FACULTY OF HEALTH SCIENCES
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PhD thesis

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Cognitive behavioural therapy versus short-term psychodynamic psychotherapy versus no intervention for patients diagnosed with hypochondriasis. Result from a randomised clinical trial

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"Phull var en af de teoretikere, der elsker deres teori så meget, at de glemmer teoriens mål – dens anvendelse i praksis; i sin kærlighed til teorien hadede han al praksis og ville ikke vide af den. Han glædede sig endda over fiasko, for en fiasko, der kom af fravigelser fra teorien i praksis, var for ham beviset på hans teoris rigtighed".

Lev Tolstoj : Krig og Fred, 1869, side 60. Dansk udgave.

CONTENTS

PUBLICATIONS

The Ph.D. thesis is based on the following publications.

I. Sørensen P, Birket-Smith M. An examination of consecutive patients diagnosed with hypochondriasis. Ready for publishing

II. Wattar U, Sørensen P, Buemann I, Birket-Smith M, Salkovskis PM, Albertsen M, Strange S. Outcome of a cognitive-Behavioural treatment for health anxiety (hypochondriasis) in a routine clinical setting. Behavioural and Cognitive Psychotherapy 2005; 33:1-11

III. Sørensen P, Birket-Smith M, Wattar U, Buemann I, Salkovskis PM. A randomized clinical trial of cognitive behavioural therapy versus short-term psychodynamic psychotherapy versus no intervention for patients with hypochondriasis. Ready for publishing

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SUMMARY

This thesis is based on an examination of 80 patients diagnosed with hypochondriasis who, in a randomised clinical trial, received either cognitive behavioural psychotherapy, short-term psychodynamic psychotherapy, or no intervention (The Kathy Trial).

The thesis consists of two theoretical chapters and 3 papers. The first chapter "The concept of hypochondriasis" connects the Kathy Trial with the current understandings of hypochondriasis. It is an overview of how hypochondriasis developed as a psychopathological concept and the ongoing discussion of hypochondriasis as a valid diagnostic entity. Focus is the present understanding and how different aspects of hypochondriasis are represented in different questionnaires and rating scales. In the chapter there is an examination of comorbidity between hypochondriasis and related conditions. The chapter contains descriptions of different models of hypochondriasis conceptualized as patterns of behaviour and cognition, and examines how these models have influenced the present diagnostic definitions and facilitated focal psychotherapy in relationship to these models. The chapter contains a description of cognitive-behavioural model for hypochondriasis with focus on the misinterpretation of bodily sensations. This model and subsequently treatment is examined clinically in this thesis. The second chapter "Randomised clinical trial as a method of evaluation of psychotherapy to hypochondriasis" describes problems in relationship to general aspects of randomised trials as method for evaluating psychotherapy, especially psychodynamic oriented psychotherapy. The point of reference is the Kathy Trial.

The 3 papers deal with different aspects of the trial. The first paper "An examination of consecutive patients diagnosed with hypochondriasis" examines the comorbidity of patients with hypochondriasis in a cross-sectional design. The purpose was to evaluate hypochondriasis as a clinical diagnose and test the hypothesis of hypochondriasis as a primary psychopathological condition. The second paper "Outcome of a cognitive-Behavioural treatment for health anxiety (hypochondriasis) in a routine clinical setting" focuses on the generalisation of a specific treatment for patients diagnosed with hypochondriasis from an academic research centre to a routine clinical setting. The third paper "A randomized clinical trial of cognitive behavioural therapy versus short-term psychodynamic psychotherapy versus no intervention for patients with hypochondriasis"

describes, analyzes, and tests the hypothesis of the randomised controlled trial. The first paper has been published; the two other papers are ready for publishing.

In the cross-sectional examination of 415 consecutive patients referred to a liaison psychiatric clinic 80 patients (19%) met the diagnostic criteria for hypochondriasis and no exclusion criteria for the randomised clinical trial. The patients had a high level of psychiatric comorbidity especially anxiety, somatisation and depression. Multiple regression analyses showed no significant relationship between a specific measure for hypochondriasis (HAI), anxiety, depression and personality patterns. The finding supports the hypothesis that hypochondriasis is a primary condition and not just a dimension by other psychiatric conditions. The limitations of the study were the selectivity of the sample, which may not be a fair representation of hypochondrial patients in general, the small sample number, and a question of the specificity of the measure of hypochondriasis.

The cross-sectional design limits the possibility for examine causal relationship between comorbide psychiatric conditions. So in the following uncontrolled pilot study we had the possibility of prospective observations. We examined if a specific cognitive-behavioural therapy (CBT) for hypochondriasis could be generalized from an academic research centre, where it was developed, to a routine clinical setting. The content of treatment and the dissemination strategy is described in the paper. But in summary, the treatment involved therapist working with the patient to develop a shared understanding of health anxiety, which required identification of the patient's idiosyncratic cognitive model for health anxiety. The therapy emphasised less threatening explanation of the patients problems. The reattribution process engaged the therapist in discussion with the patients and helped patients to carry out behavioural experiment to test alternative cognitive accounts of their problems. The treatment were adapted to fit the practise of the clinic, so part of the therapy was delivered in a group therapy setting and mindfulness were included in the treatment package. Sixteen patients were treated in the study and the size of change observed at the end of treatment was substantial and statistically and clinical significant. The changes were maintained at follow up. The result indicates a successful dissemination, but there are some factors which limits the conclusions which can be drawn. The study was not randomised, so the change could be an example of regression toward the means. The patients had a high level of psychiatric comorbidity with a high proportion of complex cases, so it is not clear, alone on these findings, if the significant improvement observed is specific for hypochondriasis or other dimensions of psychopathology are

involved. The sample number was very small, and the recruitment procedure may have resulted in an atypical sample with a high level of functioning, which partly compromises the conclusion.

The pilot study was used to develop a more comprehensive randomised clinical trial (RCT) for a firmer conclusion of the effectiveness of the treatment. The result of the pilot study was used in a power calculation and an estimation of sample size. Consecutive patients referred to a liaison psychiatric clinic were examined. A total of 176 patients were assed for eligibility, where 80 patients fulfilled the diagnostic criteria for hypochondriasis and no exclusion criteria. These 80 patients were randomised to either a specific cognitive behavioural therapy developed for hypochondriasis, short-term psychodynamic psychotherapy (SPP), or waiting-list (no intervention). The patients on waiting-list were subsequently randomised to the two active treatments to enhance the power of the between treatment comparison. All randomised patients were assessed post treatment and at six and twelve month follow up. Two primary outcome measures were defined before the trial took place, which represented changes in specific and general psychopathology. We used the Health Anxiety Inventory (HAI) for health anxiety and Hamilton Anxiety Rating Scale (HAM-A) for general anxiety. The assessors of HAM-A were blinded for patients' group allocation.

The short-term psychodynamic therapy emphasized clinical principles such as neutrality and free associations and avoided explicit and active challenge of patient's belief about health and disease, which were reserved for CBT. The key therapeutic features were the therapeutic relationship, the patient's interpersonal interaction, recognition of patterns or themes in the patient's functioning, and the development of an understanding of unconscious fantasies. We developed a rating system identifying component unique to and shared by the two treatment modalities, which we used to control the fidelity for both treatments.

The null hypothesis was divided in to two parts. Firstly, that no difference was found between the 3 intervention groups post treatment, and secondly that no difference were found at follow up for the two active treatment groups. We used a modified intention to treat analysis with post-randomisation exclusion unrelated to non-compliance, withdrawal or losses to follow up, and different types of analysis of variance to test the null hypothesis. For the missing cases we used last-observation-carried forward. A total of 62 patients (68%) completed treatment. Those patients who received CBT did significantly better on all measures compared with the waiting list control group and on

health anxiety and depression measures compared with SPP group, but not on anxiety measures. The SPP group did not show significant improvement relative to waiting list. The follow up analysis showed that CBT did significantly better overall except the HAM-A, however significant interaction suggested that the difference in both anxiety measures was reduced at the final follow up point. The treatment effect of CBT was comparable with previous large-scale controlled trials, while the psychodynamic treatment was not found to have any specific effect in the present study.

Different questions are raised in the thesis about the design of the RCT and these findings. One issue is the external validity of the trial and if the patients is a fair representation of hypochondrial patients. Another issue is the question of a successful implementation of the two treatment modalities, so the treatments were well represented in the trial. We used a specific dissemination strategy for CBT, while the SPP was seen as a available, credible and well-conducted therapy, where the therapeutic engagement did not use components of CBT, enhancing the distinctness of the different treatments and therefore the comparison. We did not use a focal psychodynamic treatment but emphasised general clinical principles for psychodynamic treatment. It is possible that the effectiveness of the treatment may have been underestimated as treatment was too short for a non-focal psychodynamic treatment, and therefore not a fair representation of psychodynamic therapy.

But the result of this trial is highly significant and a confirmation of findings from previous trials, that a specific cognitive-behavioural treatment with focus on the misinterpretation of bodily sensations is benefical for patients suffering from hypochondriasis. These results strengthen hypochondriasis or health anxiety as a useful clinical concept. Further trials are recommended to examine the long-term effect of the treatment with less selected participants. New outcomes measures are recommended which reflects the patients' levels of functioning and use of health services. The psychodynamic treatment did not have a specific effect, but a short-term treatment demand a focal approach, which was not available. A psychodynamic model using attachment patterns as a framework is under development and could be useful for future trials.

THE CONCEPT OF HYPOCHONDRIAS

I have chosen to present an overview of hypochondriasis from a psychiatric, cognitive-behavioural, and psychodynamic perspective. This approach is motivated partly by the existing literature and empirical research and partly by the Kathy trial, where these three points of view meet. This division is incomplete and especially the psychiatric and cognitive-behavioural understandings mix.

Different conceptualisations of hypochondriasis

Hypochondriasis has been conceptualised as a pattern of symptoms and cognition, which have provided the items and definitions of hypochondriasis in DSM-IV(1) and ICD-10(2). This approach has been the foundation of an empirical research into the demarcation and definition of hypochondriasis, and the examination of the concept of primary hypochondriasis as independent of other psychopathological conditions. The different models of hypochondriasis have formed specific psychotherapeutic strategies with the focus on behaviour, symptoms, and cognition. These specific treatment models have resulted in a number of controlled trials concerning the treatment of hypochondriasis. Hypochondriasis has played a continuous but peripheral role in the psychoanalytic development, and the concept has been difficult to differentiate from other conditions, in which bodily symptoms are best understood in a psychopathological framework. No specific model of hypochondriasis exists in a psychoanalytic understanding, where hypochondriasis is understood as a more general manifestation seen in different conditions. No focal psychodynamic treatment exists for hypochondriasis, and hypochondriasis has been regarded as a severe condition related to schizophrenia (3-5). This approach can be seen as in opposition to a more pragmatic understanding and treatment of hypochondriasis with focus on symptoms and cognition.

In Table 1 different understandings of hypochondriasis are shown. Kenyon's critique (6) of hypochondriasis as a diagnostic entity was a reaction to the different and contradicting understandings of hypochondriasis. His solution was to reject primary hypochondriasis as a diagnostic entity, and he concluded that hypochondriasis could be explained as another mental disorder mostly mood disorder. The table illustrates the problem that Kenyon emphasises, with many potential contradicting models of hypochondriasis. But the solution, which developed in the psychiatric diagnostic understanding, was the definition and operationalized criteria in DSM-III, where hypochondriasis was included as a diagnosis in the section of somatoform disorders. A

Table 1. Different conceptualisations of hypochondriasis

	Conceptualization	Operationalisation	Treatment
Gilliespies	Hypochondriasis is a separate category, defined as a mental preoccupation with a real or suppositious physical or mental disorder; with a discrepancy between the degree of preoccupation and the grounds for it, so that the former is far in excess of what is justified; and an affective conviction and consequent concern, and with indifference to the opinion to the environment, including irresponsiveness to persuasion	None	No specific treatment for hypochondriasis.
Kenyon	Different understandings of hypochondriasis excist, which were mutual contradicting and couldn't cover the same conditions. There no jusitification for hypochondriasis as an independent nosological condition. Primary hypochondriasis do not exists and it could be explained as a mood disorder. No model is presented to examine hypochondriasis conceptually or empirically.	None.	Treatment of primary conditions, no specific treatment for hypochondriasis.
Pilowsky	Primary hypochondriasis exists. Three factors counts for more than half the variance of hypochondriasis: bodily preoccupation, disease phobia and disease conviction. Hypochondriasis is better conceptualised as abnormal illness behaviour, which includes psychiatric and psychosomatic disorders. The understanding includes sociological models about illness behaviour and the interaction between the doctor and the patient.	1) Whiteley Index distinguishes hypochondrial from non-hypochondrial. 2) Illness Behaviour Questionnaire (IBQ) examines aspects of illness behaviour.	No specific treatment for hypochondriasis.
Kendell	Hypochondrias has a high comobidity with anxiety and depression, but with hypochondriasis as its most important constituent. There is a change from a psycho-physiological concept with dissociation between emotions and bodily perceptions to the criteria in DSM-III with assumption about the development of hypochondriasis from precipitants such as depression, anxiety and somatization. Different psychological processes are mentioned but no comprehensive model is developed.	Illness Attitude Scale (IAS). Focus on attitudes and beliefs associated with hypochondriasis both diagnostic and severity aspects. Consists of 8 scales such as illness worry, health habits, disease phobia and thanatophobia.	Explanatory therapy has won a certain distribution. But with no clear reference to a specific model of hypochondriasis or therapy. Seems eclectic. Has been examined in controlled trials
Barsky	Barsky conducted an extensive empirical and conceptual research through 3 decades with concern about internal validity, classification and comorbidity of hypochondriasis. His conceptual basis was the definitions in DSM-III/IV. His understanding has changed to a cognitive conceptualisation, questioning if hypochondriasis form a discrete category. He developed the idea of somatic amplification which he sees as important element in developing and maintaining hypochondriasis	1) Structured diagnostic interview for hypochondriasis included in SCID-I. 2) Somatosensory Amplification Scale measures the intensity of somatic amplification.	1)Cognitive-educational group treatment focused on abnormal somatic sensation. 2)Individual, manualised cognitive behavioural therapy, which targets amplification. Has been examined in controlled trial.
Noyes	Uses the definition and conceptualisation in DSM-IIIR/DSM-IV. He studied the comorbide relationship with conditions such as panic disorder, and validated and developed internal characteristics such as fear of death, interpersonal aspects in framework of personality dimension/ disorder, and childhood experiences. He developed an interpersonal model for hypochondriasis based on attachment theory. The model was validated empirically and related hypochondrias to personality dimensions such as neuroticism and negative affectivity.	1) Health Attitude Survey (HAS) evaluation of somatization; focus on dissatisfaction with health problems. 2) Fear of Death Scale, correlated with measures of hypochondriasis.	Proposes psychotherapeutic treatment directed towards attachment problems.
Stracevic	Integrated psychodynamic ideas with psychiatric diagnostic understanding of hypochondriasis as nosologic entity. He developed a substantial conceptual progress for hypochondriasis but without corresponding empirical research. He conducted trials about the relationship between hypochondriasis and different disorders of anxiety	None	Uses psychodynamic approaches but no focal therapy or model. Analyses explanatory therapy from a psychodynamic point of view, but no systematic examination.
Salkovskis, Warwic and Clark	In a cognitive framework misinterpretation of bodily sensations is a fundamental psychological mechanism for hypochondriasis. There is no direct amplification but longer-term behavioural feedback mechanism such as checking parts of the body and seeking reassurance among others. The behaviour is established to prevent exposure to anxiety provoking cues. The mechanism is seen in continuum from mild concern to more extreme cases that will receive a diagnosis of hypochondriasis. The label of health anxiety is preferred. Treatment is directed at evaluating alternative, non-threatening explanations.	Health Anxiety Inventory (HAI) Discriminates between hypochondriasis and anxiety. Sensitive to normal levels of health concern as well as clinical hypochondriasis. Is a relative independent factor in hypochondriasis.	A specific cognitive-behavioural therapy is an integrated part of the model. The treatment is focused on misinterpretation and the behaviour related to health anxiety. The treatment has been examined in controlled trials
Fink	Proposes to deconstruct our current diagnostic definition based on the high comorbidity between hypochondriasis and other somatoform disorders and establish new diagnostic definitions with better discriminatory power.	New operationalised criteria for hypochondrias with rumination of health and disease as most important item.	None

consensus was established which accepted this definition as a foundation for empirical research, although hypochondriasis as a diagnostic entity continuously has been criticized proposing new conceptualizations.

