

RUNNING HEAD: SYMPTOMS OF HYPOCHONDRIA IN OCD

Symptoms of hypochondriasis in Obsessive-Compulsive Disorder:
Prevalence, Treatment Outcome, and Relationship with Metacognition

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Synne Borgejordet

Norwegian University of Science and Technology

Authors Note

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Abstract

The study aimed to examine symptoms of hypochondriasis in relation to obsessive-compulsive disorder (OCD), by investigating the prevalence of hypochondriacal symptoms in OCD, the effect hypochondriacal symptoms has on OCD treatment outcome, and how OCD treatment affects hypochondriacal symptoms. Another objective of the study was to explore the role of metacognition in symptoms of hypochondria. A sample of 313 patients with a primary diagnosis of OCD and 382 community controls were compared on the Whiteley Index of hypochondriasis, the Metacognitions Questionnaire, and the Yale-Brown Obsessive-Compulsive Scale. Results showed that the prevalence of hypochondriasis symptoms was 30.7% in the OCD sample, significantly higher than in the control group. Furthermore, symptoms of hypochondria did not have a significant effect on OCD treatment outcome. Hypochondriacal symptoms improved moderately following OCD treatment. Finally, symptoms of hypochondriasis were found to be moderately correlated with metacognition and improvement in hypochondriasis was associated with changes in metacognitive beliefs. Implications for treatment and future research is discussed.

Keywords: hypochondriasis, obsessive-compulsive disorder, comorbidity, and metacognition.

Hypochondriasis describes a condition where one is preoccupied with fears and ideas of having a serious disease due to misinterpretation of somatic symptoms (American Psychiatric Association [APA], 1994). The preoccupation lasts for at least six months and persists despite medical reassurance that there is no underlying disease warranting the experienced distress. Because those who have hypochondria are usually disinclined to regard their problems as anything other than physical, they typically surface in primary and specialty medical settings rather than in psychiatric clinics. Moreover, they usually make frequent telephone inquiries and office visits concerning relatively minor or undetectable symptoms, seeking constant reassurance that they are not severely ill (Abramowitz, 2005). The prevalence of hypochondriasis in the normal population is unknown, but in general medical practice it has been reported to be between 4 and 9% (APA, 1994). The relatively high prevalence rate, reluctance to accept referral to psychiatric clinics, and excessive health care utilization have made hypochondriacs a social concern both from a public health, and cost effectiveness perspective (Greeven et al., 2007).

For a long time, hypochondriasis had a reputation of being chronic and relatively refractory to treatment (Greeven et al., 2007). However, relatively recent advances in cognitive-behavioral conceptualizations have led to more effective management (Beck, Emery, & Greenberg, 1985; Bouman & Visser, 1998; Speckens et al., 1995; Warwick & Salkovskis, 1990). According to the cognitive-behavioral model, a hypochondriacal “loop” starts out with bodily sensations triggering catastrophic misinterpretations. These misinterpretations are hypothesized to elicit anxiety. The anxiety is in turn assumed to initiate maladaptive hypochondriacal behavior such as checking, reassurance seeking, and avoidance of disease related stimuli. Furthermore, hypochondriacal behavior is thought to result in selective attention towards bodily sensations and disease-related stimuli, which further increases anxiety and causes more bodily symptoms. Essentially, the hypochondriac ends up being caught in a vicious circle where the problem is maintained by erroneous beliefs about health as well as maladaptive behavioral responses. Cognitive-behavioral therapy (CBT) is currently established as the leading treatment for hypochondriasis (Kroenke, 2007; Seivewright et al., 2008). Predominantly, CBT treatment constitutes techniques helping patients recognize and modify erroneous beliefs about illness, such as “all bodily sensations are signs of serious illness” (Abramowitz, 2005). Furthermore, CBT attempts to eliminate behavioral responses considered barriers to self-correction of faulty beliefs.

The observation of overlapping features in hypochondriasis and obsessive-compulsive disorder (OCD) has resulted in an upsurge of interest in how the two disorders are related

(Savron et al., 1996). OCD is characterized by the presence of obsessions and compulsions (APA, 1994). An obsession is a thought, image or impulse that repeatedly appears in a person's consciousness in a stereotypic form. Obsessions are experienced as anxiety provoking because their content is usually violent or obscene, or perceived as meaningless. Examples of obsessions are impulses to harm one's own children, and thoughts of contamination. The person experiencing obsessions usually attempts to ignore or neutralize them by respectively avoiding triggers, using thought suppression, or performing a compulsion. A compulsion is a specific behavior or mental act, which has to be performed in a rigid manner in response to an obsession. Examples of compulsive behaviors are checking, ordering, and washing, while compulsive mental acts can be counting, praying, or silently repeating words. The purpose of a compulsion is to prevent anxiety or a feared event from happening, both usually having their origin in a previous obsession. However, the behavioral or mental act of a compulsion will usually be either clearly excessive or not realistically designed considering what the person wants to prevent.

Hypochondriasis resembles OCD in that anxiety is evoked by specific reoccurring intrusive thoughts or ideas and reduced by ritualistic reassurance seeking, and symptom checking (Fallon, Javitch, Hollander, & Liebowitz, 1991; Neziroglu, McKay, & Yaryura-Tobias, 2000; Warwick & Salkovskis, 1990). Furthermore, patients with OCD often fear illness, injury or contamination, which is considered the core theme of the hypochondriasis diagnosis (Savron et al., 1996). In fact, 55% of OCD patients have been reported to have contamination obsessions, while 34% are suggested to have somatic obsessions (e.g. fear of breast cancer, with a resulting compulsion to check for this) (Rasmussen & Tsuang, 1986). Furthermore, 47.6% of OCD patients conduct washing/cleaning compulsions (Ball, Baer, & Otto, 1996) typically associated with contamination-related obsessions (Tolin & Meunier, 2008).

However, there are also important differences distinguishing hypochondriasis and OCD. The main difference is that obsessions related to illness and disease are typically characterized by fear of getting sick in the future, rather than having become ill already, which is the hypochondriac's conviction (Fallon, Klein, & Liebowitz, 1993). Also, OCD patients generally have more insight in their illness than hypochondriacs, being aware that their thoughts and actions are unrealistic (Neziroglu et al., 2000).

