Hypochondriasis. diagnostic issues and treatment.

Greeven, J.F.

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Introduction to Hypochondriasis

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Anja Greeven, Anton .J.L.M. van Balkom, & Philip Spinhoven (in press)

Chapter 1

Introduction

Patient B is a 23-year-old female who fears that she suffers from a serious physical illness and has done so since the age of 16. This fear began after a period of stomach aches. Because of her fears, she regularly checks her stool for blood and avoids certain foods. After the death of a friend from a brain tumour two years ago, this fear of having a serious illness has increased. B has searched for the specific symptoms of colon cancer on the internet with the hopes of detecting the fatal disease at an early stage. In addition she also visits her general practitioner approximately twice a month. She regularly requests referrals to a specialist and has visited with one a few times in the last years. Despite test results and regular control of stool samples, B is not reassured. She is unable to stop thinking about colon cancer. The next time B requests a referral to a specialist from her general practitioner, he discusses the diagnosis of hypochondriasis with her. He points out that the past referrals have not reassured her, despite the fact that she does not have any physical abnormalities. The general practitioner refers B to a psychologist who can learn B to better control her fears by means of cognitive behavioural therapy. When B meets with the psychologist it appears that she, in addition to hypochondriasis, also presents with symptoms of depression. B appears to suffer more from the hypochondriasis than the depressive symptoms. Therefore both B and the psychologist decide that the therapy will focus on hypochondriasis. To get a better picture of the current problematic symptoms, B is asked to keep a diary. She is also asked to register her emotions. The therapist evaluates along with B, the level of reality which her hypochondriacal thoughts actually have. These

thoughts, which are often automatic, are disputed in a critical manner in a questioning conversation and replaced with more functional thoughts. B notices her that fears lessen. Learning relaxation exercises, she reduces the general tension, which is a result of her fears. By means of homework assignments, she is stimulated to not check her stool and to develop activities which distract her from her hypochondriacal thoughts. She also learns to consult the internet on a less frequent basis. Gradually, her fears reduce and it is no longer necessary to seek reassurance with her general practitioner. In a period of four months B has received ten CBT sessions, first weekly and then less frequently. When B visits her general practitioner one year later, she reports that she no longer suffers from hypochondriacal fears.

Phenomenology

Historical Perspective

The term hypochondrias stems from ancient times. Hypochondriasis was seen as an illness of one of the organs located high in the abdomen (such as the liver, spleen and stomach). This definition of hypochondriasis remained in use until the 4th century. After that, a physical explanation became coupled to that of mental well-being. Due to an infection in the digestive channel, black gall mixed with blood, which would develop in sad mental feelings and anxiety regarding serious physical illness. Until the 17th century hypochondriasis was viewed as a sort of melancholy ('melancholia hypochondriasis') and as the male counterpart to hysteria, which was believed to exist only in women (Ladee, 1961). After this, gradually more

distance has been taken from the physical explanation for hypochondriasis and eventually it has been accepted as a mental disorder in which a constant preoccupation with physical health and incorrect interpretations of physical symptoms are the main problem.

Clinical Description and Diagnosis

According to the fourth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; APA, 1994), certain criteria must be met in order to diagnose someone with hypochondriasis These criteria are presented in table 1. When not all of the criteria for the diagnosis are met (i.e. a patient has a history of physical complaints but can be reassured and despite these anxieties the patient can function well), then one speaks of 'abridged hypochondriasis.' A different form of 'abridged hypochondriasis' is transient hypochondriasis in which the fear of illness is significantly shorter than 6 months. A well-known example of this is when medical students experience short periods of anxiety regarding the disease they are currently studying (Gurejee, Ustun & Simon, 1997).

Table 1: DSM-IV criteria for hypochondriasis

300.7 Hypochondriasis
A. Preoccupation with fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms.
B. The preoccupation persists despite appropriate medical evaluation and reassurance.
C. The belief in Criterion A is not of delusional intensity (as in Delusional Disorder, Somatic Type) and is not restricted to a circumscribed concern about appearance (as in Body Dysmorphic Disorder).
D. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
E. The duration of the disturbance is at least 6 months.
F. The preoccupation is not better accounted for by Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Panic Disorder, a Major Depressive Episode, Separation Anxiety, or another Somatoform Disorder.
Specify if:
With Poor Insight: if, for most of the time during the current episode, the person does not recognize that the concern about having a serious illness is excessive or unreasonable