The following is not a comprehensive overview of hypochondriasis but an illustration of different and sometimes conflicting understandings. I have chosen to concentrate on different researchers who play an important role in the scientific development of hypochondriasis as a psychopathological concept, and who have influenced the current diagnostic definitions. I review the cognitive model of hypochondriasis, which has a decisive influence on the understanding and treatment of hypochondriasis or health anxiety, which are the preferred term. I will present different psychoanalytic approaches to hypochondriasis as psychopathological concept, but no unifying model, which do not exist in the psychoanalytic understanding. I will present the current diagnostic definitions of hypochondriasis and different aspects of the condition, which have been reflected in a number of questionnaires used in empirical research. I review the research of comorbidity of hypochondriasis, which can be seen both as an examination of primary hypochondriasis as a valid clinical entity, and an examination of internal items of hypochondriasis such as the rumination and fear of diseases and preoccupation with somatic sensations.

Hypochondriasis as a psychopathological concept

In the history of medicine, different understandings of hypochondriasis have developed and have existed simultaneously. Hypochondriasis developed theoretically in opposition to hysteria and melancholia, and hypochondriasis as a pathological condition was part of Galen's pathology (100 AD) of organs and of humeral pathology. In this manner it was not seen strict as a mental disorder, probably because the separation between mental and organic diseases was not part of medical thinking. In the 18.th century hypochondriasis was understood as a general affection of the nervous system, and Cullen (7) assigned hypochondriasis to one form of neurosis in his nosological classification. In the late 19.th century hypochondriasis became associated with morbid preoccupation with health, and its place in a systematic classification of mental disorders was the cause of controversial discussions. A part of this discussion was if hypochondriasis should be classified as a neurotic or psychotic condition and whether it was an entity in its own right. Kraepelin (1919, 1921) considered it part of a psychotic syndrome and described hypochondrial symptoms in depression, dementia praecox, and paranoia (8). Bleuler (1924) did not recognize

hypochondrias as a disease. Jaspers (1962) perceived hypochondriasis as a reflective personality with self-scrutiny, and expectation and dread of disorders of the bodily functions.

In 1928 Gilliespie (9) supported the idea of hypochondriasis as an separate category. He defined hypochondriasis as a mental preoccupation with a real or suppositious physical or mental disorder; with a discrepancy between the degree of preoccupation and the grounds for it, so that the former is far in excess of what is justified; there is an affective conviction and consequent concern with indifference to the opinion to the environment, including irresponsiveness to persuasion. Gilliespie has influenced later conceptualizations of hypochondriasis and different aspects of his definition can be seen in our contemporary diagnostic systems, such as the persistent preoccupation with the possibility of having a serious physical disorder with no adequate physical explanation, and the refusal to accept reassurance. Additional traits such as the fear or phobia of diseases and the relationship between the preoccupation and different models of bodily sensations are not seen in Gillespie's definition. His understanding is closer related to a paranoid ideation of harbouring a serious disease, than the contemporary understanding of hypochondriasis.

The Danish psychiatrist Bjerg-Hansen (1976) dealt with hypochondria paranoia as a nosological entity (10). He discusses the problems with separating delusional and non-delusional hypochondriasis and is critical towards Gilliespie's understanding of hypochondriasis because Gilliespie do not separate his definition of hypochondiaisis from schizophrenia and paranoia. Bjerg-Hansen's own definition of hypochondriasis is inspired by German and French psychiatry, which is beyond the scope of this thesis. But Bjerg-Hansen defines hypochondriasis as a syndrome characterized by the preoccupation of having a problem in relation to the personality or the self. He does not incooperate the fear or idea of a serious disease in his definition, and his understanding is related to Ladee's and Rosenfeld's view of hypochondriasis (5;7).

Rejection of primary hypochondriasis

In 1966 Kenyon (11) questions the nosological value of hypochondriasis and focuses on the conflict between primary and secondary hypochondriasis. He rejects the existence of primary hypochondriasis independent of other psychiatric disorders, and sees hypochondriasis as an expression of a mood disorder. In a retrospective study he examines the case notes of 512 patients with primary or secondary hypochondriasis (12). Primary hypochondriasis was defined as

conditions with no other psychiatric diagnose, or condition where hypochondriasis was found to be the primary condition. There was no striking difference between these two groups, and Kenyon concludes, that it was not possibly to differentiate patients, who had hypochondriasis as a primary diagnosis from patients, who had hypochondriasis as a secondary diagnose. He therefore sees no justification for hypochondriasis as a separate diagnosis, and concludes that it is part of another syndrome, most commonly an affective one, as both groups had a high incidence of anxiety and depression. Different questions can be raised in relationsship to Kenyons study. Kenyon made no clinical evaluation of the patients. He describes differences between the two groups, but does not pay attention to this in the discussion. There is no differentiation between patients with ruminations and ideas of disease, and patients suffering from medically unexplained symptoms, what we today see as a somatoform disorder. It is a highly selective group of in-patients in a psychiatric ward, and Kenyon does not discuss the generalizability of his findings. These quistions seem weaken Kenyon's conclusions.

Kenyon does not offer a new conceptualization or model for hypochondriasis, which could be examined empirically, but only demolish the concept of primary hypochondriasis. Kenyon's understanding of hypochondriasis therefore leaves no space for a conceptual or empirical development, but his opinion is repeated in the following and ongoing discussion of hypochondriasis.

The illness behaviour

In 1967 Pilowsky supports the idea of primary hypochondriasis in his empirical and theoretical work, but develops a broader concept of hypochondriasis by including a sociological model of illness behaviour and the interaction between the doctor and his patient. Pilowsky developed the Whiteley Index Questionnaire (13), partly as a reaction to previous empirical and statistical approach to hypochondriasis using Minnesota Multiphasic Personality Inventory (MMPI) (14;15). He stated that MMPI is a symptom inventory and included items concerning the individuals' attitude and reaction to disease in the Whiteley Index Questionnaire, which are missing in MMPI. The questionnaire was given to 200 patients under care of the department of psychiatry, where 100 had been diagnosed of having hypochondrial features. The responses were used in a factor analysis. In the questionnaire no specific symptoms were included as neither the nature nor the numbers of symptoms were considered a major feature in the diagnosis of hypochondriasis. The factor analysis

revealed three factors accounting for more than half the variance: bodily preoccupation, disease phobia, and disease conviction. These three factors are reflected in the current conceptualisations of hypochondriasis and the Whitley Index Questionnaire has been widely used in empirical research and clinical work about somatoform disorders and hypochondriasis.

Pilowsky (16) diagnosed 147 patients with either primary or secondary hypochondriasis over a 2-year period in a psychiatric department as part of a routine clinical practise. The patients were both in-patients and outpatients. Hypochondriasis was defined as an unjustified concern with health or disease with only temporary response to reassurance. For patients with primary hypochondriasis no other diagnosis could be made. If anxiety or depression were present more than a mild degree, the patients were classified with secondary hypochondriasis. Pilowsky underlines that primary and secondary do not imply assumptions about aetiology and psychogenesis. These two clusters show different clinical presentation and course and indicate that they are independent syndromes.

Pilowsky (17) develops in 1970 a model for abnormal illness behaviour, where he includes his understanding of hypochondriasis. He discusses abnormal illness behaviour in relation to "the sick role" as a sociological concept, where a social system cannot function without explicit norms in relation to illness and disability, which is a condition that the individual may be granted. Usually the doctor is the appointed agent of the society, to whom the patient is obliged to cooperate to achieve and obtain the role and the privileges of the sick. Abnormal illness behaviour is invoked when the doctor do not find the sick role the patient adopts as appropriate to the perceived objective pathology, when there is a discrepancy between the organic pathology and illness behaviour. Abnormal illness behaviour covers the nature of disagreement between a patient and the doctor, the phenomenology of the patient's experience, and the patient's capacity to participate in the doctor-patient negotiations process. Normally the patient and the doctor work towards congruence in understanding the problems that makes the patient seek medical help. In abnormal illness behaviour this capacity to negotiate is invalidated. The model of abnormal illness behaviour may be used in a wide range of conditions. It is possible to cover aspects such as illness affirming, illness denying, as well as conscious or unconscious motivations. The Illness Behaviour Questionnaire (18) is a further development of the Whiteley Index Questionnaire, which evaluate the patient's abnormal illness. It is a self-report instrument with 62 questions covering different domains. The domains developed as the result of a component analyses. Domains cover such areas as hypochondriasis, disease

convictions, affects inhibition, and disturbance. Pilowsky later developed a clinical assessment instrument to abnormal illness behaviour (19). Both instruments have been validated for reliability and construct validity in published works, but have not won the same dissemination as the Whiteley index.

Pilowsky's concept of abnormal illness behaviour has brought a sociological and interpersonal perspective into the considerations of hypochondrial condition. Though he states that in some cases the patients' symptomatology and behaviour is evidence of psychopathology, he does not analyse this understanding. The model leaves you with no clear answer of which condition is an expression of illness behaviour and which condition is an expression of psychopathology.
Pilowsky used his understanding of abnormal illness behaviour in the hysteria controversy (20) in the 1960s, where Slater (21) in a follow-up study found that the only common feature in hysterical patients, were the fact that they were patients. The diagnosis of hysteria was seen as disguise for medical ignorance and source of clinical error. Walshe's reaction to this statement was (22) that when doctors have difficulties managing a condition, they suggest there is no illness, and he criticized Slater for not offering a discussion of the psychopathology of hysteria. Pilowsky brought a sociological perspective into the considerations of conditions such as hysteria and hypochondriasis. The core of abnormal illness behaviour is the disagreement between the doctor and the patient of the nature of the sick role to which the patients feel entitled, and Pilowsky proposes a conceptual transition from hysteria over somatization to abnormal illness behaviour. This transition has taken place and abnormal illness behaviour has to some degree been integrated in our present diagnostic definition of somatization disorders and hypochondriasis. Pilowkys earlier understanding of hypochondriasis has been widely used, but his conceptualization of the relation between the doctor and the patients has not disseminated to the same degree.

Ilness Attitude Scale (IAS) and explanatory therapy

Kellner is an important figure in the empirical, psychiatric research in hypochondriasis. In the 1960s and the 1970s he developed his own specific psychotherapeutic method of treating patients with hypochondriasis labelled explanatory psychotherapy (23), and developed a questionnaire scale for hypochondriasis, the Illness Attitude Scale (24). His understanding of hypochondriasis was not expressed in a specific model, and he took different positions for the relationship between hypochondriasis and mood disorders. His later view was that hypochondriasis is a primary

condition and he used the conceptualization of DSM-III, when this was published (25). He divided psychosomatic disorders into psychosomatic diseases and psychophysiological disorders (26). A psychophysiological disorder is a disturbance of function without damage to the tissue, and hypochondriasis is seen as a psychophysiological disorder. In Kellners understanding patients with psychophysical disorders do not comprehend the relationship between emotions and somatic symptoms, and Kellner perceives hypochondriasis as a neurotic disorder. His conceptualisation of a psychophysiological disorder resembles Freud's concept of actual neurosis (27). Kellner introduces different cognitive and behavioural elements in his understanding such as selective perceptions and conditioned learning.

It is Kellner's opinion that psychotherapy is the treatment of choice for psychophysiological disorders, especially conditions with hypochondrial elements. Patients with somatic functional symptoms do not respond well to insight psychotherapy but to explanatory therapy, because the patients do not understand the relationship between emotions and somatic symptoms. The initial aim of explanatory therapy (28) is to persuade the patient that his belief is false, so it no longer reinforces psychopathology. It is essential that the patients gain the conviction that their symptoms are innocuous before therapeutic progress can be made. The alliance is established partly by persuading the patients that his hypochondrial symptoms are false. Explanatory therapy focuses on accurate information, clarification, and repetitions as means to explain psychological processes. The principles of selective perception and conditioning are explained, and the patient is told how his constant awareness of part of his body is a learned condition. It is emphasised, that this information must be given repeatedly. The therapist must accept the patient unconditionally and it is important to empathize with the patients' problems. Suggestions coupled with accurate information about the patient's condition and examples from the patient's history should be employed to illustrate and underline the point. Examples are given to the patient of how to practise improve sensory perception, and how it is important to make the patient understand that the unlearning of a habit will take time.

Kellner (29) treated 36 hypochondrial patients with explanatory psychotherapy. The patients were selected if they were convinced that they suffered from a psychical illness for more than 6 months The patients were followed up till 2 years. Twenty-five patients showed an improvement at the end of the therapy.. Most patients had a symptom relief. The differentiation between somatic symptoms

with hypochondrial belief and depressive delusional belief is emphasized as essential and difficult, and several patients were excluded because of an undetected endogenous depression.

In continuation of his empirical research, Kellner uses the definition of hypochondriasis from DSM-III (30). He develops a questionnaire scale, the Illness Attitude Scale (IAS) (31), which enables an operationalisation of the diagnosis and a measure of the severity of hypochondriasis. IAS consists of eight scales with three questions each. The questions are rated on a five-point Likert scale. The scales employ topics such as illness worry, concern about pain, health habit, disease phobia, hypochondrial belief, and thanatophobia (fear of death). The items were constructed from statements made by patients with disease conviction and hypochondrial behaviour, and the scales should reflect the patient's attitude, fear, and belief of disease. The aim was to construct scales that were limited to attitudes and beliefs associated with hypochondriasis and not include items, which occur in other conditions. Kellner examined the construct validity and test-retest correlation in two groups of patients (24;32).