The similarities and differences between hypochondriasis and OCD have resulted in a debate regarding whether they're related or not. In conjunction with this, it's discussed if hypochondriasis might be best defined as a subtype of OCD (focused on illness fears and

bodily sensations), or should belong to the obsessive-compulsive spectrum, which is a collective term for disorders seemingly closely related to OCD (Fallon et al., 1991; Fallon, Qureshi, Laje, & Klein, 2000; Neziroglu et al., 2000). These questions have led to substantial research in the field addressing the link between hypochondriasis and OCD, and possible treatment implications of a potential connection. In particular, research has addressed whether what we know about treating OCD can be of help in curing hypochondria, because treatment research and success was obtained in OCD prior to that of hypochondriasis (Fallon et al., 1993). Conducting studies on hypochondriasis applying methods found effective in OCD treatment has been profitable and to a great extent contributed to our current knowledge base of hypochondriasis (Neziroglu et al., 2000).

Prevalence of symptoms of hypochondriasis in patients with OCD

Patterns of comorbidity between hypochondriasis and OCD are among the central matters in research addressing the relationship between the two (Fallon et al., 2000; Neziroglu et al., 2000; Savron et al., 1996). Most studies have investigated prevalence rate of OCD in patients with hypochondriasis rather than the reverse comorbidity. Currently, findings on the prevalence of hypochondria in OCD patients are inconclusive, but the most recent and methodological robust studies suggest a prevalence rate of 13-15%, significantly higher than that in controls.

One Indian study investigating the prevalence of hypochondriasis in 231 OCD subjects (Jaisoorya, Janardhan Reddy, & Srinath, 2003), revealed a significantly higher prevalence among the clinical sample compared with a control group consisting of relatives to neurologically ill patients ($n = 200$). Here, the prevalence rate in OCD patients was found to be 13%, while 0% of the controls exhibited symptoms of hypochondria. In their study, adults and children were mixed. Adult patients and controls were assessed for hypochondriasis using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), and the children were measured by means of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS).

Another study examining the relationship of OCD to possible spectrum disorders, found similar results as the study from India mentioned above (Bienvenu et al., 2000). Results revealed that the prevalence of hypochondriasis was significantly higher in a group of 80 patients with OCD compared to 73 healthy controls. Hypochondriasis was measured using the Schedule for Affective Disorders and Schizophrenia - Lifetime Anxiety Version, while

collateral information for diagnostic evaluation was gathered from knowledgeable informants associated to each subject. The lifetime prevalence of hypochondriasis was reported to be 15% in the OCD patient sample, and 0% in the control group. Additionally, hypochondriasis occurred more frequently in first-degree relatives with OCD, regardless of whether case probands had hypochondriasis or not.

Two studies with smaller sample sizes have found different results. One trial measured hypochondriacal symptoms by administering the subscale “hypochondriac beliefs” – a part of the Illness Attitude Scale to 30 patients with DSM-IV OCD and 30 socio-demographically similar controls (Savron et al., 1996). In this study, three from the patient group (10%) and two from the control group (6.67%) displayed characteristic hypochondriacal responses. Furthermore, unpublished observations also found that the hypochondriasis prevalence rate in OCD patients was not significantly different from that of controls (Fallon et al., 2000). Here, the Whiteley Index (WI) was administered to 17 patients with DSM-III-R hypochondriasis, 21 with DSM-III-R OCD, and 24 controls. As expected, hypochondriasis patients had a significantly higher degree of hypochondria than both OCD patients and controls. Comparing the results for the OCD patients and controls, patients with OCD had a 16.7% prevalence rate of hypochondriasis, not significantly greater than the 10% prevalence rate found in controls. The applied cutoff score was not accounted for.

In summary, previous research show that prevalence rates of hypochondriacal symptoms in patients with OCD range from 10 to 16.7%. Only half of the existing studies suggest a significantly greater prevalence rate in OCD patients compared to controls, possibly due to limited sample sizes. Finding out whether symptoms of hypochondriasis are more common amongst those with OCD, and identifying the exact hypochondriacal symptoms prevalence rate in the OCD group is important for a better understanding of both hypochondriasis and OCD. Furthermore, prevalence findings can imply what future research is needed. If the prevalence is established to be high, it will be necessary to further examine how hypochondriasis and OCD affect each other, whether they can be explained by common mechanisms, and how knowledge on these matters can contribute to efficient treatment.

Do symptoms of hypochondria affect OCD treatment outcome?

Empirical research show that the presence of comorbid disorders in mental illness may affect treatment response negatively by adding to patient distress (Abramowitz, Franklin, Street, Kozak, & Foa, 2000). Looking at OCD in particular, most patients with OCD have at

least one co-morbid mental disorder (Steketee, 1993), and at least 50% have a comorbid axis I disorder (Tükel, Polat, Özdemir, Aksüt, & Türksoy, 2002). Comorbid conditions in OCD have been found to raise anxiety and depression levels in OCD patients, and most importantly, the severity of obsessions and compulsions, as well as lowering patient insight (Tükel et al., 2002). In compliance with these findings, those who are non-responsive to OCD treatment are more likely to meet criteria for comorbid axis I or axis II disorders (Pallanti et al., 2002).

The effect of comorbid symptoms of hypochondria on OCD treatment outcome has never been examined. However, previous studies have addressed treatment effects of several other coexisting mental disorders. Overall, comorbid severe depression (Abramowitz et al., 2000; Keeley, Storch, Merlo, & Geffken, 2008; Steketee, Chambless, & Tran, 2001), generalized anxiety disorder (Hansen, Vogel, Stiles, & Götestam, 2007; Steketee et al., 2001), and panic disorder (Hansen et al., 2007) seem related to poorer OCD treatment outcome. Results regarding comorbid personality disorders are somewhat inconclusive, but it seems that especially schizotypal personality disorder hinder the treatment of OCD (Baer et al., 1992; Cavendish et al., 1997; Dreessen, Hoekstra, & Arntz, 1997; Fricke et al., 2006; Haan et al., 1997; Hansen et al., 2007; Moritz et al., 2004). Finally, comorbid tic disorder does not seem to influence the outcome of OCD treatment (Himle, Fischer, Van Etten, Janeck, & Hanna, 2003). In general, it seems like some comorbid disorders could have a negative effect on OCD treatment response. Nevertheless, it remains unknown how results of OCD treatment are affected by symptoms of hypochondriasis in particular.