The criteria which must be met to diagnose hypochondriasis according to the ICD-10 involve two main areas which are the same as the DSM-IV (see table 2). Firstly, the patient must constantly be convinced that he or she has one or two physical illnesses where at least one has a specific name. If the patient has more than two physical illnesses which cannot be named, according to the ICD-10, the diagnosis of hypochondriasis cannot be given (in contrast to the DSM-IV). Second, the patient who is preoccupied with perceived imperceptions of the body is also diagnosed with hypochondriasis, while these patients meet the DSM-IV criteria for a diagnosis of body dysmorphic disorder (World Health Organization, 1992).

Table 2: ICD-10 criteria for hypochondriasis
F45.2 Hypochondriacal disorder
The essential feature is a persistent preoccupation with the possibility of having one or more serious and progressive physical disorders. Patients manifest persistent somatic complaints or a persistent preoccupation with their physical appearance. Normal or commonplace sensations and appearances are often interpreted by patients as abnormal and distressing, and attention is usually focused upon only one or two organs or systems of the body. Marked depression and anxiety are often present; and may justify additional diagnoses.

To establish a diagnosis of hypochondriasis, several structured interviews are available. The nature and severity of hypochondriacal symptoms can be assessed with several questionnaires. In table 3 a summary of questionnaires available in the Dutch language and their accompanying psychometric qualities is given. Each instrument is evaluated as to which characteristics of hypochondriasis it measures. The table differentiates between illness fear (IF); illness beliefs (IB); safety behaviour (SB); and disruptive effects (DE); and finally experiencing physical symptoms (PS). Assessor-ratings covering most sub-fields are still missing.

Table 3: Review of structured interviews and questionnaires for hypochondriasis and the psychometric qualities of each questionnaire

	IF	IB	SB	DE	PS	Examples of psychometric properties
Structured Interviews						
Composite International Diagnostic Interview	X	X		X		Inter-rater reliability: 90% agreement (k=0.72) for DSM-III-R diagnosis; high convergent validity with clinical opinion
Structured Clinical Interview for DSM-IV	X	X				Reliability: k = 0.57 for DSM-III-R diagnoses over 24 hours to 2 weeks
Structured Diagnostic Interview for Hypochondriasis	X	X				Inter-rater reliability: 96% agreement for DSM-III-R diagnosis; convergent validity with Whiteley Index/ Somatic Symptom Inventory: *r* = 0.78
Self-report questionnaires						
Whiteley Index (WI)	X	X				Cronbach's alpha: 0.80; test-retest r: 0.90 (4 weeks); convergent validity with IAS en SAS satisfactory; sensitivity: 87%; specificity: 72%

	IF	IB	SB	DE	PS	Examples of psychometric properties
Illness Attitude Scales (IAS)	X	X	X	X		Cronbach's alpha: 0.76 (subscale: Illness Behavior), 0.85 (subscale: Health Anxiety); test-retest: 0.87 (subscale: Illness Behavior), 0.93 (subscale: Health Anxiety) (1 to 4 weeks); convergent validity with WI en SAS satisfactory; sensitivity: 53% (subscale: Illness Behavior), 79% (subscale: Health Anxiety); specificity: 84% (subscale: Illness Behavior), 64% (subscale: Health Anxiety
Somatoform Amplification Scale (SAS)					X	Cronbach's alpha: 0.77; test-retest: 0.87: convergent validity with WI and IAS: satisfactory; sensitivity: 58%; specificity: 55%
Reassurance Questionnaire			X			Cronbach's alpha: 0.83; test-retest: 0.85 (4 weeks); convergent validity with WI and IAS and SAS: sensitivity; sensitivity: 76%; specificity: 76%

	IF	IB	SB	DE	PS	Examples of psychometric properties
Maastrichtse Eigen Gezondheids-Attitude en Hypochondrieschaal (MEGAH)	X	X	X			Cronbach's alpha 0.91 for the complete scale; test-retest: 0.91; sensitivity: 74%; specificity: 93%

Note: IF = Illness Fears; IB = Illness Beliefs; SB = Safety behaviours; DE = Disruptive effects; PS = Physical symptoms

Sources: Andrews & Peters, 1998; Segal, Hersen & van Hasselt, 1994; Barsky, Wyshak, KLerman & Latham, 1990; Speckens et al. 1996; Speckens et al. 2000; Schmidt & Lousberg, 1992.