Kellner (33) examined 20 depressive patients according to DSM-III before and after treatment with amitriptylin and measured the degree of hypochondrial symptoms using the scores on IAS. One third of the patients had hypochondrial symptoms before treatment, which was reduced significantly to a level that matched healthy controls after treatment. The conclusion was that melancholia is one of the causes of the hypochondrial syndrome, which can be effectively treated. The findings do not necessarily support the view that hypochondriasis is a depressive equivalent, however, it supports the view that hypochondriasis can be a masked depression. In a review Kellner (34) defines hypochondriasis as false belief of disease or exaggerated fear of acquiring a disease. He understands the condition as spectrum from transient states reassured by physician's over a persistent or distressing disease phobia to an unfounded conviction of having a fatal disease. A common sequence of hypochondrial reaction is a stressful event with anxiety and depression and subsequently the idea of having a disease. If the idea persists a vicious circle develops with anxiety, selective perceptions, and misunderstanding of bodily sensations. These processes interact with the patients' personality. The diagnostic difference between primary and secondary hypochondriasis can be difficult to establish.

Kellner (35) used IAS to compare a group of medical patients referred to a consultation service with a control group of medical patients. The groups differed in psychiatric measures but both scored high in hypochondrial tendencies. This finding indicates that physically ill patients with hypochondrial features are not referred preferentially to psychiatric consultations service, possibly because the physicians are unaware of the patients' fears. In a subsequently examination from 1989 a total of 21 psychiatric outpatients with the DSM-III diagnose of hypochondriasis were matched with a group of non-psychotic, psychiatric patients, and a group of patients from a family practise (25). The hypochondrial patients were selected because they had fears, belief, and attitude toward disease as defined in DSM-III. The hypochondrial patients rated their somatic symptoms and anxiety as more severe than other psychiatric patients. So somatic symptoms, anxiety, and depression are important constituents of the psychopathology of hypochondriasis, and anxiety and depression may initially have been precipitants but later in hypochondriasis appear as coexistent disorders. The findings does not support the idea that hypochondriasis is a defence against anxiety or an expression of a masked depression, which is a revision of Kellner's earlier understanding. The self-reported attitudes expressed in IAS suggests a syndrome with features that is consistent with DSM-III description of hypochondriasis (36). There were conspicuous differences between the hypochondrial patients and the control groups, where hypochondrial patients reported substantially more thanatophobic features than other participants.

In a subsequent study Kellner (37) compared 100 consecutive non-psychotic patients in a psychiatric outpatient clinic with 100 patients matched accoding to age, sex, and social status from a general practise. The findings confirm previous studies that hypochondrial attitudes are associated with anxiety and depression. But in a stepwise linear regression self-rated depression did not appear as a predictor of hypochondrial attitudes, while anxiety was a significant predictor for hypochondrial attitudes in the group of female psychiatric patients, and somatization was a significant predictor in the group of male patients. Kellner proposes that persistent functional somatic symptoms or undue attentions to bodily sensations may induce hypochondrial ideas. If the bodily symptoms are the somatic manifestation of anxiety this may induce self-observation and selective perception motivated by fear. The association between hypochondrial fears and depression was secondary because of the frequent coexistence with anxiety and somatization. This finding do not support the hypothesis that hypochondriasis is a manifestation of a mood disorder. It indicates

that patients who fear a disease are more anxious, while patients who falsely belief they have a disease have more somatic symptoms.

In the 1980's and the 1990's several research groups have used the understandings and concept developed by Kellner, and IAS has received some distribution. Fava (38;39) integrated different aspects from Kellner's theory in his research on the comorbidity of hypochondriasis, depression, and anxiety disorders. Fava stated that the concept of abnormal illness behaviour can be used as framework for diagnostic differentiation between hypochondriacal beliefs and disease phobia (40), and Fava examined (41) Kellner's therapeutic intervention, explanatory psychotherapy, under controlled conditions.

Starcevic (1989) integrated psychodynamic ideas with psychiatric diagnostic understanding of hypochondriasis. He facilitated a conceptual progress for hypochondriasis but without corresponding empirical research. Starcevic was in the beginning of the 1990's a part of Kellner's research team, where he conducted trials on the relationship between hypochondriasis and disorders of anxiety (42-44).

Noyes (1994) has made comprehensive research into the comorbidity between hypochondriasis and anxiety disorders (45-48), and in this connection used (49) the concept of tantrophobia from IAS and developed a fear of death scale, which was highly correlated with measures of hypochondriasis. Noyes proposed hypochondriasis placed under the anxiety section in DSM-V(50), and used an interpersonal model for hypochondriasis (51) based on attachment theory. He proposed that patients with hypochondriasis have anxious and insecure attachment styles that lead to maladaptive communication of their needs. He examined this model empirically (52) with measures for attachment style and personality dimensions, and found a relationship between different attachment styles and hypochondrial symptoms. According to the interpersonal theory insecure attachment is manifested in maladaptive interpersonal behaviour with a significant association with personality dimensions such as neuroticism. Noyes (2005) examined personality dimensions for hypochondrial patients in different trials (53;54), which seems to confirm his interpersonal model of hypochondriasis.

Bodily amplification and hypochondriasis as a psychopathological entity

During 25 years of research Barsky has established an impressive line of empirical work about hypochondriasis. He has examined central aspects of hypochondriasis and maintained the position that hypochondriasis is a specific psychiatric diagnosis. He has empirically validated the internal characteristics of the diagnostic concept and diagnostic items, and found internal, external, and construct validity for the disorder (55).

Barsky examined many different statements and hypothesis about hypochondriasis and he has adopted and developed new conceptualizations about the condition in an original thinking and expression. Most of Barskys empirical research is based on the same setting and instruments in a cross-sectional case-control study of patients meeting the diagnostic criteria for hypochondriasis and non-hypochondrial comparison patients. The patients are recruited from a medical outpatient clinic. This raises the problem of generalizability of Barsky's findings.

In an overview, Barsky presents different models of hypochondriasis conceptualised as a psychiatric syndrome (56). Hypochondriasis can be seen either as psychopathological entity, or a cluster of illness attitudes and behaviours that are not psychiatric in nature. He states that our lack of knowledge about hypochondriasis reflects modern medicine's emphasis on disease rather than illness. Hypochondriasis can be conceptualized as chronic psychopathological condition epitomized in DSM-III, where it is defined as an unrealistic interpretation of ones bodily sensations leading to fear or belief that one has a seriously disease. The disorder may be primary and occur without any other mental disorder or secondary in the course of preexisting psychiatric disorder. A central question for Barsky is the understanding of primary hypochondriasis (57), where no other psychiatric disorder is present or is independent of hypochondriasis. Secondary hypochondriasis occurs as an ancillary feature of a more pervasive and dominant condition such as a life-threatening disease or secondary to other psychiatric disorders. Primary and secondary hypochondriasis does not refer to any presumed cause-and-effect relationship. The question is to what degree hypochondriasis occurs in the absence of other psychopathological features and the extent of overlap to anxiety disorders, depression, and somatization disorder. Patients with panic disorder have elevated hypochondrial fears, and cognitive researchers propose (58) a similarity in the two conditions in form of misinterpretation or catastrophizing of somatic symptoms. There is a

correlation between depressive symptoms and the degree of hypochondriasis both in hypochondrial and depressive groups (59).

Barsky increasingly use a cognitive understanding of hypochondriasis (60), and defines hypochondriasis as an enduring attitude toward health, concern of the body, and belief of disease. Barsky (61) proposes that hypochondrial patients mistakenly believe good health to be a symptom-free state, and that symptoms indicate disease. This belief system could be seen as a cognitive schema that guides perception, memory, inference and judgment. This latent cognitive schema about health and disease can be activated by current life stresses (62). The schema is supported by confirmatory bias that leads hypochondrial individuals to attend selectively to information supporting this schema and ignores disconfirmatory information. The model explains the refractoriness to medical reassurance in hypochondrial individuals.

The concept of bodily amplification has a central position in Barsky's work. He introduced the concept early in his theoretical development, examining different psychiatric aspects about patients in primary care, and the reasons why they visit doctors (63;64). The concept is incorporated in the DSM-III-R (65), which emphasises the interpretation of physical signs and sensations as evidence of physical illness. According to Barsky (66) this statement is consistent with a model where hypochondrial patients amplify benign somatic sensations, and misattribute them as serious disease. He understands these intense and disturbing bodily sensations as the foundation for the misinterpretation. In this conceptualisation the other clinical characteristics are secondary phenomena. He supports his model on findings in experimental and cognitive psychology. He develops a self-report instrument that reflects the patients' perceptual dysfunction and bodily amplification. Somatization was assessed independently by a combination of items from MMPI and Symptom Checklist-90-Revised (SCL-90R) (67). By the use of stepwise regression analysis Barsky's concept of amplification was the most powerful predictor of hypochondriasis, expresses by the Whitely Index. Amplification plays a lesser role in bodily preoccupation than in disease fear and conviction. So the work suggests a relationship between amplification and hypochondrial attitudes, which support the hypothesis that misattribution of benign sensations is important in the development of hypochondriasis. Barsky continues to use this self-report rating system in a number of scientific papers, and develops a cognitive treatment for hypochondriasis (68) with amplification as a central target for the therapy. This treatment approach had recently been examined under controlled conditions (69). Other research teams has used the understanding and instrumentation of

bodily amplification to some degree and examined the reliability and validity, which were found satisfactory (70). Barsky's conceptualization of the relationship between bodily sensations and hypochondriasis emphasise other aspects than Salkovskis and Clark's (71) cognitive understanding of health anxiety, which see misinterpretation of bodily perceptions as the central psychological mechanism.

In the following I have chosen to refer in more details to some of Barsky's works. In the first examination of the concept of hypochondriasis from DSM-III, Barsky develops a screening method to hypochondriasis for outpatients in a medical clinic so he was able to separate 2 groups of patients with and without hypochondrial features and compare these groups with different variables (72). This design and screening method has been the foundation of most of Barsky's empirical research. Barksy discus if a hypochondrial attitude constitutes a psychiatric disorder or is an intensification of a normal concern of health. With the criteria of hypochondriasis in DSM-III as a basis and in integration with the existing literature and clinical surveys eight criteria for hypochondriasis were formulated: disease conviction, disease fear, bodily preoccupation, somatic symptoms, illness and sick role behaviours, disability, absence of medical disease, and absence of other psychiatric disorders. The first three criteria were developed from the Whiteley index (73) while the fourth criterion of hypochondriasis was drawn from the SCL-90R and other scales designed to measure somatization. These criteria constituted a self-report questionnaire, while the following three criteria were obtained by a structured interview and medical record audit. The absence of other psychiatric disorders was obtained by the use of Beck Depression Inventory. Participants were obtained from a general medicine outpatient clinic and consisted of patients attending on randomly chosen days at varying times. A total of 111 patients were asked to participate, and 92 patients completed the assessment. By the use of the above mentioned criteria, an examination of hypochondriacal attitude and somatic symptoms showed no evidence of bimodal distribution. Using stepwise multiple regression it was shown, that for both hypochondriacal attitude and somatic symptoms the degree of depression emerged as the most powerful predictor. The patients show varying degrees of hypochondriacal attitude and somatic symptoms, but the design of the study did not allow for a clinical diagnose of hypochondriasis. At that time no structured interview using DSM-III criteria for hypochondriasis existed. A limitation of the study is that only Beck Depression Inventory is used as a clinical tool for investigation of psychiatric comorbidity. The score for depression is the strongest predictor for hypochondriacal attitude and somatic symptoms, but no other score for psychiatric

comorbidity is part of the study. The Participants of the study were outpatients from a medical clinic, and medical diagnoses were obtained from reviews of the medical record. It is an inaccurate finding, and the nature of the hypochondriacal attitude is uncertain. There is no information about how long this attitude has lasted, and if it developed in connection with a known medical disease. But hypochondriacal attitude was not significantly related to the number of medical diagnosis in the record. In spite of these limitations the conclusion of the study is that the essential core of the hypochondriacal syndrome appears to be a complex of attitudes associated with somatic complains. A complex that is consistent with hypochondriasis defined in DSM-III. We do not know how well the findings can be generalized to the whole population. And we do not know if these cross-sectional findings represent enduring and stable traits, or how much a psychosocial event of being a medical patient influences the findings. In addition it is medically ill patients, without information about the extent of medically comorbidity.

According to Barsky we have an agreement on the diagnostic criteria for hypochondriasis, and the disorder appears to have internal, external, and construct validity, But we lack an instrument to establish validity and reliability for diagnosing hypochondriasis (55). Barsky developed a structured interview to diagnose hypochondriasis, which could be used as module for the Structured Clinical Interview for DSM-III.(74). Patients scoring above a cut-off score for hypochondriasis were divided into interview-positive and interview-negative patients. There was a high interrater agreement on the diagnostic criteria and the diagnosis of hypochondriasis; but there is no data of which criteria the interview-negative patients did not fulfil. There was a reasonable concordance between the interview and other independent measures of hypochondriasis, and a reasonable concordance with external characteristics such as social functioning and daily living. This was seen as support for an external validity of the interview, which demands an implicit assumption that hypochondrial patients have a lower level of functioning. The relationship between a diagnostic instrument and a diagnostic construct is discussed, and it is stated that hypochondriasis is apparently a dimensional rather than a categorical variable. It causes empirical problems to transform the dimensional construct into a categorical construct. It is difficult to separate the validity and reliability of the diagnostic construct from the instrument that measures it. The instrument cannot be seen in isolation from the disorder and the stability and validity of hypochondriasis remain unclear as a nosological entity.

To determine the psychiatric comorbidity in a representative sample of DSM-İII-R hypochondriacs from an outpatients medical setting Barsky (57) used the screening questionnaire from earlier investigations. A total of 8.9% attendees on randomly selected days the exceeded cut-off score and 41 were diagnosed with DSM-III-R hypochondriasis with a structured interview. A group below the cut-off screening was randomly selected to constitute the comparison group. Psychiatric disorder was assessed with the Diagnostic Interview Schedule (DIS) (75). Only 11.9% of DSM-III-R hypochondriacs had no other Axis I lifetime diagnosis and only 21.4% had no concurrent Axis I diagnosis. Sixty-three percent had a personality disorder caseness according to Personality Diagnostic Questionnaire (76) The prevalence of DSM-III-R somatization disorder was 21.4% and the prevalence for sub-threshold somatization disorder was 39.0%. The bodily symptoms of somatization disorder were not significantly correlated with hypochondrial attitudes. The morbidity of DSM-III-R hypochondriacs was significantly greater than the comparison group. As only one fifth of the patients were without concurrent Axis I diagnosis, the rate of primary hypochondriasis, understood as no other co-morbid Axis I conditions, was low, and the overlap to anxiety and depression were extensive. Few features distinguish hypochondriasis with anxiety and depressive disorder from patient without comorbid disorders. This is consistent with Kenyon's previous investigations (16). In secondary hypochondriasis a higher degree of fear of disease was found, which may reflect the prevalence of anxiety disorder in these patients, while disease convictions was not significantly higher in secondary hypochondriasis.