Changes in hypochondriacal symptoms following OCD treatment

Treatment studies have never addressed whether OCD treatment affects hypochondriacal symptoms. However, a variety of treatment trials have been conducted to see if effective OCD treatment strategies adapted to address hypochondriacal symptoms are effective for hypochondriasis (Fallon et al., 1993). Looking into existing research on hypochondriasis, cognitive-behavioral strategies applied effectively in OCD treatment (Marks, 1981; Salkovskis & Warwick, 1985) have been successfully implemented in the treatment of hypochondriacal patients (Fallon et al., 1993; Salkovskis & Warwick, 1986; Warwick, Clark, Cobb, & Salkovskis, 1996; Warwick & Salkovskis, 1989; Yaryura-Tobias & Neziroglu, 1997). Although previous studies have tried out treatments of hypochondriasis inspired by strategies from OCD treatment, hypochondriacs have traditionally been treated

with manuals adapted specifically to hypochondriasis. Moreover, several studies suggest that pharmacotherapy reduce hypochondriacal symptoms when applying dosage levels of serotonergic medication originally modeled after studies of OCD (Bodkin & White, 1989; Fallon et al., 1991). If strategies utilized in the treatment of OCD also can be successfully applied in hypochondriasis treatment, one might think that OCD treatment could improve symptoms of hypochondriasis.

Metacognitions and symptoms of hypochondria

The CBT-model is currently established as the leading conceptualization of hypochondriasis. Recently however, it's been suggested that psychopathology due to disturbance in metacognitions might necessitate a different understanding of the condition (Bailey & Wells, 2013a, 2013b). Metacognition is known as the appraisal of thought content or cognitive processes (Flavell, 1979). Furthermore, metacognition is assumed to comprise knowledge (beliefs), processes, and strategies that appraise, monitor or control cognition (Wells, 2000). In other words, metacognitions reflect attitudes about one's own cognitions such as believing that one should control thinking, that worrying is dangerous, or that one is bad for thinking certain thoughts. These attitudes will in turn affect how a person experiences their cognitive process, and strategies applied with the purpose of creating harmony between thoughts, and thoughts about thoughts. According to metacognitive theory, the cause of psychopathology is not to be found in the content of thought, but in the individual's metacognitions.

Metacognition is predicted to be particularly important for emotional distress in disorders characterized by uncontrollable cognition (Wells, 1995). OCD is a disorder presenting uncontrollable cognition, characterized by unwanted, recurrent, and persistent obsessions (APA, 1994). Thus, metacognitions should be important in the psychopathology of OCD, which also seems to be the case (Janeck, Calamari, Riemann, & Heffelfinger, 2003; Ladouceur et al., 1995; Ladouceur, Rheaume, & Aublet, 1997; Lopatka & Rachman, 1995; Rachman & de Silva, 1978; Solem, Håland, Vogel, Hansen, & Wells, 2009; Wells, 1995). Considering that metacognition is theorized especially influencing in disorders where uncontrollable cognition is prominent, the same should be true for hypochondriasis.

Comparisons between metacognition and established hypochondriacal symptom correlates demonstrate that metacognition has superior predictive power over other influential variables in hypochondria (Bailey & Wells, 2013a). One study looked at somatosensory

amplification, illness cognition, and neuroticism relative to metacognition. An analogue sample of 351 completed a questionnaire battery revealing that metacognitions correlated significantly and positively with hypochondriacal symptoms. Three metacognitive dimensions, namely negative beliefs about the uncontrollability of thoughts and danger, cognitive confidence, and beliefs about the need for control, accounted better for substantial variance than the control variables. Moreover, controlling for metacognition also had a large effect on the strength of the relationship between cognition and hypochondriacal symptoms.

A clinical case study provides preliminary evidence for the effectiveness of applying a metacognitive treatment protocol when treating hypochondriasis (Bailey & Wells, 2013b). Here, an A-B single case series methodology (N=4) with a six month follow up was implemented. Therapy was guided by a generic metacognitive therapy (MCT) treatment plan. The hypochondriacs' metacognitions demonstrated clinically significant improvements after treatment, and MCT resulted in reduction of hypochondriacal symptoms. Patients maintained recovery status at follow up.

A study investigating worry and metacognitions in hypochondriasis found hypochondriacals to be more concerned about the lack of control, and the excess of interference they experienced in relation to illness-worry (Bouman & Meijer, 1999). Meta-worry appeared to be the best predictor for hypochondriasis, as well as an increased awareness of one's thoughts. Superstitious beliefs about worry have also been revealed by worry being assumed to provide protection from "tempting fate" (Wells & Hackmann, 1993). In summary, existing research points towards metacognition as an important component in accounting for the psychopathology of both OCD and hypochondriasis. Thus, metacognition seems to be significant regardless of diagnosis, and possibly a sustaining factor in both disorders.

Aims and hypotheses of the current study

There are four aims of the present study. First, the intension is to further examine the prevalence of hypochondriacal symptoms in patients with OCD. Overall, most studies have addressed prevalence rate of OCD symptoms in patients with hypochondriasis, rather than the reverse comorbidity. Currently, studies examining the prevalence of hypochondriacal symptoms in patients with OCD are few with relatively small sample sizes. Existing findings are inconclusive, but the robust and most recent studies are consistent suggesting that the prevalence rate is in the range of 13-15%, and significantly higher than in controls. In light of

previous research, the first hypothesis in this study will be: There is a higher prevalence of symptoms of hypochondria in patients with OCD than in controls, and the prevalence rate is in the range of 13-15%.

A second aim of the study is to explore whether patients' obsessive-compulsive symptom levels after OCD treatment are affected by symptoms of hypochondriasis at treatment startup. This has not been done prior to the current study. However, regarding comorbidity effects of other Axis I disorders, comorbid hypochondriacal symptoms could have a similar influence. The hypothesis tested, will therefore be that comorbid symptoms of hypochondriasis in OCD patients predicts poorer OCD treatment outcome.

Third, this paper aims to examine if changes in hypochondriacal symptoms follow OCD treatment. Due to lack of previous research on this matter, a hypothesis has not been formulated.

The fourth and final purpose of the current study is to further investigate whether metacognitions is related to symptoms of hypochondria and if change in metacognitions is related to symptom reduction. Considering previous research suggesting that metacognition is related to, and plays an important role in hypochondria, we expect compatible findings. Thus, our last hypothesis is that metacognition is related to hypochondriacal symptoms, and furthermore, change in metacognitions is related to improvement in symptoms of hypochondria.