Demographic characteristics, prevalence and course

The prevalence of hypochondriasis varies per setting. Percentages based on DSM III-R criteria are higher for somatic settings (approximately 8%) than for psychiatric areas (approximately 4.5%) and the general population (approximately 4.5%) (Creed & Barsky, 2004). There are two main reasons for the high prevalence in somatic settings. First, it is inherent in the disease to not seek psychiatric help, but rather medical help. After all, the hypochondriacal patient is convinced that he or she suffers from a serious physical illness. Secondly, innocent physical ailments might be paired with extreme fear which is exaggerated in light of the severity of this specific ailment and can take on the form of hypochondriasis.

Until recently there was little research directed at the risk factors of hypochondriasis. In one retrospective study it was demonstrated that within the somatic setting patients with hypochondriasis reported more frequently

damaging events during their youth such as serious illnesses of themselves or significant others, when compared to non-hypochondriacal medical control patients (Noyes et al., 2002).

Four longitudinal studies showed that the course of hypochondriasis was chronic for a large group of patients. The number of untreated patients who continued to have hypochondriacal complaints 6 to 12 months after the first assessment varied between 34% and 67% . A more chronic course was predicted by more severe hypochondriacal symptoms, more comorbid anxiety and depressive symptoms and more use of the health care system at the first assessment (Robbins & Kirmayer, 1996; Barsky, Fama, Bailey, & Ahern, 1998; Noyes et al., 1994; Simon, Gureje & Fullerton, 2001). The frequency of hypochondriasis increases with age. A possible explanation for this is that older people are more sensitive to the development of hypochondriacal symptoms as they are, in general, more socially isolated, have more psychiatric complaints and experience an overall physical decline (Asmundson, Taylor & Cox, 2001, p.13-14).

A couple of studies in a somatic setting investigated whether patients with hypochondriasis have a higher or lower comorbidity with other psychiatric problems when compared to non-hypochondriacal medical control patients (Barsky, Wyshak & Klerman, 1992; Noyes, Kathol, Fisher & Phillips, 1994). Patients with hypochondriasis had more additional psychiatric diagnoses than non-hypochondriacals, 88% versus 51% in Barsky et al. (1992) and 62% versus 30% in Noyes et al. (1994). In particular, the comorbidity for hypochondriasis with depressive, panic, generalized anxiety and somatoform disorders appeared to be high. Obsessive-compulsive disorder (OCD) was diagnosed in 10% of the patients with hypochondriasis

in the study by Barsky et al. (1992). In Noyes et al. (1994) none of the patients with hypochondriasis were diagnosed with OCD. The results from both studies are summarized in table 4.

Due to the high comorbidity rates of hypochondriasis with other axis I disorder the validity of hypochondriasis as a primary disorder has long been disputed. Barsky et al. (1992) emphasized that hypochondriasis could best be described as a secondary disorder following a primary anxiety or depressive disorder. A number of factor analytical studies have, however, shown that hypochondriasis is a valid and reliable diagnosis based on a number of consistent characteristics (physical complaints, illness conviction and anxiety regarding illness) which have a very close relationship (Pilowsky, 1967; Barsky, Wyshak & Klerman, 1986).

Table 4 Comorbidity identified in patients with DSM-III-R hypochondriasis

	Barsky et al. (1992)			Noyes et al. (1994)		
	hypochon-driasis n = 42 (%)	control subjects N = 76 (%)	*p*	hypochon-driasis n = 50 (%)	control subjects n = 50 (%)	*P*
Major depression	43	18	.005	38	16	.005
Dysthymic disorder	45	9	.0001	8	2	ns
Panic disorder	17	3	.01	16	6	ns
Generalized anxiety	71	28	.0001	0	0	ns
Phobic disorder	43	21	.05	6	2	ns
Obsessive-compulsive disorder	10	2	.05	0	0	ns
Alcohol abuse/depen-Dence	10	18	ns	14	12	ns
Drug abuse/depen-Dence	12	4	ns	8	10	s
Somatization disorder	21	0	.0001	7	0	—
Any depressive disorder	55	20	.0001	44	18	.005
Any anxiety disorder	86	36	.01	22	6	.05
Any substance use disorder	17	20	ns	20	18	ns