Hypochondriasis is generally thought to be a chronic and stable condition with low spontaneous remission rate, but the empirical evidence supporting this understanding is sparse. Barsky (77) conducted a 4- to 5-year prospective, case-controlled study of DSM-III-R hypochondriasis. He used the same settings as in previous studies by screening consecutive patients from a primary care clinic by the screening instrument described earlier. The patients above a predetermined cut-off for hypochondrial symptoms underwent a diagnostic interview. A group below the cut-off was chosen as a comparison group. The hypochondrial group improved considerably but 63,5% still met the DSM-III-R criteria for hypochondriasis at follow-up. Hypochondrial symptoms and somatization declined significantly during time. It was suspected, that at the time the patients were enrolled in the clinic, they were likely to have an illness period, and this was less likely at follow-up. But the hypochondrial sample still remained more hypochondrial than the comparison group. The medical morbidity increased in both groups but did not differ. The mortality of the hypochondrial group was

one half of the comparison group. The body amplification score did not change over 4 to 5 years, even though hypochondrial symptoms and somatization did decrease. Remission was more closer associated with a decline in somatization than in hypochondrial attitudes. An unstructured interview revealed that clinical improvement appeared to result from environmental factors more than cognitive shifts, where some had substituted elaborated self-treatment regiments for former extensive pursuit of medical care, providing a comforting sense of self-control over health.

Barsky has over the years consolidated his understanding of hypochondriasis as an enduring attitude toward health, concern of the body, and belief of disease. He uses the definition in DSM-III-R and DSM-IV in his empirical approach, and in that way underline hypochondriasis as a psychiatric disorder. He uses a cognitive model for characterizing the belief system of the hypochondrial patient and focuses on bodily amplification as an important feature. He developed a cognitive-educational treatment based on his model that targets the cognitive and behavioural amplification of benign bodily symptoms, and the treatment has been examined in a randomised controlled design, which indicates that it is an effective treatment. Barsky is therefore an important contributor to the present understanding of hypochondriasis, and he has incorporated factors into a comprehensive model of hypochondriasis, which serve as a platform for psychiatric and cognitive-behavioural interventions (78).

Misattribution, reassurance, and hypochondriasis as a cognitive construct

In a series of important papers in British psychiatry and psychology a powerful and quick conceptual development of hypochondriasis in a cognitive framework took place. This understanding was based upon experimentally validated principles of assessment and treatment. Methods and understanding were implemented from models of anxiety and obsessive disorders. This development was succeeded by controlled trials validating treatments based on these understandings. It is my impression that the 3 most important papers in which the fully developed model was introduced were published in 1989.

David Mechanic's model of hypochondriasis is related to social sciences and focus on the social context where abnormal illness behaviour develops (79). The approach is learned behaviour and offers therapeutic possibilities in terms of learning theory, which is used in cognitive-behavioural understanding of hypochondriasis. A central psychological mechanism is the tendency to perceive

signs and symptoms as more dangerous than the really are. David Mechanic conceptualised hypochondriasis as a process of misattribution, where patients appraise bodily perception as different from normal sensation. This transition from ordinary bodily sensation to symptoms is frequently observed with medical students. Factors contributing to this development include social stress, anxiety, bodily symptoms, and new incomplete information about diseases. The students' access to more detailed medical information contributes to the attributing process. It is understood as a reconcepetualisation in the context of newly developed information about diseases. There have been speculations about this transient phenomenon – all doctors are not hypochondrial – and it is proposed that the correction of the misattribution is a more exact and clearer knowledge, which facilitates the disconfirmation of the earlier misattribution. Mechanic analyses the conditions under which such misattribution may occur. He points to the idiosyncrasies in hypochondrial developments and the perception of personal vulnerability. He discusses the process of identification and points to different modes of interaction, where identification might be important. He emphasizes the observation that hypochondrial patients are not easily reassured and propose an understanding of the mechanism of disconfirmation. He finds that non-specific reassurance provides no alternative framework for the hypochondrial patient to understand his symptoms and fear, and it is Mechanic's opinion that credible instruction would help the hypochondrial patient to avoid attributional errors. A general reassurance should not stand alone. Frequently, the interpretation is provisional and vague. Balint (80) indicates that the patients' attribution is readily changed by the physician's suggestions and may alleviate the patient's distress, if the physician's can provide a benign interpretation, that are credible and can reassure the patient and bolster his sense of mastery. The suggestion must be credible and consistent with the patient's experiences and expectations to the future. The alternative attribution must relieve the patients' anxiety and be culturally and psychologically acceptable. A specific treatment model of disconfirmation has developed in the cognitive understanding of hypochondriasis and health anxiety. But it is a model where the psychological process of identification and the patients' idiosyncrasies are neglected.

In a conceptual paper Mayou (81) sees hypochondriasis as an arbitrary syndrome defined by consultation behaviour and not true phenomenology. He understands hypochondriasis as a reaction like anxiety, ranging from normality to disabling severity. He focuses on disease phobia and recognizes that this entity is difficult to differentiate from hypochondriasis, and describes disease phobia as a persistent unfounded fear not allayed by reassurance. He proposes a mechanism of

interpretation of bodily symptoms, which he connects to disease phobia and hypochondriasis. Misinterpretation is important and determines the psychological reaction and disability.

One of the key figures in this approach was Paul Salkovskis. He emphasizes (82) the importance of considering psychological processes rather than diagnostic categories. The approach is psychophysiological using a stimulus-response model: a particular type of stimuli or psychological reaction produces a characteristic physiological reaction. It is an important principle in the cognitive-behavioural approach that the patient's problem should be positively formulated in psychological terms and not relies on diagnosing psychological problems by exclusion. The psychological conceptualisation is crucial and is the primary approach, while medical diagnostic groups have secondary considerations. The approach should be realistic and provide a context for a working cognitive-behavioural hypothesis, which is formulated by identifying factors currently maintaining the patient's problem and the distress experienced. Salkovskis and Warwick (71) introduce a specific cognitive-behavioural approach to hypochondriasis. They prefer a less ambiguous label as health preoccupation and see the phenomenon as following a continuum from mild concern to a more extreme cases who will receive a diagnose of hypochondriasis. They point to two key elements of hypochondriasis: preoccupation with bodily health out of proportion to existing justification and the pursuit of reassurance. They differentiate morbid health preoccupation from obsessional thoughts. Morbid preoccupation is repetitive, excludes other mental activities, and is associated with disturbed mood. The preoccupation is consistent with the personality and experience of the individual and is regarded as sensible. The preoccupation of health is a key cognitive element, and the behavioural components such as seeking reassurance from physicians and checking on bodily states serves as an important maintenance functions. This is illustrated with two cases where the behavioural treatment consists in not giving the patients reassurance, which reduces hypochondrial symptoms and behaviour. The alleviation of anxiety by reassurance was transient and resulted in long-term worsening. There is a similarity to obsessive-compulsive disorder where avoidance behaviour in a similar way maintains the anxiety. So the unrealistic fear of hypochondriasis persists not despite medical reassurance but because of repeated medical reassurance.

Warwick and Salkovskis (83) defend the position that hypochondriasis can be primary and can develop independent of other disorders. They question the method Kenyon (84) used to refuse the

concept of primary hypochondriasis. They stress the importance of considering the chronological development in primary-secondary distinction, where the existence of a primary hypochondriasis is followed by a secondary depression, and the authors make an analogy to obsessive-compulsive disorders and the existence of a primary obsessive-compulsive disorder. It is proposed that misinterpretations of bodily sensations are a fundamental psychological mechanism in both hypochondriasis and panic attacks. Such misinterpretation may lead to avoidance of situations, which are likely to trigger panic attacks. The avoidance represents the patient's attempt to deal with the catastrophe, which he perceives as being about to occur. In hypochondrial fears the anticipated harm is perceived as much less imminent. This gives the hypochondriacal patient more time to prevent the anticipated disaster by seeking medical attention. Patients with panic attacks tend to misinterpret autonomic symptoms, which provide an obvious feedback mechanism where anxiety may rapidly escalate. In hypochondrial problems, the symptoms misinterpreted a not likely to by subject to such direct amplification as in panic attacks, and the feedback mechanism is more behavioural and longer term by maintaining focus on particularly parts of the body with repeated checking or physical manipulation. The panic patient believes that the catastrophe is happening already, whereas the hypochondrial patients tend to believe that the symptoms indicate a more insidious course.

Warwick and Salkovskis express that from a cognitive perspective anxiety occurs, when a particular situation is experienced as a threat, and the patient's ability to cope with the situation is doubted (83). So the cognitive hypothesis of hypochondriasis proposes that bodily signs are perceived as more dangerous, than they really are, and a particular illness is believed to be more probable than it really is. At the same time the patient is not able to prevent this illness and is unable to affect the course. They propose that hypochondriasis develops as knowledge about past experiences of illness (83). This knowledge leads to a specific assumption about symptoms, disease, and health behaviour, which is learned from a variety of sources and consist of both previous experiences and present information. Once a critical incident has resulted in a particular assumption, this leads to confirmatory bias in the patients thinking. When health anxiety has developed other mechanisms may be involved in maintenance of the problem. Anxiety results in physiological arousal where patient misinterpret autonomic symptoms as further evidence of physical disease. Selective attention to illness related information such as perception of bodily change leads to bias about information of disease. Behaviour designed to avoid, check for, or exclude physical illnesses will

maintain anxiety. Unlike the more immediate misinterpretation of panic patients, the misinterpretation of hypochondrial patients allow more scope to seek safety by attempts to obtain a medical resolution of the perceived threat to their health. The patients will not seek consultation because of inconvenience of the symptoms, but due to the presence of a variety of anxiety provoking cognitive events and intrusive thoughts about the causes of the symptoms. So cognition has a major impact on illness related behaviour. This behaviour is established to prevent exposure to anxiety provoking cues, and includes a wide range of actions intended to check on health state. Checking and reassurance focus patient's attention on their fears and prevent habituation to the anxiety-provoking stimuli (82). Avoidance behaviour maintains the patients' preoccupation with disease by preventing the patient to obtain information, which contradicts his interpretation of the symptoms. Reassurance-seeking behaviour can have the same effect as obsessional checking. It focuses attention on patients' worries reducing anxiety on short term, but increase preoccupation in the long term. Patients anxious about their health respond differently to health reassurance, which becomes counterproductive as the patients selectively attend to and misinterpret the reassurance itself. Some behaviour may have a direct effect on the patient's symptoms as repeated rubbing of a painful area to check if it still is painful.

As part of the assessment and treatment abnormal behaviour has to be identified and modified (82). Reassurance function as a compulsive ritual should be prevented and used as basis for re-attribution. Warwick and Salkovskis disagree with the view presented by Kellner (85) and Pilowsky (86), whose treatment strategies consist of repeated reassurance. They postulate that this most unlikely will be a useful strategy with patients fulfilling the diagnostic criteria for hypochondriasis. There is a need for a more careful definition of reassurance, as reassurance can be offered in a variety of ways, some helpful, and some may increase anxiety. Salkovskis (82) extent this understanding of health anxiety to a larger group of patients, who have disturbed experience and perception of the body. He sees the model as applicable on a wide scale of psychosomatic problems and it should be of central importance in psychological medicine.

In a short and consice paper Warwick (87) describes the treatment, which is based on this cognitive conceptualisation of hypochondriasis and health anxiety. The treatment is directed at evaluating alternative, non-threatening explanations for the patients with health anxiety. The explanation must provide the patient with a credible and normative account of his idea of disease. The treatment model is elaborated by Salkovskis (82) with more clinical details to illustrate the treatment and

establish a more feasible impression for the reader. Assessing belief is an important part of the initial assessment for hypochondrial patients. What goes through the patient's mind, when symptoms are at their worst? The patient can have an active avoidance trying to suppress thoughts of disaster. The effect of cognitive avoidance can be an unpleasant breakthrough of terrifying thoughts. The patients can have exaggerated dysfunctional belief about health and illness and use an overinclusive cognitive style. Behaviour, which is the consequence of the patient's symptoms and anxiety, are assessed in detail. This includes both actions of what the patient actually does and less voluntary actions such as focusing on the body. Reassurance seeking from medical or non-medical sources and behaviour that anticipates symptoms, anxiety, and associated thoughts should especially be assessed. If the patient habitually avoids a certain activity, they cannot identify associated thoughts. People with hypochondriasis often report few identifiable negative thoughts. According to Salkovskis the therapeutic target is to engage the patient sufficiently to be able to assess the problem collaboratively (82). But no treatment should be offered until the therapist has reached a positive psychological formulation of the patient's problem. On the basis of a preliminary understanding of the problem, the therapist summarises what the patients have said, emphasising the patients symptoms, thoughts, belief and behaviour, and present the conceptualization in these terms. The therapist and patient must agree on treatment goals. The therapist attempts to arrive at a psychological formulation for treatment of the patient's problem. But the patient may have a different set of goals. He might see the therapist as a new source of expert reassurance or regard the therapist as a potentially ally in the attempt to rule out physical illness, or to have his belief of medical basis of his problems accepted as true. These different expectations should be reconciled if treatment is to be effective. The therapist accepts, that the patient experiences physical symptoms and believe that they are due to a serious physical illness. The therapist explains that there may be alternative explanations of the observation the patient has made, and treatments involves evidence of alternative understanding and include use of specific tasks to test these explanations. The patients are not asked to give up their view of their problems, but to test an alternative for a given period. A combination of discussing the basis of negative belief, self monitoring, and behavioural experiments is applicable to a wide range of symptoms involving anxiety and depression, as a reaction to physical symptoms and fear. Ratings indicate how successful a belief change has been. In situations, where the hypochondrial believes are present, the negative thoughts should be identified and challenged, as disconfirmations in this situation will

have the biggest impact on the patient's behaviour. Behavioural experiments are a powerful way of changing patients' belief about the origin and nature of symptoms.

Salkovskis states that the aim is to demonstrate to the patient, that others factors is responsible or can influence their symptoms (82). Behaviours involved in somatic problems are perceived as having a preventive function. They are therefore relatively difficult to modify without attention to the underlying beliefs. When illness behaviour is prominent, the treatment goal is to elicit and demonstrate the role of behaviour in maintaining anxiety, preoccupation, and physiological disturbance. The use of questions as part of a guided discovery can be helpful, and direct demonstrations are convincing by showing that changing behaviour affects symptoms. The patient and therapist design experiments to test the patients' belief, that behaviour keeps them safe from serious harm, and to see if behaviours the patients' think relieves symptoms, really do so. The way the patients seek reassurance can vary such as causal conversations and bodily checking. It is helpful to devise behavioural experiments to demonstrate the effect of reassurance, which can function as an engagement strategy. Self-monitoring of anxiety about health, specific illness-related thoughts, and need for reassurance is regularly rated on a scale. The self-monitoring is used as basis for discussion about the way reassurance keeps anxiety going, and to engage the patient in treatment and establish a collaborative relationship. It provides a rationale for controlling reassurance seeking, and helps the patient to tolerate the initial anxiety caused by the behaviour change.