Method

Procedure

The study was conducted in Norway, and included 313 patients with OCD, and 382 community controls. Between 2007-2013, OCD patients were recruited mainly through referrals from general practitioners or psychiatric outpatient clinics. Whereas 216 of the patients were inpatients at St. Olav's Hospital in Trondheim, 32 were outpatients from a multisite study on exposure and response prevention for OCD, and 63 were outpatients at Sørlandet Hospital in Kristiansand (Håland et al., 2010). The main criterion for inclusion in the study was having a primary diagnosis of OCD. At St. Olav's Hospital, this was obtained with the Anxiety Disorder Interview Schedule (ADIS-IV; Brown, DiNardo, & Barlow, 1994), while the Structured Clinical Interview for DSM-IV (SCID-1; First, Spitzer, Gibbon, & Williams, 1995) was used at Sørlandet Hospital, and in the multisite study. The same

exclusion criteria were applied across patient groups. Furthermore, patients using anti-obsessional medication were encouraged to maintain stable dosage levels throughout treatment. A description of the patient sample's demographic information is provided in table 1. Within the patient sample, 43.6% of the St. Olav's patients, 47.8% of the multisite patients, and 40.3% of the patients at Sørlandet received disability benefits. The remaining patients were either full-time students, part-time students, working full-time or part-time. The data collection was conducted by having patients fill out self-report inventories pre, and post-treatment. Participants were measured on OCD symptoms, hypochondriacal symptoms, depressive symptoms, and metacognitive domains considered important in exploring, and conceptualizing psychopathological processes.

Healthy controls were recruited through social media. The author encouraged acquaintances to respond to an internet survey consisting of the Yale –Brown Obsessive-Compulsive Scale-Self-Report (Y-BOCS-SR), the Whiteley Index (WI), and the Metacognitions Questionnaire (MCQ-30). Of the 453 participants responding to the invitation, 382 completed the WI, 326 the MCQ-30, and 280 the Y-BOCS. A description of the control sample's demographic information is provided in table 1. Among the controls, 45.5% were full time students, 43.5% in full time jobs, and 0.2% were retired. Furthermore, 8.8% were either in part time jobs or preoccupied with part time studies, while 2.0 % received disability benefits. In addition to the demographic descriptions of both patient and control sample, table 1 presents a comparison of the samples.

Table 1

Demographic Descriptions and Comparisons of the Patient and Control Sample

	OCD	Controls	<i>p</i>
N	313	453	
Age	34.55 (11.98)	26.15 (5.72)	.000
Age at debut of OCD	18.50 (9.41)		
Female gender	65.4%	55.2%	.000
Civil status			.000
Single	51.9%	42.6%	
Married/cohabitant	44.0%	56.5%	
Separated/divorced	4.1%	0.9%	
Disability benefits	40.3-47.8%	2.0%	.000

Note: OCD = Obsessive-Compulsive Disorder.

Treatment and therapists

Patients were enrolled in different treatment programs based on exposure and response prevention (ERP) for OCD (Foa & Kozak, 1997). These programs involved psycho-education, construction of a symptom hierarchy, ERP, and maintenance/relapse prevention. However, the treatment was given in different formats. The majority of St. Olavs patients received inpatient treatment, those from Sørlandet Hospital underwent outpatient group therapy, and the multisite patients were given outpatient treatment. The group therapy was based on a manual on OCD group therapy, developed by Krone, Himle, and Nesse (1991). The therapists were all trained in ERP, and consisted of mainly psychologists, but also psychiatrists, and psychiatric nurses.

Measures

OCD-symptoms. OCD symptoms were measured with the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al., 1989a) or the Yale-Brown Obsessive-Compulsive Scale-Self-Report (Y-BOCS-SR; Baer, Brown-Beasley, Sorce, & Henriques, 1993; Steketee, Frost, & Bogart, 1996). Y-BOCS is a clinician-rated scale measuring severity of cardinal symptoms of OCD along dimensions of time, interference, distress, resistance, and control. The scale has ten items, and a two-factor structure (obsessions, and compulsions). Each item is rated from 0-4, higher numerical score corresponding to greater illness severity. This makes the total range 0-40. Only total scores were used in our study. Y-BOCS has good psychometric properties in terms of being a reliable, and valid instrument for assessing OCD symptom severity (Goodman et al., 1989a, 1989b) and is considered the gold standard assessment for this purpose (Grabill et al., 2008).

The Y-BOCS-SR (Baer et al., 1993; Steketee et al., 1996) is a reliable and valid measure (Steketee et al., 1996), derived from the clinician-administered version (Goodman et al., 1989a, 1989b). The Y-BOCS-SR instrument is designed with the same items and scoring system as Y-BOCS. Pearson correlations for the total score between Y-BOCS-SR and Y-BOCS interview version (Goodman et al., 1989a, 1989b) were 0.79 in a clinical population, and 0.75 in a non-clinical population (Steketee et al., 1996). The psychometric properties of the Y-BOCS and the Y-BOCS-SR have not been assessed with Norwegian samples. The Cronbach's alpha value for the total scale was .83 in the patient sample, and .87 in the control sample.

Hypochondria symptoms. The Whiteley Index (WI; Pilowsky, 1967) was utilized to measure hypochondriacal symptom severity. WI is a self-report questionnaire mapping hypochondriacal attitudes and beliefs. The scale comprises 14 items about worry and conviction about illness, attention to self and body, and reaction to doctor's reassurance. Factor analysis has extracted a 3-factor structure, subscales being disease fear, disease conviction, and bodily preoccupation (Pilowsky, 1967). Responses are scored on a five-point Likert-scale, with a total score range of 14-70. High scores reflect a greater occurrence of hypochondriacal attitudes and beliefs (Barsky et al., 1992). The WI has shown to be satisfying regarding psychometric properties, with established test-retest reliability, internal consistency, and validity (Beaber & Rodney, 1984; Hanback & Revelle, 1978; Kasteler et al., 1976; Pilowsky, 1967, 1978). The total scores and a cutoff set to 40 were applied to detect probable hypochondriacals. This cutoff was based on a previous study where a cutoff at 40 distinguished nearly all interview-diagnosed patients from controls (Noyes et al., 1993). The Cronbach's alpha value for the total scale was .92 in the patient sample, and .89 in the control sample.