Source: Asmundson, Taylor et al. 2001 (p. 142)

Differential diagnosis

Concerning the differential diagnoses, the difference between hypochondriasis and anxiety and other somatoform disorders is sometimes difficult to make. An important difference between hypochondriasis and panic disorder is that patients with panic disorder fear autonomous complaints (such as pounding chest, shaking and sweating) which are perceived as an imminent threat of an acute life-threatening somatic disorder such as cardiac arrest or a cerebro-vascular accident (Clark, Salkovskis, Ost & Breitholtz, 1997). Patients with hypochondriasis are said to be anxious of physical complaints, which they perceive as symptoms of more chronic and insidious disorders such as cancer, multiple-sclerosis or dementia (Cote et al., 1996). Contrary to patients with hypochondriasis, patients with generalized anxiety disorder worry not only about their health but also about a number of events or activities, such as, school, finance, family and work. An important difference between hypochondriasis and obsessive compulsive disorder is that in the latter the obsessions and compulsions are not limited to concerns about illness (APA, 1994). Furthermore, it has been suggested that patients with obsessive compulsive disorder in general perceive their fears as excessive or unreasonable (APA, 1994). Finally, it has been proposed that patients with OCD lack bodily symptoms accompanying their obsessions (Barsky, 1992). Despite these differences, there is also some striking phenomenological overlap between hypochondriasis and OCD in terms of obsessive thoughts followed by stereotypic rituals in order to reduce anxiety or to avert a catastrophe (e.g., Barsky, 1992; Fallon, 2000). Especially the overlap in symptom profile

between patients with hypochondriasis and OCD patients with contamination and somatic obsessions is suggested to be conspicuous (Rasmussen & Eisen, 1992). Therefore, some authors have suggested that hypochondriasis belongs to the so-called OCD-spectrum (Hollander, 1993).

Compared to patients with hypochondriasis, patients with a somatoform disorder have a great deal of physical complaints, as well as a large number of limitations as a result of those complaints; however, anxiety as a result of the catastrophic interpretation of these complaints is not the central problem (APA, 1994). This theoretical difference between hypochondriasis and the other somatoform disorders is, however, obscure between hypochondriasis and a specific functional somatic syndrome, namely non-cardiac chest pain (NCCP). Subjects with NCCP present at the department of cardiology with a wide variety of cardio respiratory symptoms (e.g. chest tightening, palpitations, and limb pain) (Bass, 1990), which cannot be explained medically but resemble cardiac problems and are therefore prone to be interpreted as signs of a serious heart condition (Eifert, 1992). As a consequence of this fearful attribution, subjects with NCCP, just like subjects with hypochondriasis, resort to anxiety-reducing behaviors in order to avert or prevent the feared catastrophe (Eifert, 1992).

Etiopathogenesis

Sigmund Freud developed one of the first modern theories of hypochondriasis in the 1900s. He saw this disorder as a very primitive defence mechanism in which "a narcissistic investment of the libido in one specific internal organ, as opposed to an external object, would alternate the

unpleasurable with the pleasurable (the equivalent of masturbation)" (Starcevic & Lipsitt, 2001, p. 187, 188). After this period, little attention was given to hypochondriasis. This ended in the 1980s with the theory of Beck, Emmery and Greenberg (1985) on the development and the maintenance of anxiety. Salkovskis and Warwick (1986) elaborated this theory to hypochondriasis and the past decades it has shown to be of great use in describing the different factors associated with the etiology and maintenance of hypochondriasis. Before discussing the cognitive theory, four other theories will be discussed; the hypervigilance model, the neurobiological model; the theory of the 'hypochondriacal personality disorder'; and the theory of somatosensory amplification.

The Hypervigilance Model

The hypervigilance model focuses on the hypochondriacal patients' constant *attention* to physical functions, as opposed to the Somato Sensory Amplification Model which implies an elevated *sensitivity* to physical sensations (Taylor & Asmundson, 2004, p. 27-29). Research by Pennebaker (1980) and Haenen, Schmidt, Kroeze, and van den Hout (1996) has demonstrated that attention given to these sensations can intensify them. Hypervigilance is caused by the catastrophic ideas that the patients with hypochondriasis have about their physical complaints. When these are perceived as threatening, the attention is automatically increased. In addition, other factors, such as a stimulant environment can distract from internal perceptions, while a less stimulating environment holds the attention towards physical sensations (Stone et al., 2000, p. 299-315).