The model and the treatment were fully developed with these papers (82;83;87). The next steps were to validate the treatment empirically and to develop treatment protocols and rating systems to evaluate the treatment efficacy and fidelity of therapist using this treatment model.

A psychodynamic concept of hypochondriasis

In a psychodynamic understanding there is not a comprehensive or unifying model for hypochondriasis, so different understandings of hypochondriasis is possible. These conceptualizations do not exclude each other, but give the possibility of a more differentiated view of the patient suffering from hypochondriasis. This enables a flexible approach but obstruct a focal psychodynamic understanding and intervention.

Freud originally classified hypochondriasis, along with neurasthenia and anxiety neurosis, as an example of an incomplete actual neurosis lying closer to psychoses, and states that hypochondria stands in the same relation to paranoia as anxiety neurosis does to hysteria (88). It was Freud's opinion that unlike the psychoneurosis the symptoms in actual neurosis was a toxic condition which could not be analyzed (89). Freud understood hypochondriasis as a narcissistic condition intimately related to paranoia (88;90). But Freud was not satisfied with the concept of hypochondriasis and saw it as a disgraceful gap in the psychoanalytic work (91). Later hypochondriasis have been recognized as playing a part in the clinical pictures of the neuroses, and the psychoanalytic work has brought forth that hypochondrial phenomena is an event in the transference (3) .

There are a few psychoanalytic papers solely concering aspects of hypochondriasis. McCraine propose an ego psychological model for hypochondrial neuroses (92), where he sees hypochondriasis as a defense against feelings of low self-esteem perceived as there being something wrong with the self. He states that on a descriptive level the patient is suffering from the idea of having a disease. The patient had sought a diagnosis as a medical sanction for a sick-role type of adjustment. This sanction provides both a hope and a relief. The hope is that what is wrong with the self has been discovered and cured medically. There is a displacement from the psyche to the soma with the transformation of an amorphous feeling that something is wrong into a somatic disease. At a dynamic level the patient is characterized by chronic anxiety and recurrent depression, activated by repetitive frustrations of basic intake and self-protective needs. McCraine proposes that the patient has no insight into the connection between his frustrations and maladaptive functioning. The neurotic symptom is a compromise solution of an unconscious conflict and hypochondriasis serves the ego function of conflict-resolution by allowing the patient to express impulses, dependency and anger without loss of self-esteem. As in all neuroses there is a depressive core in hypochondriasis. But the depressive pain is a primary reaction rather than a secondary defensive maneuver. It is a painful frustration rather than a masochistic need to suffer. Hypochondriasis serves the ego function of conflict-resolution by allowing the patient to express impulses, dependency and anger without loss of self-esteem.

Melanie Klein (in 1934) emphasized the concreteness of the unconscious phantasies as internal objects and has related the hypochondrial anxieties of the adult to early experiences of the infant, and differentiated between depressive and persecutory types of hypochondriasis (93). Herbert

Rosenfeld (in 1958) uses these metafores of unconscious phantasies as internal objects, and states that in a chronic form of hypochondriasis there is a failure of normal splitting and differentiation of good and bad objects, which results in a confusional anxiety or confusional state (5). The good parts of the self and the good objects on which the stability of the ego depends are constantly in danger of being overwhelmed by the bad parts of the self with whom they are confused. The depressive anxiety, which stimulates the reparative drive, cannot be worked through in the mind. Even if there is a concern for objects and therefore a desire for reparation, no reparation can take place because the normal split between good and bad objects are necessary in order to allow the depressive anxieties to be worked through. Rosenfeld continues in the paper that the ego succeeds in splitting of and project the confusional anxiety into the body and body organs. This splitting process does not just take place within the body and ego boundaries. There is a constant projection of internal objects such as anxieties, and parts of the self like sadism, into external objects, and the external objects are always instantly reintrojected into the body and body organs. By projecting the confusional state into the body, the ego to some extent succeeds in splitting off the sadistic forces inherent in these confusional states. But the sadistic impulses remain active and the hypochondrial patient attacks his external objects by sadistically frustrating them and finds himself bored and dissatisfied in sexual relations and sublimation such as work and interest. He attempts to withdraw to good internal objects but does not succeed. So according to Rosenberg no good objects are separate from bad ones and secondly through the introjection of sadistically attacked external objects, the patient with hypochondriasis meets the same situation. The symptoms of self-observations play an important part in the way the hypochondrial patient deals with this sadism. The obsessionally self-observing keep the persecutory and depressive anxieties within the body and off the mental sphere.

Rosenfeld concept of hypochondriasis stands in close relation to psychoses and schizophrenia, where the symptoms of hypochondriasis are defense against a severe and profound, disturbance of the self. Donald Meltzer (1964) adapted Rosenfelds understanding of hypochondriasis as severe condition and differentiated between hypochondrial symptoms and somatic delusions (3). He sees an connection with thought disorders and refers to Bions theories of alpha-functions and beta-elements (94).

Very few if any psychoanalytic studies exists reporting treatment of hypochondriasis. Nissen investigated eight patients with hypochondriasis, who received psychoanalytic psychotherapy (4). He states that hypochondrial symptoms may ensure an attempt from the patient to defend against psychic decompensation, and there is a risk of malignant regression because of instability in psychic structures. Nissen uses Rosenfeld's conceptualisation of hypochondriasis in his examination.

Current definition and understanding

The current definition of hypochondriasis in DSM-IV (1) and ICD-10 (95) are shown in table 2. The question is if these definitions reflect a more fundamental concept of hypochondriasis. Barsky stated in 1992 (55) that there is a general agreement on the diagnostic criteria for hypochondriasis, and the disorder in it self appears to have considerable internal, external, and construct validity. But in a recent paper Barsky expressed (96), that it is not clear whether these features form a discrete category, which is best conceptualized as personality characteristics, maladaptive coping styles, nonspecific symptoms of other more pervasive psychiatric disorders, or simply normal variants. So these different statements are an expression of the ambiguous understanding of hypochondriasis. There is ongoing discussion if hypochondriasis can be understood as a primary disorder, which is independent of other psychiatric disorders. With the current conceptualization in our diagnostic systems, hypochondriasis is seen as diagnostic entity placed in the section of somatoform disorders. In DSM-III the diagnosis of hypochondriasis demanded that the patient did not fulfil the criteria for a somatoform disorder, but the current criteria in both systems diminish this demand, and state that the disorder must not better be accounted for by another somatofrom disorder. This strengthens the understanding of hypochondriasis as a primary condition and is the basis for the empirical research about the subject.

Table 2: Diagnostic criteria for hypochondriasis. The items have been rearranged for comparison.

ICD-10: Hypochondriacal disorder	DSM-IV: Hypochondriasis
A persistent belief, of at least 6 month duration, of the presence of a maximum of two serious physical diseases (of which at least one must be specifically named by the patients)	Preoccupation with the fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms. The duration of the disturbance is at least 6 month
A persistent preoccupation with a presumed deformity or disfigurement (Body Dysmorphic Disorder)	The belief is not of delusional intensity and is not restricted to a circumscribed concern about appearance (as in Body Dysmorphic Disorder)
Preoccupation with the belief and the symptoms causes persistent functioning in daily living, and leads the patients to seek medical treatment or investigations (or equivalent help from local healers)	The preoccupation causes clinically distress or impairment in social, occupational, or other important areas of functioning.
There is persistent refusal to accept medical reassurance that there is no physical cause for the symptoms or physical abnormality.	The preoccupation persists despite appropriate medical evaluation and reassurance.
	The preoccupation is not better accounted for by Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Panic Disorder, a Major Depressive Disorder, Separation Anxiety, or another Somatoform Disorder.

There is consensus about hypochondriasis as a condition characterized as a mental preoccupation with a real or suppositious physical disorder, which to some degree is irresponsive to reassurance. This is reflected in the current diagnostic definitions. The aspects of anxiety as part of the diagnostic criteria are less clear. Pilowsky (97) emphasized in his empirical work disease phobia as an important part of the definition, and in the current cognitive model for hypochondriasis health anxiety is a decisive symptom, which can be differentiated from panic disorder by a less immediate misinterpretation and reassurance-seeking behaviour. There is an ambiguous relationship between anxiety and hypochondriasis in the two diagnostic systems. Health anxiety is not part of the diagnostic criteria in ICD-10, but in the clinical description it is stated (95), that hypochondriacal disorder includes nosophobia and can be differentiated from anxiety disorders, because the somatic symptoms of anxiety in anxiety disorders do not lead to a conviction about the presence of physical illness. It is mentioned that fear of the presence of one or more diseases should be classified as a hypochondriacal disorder. In DSM-IV the fear of having a serious disease is explicitly mentioned in the diagnostic criteria, but disease phobia in which the individual is fearful of being exposed to a disease should be classified as specific phobia and not hypochondriasis. In a cognitive model for

hypochondriasis misattribution and misinterpretations of bodily sensations is a central psychological process. This is reflected in DSM-IV (1) where hypochondriasis is defined as a "preoccupation with the fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms". So the manifest symptoms of hypochondriasis are due to misinterpretation of bodily sensations. If this causality is not established the patients do not fulfil the criteria for hypochondriasis. The same causality is not mentioned as part of the diagnostic criteria for hypochondriasis in ICD-10. But under clinical descriptions (95) it is stated that "commonplace sensations are often interpreted by patients as abnormal or distressing", so this misinterpretation is not causally linked to preoccupation of having a serious disease. In the definition of hypochondriasis in ICD-10 the essential feature is preoccupation or persistent belief of having a serious illness, where health anxiety has a less prominent place than in DSM-IV, even though disease phobia is included in ICD-10 but excluded in DSM-IV. Non-delusional dysmorphophobia is included in ICD-10, but excluded in DSM-IV (see table 3). In ICD-10 preoccupation and persistent non-delusional belief systems are seen as the core items of hypochondriasis, which is consistent with older conceptualizations of hypochondriasis such as the understandings of Ladee (7) and Gillespie (9). DSM-IV stresses the fear of having a serious disease and cognitive models with misinterpretation of bodily sensations as the essential part of hypochondriasis, and uses a more narrow delineation of the disease entity excluding dysmorphophobia and disease phobia.

Table 3: Diagnostic criteria for hypochondriasis

DSM-IV	**ICD-10**
Disease conviction	Disease conviction
Disease fear	Disease fear
Disease rumination	Disease rumination
Bodily amplification/misinterpretation	Bodily amplification/misinterpretation
Bodily misinterpretation →Hypochondrial symptoms (fear, rumination, conviction)	Hypochondrial symptoms (fear, rumination, convictions) → Bodily misinterpretation
	Disease phobia
	Dysmorphophobia

We have chosen to include disease phobia and exclude dysmorphophobia in our examination of hypochondrial patients. We propose that disease phobia is an important aspect of health anxiety, while dysmorphophobia are seen as a different condition related to schizotypal disorder. We examined 176 patients for hypochondrias and no patients in this selected group fulfilled the diagnostic items for dysmorphophobia.

The definitions of hypochondriasis have been represented in structured diagnostic interviews for mental disorders, which are widely used in clinical research. The definition of hypochondriasis in DSM-IV can be assed using the Structured Clinical Interview for DSM-IV (SCID-I) (98) and the definition in ICD-10 by using Schedules for Clinical Assessment in Neuropsychiatry (SCAN) (99). It is also possible to diagnose mental disorders according to the definitions in DSM-IV using SCAN. The questions and diagnostic considerations for somatoform disorders and hypochondriasis for both SCAN and DSM-IV are shown in tables 4 and 5. Both interviews start with questions about the patients' experience of their physical health and their attitude and behaviour in relation to this. There is focus on their relationship with experts and doctors. This approach allows for examination of a possible hypochondrial condition by indirectly encouraging the patient to tell about his idea and perception of health and disease, and the manner in which he experience the health system. Both diagnostic interviews have specific questions about somatic symptoms according to the definition of somatization disorder, before questions concerning items, which define hypochondriasis. In SCID-I you have a clear differentiating between Body Dysmorphic Disorders (dysmorphophobia) and hypochondriasis consistent with the diagnostic criteria in DSM-IV, the same is not the case in SCAN. In SCAN the questions, which deals with hypochondriasis, are divided into two parts. The first part is labelled hypochondriacal preoccupation and the focus is relationship with the health

Table 4: SCAN.ver 2.0. Somatization disorder and hypochondriasis

Structured Interview	Diagnostic considerations
Would you say your physical health has been excellent, good, fair or poor?	
For how long has your physical health been poor?	
In the past years, have you had any bodily aches or pains, or weakness, or physical illnesses or injuries, or disabilities that limited your activities in any way? What sort of problems have you had? Did the doctor/specialist tell you what was the matter?	- None - Describes somatic symptoms or disorder without a clear diagnosis - Has clear physical diagnosis
Have any of these problems limited your well-being or activities in the past years? How severe has the limitation on your physical activities been?	- None - Mild - Moderate - Severe
Have you had expert advise about ..(physical problems)? How much contact did you have with doctors? What did the doctor say was wrong? Are you reasonably satisfied that the problem has been thoroughly investigated and have you received good advice? Have you any more appointments with a doctor	- Satisfied that the problem has been investigated and treated with reasonable care - Says the doctors think nothing is wrong or do not take the problems seriously - Unclear whether dissatisfaction is due to overconcern or to reasonable grievance
Specific questions about physical symptoms	
To summarize then, you have symptoms of... Have you been worried that you have a serious illness(es)? Have you been distressed by these worries? Have long have you felt this way? How old when it first started?	
Variability of somatoform symptoms	
Hypochondriacal preoccupation: How many doctors have you consulted in the past 2 years? What investigations were made? What was the result? Were the doctors/healers reassuring? Why do you think there is something physical wrong? Have you been told the complaints are nervous? Does that seem likely to you? Have you been dosing your self with medications?	Consider the degree of pressure, preoccupation, repetition and distress expressed in the complaints, frequency of seeking a medical explanation in spite of reassurance - Condition not present - Present but mitigated for a time by reassurance (i.e. for a few weeks during or after investigation have been carried out) - Present and apparently unmodifiable
Hypochondriacal conviction: What do you yourself think is wrong? Do you think you have a serious disease? Or more than one (Are you upset about it?) Have you told the doctors about this (what do they say?) Did you feel relieved when they said nothing was wrong? Have you still been convinced, in spite of the experts? What medication do you take? (on doctor's prescription?) Expresses delusional conviction in face of evidence to the contrary,	Expresses delusional conviction in face of evidence to the contrary, that he or she has one or more serious diseases. Exclude fear of contracting a disease rated at (Specific phobia). Include Dysmorphophobia, and rate also at (Perceptual Disorders). Include hypochondrial delusions in context of depression

system, and the patient's understanding of himself in this respect. It includes a closed question about the idea of why something should be physically wrong. In the following part labelled hypochondriacal conviction, the questions are more open, such as the idea of having a serious disease, and how this idea influenced and was influenced by the relationship with the health system. Unfortunately this line of questions is related to hypochondrial conviction, where hypochondrial convictions are defined as having a delusional quality. It is not possible to diagnose hypochondrial

conviction of a non-delusional quality, but only hypochondrial preoccupation. It is not possible to diagnose health anxiety related to hypochondriasis or to examine the relationship between misinterpretation of bodily sensation and hypochondrial ideas, so important aspects of the concept of hypochondriasis is left out in SCAN. There are some modifications to this understanding in the Glossary (100) introducing sensations and physical signs in the relationship of symptoms, but the rigid differentiation between preoccupation and delusional convictions as the only possibilities is maintained. In the diagnostic guidelines (95) the term belief of physical illness is used, which has a more open meaning with both non-delusional and delusional aspects. This diagnostic approach is not transformed to SCAN. In SCID-I the questions specifically related to items concerning the definitions of hypochondriasis are few and very open, and encourage the patient to reflect about the idea of having a serious disease, and how this might influence the way he experiences doctors and influences his way of living. The diagnostic criteria a listed and it is possible to guide the interview so different aspects of the condition will be examined. SCID-I represent a reasonable transformation of the definition of hypochondriasis in DSM-IV to the related assessment instrument. It is a problem that it is not explicit stated, that you should differentiate between preoccupation with the fear of having a serious disease and disease phobia, which should be rated as specific phobia.