Metacognitive dimensions. The Metacognitions Questionnaire (MCQ-30; Wells & Cartwright-Hatton, 2004) was used to measure metacognition. The MCQ-30 is a 30-item self-report scale assessing a range of metacognitive domains considered important in exploring and conceptualizing psychopathological processes. These domains constitute positive beliefs about worry, negative beliefs about the controllability of thoughts and corresponding danger, cognitive confidence, negative beliefs about thoughts/need to control thoughts, and cognitive self-consciousness. Each of the 30 items has a four-point scale ranging from one to four. Higher scores reflect greater difficulties with the metacognitive dimension in question. The MCQ-30 possesses good internal consistency (Spada, Mohiveddini, & Wells, 2008) and convergent validity, as well as acceptable test-retest reliability (Wells & Cartwright-Hatton, 2004). The Cronbach's alpha value for the total scale was .92 in the patient sample, and .91 in the control sample.

Depressive symptoms. Depressive symptoms were measured with the revised Beck Depression Inventory (BDI; Beck & Steer, 1993) or the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). BDI is a self-report instrument consisting of 21 items, each employing a 4-point scale ranging from 0-3. Total scores range from 0-63, higher scores indicating greater depression symptomatology. BDI is a reliable and valid measure of depression severity (Beck, Steer & Garbin, 1988; Nuevo et al. (2009).

BDI-II (Beck, Steer, & Brown, 1996) is an upgraded version of BDI. In general, the

psychometric characteristics of BDI-II are highly congruent with those of BDI, with the correlation between BDI and BDI-II being 0.93 in a non-clinical population, despite several instrument alterations in format and content (Dozois, Dobson, & Ahnberg, 1998). The psychometric properties of BDI-II have been replicated with a Norwegian non-clinical sample (Aasen, 2001). The Cronbach's alpha value for the total scale was .83 in the patient sample.

Overview of statistical analyses

For the first analyses, the patient sample was compared to the control group on demographic variables using independent t-tests and one way ANOVA. Furthermore effect sizes and a paired samples t-test was utilized for evaluating treatment effect. To compare patients and controls on the different variables, independent t-tests were conducted, and correlation and regression analyses were used to investigate whether hypochondriacal symptoms in OCD patients affected symptoms of OCD after treatment. Finally, we calculated the correlation between metacognitions and symptoms, and rendered regression analyses to assess whether change in metacognition was related to treatment outcome measured with WI-post-treatment.

Results

Prevalence

Figure 1 and 2 illustrate the distribution of WI scores in the patient sample and control group respectively. The vertical line indicates the cut-off score of 40. Figure 1 shows that 30.7% of the OCD patients scored above the suggested cut-off on WI total at pre-treatment. Figure 2 reveals a significantly lower hypochondriacal symptoms prevalence rate of 4.7% in the control group.

Figure 1. Hypochondriacal Symptoms in the OCD Sample

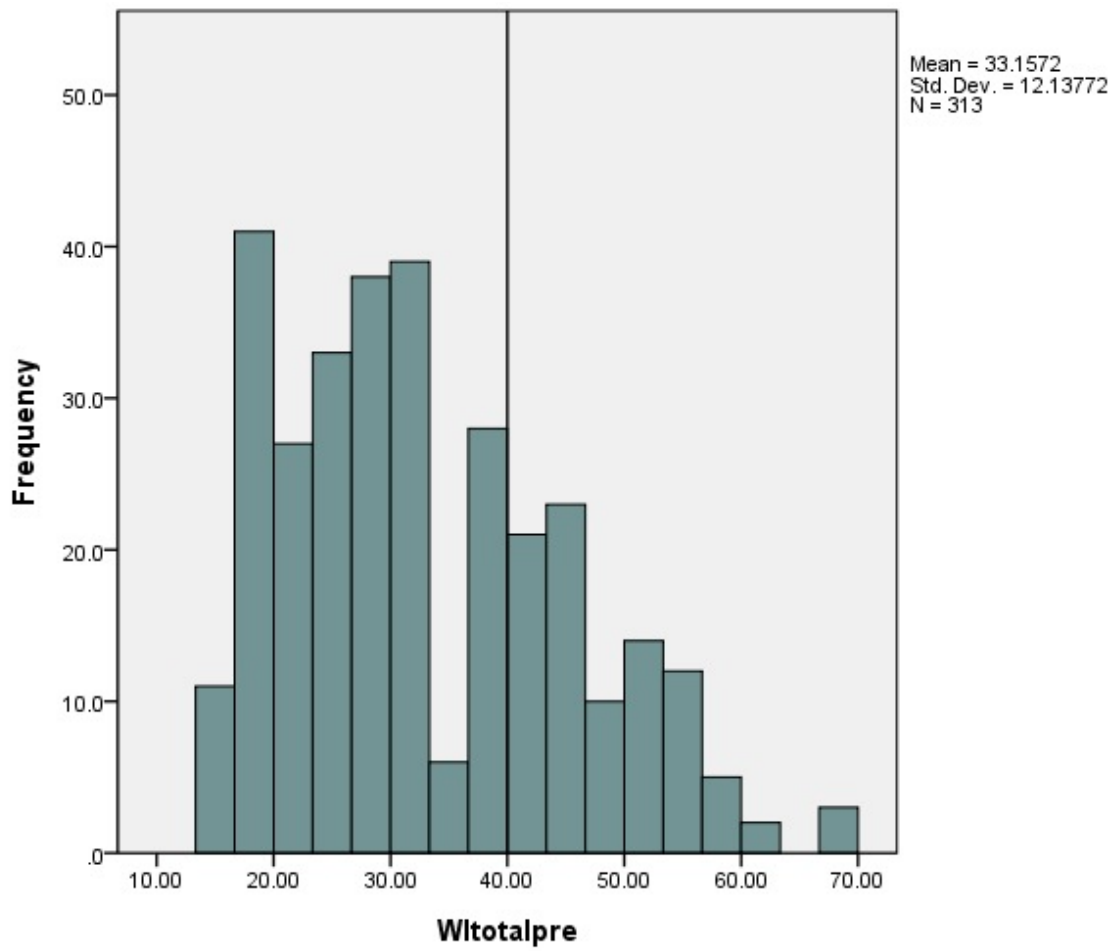


Figure 1. The vertical line represents the cut-off score (>39) for the WI questionnaire detecting probable hypochondriasis.