The Neurobiological Model

The finding that patients with hypochondriasis can be successfully treated with antidepressive drugs suggests that serotonin plays a role in the development of hypochondriacal symptoms. To date, there is little research directed to the role of neurobiology in the etiology of hypochondriasis. One of the few studies is a f-MRI study by van den Heuvel et al. (2005). In this study both a cognitive and emotional Stroop task, which consisted of OCD and panic disorder related negative words, were completed by patients with hypochondriasis, OCD, and panic disorder. This study showed that during completion of these selective attention tasks, patients with hypochondriasis and OCD used different neural areas. There was no difference found in the manner in which patients with hypochondriasis and panic disorder reacted. On the one hand, the results show a specific neurological pathway in hypochondriasis and OCD. On the other hand, it remains unclear if patients with hypochondriasis differ from those with panic disorder.

The hypochondriacal personality disorder

Tyer, Keleman, Fowler-Dixon and Ferguson (1990) criticized the diagnosis of hypochondriasis as a mental disorder or "state" and instead suggested that hypochondriasis could be considered as a personality disorder or "trait". This would be especially the case when 'hypochondriasis begins early in life, shows characteristic patterns of behavior that are socially handicapping and distressing to others, and persists throughout life'(Tyrer et al., 1990). The main difference between hypochondriasis as a mental state

disorder and hypochondriasis as a personality disorder is in the latter, 'a constant preoccupation with health-seeking behaviour as a desirable goal, and a lifestyle designed to perpetuate these ends that is strongly defended at all times' (Tyrer et al., 1999). Hypochondriasis has also been associated with traits of perfectionism and rigidity (Barsky & Klerman, 1983).

The Somatosensory Amplification Model

According to Somatosensory Amplification Model, patients with hypochondriasis have the tendency to experience normal physical sensations as intense, damaging and intrusive. As a result of this, patients conclude that these symptoms must be abnormal and pathological. These attributions subsequently lead to bodily inspection and selective attention towards symptoms, which confirm the hypothesis of the presence of a serious physical illness (Barsky, 1992). The model has been studied using the Somatoform Amplification Scale (SAS) (see table 3). Results of these studies showed that patients with hypochondriasis indeed appeared to be more sensitive in terms of *reporting* physical functions than non-hypochondriacals (Barsky, Brener, Coeytaux & Cleary, 1995). But, does that mean that they are *actually* more sensitive?

With the help of perceptual acuity studies (i.e. sensory detection thresholds) and studies of perceptual accuracy (i.e. heart rate estimation), this question has been studied a number of times. The results of these studies were, however, not as expected. Individuals with high levels of health anxiety do not appear to have a lower tolerance of pain then individuals who do not experience high levels of health anxiety.

Furthermore, there is little evidence to suggest that individuals with hypochondriasis perceive physical sensations (such as heartbeat) more accurately than psychiatric control patients. By contrast, the opposite appeared to be true; patients with hypochondriasis showed poorer result than the control group (Barsky, Cleary, Barnett, Christiansen & Ruskin, 1994; Barsky et al., 1995). These results led to the theory that hypochondriacal patients possibly have 'noisy bodies.' Implied here is the idea that normal bodily functions and mild physical complaints create 'background noise' and make it impossible to perceive physical sensations (such as heart rate) in an accurate manner (Starcevic & Lipsitt, 2001, p. 223-248). To date, there is no real physiological or neurological support for the 'noisy body' theory. The fact that patients with hypochondriasis state that they are more sensitive than non-hypochondriacal patients, while they are actually no better at distinguishing physical sensations than non-hypochondriacal patients emphasizes the importance of the cognitive aspects of this disorder.