The current definition of hypochondriasis has provided a common platform for empirical research in spite of different understandings. Research groups have been forced to use the diagnostic definitions and have tried to validate or challenge different aspects of the conditions depended of their theoretical approach. An important area of research is comorbide conditions in relationship to hypochondriasis. It is common knowledge that there is a high psychiatric comorbidity with hypochondriasis, and the condition consists of different aspects of anxiety, obsession-compulsion, and somatization. An ongoing question is if a primary hypochondriasis exists and if hypochondriasis is a valid diagnostic entity. Research in comorbide conditions is not just research about the diagnostic boundaries for hypochondriasis but also an examination of items and features, which create the current conceptualisation of hypochondriasis.

Table 5: SCID Version 2.0 (for DSM-IV) for somatization disorder and hypochondriasis

Structured Interview	Diagnostic considerations
SOMATOFORM DISORDERS Over the last several years, what has your physical health been like? How often have you had to go a doctor because you weren't feeling well (what for?) IF YES: Was the doctor always able to find out what was wrong, or were there times when the doctor said there was nothing wrong but you were still convinced that something were wrong? Do you worry much about your physical health? Does your doctor think you worry too much? Some people are very bothered by the way they look. Is this a problem for you? IF YES: Tell me about it IF NOTHING SUGGEST THE POSSIBLITY OF A CURRENT SOMATOFORM DISORDER. GO TO NEXT MODULE IF SUBJECT HAS ACKNOWLEDGED ONLY BEING BOTHERED BY THE WAY HE OR SHE LOOKS, SKIP TO BODY DYSMORPHIC DISORDER **SOMATIZATION DISORDER** Have you been sick lot over the years? IF YES: How old were you when you first started to have a lot of physical problems or illnesses	**SOMATIZATION CRITERIA** A history of many physical complains beginning before age 30 years, that occur over a period of several years..
SPECIFIC PHYSICAL SYMPTOMS Did you see a doctor about it? IF YES: What was the diagnosis (Medical condition that could account for the symptom) IF NO: Did it interfere with your life a lot	FOR EACH PHYSICAL SYMPTOM: (1) The symptom results in treatment being sought or causes impairment in social, occupational, or other important areas of functioning (2) The symptom cannot be fully explained by a known medical condition and the impairment are in excess of what would be expected (3) The symptom is not intentionally feigned or produced
SOMATIZATION DISORDER OR PAIN DISORDER CHECK AND **GO TO HYPOCHONDRIASIS** UNDIFFERENTIATED SOMATOFORM DISORDER. **GO TO HYPOCHONDRIASIS**	A. Physical complaints B. Cannot be explain by medical condition C. Clinically significant distress or impairment D. Duration least 6 month E. Not better accounted for by another mental disorder. F. The symptoms not intentionally produced.
HYPOCHONDRIASIS Do you worry a lot that you have a serious disease that the doctors have not been able to diagnose? What have the doctors told you? (When did all this begin)	**HYPOCHONDRIASIS CRITERIA** A. Preoccupation with the fears of having, or the idea that one has, a serious disease, based on the person's misinterpretation of bodily symptoms DESCRIBE: B. The preoccupation persists despite appropriate medical evaluation and reassurance C. The belief in 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. Duration of the disturbance is at least six month F. The preoccupation is not better accounted for by Generalized Anxiety Disorder, Obsessive Compulsive Disorder, Panic Disorder, a Major Depressive Episode, or another Somatofrom Disorder

Hypochondriasis and comorbide psychiatric disorders, is primary hypochondriasis a valid concept?

Patients with hypochondriasis has a high correlation with depressive, anxiety and somatic symptoms, and high rates of comorbide depressive, anxiety and somatoform disorders (101). Barksy (57) examined the comorbidity in patients fulfilling the diagnostic criteria for hypochondriasis in DSM-III, which is the forerunner of the current diagnostic definition. The patients came from a general medical clinic. The patients were compared with a random sample of outpatients from the same clinic not fulfilling the diagnostic criteria for hypochondriasis. A total of 88% of the patients had one or more additional Axis I disorders, so there was a relatively low proportion of primary hypochondriasis understood as hypochondrial conditions with no other psychiatric comorbidity. One fifth had a somatization disorder but the conditions appeared to be phenomenological distinct. Barsky findings were consistant with Kenyon examination (102) with only few differences between primary and secondary hypochondriasis. Hypochondriacal patients with comorbide anxiety and depression did not differ from patients without comorbide conditions. Barsky proposes that the nosological status of hypochondriasis should not be altered, even though the natures of hypochondriasis remain unclear. This conclusion is the opposite of Kenyon's conclusion with a similar finding.

Different ideas about the current nosological status of hypochondriasis are still being discussed. Hypochondriasis has been questioned as a diagnostic entity and favoured a dimensional view seeing hypochondriasis as an anxiety disorder which consist of a continuum from mild health concerns to more extreme cases with morbid preoccupations about harbouring a disease (71). It has been proposed to dissolve hypochondriasis as a diagnostic entity, and combine items from the definition pf hypochondriasis with items from somatoform disorder to form a new entity with rumination of health and disease as the most important item (103). In our current diagnostic systems hypochondriasis is granted legitimacy as a separate diagnostic disorder and has been included in the somatoform disorder section, but there are many phenomenological similarities and comorbidity with anxiety disorders, so it has been proposed to place hypochondriasis as a diagnostic entity in the anxiety domain (104). Noyes (105) concluded that hypochondriasis is distinct from the anxiety disorders, but because of phenomenological similarities and extensive comorbidity should probably be classified among them. Other groups have proposed hypochondriasis as part of an obsessive-compulsive spectrum (106).

Hypochondriasis and anxiety

Previous research suggested considerable overlap among the symptoms of panic disorder and hypochondriasis. Patients primarily diagnosed with hypochondriasis have a considerable comorbidity with panic disorder (46) and patients primarily diagnosed with panic disorder have a considerable comorbidity with hypochondriasis (43;107). Fava found (40) that the difference between hypochondriacal beliefs and disease phobia demanded the concept of abnormal illness behaviour as framework for differentiation. Furer (108) reported that 48% of patient with panic disorder also met DSM-IV criteria for hypochondriasis, whereas only one patients with social phobia and none of the healthy control subjects met the criteria for hypochondriasis. Hiller (109) compared patients with hypochondriasis, panic disorders and patients with both conditions. The result confirms that hypochondriasis and panic disorder, are distinguishable clinical conditions, where hypochondriasis is characterized by more psychopathology and distress. This is consisting with findings from Barksy (107) and Noyes (46). The emotional, cognitive, and behavioural reaction of panic patients is similar to those described for hypochondriacal disorder. The major difference is the episodic nature of the symptoms in panic disorder versus persisting complaints in hypochondriasis. It has been proposed that the some psychopathological process, which underlies panic disorder, is responsible for hypochondriasis, and hypochondriasis has been seen as secondary phenomena to panic disorder. This hypothesis has been confirmed by the treatment respons directed to panic attacks (110). Fava (111) described a series of patients, where hypochondriasis preceded the emergence of panic attacks. Hypochondriasis and panic disorder are associated with distorted cognitive assessment of health status, which may be accompanied by a state of higher alertness and awareness of physiological processes(50). Patients with panic attacks tend to misinterpret autonomic symptoms which provide a feedback mechanism where anxiety may rapidly escalate. In hypochondrias, the symptoms misinterpreted a not likely to by subject to such direct amplification and the feedback mechanism is more behavioural and longer term by maintaining focus on particularly parts of the body with repeated checking or physical manipulation. The panic patient believes that the catastrophe is happening already, whereas the hypochondrial patients tend to believe that the symptoms indicate a more insidious course(83). The conclusion of these conceptual and empirical research is that some overlap between the two diagnoses exists, but the conditions are distinct and have discriminant validity (107).

There is different comorbidity between hypochondriasis and general anxiety in different studies (46;57;78), but these findings have not led to any conceptual discussion of the discrimination between hypochondriasis and general anxiety. Both conditions involve excessive worry but according to DSM-IV (1) criteria for generalised anxiety disorder the focus of worry is not confined to have a serious illness. In Barsky's (57) examination of psychiatric comorbidity in DSM-III-R hypochondriasis he found the lifetime prevalence for generalized anxiety disorder was 71%, and the current prevalence 24%. Starcevic (44) compared hypochondrial symptoms in patients with generalized anxiety disorder and panic disorder, even though 30% with generalized anxiety had worries about health and illness, these patients were far less likely to harbour hypochondrial fears and belief than patients with panic disorder. They experienced fewer somatic symptoms and their worries about health and disease did not have the intrusive quality and aspects of ruminations as patients with hypochondriasis.

The definition of hypochondriasis in ICD-10 (95) emphasises the persistent belief of physical illness and refusal to reassurance as the most important items. There is no explicit description of the anxiety involved in the condition, but it is stated that marked anxiety often is present and may justify additional diagnosis, and that fears of the presence of one or more diseases (nosophobia) is included. In DSM-IV the definition includes explicit the fear of having a serious disease based on misinterpretation of bodily symptoms, but do not include a specific disease phobia where the individual is fearful of being exposed to a disease and avoidance to situations that may lead to contracting a disease. It is stated that hypochondriasis is characterized by a preoccupation that one has a disease, and the discrimination depend on the presence of disease conviction. According to Marks (112) illness phobia may be a subtype of hypochondriasis focused on a specific illness instead of multiple bodily symptoms, but it is uncertain whether it represents a separate entity. Malis (113) developed a structured interview to assess specific phobia of illness and hypochondriasis and 10 out of 21 patients who reported that illness fear bothered them met the criteria for specific phobia of illness, but 4 of these 10 patients did also meet the criteria for hypochondriasis. Benedetti (114) reported that in his sample of hypochondrial patients 50% reported illness phobia before the unset of hypochondriasis. Illness phobia is not the only factor that influence the development of hypochondriasis other premorbid features are included, and Stracevic (43) propose a cognitive structure where patients developing hypochondriasis is explanation-

seeking rather than treatment-seeking. The current findings do not confirm that illness phobia is separate disorder.

Hypochondriasis and the affective spectrum

There is a considerable comorbidity between hypochondriasis and depression (46;57). But these findings have not lead to any conceptual discussion of the relation between hypochondriasis and affective disorders. It is implicit taken for granted that these findings are an expression of high comorbidity between different nosological and psychopathological entities. Kenyons opinion that hypochondriasis always is a part of another syndrome mostly an affective one (115) has been criticized (83) and abolished. For patients with concurrent depression the onset usually followed after hypochondriasis and was associated with more severe symptoms and impairment, and the depression may result from or contribute to a greater severity of hypochondriasis. The hypochondrial concerns may be so disruptive that they lead to high levels of depression. Depressed patients are more hypochondrial and a positive correlation between hypochondrial concerns and depression has been established (116;117). But these findings have not lead to a discussion of the concept of primary hypochondriasis.

Hypochondriasis and the obsessive-compulsive spectrum

There is a considerable overlap between hypochondriasis and obsessive-compulsive disorder (OCD) (118;119) such as stereotyped repetitive behaviour as attempt to control anxiety. Based on findings (120) comparing groups of patients with OCD, with OCD and hypochondriasis, and patients with hypochondriasis, it has been proposed to include hypochondriasis in the OCD spectrum but recognizing difference between the disorders. This is in contrast to Fallon statement (121) that little comorbidity exists to support that hypochondriasis is an obsessive-compulsive spectrum disorder, and Barksy (122) who found that lifetime prevalence for OCD in hypochondrial patients was 8%. Some relationship between OCD and hypochondriasis is present but the nosological status between these disorders is uncertain based on empirical findings. Patients with OCD exhibit higher degree of compulsivity and the compulsion in hypochondriasis is of a different kind of nature, such as the contact and behaviour related to health system. The rumination of hypochondriasis is experienced less as an intrusive mental event and more as a reasonable response to a realistic health treat. In hypochondrial patients (123) there is a strong disease conviction where the essential belief is that a disease is present and compulsion revolve around the identified reality. This elevated overvalued

ideation, which is a concept in the theory of obsessive-compulsive disorders, represents a fundamental difference between hypochondriasis and OCD. In OCD compulsions revolve around preventing an obsessional idea from becoming reality, while patients with hypochondriasis see their fears as realistic (122) and their pervasive ideas of illness and health are seen as part of their personality. They express their concern in public, where OCD ruminations are held secretly and are seen as unrealistic and separate from their personality. Barsky (122) notes that the ideas of the hypochondriac constitutes an interpretive schema, where the ideas of the obsessive-compulsive patients lack an organisation principle. This distinction falls short in subgroup of patients with OCD (124), who have poor insight into the irrationality of their fears, and who perform checking behaviour without hesitation, and a subgroup of patients with hypochondriasis who have good insight but avoid doctors because of the risk of humiliation or a feeling of hopelessness.