Figure 2. Hypochondriacal Symptoms in the Control sample

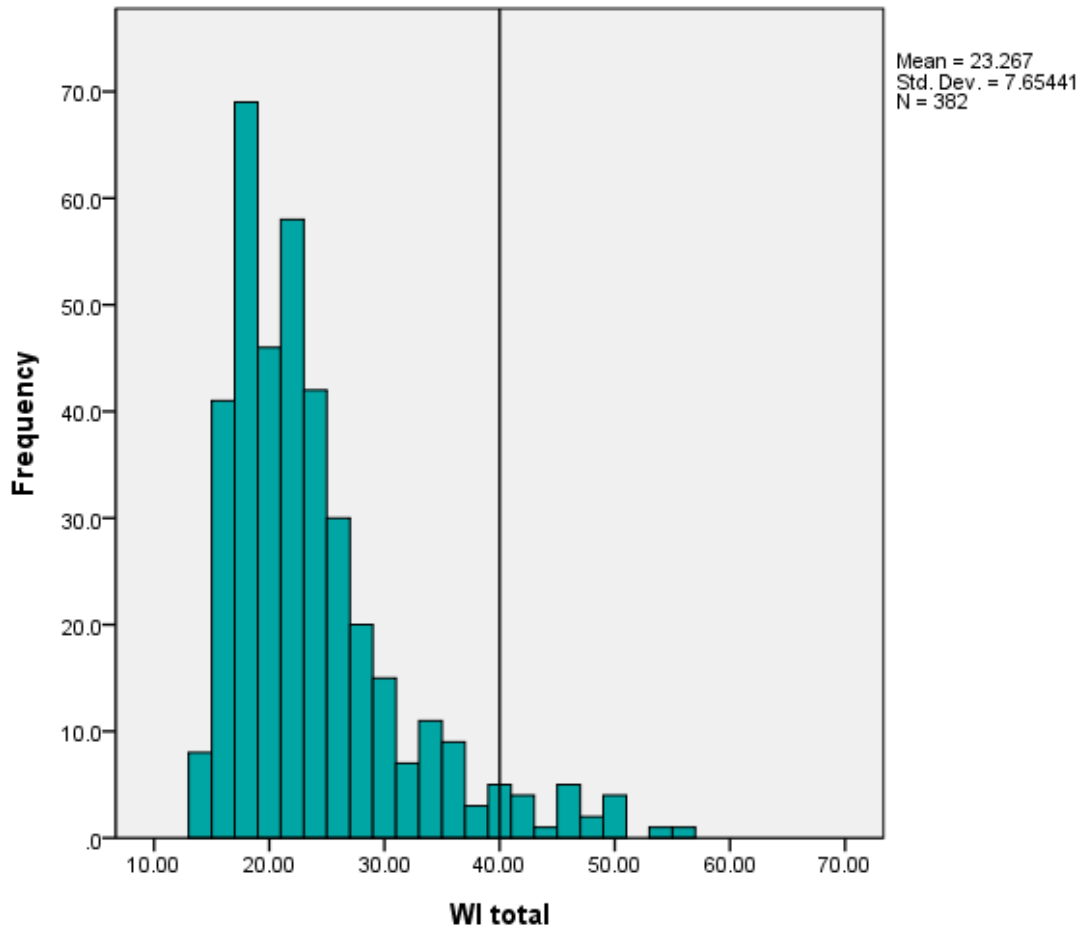


Figure 2. The vertical line represents the cut-off score (>39) for the WI questionnaire detecting probable hypochondriasis.

Do symptoms of hypochondria affect OCD treatment outcome?

Symptoms of pre-treatment hypochondria were almost significantly correlated with post-treatment Y-BOCS ($r = .12, p = .053$). A regression analysis controlling for Y-BOCS pre-treatment in step 1 suggested that pretreatment levels of OCD were significantly related to post-treatment Y-BOCS ($Adj R square = .09, p = .000$), while WI pre-treatment in step two did not add significantly to the equation ($p = .257$). In other words, there were no sign of hypochondriacal symptoms affecting OCD treatment outcome. However, by transforming WI scores into z-scores, and comparing patients with $\pm 1SD$ there is a significant difference between the high WI and the low WI group (equal to an effect size of .56). Patients with high

scores exhibit poorer YBOCS post scores (13.2, [SD = 6.13]), while those with low cores get better results on YBOCS post (9.9, [SD = 5.7]). This means that patients with a high hypochondriasis symptom level at pre-treatment exhibit more symptoms of OCD after OCD treatment, compared with those who have less symptoms of hypochondria at treatment start.

Changes in hypochondria symptoms following OCD treatment

Overall, the results showed a significant decrease in all variables after treatment. OCD treatment was effective in reducing OCD symptoms, as documented by a large effect size ($d = 2.12$). Results also revealed significant changes with large effect sizes in depressive symptoms ($d = 1.22$) and metacognitions ($d = 1.25$) following treatment. For all variables, there was found a significant difference between the samples. Table 2. Illustrates sample comparison and treatment effects on symptoms and beliefs.

The share of participants exhibiting WI scores above cutoff went from 30.7% before treatment to 9.0% after treatment in the OCD sample. Effect size calculations suggested that symptoms of hypochondria changed moderately ($d = .60$) following OCD treatment. Paired samples t-test suggested that these changes were significant. At post-treatment, the differences between the patient sample and the control sample were relatively small ($d = .25$). Paired samples t-test for WI scores from post-treatment to follow-up suggested that the changes in hypochondria symptoms were stable ($p = .892$).

Table 2

Sample Comparison and Treatment Effects on Symptoms and Beliefs

	Controls	OCD		Controls vs. OCD		Pre-post	
		pre	post	<i>p</i>	<i>d</i>	<i>p</i>	<i>d</i>
WI	23.27 (7.65)	33.16 (12.14)	25.85 (12.31)	.000	.97	.000	.60
WI*	4.7%	30.7%	9.0%				
Y-BOCS	3.74 (4.64)	24.55 (5.31)	11.95 (6.49)	.000	4.17	.000	2.12
BDI	n. a.	17.75 (9.68)	7.18 (7.46)			.000	1.22
MCQ	48.99 (12.31)	66.01 (16.21)	47.05 (14.10)	.000	1.18	.000	1.25
Pos	8.77 (3.12)	9.96 (4.31)	7.91 (3.00)	.000	.32	.000	.55
Neg	10.19 (3.52)	15.62 (4.20)	9.87 (3.66)	.000	1.40	.000	1.46
CC	9.37 (3.36)	12.21 (4.81)	9.73 (3.89)	.000	.68	.000	.57
Control	9.36 (3.15)	13.26 (4.43)	8.65 (3.06)	.000	1.01	.000	1.21
CSC	11.31 (3.58)	14.96 (4.39)	10.88 (4.17)	.000	.91	.000	.95

Note: WI = Whiteley Index, WI* = percentage above WI cutoff score, Y-BOCS = Yale-Brown Obsessive Compulsive Scale, BDI = Beck Depression Index, MCQ = Metacognitive Questionnaire, Pos = Positive beliefs, Neg = Negative beliefs, CC = Cognitive confidence, Control = Need to control thoughts, CSC = Cognitive self-consciousness.