The Cognitive-Behavioural Model

The Cognitive-Behavioural Model for hypochondriasis is based on the cognitive theories of Beck et al. (1985). The basis of this theory is that situations and stimuli are interpreted as more threatening than they actually are. In the case of hypochondriasis, this means that innocent physical sensations and other health related issues are interpreted as possible evidence of a serious physical illness. The tendency to have a faulty interpretation of health related information originates often from personal experiences, such as illness of oneself or others, and information concerning

diseases by means of the media (such as a few years ago Creutzfeldt-Jacob). These experiences lead to formulating certain assumptions regarding symptoms, diseases, healthy behaviours and healthcare. The impact of these assumptions, according to cognitive theory, is determined by the anxiety they create which is also a function of the following factors:

$$\text{Anxiety} = \frac{\text{Perceived likelihood of the disease * Perceived costs, seriousness and inconviences the disease entails}}{\text{Perceived possibilities of coping with the disease + Perception to what extent external factors may be of help}}$$

Source: Starcevic and Lipsitt, 2001 (p. 205)

According to this function, the level of anxiety concerning illness depends on the value the patient assigns to the different factors. For example, anxiety reduces when both the chance and severity of illness become smaller. As the patient begins to think that he/she can deal with the illness and he/she makes use of appropriate medical care, the fear becomes even less severe. In order to determine the severity of hypochondriacal complaints and to formulate a treatment plan, all four factors have to be taken into account.

The cognitive-behavioural model assumes that many factors play a role in the maintenance of hypochondriasis. In the first place, patients with hypochondriasis pay much attention to information which supports their ideas about a serious illness, while disregarding information which does not confirm their beliefs. This phenomenon is referred to as 'confirmatory bias.'

Selective attention is one of the first examples which come to mind of 'confirmatory bias.' Patients with hypochondriasis, as a rule, focus on physical symptoms which, according to them, confirm their beliefs that they have a serious illness and reject symptoms which are inconsistent with their beliefs. Secondly, anxiety as a consequence of inaccurate interpretation results in an inevitable stress reaction including physiological reactions (such as tense muscles and chest pains). These stress reactions confirm the idea that there is something wrong with the body and further support anxiety reducing behaviours (such as avoidance, body checking, and asking for reassurance). These behaviours do reduce anxiety in the short term, but appear to have the opposite effect in the long term. Because of the attention given, both symptoms and anxiety increase. Quickly a vicious circle develops in which anxiety and anxiety reducing behaviours alternate each other. Finally, thoughts and anxiety about possible serious physical illness might lead to depression. Depression will, in general, result in yet more unpleasant thoughts about the illness and consequences resulting from this illness (Salkovskis & Warwick, 1986).

Treatment

Pharmacological Treatment

While clinicians have acknowledged the effectiveness of drug treatments for secondary hypochondriasis (hypochondriacal symptoms which manifest themselves in primary anxiety or mood disorders), there was an attitude of pessimism towards the pharmacological treatment for

hypochondriasis as a primary disorder. This pessimism was part of a general pessimism concerning the pharmacological treatment of obsessive compulsive disorder which changed into optimism in the 1980s when serotonin antidepressive drugs appeared to be effective in patients with obsessive symptoms. Because of the similarities between OCD and hypochondriasis, approximately 10 years ago the first study took place examining the effectiveness of Fluoxetime for patients with hypochondriasis without comorbidity with depressive complaints (Fallon et al., 1993). The results of this study were promising and the past decade more uncontrolled open-label studies have shown positive results for patients with hypochondriasis to the SSRI's, Fluoxetine, Fluvoxamine and Paroxetine (Fallon et al., 1993; Oosterbaan, van Balkom, van Boeijen, de Meij & van Dyck, 2001; Fallon et al., 2003). More definite conclusions about the effectiveness of antidepressants can, however be made only once controlled studies investigating the specific effects of antidepressants over and above non-specific placebo effects are conducted. To date, there is only one placebo controlled study where patients were randomly assigned to a SSRI (Fluoxetine) or a placebo. Although the results of this study support the efficacy of Fluoxetine when compared to the placebo, these results must be interpreted with caution. The study involved a relatively small sampe (N = 25) (Fallon, 1996) and must be replicated with a larger group of patients to establish whether the positive results can be attained. In table 5 there is a review of the pharmacological trials to date. The number of patients which show a clinically significant improvement appears to be independent of the nature of the comorbidity of the disease. Both patients with affective disorders who are known to respond to SSRIs, as well as patients with other

disorders appeared to benefit from this treatment. Research shows that only 4% of patients with hypochondriasis prefer drug treatment instead of cognitive behavioural therapy (Walker, Vincent, Furer, Cox & Kjernisted, 1999). While the authors give no explanations for this finding, the anxiety about possible side-effects to pharmacological treatment could be significant. Further research is necessary to investigate whether patients with hypochondriasis can tolerate the side-effects of antidepressive drugs.