Hypochondriasis and somatization disorder

According to criteria in DSM-IV both hypochondriasis and somatization disorder are characterized by multiple somatic symptoms. A hierarchical rule where somatization disorder excludes hypochondriasis was eliminated from DSM-III-R, but existed in DSM-III. Different studies (125-127) have found a relatively high comorbidity between hypochondriasis and somatization disorder and especially sub-syndromal somatization disorder. Barsky (57) found a prevalence of somatization disorder at 21% in a group of medical outpatients with hypochondriasis, Noyes (46) found a lower rate (7,4%) in a similar cohort, but a third of the hypochondrial patient met less stringent criteria for somatization disorder. The findings indicate a high comorbidity between the two conditions but the relationship is not well understood (128). Though many patients with a somatoform disorder worried about their health not all did (129), and though many patients with hypochondriasis have somatic symptoms not all of them have (45). So the overlap between these disorders a considerable but not complete. Barsky proposes (57) that different conceptual conditions is describing the same clinical entity. But it seems to be a more widely distributed impression in the scientific literature, that these conditions can be differentiated. It has been proposed that patients with hypochondriasis had poor health habits and are profound explanation-seekers rather than treatment-seekers, and patients with hypochondriasis as explanation-seekers are seeking help and then rejecting the assistance as ineffective (43;107). Hollifield (130) has in a recent study confirmed this understanding. In an additional study (131) it was found that that the variance in aspects of personality and attitude to the self were associated to high somatic complains but not hypochondriacal symptoms. Hiller (78) studied 570 subjects with psychophysical disorders. A

subgroup of 69 patients meet the current criteria for hypochondriasis and the group difference remained after controlling statistically for somatization. Hiller concluded that hypochondriasis is not merely a secondary attribute of somatization. Psychological mechanisms such as false causal attribution and unrealistic health attitudes are more important for the development of hypochondriasis than medically unexplained somatic symptoms. Fink (103) screened more than 1000 consecutive patients consulting non-specialized primary care physician and a subgroup of 701 patients were interviewed with SCAN. The key items included in the DSM-IV hypochondriasis diagnosis were also common among patients with other somatoform diagnoses. Based on a latent class analysis Fink proposes a new diagnosis for hypochondriasis with better discriminatory power, where rumination about harbouring an illness is the most important item. So Fink proposes to deconstruct our current diagnostic definition based on the high comorbidity between hypochondriasis and other somatoform disorders and establishes new diagnostic definitions with better discriminatory power. The question is if this nosologic rearrangement is based on different psychopathological entities. Some aspects or items of hypochondriasis are missed in the analysis, such as preoccupation or fear of having a disease based on the misinterpretation or amplification of bodily symptoms, the aspects of health anxiety in hypochondriasis, and the interaction between reassurance, consistent disease preoccupation and abnormal illness behaviour. These items represent valuable information related to our current conceptualization of hypochondriasis.

Hypochondriasis and personality disorder

Patients with hypochondriasis have a higher rate of personality disorder than non-hypochondrial patients (66;132). In an early conceptualization (1966) of hypochondriasis by Ey (133) the condition is seen as a pathological form of human existence. Ey proposes that all men are preoccupied with health. In the same measure as existence is a problem, the feelings of anxiety that are attached to the body, to life and the death of the body, become embodied in the human condition, so patients with hypochondriasis share their problems with whole humanity. The difference is that patients with hypochondriasis do not have the illness, they think they have. This fact hides the real mental illness they have, and being unaware of their mental illness, they live a false illness. This understanding of hypochondriasis as a discrete psychopathological condition is difficult to discriminate from the personality.

In a more modern understanding hypochondriasis has been conceptualized as a personality characteristic more than a circumscribed, discrete illness, where hypochondriasis is a way of

interpreting ones experience and thinking of health and diseasev(57). Starcevicv (134) proposes a relationship between obsessive-compulsive personality disorder and hypochondriasis, where the self-doubt of the obsessive-compulsive personality disorder and the disease suspicion reflects a basic perception of oneself as vulnerable and insecure. The hypochondrial patient lacks a sense of somatic security. The experience of the body as the source of danger leads to a detachment of the somatic representation from the self-representation. If the perceived threat continues to arise from the bodily, it will guide the psychopathological regression in the direction of hypochondriasis regardless of the context of the personality structure. Patient with hypochondriasis have an obsessive cognitive style with excessive need for control and poor tolerance for uncertainty and ambiguity. For patients with obsessive-compulsive personality disorder this cognitive style is more generalized in scope and of more psychological nature. In hypochondriasis it is constricted to ideas of disease and health. Hypochondriasis is seen as more pervasive and incapacitating than obsessive-compulsive personality disorder and closely related underlying psychopathology.

Primary hypochondriasis has been conceptualized as a somatic style with hypochondrial episodes occurring secondary to other psychiatric disorder or major life stress. Bass and Murphy (135) suggest that somatoform disorder and hypochondriasis may be more accurately conceptualized as maladaptive personality traits than as axis I diagnoses, according to DSM-IV. Some of the most salient aspects of somatization disorder are interpersonal in the way the patient presents themselves and the ways the doctors and other respond to them (136). Only a few empirical studies examine the prevalence of axis II diagnosis in hypochondriasis. Barsky (57) compared a group of patients with DSM-R-III hypochondriasis with a random sample of outpatients both groups taken from a general medical outpatients clinic. The patients with hypochondriasis had a 3 time higher personality disorder caseness measured with Diagnostic Interview Schedule and self-report questionnaires than the control group. Tyrer (137) found empirical support for a hypochondrial personality disorder which high score on scales measuring anxiety and dependence. In a follow up study (138) the hypochondrial personality disorder was a strong negative prognostic indicator, which support the assumption of the hypochondrial personality disorder as a valid clinical diagnosis. In this understanding hypochondriasis is a personality disorder among neurotic psychiatric patients characterized with preoccupation and rigid belief about health. Other research groups found that patients with high hypochondrial responses were high on neuroticism and low on extraversion measured with NEO five factor inventory (131). In non-clinical population Watson (139) found a

close relationship between hypochondriasis and negative affectivity understood as a tendency to experience negative emotions and overreacting to stress. Ferguson (140) observed a close relationship between emotional stability or neuroticism, which is a domain from the five-factor model, and hypochondriasis. To examine the relationship between personality dimension, hypochondrial concerns and somatic symptoms Noyes conducted (141) a factor analyses which identified separate dimensions. A multiple regressions models determined the proportion of variations in these measures of somatic distress explained by personality scales. Negative temperament or neuroticism is a strong predictor of hypochondriacal concern measured by SNAP and the Whiteley Index. This finding is consistent with the literature showing a relationship between neuroticism or negative emotionality and hypochondriasis (142). This replicate findings from non-clinical samples (143;144). Patients with negative temperament are prone to find bodily sensations noxious and interpret them as signs of serious illness, and negative temperament may represent a trait-like vulnerability to hypochondriasis. Hypochondriacal concerns and somatic symptoms appear to be related to non-specific trait distress and not to specific personality features. Three specific trait scales in SNAP showed correlation to hypochondrial concerns: Mistrust, Low Self-Esteem and Eccentric perceptions. Mistrust assesses a sense of injustice and alienation, and patients with hypochondriasis has been described as resentful and mistrustful (145). The eccentric perception scale measures unusualness of perceptions and beliefs. The patients report perceptual disabilities and an atypical worldview, feature fund among the schizoid personality disorder. The relatively high correlation suggest a link with hypochondriasis, and point to a relationship with depersonalization (7;146). Alternatively Eccentric Perception may represent metaphysical and superstitious beliefs held by hypochondrial persons (147).

These findings show a correlation between hypochondriasis and different measures of personality disorders or personality traits. It is shown that hypochondriasis have features in common with the present conceptualisation of personality disorders, such as a pervasive and long-lasting features of interpreting health and the body and way the patients relates to doctors and health personal. Hypochondriasis is found to correlate with specific patterns of personality traits such as negative temperament or neuroticism, which is a strong predictor of hypochondrial concerns, but other personality dimension, such as mistrust and eccentric perception may remain important.

The concept and measurement of different features of hypochondriasis

Instruments measuring different features of hypochondriasis are shown in table 6 (55;148-151). It is an illustration of different and maybe conflicting understandings and how research groups focus on specific aspects of hypochondriasis. The definition of DSM-IV unites these research groups, but the question is if this pattern of symptoms, cognition and behaviour is a representation of a fundamental psychopathological concept. There seems to be a consensus about preoccupation with health and disease as central feature in hypochondriasis, which were confirmed in Pilowskys (152) empirical research. Pilowsksy model reflects a contemporary understanding from David Shapiro (153), where hypochondriasis could be seen as neurotic style with patterns of characteristic functioning including ways of thinking, perceiving, experiencing emotions, and specific modes of activity. These stable patterns or "forms of minds", a kind of gestalt, were regarded as general mode of functioning and were distanced from an ego-psychological model of drive-inhibiting mechanism. Pilowskys developed the Whiteley Index as part of his empirical research and the questionnaire addresses internal features of hypochondriasis such as bodily preoccupation, disease phobia, and conviction of presence of disease. This line of work was continued with Kendell and the Illness Attitude Scale (24), which reflects the patients' attitude, fear, and belief of disease, but also introduced other aspects in conceptualisations of hypochondriasis such as the fear of death (tantrophobia), health habits and concern about pain. Kellner conceptualised hypochondriasis as neurotic disorder, where the patients did not comprehend the relationship between emotions and somatic symptoms. He introduced different cognitive and behavioural elements such as selective perceptions and conditioned learning. The cognitive model of hypochondriasis developed by Salkovskis, Warwick, and Clarck has shown to be coherent and influential, and has affected the definition of hypochondriasis in DSM-IV. The model propose cognitive process of misattribution of bodily sensation as the primary element of hypochondriasis, which causes the symptoms, affect, and behaviour of the patient (82). The model resembles Pilowsky and Kellners understanding with a focus on immediate observable phenomena. Two questionnaires for measuring the cognitive model of hypochondriasis are available (149;151) and both instruments have received some dissemination. Both scales are based on the cognitive theory of health anxiety and comparative studies are probably needed. Barksy has over the years consolidated his understanding of hypochondriasis as an enduring attitude toward health, concern of the body, and belief of disease.

Table 6: Different measures of hypochondriasis and related items

Scales	Target	Format	Construct assessed
Whiteley Index	Hypochondriasis	Questionnaire	Includes item that do not directly measure hypochondriasis. Consist of 3 factors: bodily preoccupation, disease phobia, and conviction of presence of disease.
Illness Behaviour Questionnaire (IBQ)	Abnormal illness behaviour	Questionnaire	Consist of 7 scales: general hypochondriasis, disease conviction, psychological versus somatic perception of illness, affective inhibition, affective disturbances, denial, and irritability.
Illness Attitude Scale (IAS)	Hypochondriasis	Questionnaire	Consist of 7 scales: worry about illness, concerns about pains, health habits, hypochondrial belief, thanatophobia, disease phobia, and bodily preoccupation.
Structured Diagnostic Interview for Hypochondriasis (SDIH)	Hypochondriasis	Structured interview	Structured interview designed to be administered by mental health professionals with clinical interview experience and knowledge of psychopathology. The sequences of questions follow the diagnostic tree of DSM-III-R and cover the diagnostic criteria for hypochondriasis from DSM-III-R. It is part of the Structured Clinical Interview for DSM-IV
Somatic Symptom Inventory	Somatic symptoms	Questionnaire	Scale for somatic symptoms associated with hypochondriasis. Consists of items from MMPI hypochondriasis scale and SCL-90 somatization scale. Responses are obtained on 5-point linear scale.
Somatosensory Amplification Scale	Somatosensory Amplification	Questionnaire	Five-item self-report instrument scored on a Likert scale concerning the sensitivity to a range of normal bodily sensation. The scale is based on a model of hypochondriasis as an enduring attitude toward health, concern of the body, and belief of disease, where amplification of bodily sensations is an important causal factor.
Health Anxiety Questionnaire (HAQ)	Health anxiety	Questionnaire	21-item measure developed to identify individuals with high levels of health anxiety. Responses are obtained on 4-point Likert scale. The scale is based on a cognitive-behavioural analysis of health anxiety, it intend to reflect symptoms of health anxiety and is able to discriminate between hypochondrial and non-hypochondrial patients with similar high levels of general anxiety and depression. Factor analysis suggests four groups of items. Health worry and preoccupation, fear of illness and death, reassurance seeking behaviour, and interference with life.
Health Anxiety Inventory (HAI)	Health anxiety	Questionnaire	18-item self-rated measure of health anxiety. Response obtained on 4-point Likert scale. It is described as a continuous measurement of health anxiety that is sensitive to normal levels of health concern as well as clinical hypochondriasis. The item chosen are closely based on the cognitive theory of health anxiety. Data suggest that HAI is relative independent factor in hypochondriasis consistent with current cognitive theories. It discriminates patients diagnosed with hypochondriasis and patients with anxiety disorders; it is sensitive to treatment effect.
Fear of Death Scale	Fear of Death	Questionnaire	27-item scale to assess physical, interpersonal, and existentialistic aspects of fear of death. Response are obtained on a 5-point linear scale

He uses a cognitive model for characterizing the belief system of the hypochondrial patient and focus on bodily amplification as a decisive feature. Barsky therefore emphasise other aspects of hypochondriasis, than the cognitive model of misattribution and misinterpretation. Barksy developed (66;148) a scale to measure the degree of amplification understood as the sensitivity to a range of normal bodily sensation, and a scale to a measure the tendency to express emotional dysphoria as somatic symptoms (72). These scales have been used as a screening instrument for hypochondriasis in different populations.