Metacognitions in symptoms of hypochondria and OCD

As can be seen in table 3, pre-treatment correlations suggested that WI was moderately correlated with depression and metacognitions. All metacognitive dimensions were positively correlated with symptoms of hypochondriasis. The dimension “beliefs about the controllability of thoughts and corresponding danger” had the highest correlation with WI.

“Need to control thoughts” had the second highest correlation, while “cognitive confidence” and WI were most weakly correlated. Y-BOCS showed a weak positive correlation with WI, and a weak to moderate positive correlation with metacognitions. The correlations showed a similar pattern at post-treatment. Change-score correlations revealed similar patterns and changes in metacognition were related to both changes in symptoms of OCD, depression, and hypochondriasis. Y-BOCS scores at post-treatment were more strongly correlated with the other measures than at pre-treatment, probably due to increase in range of Y-BOCS scores. The correlation patterns observed in the patient sample were similar to the pattern observed in the control sample as displayed in table 4.

A regression analysis entering WI pre at step 1 and MCQ change on step two suggested that pre-treatment levels of hypochondria explained 35.5% of the variance in post-treatment WI. Changes in metacognition added significant variance and explained an additional 11.8% of post-treatment scores in WI. Adjusted R square for the model was .47.

Table 3

Correlations Between Symptoms and Metacognitive Beliefs in the OCD Sample

Pre-treatment correlations									
	WI	Y-BOCS	BDI	MCQ	Pos	Neg	CC	Control	CSC
WI		.16**	.43**	.46**	.31**	.48**	.23**	.39**	.30**
Y-BOCS			.34**	.26**	.13*	.35**	.09ns	.24**	.18**
BDI				.42**	.17**	.40**	.30**	.40**	.27**
Post-treatment correlations									
WI		.33*	.41**	.49**	.35**	.47**	.33**	.41**	.36**
Y-BOCS			.50**	.59**	.37**	.62**	.40**	.47**	.45**
BDI				.52**	.32**	.51**	.41**	.42**	.39**
Change-score correlations									
WI		.25**	.38**	.48**	.14ns	.44**	.35**	.34**	.41**
Y-BOCS			.33**	.38**	.14*	.43**	.25**	.22**	.33**
BDI				.46**	.15*	.34**	.41**	.45**	.33**

Note. WI = Whiteley Index, Y-BOCS = Yale-Brown Obsessive Compulsive Scale, BDI = Beck Depression Index, MCQ = Metacognitive Questionnaire, Pos = Positive beliefs, Neg = Negative beliefs, CC = Cognitive confidence, Control = Need to control thoughts, CSC = Cognitive self-consciousness, * = $p < 0.05$, ** = $p < 0.01$.

Table 4

Correlations Between Symptoms and Metacognitive Beliefs in the Control Sample

	Y-BOCS	MCQ	Pos	Neg	CC	Control	CSC
WI	.43	.47	.22	.53	.23	.37	.35
Y-BOCS		.56	.43	.55	.27	.41	.39

Note. WI = Whiteley Index, Y-BOCS = Yale-Brown Obsessive Compulsive Scale, MCQ = Metacognitive Questionnaire, Pos = Positive beliefs, Neg = Negative beliefs, CC = Cognitive confidence, Control = Need to control thoughts, CSC = Cognitive self-consciousness, $p = <0.01$ for all correlations.

Discussion

Prevalence of symptoms of hypochondriasis in patients with OCD

Symptoms of hypochondria were more common in OCD patients compared with that of the community sample. However, results revealed a prevalence rate of 30.7%, which is more than twice as high as foreseen based on previous research (Jaisoorya et al., 2003; Bienvenu et al., 2000).

Perhaps the WI cut-off applied in this study detecting probable hypochondriacals was set too low, creating a falsely high prevalence of hypochondriacal tendencies among the participants. On the other hand, the cutoff in this trial was based on the one set in a previous study where the researchers successfully distinguished nearly all interview-diagnosed patients from controls (Noyes et al., 1993). In other words, the current cut-off should be relatively precise in detecting symptoms of hypochondria.

In the two previous studies that are emphasized here, SCID-I, K-SADS (Jaisoorya et al., 2003), and SADS-LA (Bienvenu et al., 2000) were used to measure hypochondriasis. It's possible that the WI questionnaire generally tends to give a higher prevalence rate than the diagnostic interviews used in the other studies. According to Fallon et al. (1993), the WI questionnaire clearly detects fear of *having* a serious disease rather than fear of getting one in the future, which is one of the main differences between hypochondriasis and OCD. However, the results might be due to the WI separating poorly between hypochondriasis and health-related obsessions in OCD after all.

Certain OCD subtypes seem to be more closely related to comorbid hypochondriasis than others (Lochner et al., 2005; Abramowitz, McKay, & Taylor, 2008). For example,

Abramowitz and colleagues (1999) compared OCD patients with and without health concerns. Results revealed that OCD patients with health concerns scored higher on contamination obsessions and washing compulsions, whereas the “pure” OCD patients were more engaged in harming obsessions and checking compulsions. Regarding this, an especially high share of the OCD subtype themed around contamination and decontamination in the patient group, would most likely contribute to increase the number of patients also exhibiting hypochondria.

Another possible explanation for the high prevalence estimate could be that depressed individuals exhibit mood-congruent recall (Matt, Vázquez, & Campbell, 1992). This means that stimuli of the same affective valence as their present mood are learned and retrieved better than stimuli of valence disagreeing with this mood. Thus, when depressed patients rapport their hypochondriacal symptoms, it's possible that they're biased towards an exaggerating reporting style creating inflated scores.

Looking at the correlation between the hypochondriacal symptoms and other symptomatology, symptoms of hypochondria exhibited a closer relationship with depressive symptoms than those of OCD. It could be that the high prevalence rate is a result of high symptom pressure in general. Considering the previously mentioned similarities between hypochondriasis and OCD however, the disorders are likely to co-occur due to similar underlying mechanisms.