Table 5: Studies of the effectiveness of SSRIs and hypochondriasis

authors	weeks, doses (≥mg) and design	N	comorbidity	duration (years)	drop-out	results and conclusions
Fallon et al. (2003)	12 weeks, Fluvoxamine (≤ 300) pretest – posttest	18	79% of all patients; 50% depressive episodes; 21% panic disorder; 21% GAS	?	29%	Response rate was 73% (N = 8 out of 11) for subjects who completed at least 6 weeks of therapy and 57% (N = 8 out of 14) for the intent-to-treat sample. Fluvoxamine is active and well tolerated
Oosterbaan et al. (2001)	12 weeks, Paroxetine (≤ 60) pretest – posttest	11	55% Pain disorder; 27% GAS; 1% Undifferentiated somatoforme disorder	15	18%	At post-treatment measurement 89% clinical improvement (N = 8 out of 9) with 5 subjects scoring within norms for healthy population. Paroxetine is active and well tolerated
Fallon et al. (1996)	12 weeks, open-label trial Fluoxetine vs placebo	20	Unknown	11	20%	67% (N = 8 out of 12) responded after 12 weeks of treatment with Fluoxetine. 50% in the placebo condition (N = 4 out of 8) experienced significant improvement. Fluoxetine is active and well tolerated
Fallon et al. (1993)	12 weeks, Fluoxetine (≤80) Pretest – Posttest	16	panic disorder, social phobia, OCD	?	14%	63% responded after 12 weeks of treatment (N = 10 out of 16). Fluoxetine is active and well tolerated

Note: In the studies of Fallon et al. a patient who was virtually symptom free was considered responder

Psychotherapy

While hypochondriasis has long been viewed as a difficult to treat disorder, results of randomized studies conducted in the last few years have shown the efficacy of cognitive behavioural therapy (CBT). Patients with hypochondriasis appeared to have significantly fewer complaints after treatment than those patients on waiting lists. These effects seem to remain in the long term. In most studies CBT is given on an individual basis; however group therapy also appears to be both acceptable and effective. In addition, group therapy appears to be cost effective, and has some other non-specific advantages, such as group support and recognition (Bouman, 2002).

CBT has been derived from the cognitive behavioural model of Beck et al. (1985). In this therapy attention is given to basic assumptions which form the core of the anxieties of health anxiety. The ultimate goal of therapy is to find an alternative and less threatening explanation for the worries and concerns as well as reducing the safety-seeking behaviours, such as checking. The most important components of CBT for hypochondriasis are:

- Identifying dysfunctional automatic thoughts and basic assumptions ('I have stomach cancer')
- Disputing automatic dysfunctional automatic thoughts and basic assumptions ('What is the proof supporting or not supporting this interpretation of stomach cancer')
- Formulation of more realistic thoughts ('Stomach complaints from spoilt food')

- Formulating behavioural experiments to test both the credibility of the automatic thoughts as well as testing the alternative thought patterns (Changes in eating patterns)
- Exposure and response prevention (Watching a program on cancer and not asking for reassurance)
- Relapse prevention (Creating a safety net to help reduce anxiety if and when anxiety related to cancer returns, as well as developing alternate methods of dealing with anxiety)

One study compared the effectiveness of CBT and stress management (Clark et al., 1998). Stress management was based on the rationale that some people react to stress with physical symptoms. Patients were taught to deal with stress with the underlying idea that the physical complaints and anxiety regarding illness would be reduced. The ingredients of stress management were not the same as the ingredients of CBT. Results collected directly post treatment showed CBT to be more effective than stress management. A follow-up assessment one year after treatment showed similar levels of improvements for both forms of therapy. According to the authors the strength of stress management lies in its clear and easily acceptable explanations for the patients' symptoms and concerns. Secondly, many of the intervention methods in stress management, such as relaxation exercises leaded to a reduction in physical complaints (Clark et al., 1998).

The relative effectiveness of two different techniques as used within CBT has also been studied (Visser & Bouman, 2001). One group of patients received cognitive therapy, while another group of patients received behavioural therapy (exposure in vivo and response prevention) (Visser & Bouman, 2001). Cognitive therapy appeared as effective as exposure in vivo

and response prevention and both proved more effective than no treatment (waiting list control group) (Visser & Bouman, 2001). Table 6 provides a summary of the above mentioned studies. The table also shows the effectiveness of CBT in the long term. Further the Barsky and Ahern (2004) study shows that also CBT with a relative small number of sessions yields positive results.

