temporal gyrus, right inferior frontal gyrus, and right anterior insula—whereas harm/checking-related anxiety correlated with activation of the lateral prefrontal cortex, putamen, globus pallidus, and left thalamus [35]. This study was compromised by the fact that not all the patients with OCD had the symptoms the investigators were attempting to provoke, so not all of them became anxious or symptomatic during the provocation.

The same research group then provoked hoarding/saving symptoms in OCD patients with and without prominent hoarding symptoms and normal controls by having them view pictures of commonly hoarded objects while imagining that these objects belonged to them and that they “must throw them away forever” [36•]. OCD patients with prominent hoarding symptoms showed significantly greater activation of the bilateral frontal pole and anterior mPFC than nonhoarding OCD patients and controls and greater cerebellar activation than controls. Hoarding and nonhoarding OCD patients showed significantly less activation of the left OFC than controls. Across all OCD patients studied, provoked hoarding-related anxiety correlated with activation of the left ventromedial PFC, right ventrolateral PFC, bilateral hippocampus and mesial temporal cortex, right amygdala, left thalamus, bilateral sensory-motor cortex, and bilateral cerebellum. Provoked anxiety was negatively correlated with activation of the left dorsal ACC, bilateral temporal cortex, dorsolateral PFC, and various parieto-occipital cortical regions. Hyperactivation of the ventromedial PFC, a region involved in decision-making about potential gains and losses, may reflect compulsive hoarders’ greater difficulties in deciding upon the value or importance of objects they were imagining having to discard, whereas relative underactivation of the dorsal ACC, dorsolateral PFC, and parieto-occipital cortex may reflect deficient emotional regulation and planning abilities [36•].

Neuropsychological studies

Compulsive hoarders often report problems with attention and memory and appear to have some neurocognitive deficits. Compared with normal controls, compulsive hoarders had worse delayed visual and verbal recall and used less effective organizational strategies for visual recall [34]. Hoarders also reported significantly less confidence in their memory and more catastrophic assessments of the consequences of forgetting. In addition, compulsive hoarders have been found to have slower reaction time, greater impulsivity, and worse spatial attention than clinical comparison patients and normal controls [37•]. Hoarding OCD patients reported significantly more difficulty making decisions than nonhoarding OCD patients [2,4•] or healthy controls, who did not differ from each other [2]. OCD patients with prominent hoarding symptoms showed impaired decision-making performance and a qualitatively different pattern of autonomic skin conductance responses during a gambling task, whereas low-hoarding or nonhoarding OCD patients showed normal performance [31•].

Together, the results of neuroimaging and neuropsychological studies demonstrate that the neurobiology of compulsive hoarding is distinct from that of nonhoarding OCD. The pathophysiology of compulsive hoarding involves abnormalities in the neural systems mediating attention, decision-making, and emotional regulation. Along with genetic findings [8,25••] and the taxometric study showing that compulsive hoarding is a discrete categorical entity [16••], these data suggest that compulsive hoarding should be classified as a separate disorder with its own diagnostic criteria in the *DSM-V*.

Treatment of Compulsive Hoarding Pharmacotherapy

Some, but not all, studies investigating the influence of OCD symptom factors on treatment response have found that hoarding symptoms were associated with poor response to pharmacotherapy with serotonin reuptake inhibitors (SRIs). One small study found that nonresponders to paroxetine or placebo for OCD were significantly more likely to have hoarding symptoms than responders [38]. In a case series of 18 compulsive hoarders treated openly with a variety of SRIs, only one patient had an adequate response, eight had partial responses, and nine had no response [22]. Higher scores on the hoarding symptom factor predicted poorer response in an analysis of placebo-controlled trials of SRI treatment for OCD patients (after controlling for baseline severity) [39]. A recent study found that high scores on a hoarding/symmetry factor predicted worse outcome in a double-blind trial of citalopram versus placebo for OCD [40].

However, several other studies that examined OCD symptom factors and treatment response did not confirm this association. Instead, sexual/religious obses-

sions uniquely predicted poorer long-term outcome after SRI treatment in one study [41] and were the only OCD symptoms that were significantly more common in treatment-refractory OCD patients than in treatment responders in another [42]. Poor insight and somatic obsessions were significantly more common in nonresponders to SRIs than responders in one study [43], whereas sexual obsessions, washing compulsions, and miscellaneous compulsions predicted nonresponse to SRIs in another [44]. These studies all found no significant effect of hoarding/saving symptoms on response to SRI treatment. In addition, a family study that compared hoarding OCD patients with nonhoarding OCD patients found that a very similar proportion of patients in the two groups reported response or remission with SRI treatment [5]. Thus, compulsive hoarding does not appear to be a consistent predictor of poor response to SRI medications.