Do symptoms of hypochondria affect OCD treatment outcome?

Hypochondriasis was not found to affect OCD treatment outcome negatively. This is inconsistent with the general negative effects often found for comorbidity in treatment of emotional disorders and OCD more specifically (Abramowitz et al., 2000; Hansen et al., 2007; Keeley et al., 2008; Steketee et al., 2001; Tükel et al., 2002). However, when comparing high and low scorers on the WI there was a difference in treatment outcome for the two groups similar to previous findings on the role of depression in OCD (Abramowitz et al., 2000). Patients with high scores on the WI had significantly higher Y-BOCS post-treatment scores than the low group and these patients also exhibited higher symptom levels of OCD, depression, and metacognitions when starting treatment.

Perhaps sub-clinical symptoms of hypochondriasis do not hinder OCD treatment while severe OCD- and hypochondriasis symptoms could interfere with treatment. This could be due to general symptoms severity and because of how the two disorders overlap and share

common underlying mechanisms such as metacognitions. As shown in this study both OCD and hypochondriasis were related to metacognitive beliefs which were significantly reduced following treatment. Also, as mentioned initially, hypochondriasis resembles OCD in that evocation and regulation of anxiety unfold much like obsessions and compulsions (Fallon et al., 1991; Neziroglu et al., 2000; Warwick & Salkovskis, 1990). Furthermore, the core characteristic of hypochondriasis (fear of illness) is a common theme of obsessions and compulsions (Savron et al., 1996). Taken together, this might make the patients with hypochondriasis symptoms able to generalize what they've learned about dealing with intrusive thoughts and coping strategies.

Changes in hypochondriacal symptoms following OCD treatment

The findings suggest a significant improvement in hypochondriacal symptoms of moderate strength following OCD treatment. This is in line with numerous controlled studies have shown that manual based treatment directed towards treating one particular disorder can lead to symptom improvement in other disorders as well (Wilson, 1997). As mentioned previously, there are significant overlaps between hypochondriasis and OCD (Fallon et al., 1991; Neziroglu et al., 2000; Savron et al., 1996; Warwick & Salkovskis, 1990). Considering these overlaps, it's possible that they might cause OCD treatment to be effective on hypochondriacal symptoms as well. The finding that symptoms of hypochondria and OCD are both related to metacognitions is in line with metacognitive theory as one would expect that symptoms of emotional disorder decrease when dysfunctional metacognitive beliefs are adjusted. Another possible explanation for these findings is that health related intrusions and behaviours have been addressed during the treatment. This might be due to health personnel viewing hypochondriacal symptoms as part of- or related to the OCD.

Metacognitions in symptoms of hypochondrias and OCD

Hypochondriacal symptoms were moderately correlated with metacognition, and changes in metacognitions explained of a significant proportion of hypochondriacal symptoms at post-treatment. These results correspond with existing research (Bailey & Wells, 2013a, 2013b; Bouman & Meijer, 1999; Solem et al., 2009; Wells & Hackman, 1993). Two particular metacognitive domains that could be relevant in a metacognitive model of hypochondriasis were identified. These are “negative beliefs about thoughts concerning uncontrollability and danger”, and “negative beliefs concerning the consequences of not

controlling thoughts”. The strong correlations between these two metacognitive dimensions and hypochondria is in accordance with the previous study reporting hypochondriacals to be more concerned about the lack of control, and the excess of interference experienced in relation to worry (Bouman & Meijer, 1999). However, here hypochondriasis was found to be a dimension best predicted by specific meta-worries about lack of control over thoughts about illness, as well as a cognitive self-consciousness, suggesting the existence of disorder-specific meta-worries.

The design of this study does not allow for conclusions concerning causality between change in metacognitions and symptom improvements during treatment. In accordance with the metacognitive model (Wells, 2000), improvement in metacognitions might have led to improvement in hypochondriacal symptoms. It could however, be that improvements in symptoms of hypochondriasis post OCD treatment caused a change in metacognitions. Also, an undetected/unknown factor accounting for both reduction of hypochondriasis symptoms and improved metacognitive functioning might be present.

Strengths and limitations

The strengths in this study were that diagnostic interviews were applied detecting OCD before inclusion in the patient sample, that there was a relatively large sample size, that a community group matching the size of the patient group was applied, and that two out of four problem formulations had not been addressed in previous research.

Limitations are that no conclusions about causation can be made, that data were collected using self-report measures, and that follow-up data were not included. Furthermore, the control group was a convenience sample, which did not match the patient sample demographically. Other limitations are that the study also lacked a control group with a different psychopathology, that our patient sample consisted of patients from three different ERP-treatment programs, and that there was no adherence measurement. A final limitation of these findings is that the diagnoses described in the present study were based on DSM-4, rather than DSM-5 criteria (APA, 2013). Diagnostic criteria for OCD did not change appreciably from one edition to the next, but the diagnosis of hypochondriasis has been eliminated as a disorder. Two diagnoses, namely illness anxiety disorder and somatic symptoms disorder are assumed to cover the former hypochondriasis diagnosis (Changes DSM 5). Because these are new as of DSM-5, we cannot be sure of the transfer value of the research that’s been done in this thesis.

Conclusion and Clinical Implications

The present study confirms that there is a relatively high occurrence of comorbid hypochondriasis in OCD. This implies that future research is needed to further examine how hypochondriasis and OCD affect each other, whether they can be explained by common mechanisms, and how knowledge on these matters can contribute to efficient treatment of both OCD and hypochondriasis. Findings suggest that patients enrolled in OCD treatment programs seem able to profit from treatment despite having comorbid hypochondriasis, unless the degree of hypochondriasis is fairly severe. In fact, most patients receiving treatment for their OCD who also suffer from hypochondriasis actually seem to exhibit improvements in both conditions. This suggests that there might be common mechanisms causing both OCD and hypochondriasis, and considering the results of the current study, these mechanisms might take the form of metacognitions. The clinical implications of these findings are that comorbid hypochondriasis should not be an exclusion criteria from being enrolled in OCD treatment, except for cases of particularly severe hypochondriasis where enrollment is dissuaded. Actually, if a person suffers from both OCD and symptoms of hypochondriasis, OCD treatment could be cost effective considering that both conditions might improve. Furthermore, examining and focusing on improving metacognitions in patients with OCD, hypochondriasis or both is advised.

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