Table 6: Studies of the effectiveness of CBT for hypochondriasis

authors	design and N	number of sessions in minutes	comorbidity	duration in years	drop-out	follow-up in months	results and conclusions
Barsky et al. (2004)	CBT vs. waiting list control (WL) (n = 187)	6 x 90	?	11	25%	12	CBT>WL with regards to hypochondriasis and social functioning
Visser et al. (2001)	Cognitive therapy (CT) vs. exposure and response prevention (ERP) vs. waiting list control subjects (n= 78)	12	50% total; 31% panic disorder; 23% GAS; 13% OCD	6	28%	7	CT=ERP>WL for all results
Clark et al. (1998)	CT vs. stress managements (SM) vs. WL (n = 48)	16x 60	?	4	4%	12	Post-test: CT> SM for all measures of hypochondriasis, CT=SM for all measures of mood. Both CT and SM > WL Follow-up: CT = SM
Warwick et al. (1996)	CBT vs. WL (n = 32)	16 x 60-	?	7	6%	3	CBT > WL

Chapter 1

Objectives and outline of the thesis

The present thesis can be subdivided in three connected parts; the first part deals with diagnostic issues, the second with treatment of hypochondriacal complaints and the third focuses on the development of a new questionnaire assessing the different features of hypochondriasis. Because all chapters in the present thesis have been submitted as separate publications, it is unfortunately inevitable to prevent some overlap between the different chapters.

Diagnostics

The first two chapters connect to the paragraph on the differential diagnoses and add to the discussion whether hypochondriasis should be placed within the OCD-spectrum.

In chapter 2 data will be presented on the overlap in hypochondriacal and (the presence and fear of) bodily symptoms between hypochondriasis and OCD and vice versa. Both patients with hypochondriasis as well as OCD subjects fulfil the diagnostic criteria of the DSM-IV. After this, the group of OCD patients was subdivided in a group with and without contamination obsessions and cleaning and washing compulsions. Both OCD subgroups were phenomenologically compared to each other and to hypochondriasis. The aim of subdividing the groups was to find out whether this specific OCD subgroup indeed shows more symptomatic overlap with hypochondriasis than other OCD subgroups, as has been suggested by Rasmussen and Eisen (1992).

In the third chapter the attention is shifted from phenomenological issues towards meta-cognitive differences between patients with hypochondriasis and OCD patients with somatic obsessions. In this chapter an explorative attempt is made to translate the theoretical differences as suggested by Barsky (1992) in empirical research questions in order to examine whether patients with hypochondriasis indeed experience and perceive their health-related thoughts and behaviours in a different manner (i.e. appropriate and shameful) than OCD patients with somatic obsessions.

In the fourth chapter the focus is placed on the overlap between hypochondriasis and a specific somatoform disorder, namely non-cardiac chest pain (NCCP). The extent of health anxiety, comorbidity, and personality characteristics between both groups is investigated. In addition, this chapter also concentrates on an etiological issue; a second objective of chapter 4 is namely to assess whether personality characteristics like harm avoidance, self-directedness and cooperativeness predict the presence of hypochondriasis.

Treatment

Although there is some preliminary evidence that pharmacological treatment might be effective in treating hypochondriacal complaints, the specific efficacy of SSRI's (selective serotonin reuptake inhibitors) still needs to be investigated. In chapter 6 the results of the first randomized controlled trial (RCT) comparing the relative efficacy of a pharmacological treatment with a SSRI (Paroxetine), cognitive behavioural therapy and a pill-placebo

will be presented. Chapter 7 focuses on the naturalistic long-term follow-up of CBT and Paroxetine, which is up to 6 years.

Assessment

Until now only self-report questionnaires are available for assessing hypochondriacal complaints, most of them not covering the full spectrum (see the paragraph on Clinical Description and Diagnosis). Therefore, comprehensive clinician-rated interviews can be of additive value. The Y-BOCS, the golden standard in the assessment field of OCD, was adapted for hypochondriasis. In the seventh chapter, the psychometric properties of this first clinician-administered scale for hypochondriasis will be reported.

Summary and Discussion

Chapter 8 provides a short summary of the main results followed by an extensive discussion on the methodological issues, the implications of the results for theory and clinical practice ended by some suggestions for future research.