Only one study to date has prospectively and quantitatively measured response to standardized pharmacotherapy in compulsive hoarders [45••]. Thirty-two patients with the compulsive hoarding syndrome and 47 nonhoarding OCD patients were treated openly with paroxetine monotherapy (mean dose, 41.6 ± 12.8 mg/d) for 12 weeks. The severity of compulsive hoarding symptoms was specifically quantified before and after treatment using the UCLA Hoarding Severity Scale [45••]. Compulsive hoarders responded as well to paroxetine as nonhoarding OCD patients, with significant and nearly identical improvements in OCD symptoms, depression, anxiety, and overall functioning. A similar proportion of hoarding and nonhoarding OCD patients were strong responders (28% vs 32%) and partial responders (22% vs 15%). The proportion of dropouts was also similar (22% vs 15%). Compulsive hoarders who completed treatment showed a mean 31% decline in Yale-Brown Obsessive Compulsive Scale scores. Hoarding/saving symptoms improved as much as other OCD symptoms. No correlation was found between hoarding severity and treatment response. These results suggest that SRI medications are just as effective in compulsive hoarders as they are in nonhoarding OCD patients [45••].

Cognitive-behavioral therapy

Hoarding symptoms have been associated consistently with poor response and premature dropout from cognitive-behavioral therapy (CBT) for OCD. OCD patients with prominent hoarding symptoms have been found to be more likely than nonhoarding OCD patients to drop out of CBT prematurely [46], less likely to respond to outpatient CBT [46,47], and less likely to respond to intensive inpatient CBT [48]. Higher scores on the hoarding symptom factor predict premature dropout [46] and nonresponse to CBT [48].

Frost and Hartl [9] developed a CBT treatment strategy based on their cognitive-behavioral model of compulsive hoarding. Their approach includes cognitive restructuring, decision-making training, and exposure and response pre-

vention involving discarding of saved clutter. They treated 14 unmedicated compulsive hoarders with 26 individual sessions of CBT—including frequent home visits—over 7 to 12 months [49•]. In the 10 patients who completed treatment, significant pretreatment to post-treatment decreases were noted in hoarding severity and clutter but not in global clinical severity. Treatment completers showed a mean 28% decline in hoarding severity. After treatment, 50% of treatment completers were rated as “much improved” or “very much improved” [49•].

Our group developed an intensive, multimodal treatment protocol for compulsive hoarding based on Frost and Hartl’s [9] model but modified it for use in a short-term, intensive treatment setting; we broadened it by including medication treatment, structuring daily activities, and involving families in treatment [10]. We studied 190 patients with OCD, 20 of whom had the compulsive hoarding syndrome. All patients were treated for 6 weeks in a Partial Hospitalization Program with intensive, daily CBT; the vast majority received medication. Although previous trials of SRIs or outpatient CBT had failed in most of the compulsive hoarders, they showed significant improvement, with a mean 35% decrease in OCD severity. A total of 45% of hoarders were classified as responders to treatment. However, nonhoarding OCD patients had significantly greater improvement, with a mean 46% decrease in OCD severity [10].

Unfortunately, there have been no controlled trials of pharmacotherapy or CBT for compulsive hoarding. Based on the open trials summarized previously, it appears that SRI medications and CBT are effective treatments for compulsive hoarding, but combined, multimodal treatment is more effective than medication or CBT alone.

Conclusions

The evidence summarized in this article strongly suggests that compulsive hoarding syndrome is a discrete entity with a unique profile of core symptoms, associated features, genetic markers, and neurobiologic abnormalities that differ from those of OCD. Therefore, compulsive hoarding syndrome should be listed as a separate disorder in the *DSM-V*, with its own diagnostic criteria.

There is much we do not know about compulsive hoarding. No epidemiologic studies of compulsive hoarding exist, so we do not know its prevalence or gender distribution. However, it is likely to be much more common than previously thought. No studies have examined brain structure, neurochemistry, immune function, or non-SRI medications in compulsive hoarding.

Prior studies of OCD had important flaws that seriously limited their applicability and usefulness regarding compulsive hoarding. The *DSM-IV* criteria for OCD do not even mention hoarding. Most prior studies of OCD used diagnostic or screening instruments that excluded the many patients with compulsive hoarding but no other

OCD symptoms. Many neurobiologic studies of OCD have not included hoarders. Most treatment studies of OCD have not focused on treatment of hoarding symptoms. Even the many studies comparing OCD patients with and without hoarding symptoms examined a phenotype defined by the presence of any hoarding symptoms rather than clinically significant compulsive hoarding. Hence, differences between groups were likely diluted by the inclusion of patients in the “hoarding OCD group” who had only mild hoarding symptoms [20]. Future studies should examine the more well-defined categorical phenotype of compulsive hoarding syndrome to improve their chances of identifying its specific genetic and neurobiologic substrates.

Much more research needs to be done to elucidate the epidemiology, etiology, and pathophysiology of compulsive hoarding. We must determine its prevalence and gender ratio in population-based studies, not just in clinical samples or patients with OCD. We must investigate compulsive hoarding in older adults and determine whether late-onset hoarding differs from early-onset hoarding. We must determine what proportion of compulsive hoarders have structural brain lesions and how they are involved in the pathophysiology of compulsive hoarding. Neuroimaging studies must identify biologic markers that may aid in diagnosing compulsive hoarding and providing targets of treatment. Most importantly, better treatments must be developed for this common and disabling disorder.

Disclosure

No potential conflict of interest relevant to this article was reported.

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