In a phenomenological orientation hypochondriasis is seen as a disturbance in the normal givenness of the body (154), where the world as the intended object of experience through the body is changed, so the body itself is the intended object of experience. The hypochondrial patient is exploiting his own experience to be continually directed toward his own body to obtain control of the body. This crisis in experience is based on a deep-rooted unresolved fear of death, and the fact that the body will die and existence terminated. Tantrophobia or death concern is an important characteristic of hypochondriasis, and a subgroup of patients with hypochondriasis report distressing thoughts of death (49). In a psychoanalytic understanding many anxieties, obsessions, and other neurotic symptoms are related to the fear of death and its symbolic equivalents (155), and through an ambivalent identification with an important other, hypochondriasis can be seen as defence against the fear of death (156). In a psychoanalytic conceptualisation the fear of death(42) is an inadequate development of a mechanism for neutralization of primary forms of anxiety, where immature ego-experience, which are a threat to the integrity, contributes to a pathological fear of death. Higher level of anxiety may be dominant, and there is a vulnerability invalidating the regulation of the primary anxiety. This vulnerability may represent aspects of ego weakness and impaired ego-function especially defensive, symbolic and cognitive function. Kellner's illness attitude scale contain three tantrophobic items and in a recent investigation a relationship between hypochondrial concerns and fear of death have been shown (157). Noyes developed a fear of death scale (49) and with his findings confirms, that fear of death is an integral part of hypochondriasis. In contrast to these internal features of hypochondriasis stands the behaviour related with hypochondriasis. The interpersonal aspect is an essential part of the understanding of hypochondriasis and it is reflected in two items in the diagnostic definition. Firstly, that the preoccupation persists despite appropriate medical evaluation and reassurance, and secondly, that the preoccupation causes a significant impairment in important areas of functioning. In the

cognitive model for hypochondriasis the patients' tendency to seek reassurance is seen as an important psychopathological feature, which consolidates the disorder and some assessment scales rate the reassurance behaviour driven by the anxiety and compulsion. Pilowsky (158;159) includes a sociological model in his understanding of illness behaviour and the interaction between the doctor and his patient, where abnormal illness behaviour can be seen as a sociological concept, where a social system cannot function without explicit norms in relation to illness and disability. The concept of abnormal illness behaviour covers the nature of disagreement between a patient and the doctor, the phenomenology of the patient's experience, and the patient's capacity to participate in the doctor-patient negotiations process. Normally the patient and the doctor work towards congruence in understanding the problem, but in abnormal illness behaviour this capacity to negotiate is invalidated. Pilowsky concept of abnormal illness behaviour has brought a sociological and interpersonal perspective into the considerations of hypochondrial conditions. Pilowsky developed the Illness Behaviour Questionnaire (IBQ) (19) as a measure, which integrate the patients' illness behaviour with other features of hypochondriasis, but the questionnaire has never won the same distribution as the Whiteley Index. Cognition and the related anxiety has a major impact on illness related behaviour (82), and the avoidance behaviour maintains the patients' preoccupation with disease by preventing the patient to obtain information, which contradicts his interpretation of symptoms. Reassurance-seeking behaviour can have the same effect as obsessive checking. It focuses attention on patients' worries and reduces anxiety on short term, but increases preoccupation in the long term. Patients anxious about their health respond differently to health reassurance, which becomes counterproductive as the patients selectively attend to and misinterpret the reassurance itself. Checking and reassurance focus patient's attention on their fears and prevent habituation to the anxiety-provoking stimuli. Reassurance as a compulsive ritual should be prevented and used as basis for re-attribution. Non-specific reassurance provides no alternative framework for the hypochondrial patient to understand his symptoms and fear. Appropriate reassurance is defined (160) as the provision of new information that is relevant to the patient's clinical condition, where the suggestion must be credible and consistent with the patient's experiences and expectations to the future. Balint (80) indicates that the patients' attribution is readily changed by the physician's suggestions and may alleviate the patient's distress, if the physician can provide a benign interpretation, that are credible and can reassure the patient and bolster his sense of mastery. Balint uses the concept the doctor's apostolic function, which is the clinicians' individual way of dealing with his patients, and the clinicians' idea of how a patient

should behave when ill. There is a compromise between the patients demand and the clinicians' conviction and response, and therefore a limitation of the clinicians' elasticity, and the way it affects his practise. Starcevic (161) emphasises that hypochondriasis exists in an interpersonal context and a negative response to reassurance is paradigmatic of hypochondriasis. He introduces the possibility to analyse the role and meaning of reassurance in hypochondriasis. The response to reassurance gives clue to the pathology of hypochondriasis. The hypochondrial patient is coloured by a basic mistrust, and the need to be reassured do not correlate with the ability to accept reassurance. Starcevic states (162) that reassurance constitutes a treatment modality for hypochondriasis, and he recommends an individualized strategy and identification of patients with depression-prone neurotic-spectrum character difficulties, as persons who will benefit from reassurance. This understanding is in conflict with the cognitive method, which sees reassurance as antitherapeutic because reassurance maintains hypochondriasis. Starcevic challenges this understanding, and states that reassurance constitute a treatment modality for hypochondriasis. In explanatory therapy, developed by Kellner (163), reassurance is seen as a therapeutic intervention and the initial aim is to persuade the patient that his belief is false, so it no longer reinforce psychopathology. The alliance is established partly by persuading the patients that his hypochondrial symptoms are false.

Different models of hypochondriasis appear in this empirical and conceptual development. We have a model of hypochondriasis, which consists of a cognitive schema with misinterpretation and amplification of bodily perceptions as central psychological processes. This schema guides perception, memory, inference and judgment. This model is closely related to the diagnostic definitions of hypochondriasis, which consist of specific patterns of symptoms, cognition, and behaviour. In connection with the diagnostic definitions the model has served as an important foundation for empirical research and development of specific psychotherapeutic treatments, which have been examined under randomized clinical conditions. Secondly, we have an older understanding of hypochondriasis, where the psychopathology is seen as a result of an individual's failure to find a meaningful existence. This understanding develops from a phenomenological and psychoanalytic tradition, but has a present conceptualization in the interpersonal model of hypochondriasis (52), where the relationship between the patients and the physician is seen as a central feature related to the attachment model of human development.

RANDOMISED CLINICAL TRIALS AS A METHOD OF EVALUATION OF INTERVENTIONS FOR HYPOCHHONDRIASIS

In the following I will comment on different more general aspects of randomised clinical trials as method for evaluating psychotherapy, especially psychodynamic oriented psychotherapy. The point of reference is the trial, which is presented in the enclosed paper. I will focus in detail on some problems in relationship to the randomsised trial, which is the empirical foundation for the paper, and relate this work to other published works of psychotherapeutic treatment to patients with hypochondriasis. Gabbard (164) proposes hierarchies of evidence for the effect of psychotherapeutic treatments with randomised trials as the most rigorous and important design. He emphasizes that psychoanalysis focus on an intrapsychic domain, which is not necessarily compatible with controlled observations and testable hypotheses. Psychoanalysis has therefore deprived itself of the interplay between data and theory that has contributed to the growth of modern science.

The scientific foundation for randomised controlled trial

A clinical trial is a planned experiment on human beings designed to evaluate the effectiveness of one or more forms of treatment. The key idea is to compare groups of patients who differ only with respect to the treatment. If the groups differ in some other way, then the comparison of treatment may become biased. Difference between groups may arise by chance. To control for this problem in small populations stratified randomisation should be used. The statistical methods are based on what is expected to happen in random samples from populations with specified characteristics, and in the statistical analysis you use null hypothesis testing, which gives you the probability for a given result, if there is no difference between randomised groups (165). In Kathy the randomisation allocated patients to 3 groups. Two groups receiving different forms of psychotherapy and one group were on a waiting list. The null hypothesis in this trial was divided in two parts. Firstly that no difference were present between the 3 intervention groups at 0 month after treatment, and secondly that no difference was present between the two groups receiving Cognitive behavioural Therapy (CBT) and Shortterm Psychodynamic Psychotherapi (SPP) 0, 6, and 12 months after treatment. The statistical analysis used to test the null hypothesis for the first part of the trial was a one-factor analysis of variance for the 3 intervention groups 0 month after treatment (three group analysis), and the result of the analysis the probability of the given finding with no difference

between the groups. The statistical analysis for the second part of the hypothesis testing (two group analysis) was a two factor mixed design, repeated measure analysis of variance with outcome variables after treatment as dependent variables, the two treatment groups as between-subject factors and time 0, 6 and 12 months after treatment as within-subject factors. The result of the analysis was the probability of the given findings with no difference between the groups receiving psychotherapy. In the paper "A randomized clinical trial of cognitive behavioural therapy versus short-term psychodynamic psychotherapy versus no interventions for patients with hypochondriasis", we did not use a null hypothesis, but the alternative hypothesis, which constituted our expected findings.

Explanatory or pragmatic trials trials

Two different types of randomised trial have been proposed called explanatory and pragmatic trials. Explanatory trials generally measure efficacy under ideal conditions in a carefully controlled setting. The patients are carefully selected and the eligibility criteria are narrow to recruit a homogeneous sample of participants to minimize the impact of extraneous variation. Explanatory trials examine new types of interventions and are not only hypothesis testing but also hypothesis generating. Pragmatic trials measure effectiveness such as the treatment procedure in routine clinical practice, they are normally on a larger scale with more typical patients and therapists. More inhomogeneous populations are accepted by the use of wider eligibility criteria. To ensure generalization the patients should represent the population to whom the treatment should be applied, and the results in the pragmatic approach are easier to generalize by the use of broader eligibility criteria and a population, which resemble the patients in a clinical practice. A pragmatic approach has a higher external validity by providing a more correct basis for generalization to other circumstances (166). The interventions should be described precisely, but in pragmatic trials this does not mean that the treatment is offered to each patient. It is the management protocol, which is the subject of investigation. In pragmatic trials blinding of patient and clinician is not always possible, and clinician and patient's bias is part of the response to the treatment and included in the overall assessment. The treatment response is the total difference between two treatments, which included treatment and placebo response. This reflects the clinical response in clinical practice. Pragmatic trials are designed to reflect the realities of clinical practice (167) and to inform if the treatment is applicable to clinical practice and if the treatment works.

In the Kathy trial we examined in a clinical setting a specific cognitive-behavioural psychotherapy for hypochondriasis (CBT). We used a dissemination strategy where the treatment was implemented in a clinical setting by training and supervision of the therapists involved. The treatment was established in a pilot study and the dissemination procedure was finished before the trial started. As a comparison we used short-term psychodynamic psychotherapy (SPP) and waiting list control. The waiting list can be seen as treatment as usual, as no specific treatment for hypochondriasis was available before this trial in Denmark. In a pragmatic approach these 3 interventions can be seen as complex interventions (168), where the principle of double blinding of patients and clinician is not possible, and the resulting bias is part of the treatment response. The trial took place in a secondary health care clinic, specialized for patients with psychosomatic problems understood as comorbide mental disorders or medically unexplained complaints. The clinic offers consultations and outpatient treatment. Patients are referred from general practice and from primary and secondary healthcare settings. The clinic cooperated with a clinical centre specialised in cognitive behavioural treatment for anxiety disorders, and in cooperation with this centre implemented the cognitive behavioural treatment for patients with hypochondriasis. The patients were referred for treatment and only in a few referrals there was knowledge of an ongoing trial. We had wide eligibility criteria accepting patients with comorbide mental disorders, but with health anxiety (hypochondriasis) as their main problem. A total of 415 patients were referred to the clinic in the inclusion period. A psychiatrist saw all patients, and all patients were offered treatment. A total of 176 patients were assessed for eligibility in the Kathy trial, but 91 patients did not meet inclusion criteria. Eighty-five patients fulfilled the inclusion criteria, but 5 patients declined to participate. The trial was therefore an integrated part of a general health care system and can in this manner be seen as a pragmatic randomised clinical trial. The trial took place in a routine clinical setting, which facilitates the generalizability of the result and strength the external validity. On the other hand the trial introduced a short-term psychodynamic psychotherapeutic treatment for patients with hypochondriasis. This psychotherapeutic approach for hypochondrial patients has not been reported systematically. So the trial introduced a new treatment and leaves you with the possibility of examining difference between different psychotherapeutic interventions. In this manner the trial have explanatory and hypothesis generating features. There was no significant difference between the dropout rates, which could have invalidated this analysis.

Randomisation

It is crucial that the assignment of participants in different groups in randomised trials is established on basis of a chance process characterized by unpredictability (166). It is necessary to avoid selections bias and to secure that the groups only differ by the intervention type, which is the condition for a null hypothesis testing. In spite of randomisation the possibility of difference between groups may arise by chance in small populations. In the Kathy trial we therefore used a restricted randomisation and decided on stratification based on gender and degree of depression measured by the sum-score on Hamilton Rating Scale for Depression (HAM-D). Results suggest (169) that small trials overestimate intervention benefits, if the generation of allocation sequence or allocation concealment were inadequate. We used computer-generated permuted blocks as the random allocation sequence, where the block sizes were concealed until the end of the trial. The allocation concealment was obtained by centralised telephone randomisation at the Copenhagen Trial Unit (CTU). Half the patients were randomised to a waiting list and the other half randomised to either CBT or SPP. The randomisation ratio was 1:2:1. After 6 months, the participants on waiting list were re-randomised to either CBT or SPP using the same generation and concealment strategy.

Blinding

In controlled trials blinding refers to keep patients, investigators, clinicians (therapists), outcome assesors, data manager, statisticians, and conclusion drawer unaware of the assigned interventions, so they will not be influenced by the knowledge of what group the patient are allocted to (166). Due to the character of the intervention in the Kathy trial, it was not possible to blind the patients and therapists to group allocation. It has been recognized in pragmatic randomised trials, that blinding is not always possible in complex interventions, and we accepted that a bias is introduced, as patients and therapist were aware of the treatment (170). In the Kathy trial a new treatment for health anxiety was announced in the media so a notion might had developed, that an effective treatment existed, which could influence the patient's and the therapist's attitude to a given treatment, and affect the refusal rate and the course of treatment. We were unable to examine this bias in the present trial, as we have no systematic information about the patients' expectation to the allocated treatment, but results suggest that small trials without double blinding overestimate interventions benefits significantly (169). Blinding is important in assessment when outcome measures involve some subjectivity. One of the primary outcome measures in the Kathy trial was Hamilton Anxiety

Rating Scale (HAM-A) (171), and one of the secondary outcome measures Hamilton Rating Scale for Depression (HAM-D) (172). The assessors were blinded to group assignment for the duration of the study. There has been some focus on the current lack of reporting success of blinding in controlled trials and its potential effect on study results (173). In the Kathy trial the blinding was evaluated, as the assessor was asked to guess the treatment the patient had received. They guessed the treatment high above chance ($X^2 = 25.1$, $P<0.0001$), and the blinding was therefore compromised. This represents a potential risk of bias and in this respect the trial can be regarded as un-blinded, though the assessors were in doubt and independent of the trial and the treatment. The patients were instructed not to tell the assessor what treatment they received. The reason for the compromised blinding was probably that the patients revealed the treatment unwillingly by the manner they answered the questions concerning different symptoms. The persons involved in encoding the data were blinded to the group allocation, but there was no blinding in the data analysis process.

Intention to treat, post-randomisation exclusion, and drop-outs

By the principle of intention to treat, the analyses in a randomised clinical trial compare patients according to the group, to which they were randomly allocated, regardless of compliance or withdrawal from the study. Intention to treat limits arbitrary or ad hoc sub groups in the trial and minimise the influence of withdrawal and allows for the greatest generalizability (174;175). Post-randomisation exclusions unrelated to non-compliance, withdrawal or losses to follow up are not in conflict with these principles of intention to treat.

If you accept post-randomisation exclusion in a randomised trial, you have to avoid bias and minimise random error. There are three situations where post-randomisation is a possibility (176). These situations should if possible be avoided by rigorous design and pre-testing of the study protocol:

- Patients mistakenly included who did not meet inclusion criteria.
- Poor or excessively broad eligibility criteria.
- Premature randomisations of patients into clinical trial, where clinical circumstances evolve so the patients never receive the intervention.

If a number of patients who were randomised and included in trial subsequently were excluded, the exclusion could transform a null result into a positive one and could create a misleading impression

of the overall effect of the treatment on the population it was applied. It is recommended that an independent adjudication committee blinded to treatment and outcome should systematically review each patient.

In the Kathy trial an independent group, who was blinded to treatment and outcome of the patients, evaluated all patients included in the trial. Four patients who were included were subsequently by this independent group found not to meet inclusion criteria. Two had previously received CBT, and 2 were not diagnosed correctly: one patient was psychotic, and one patient had a severe personality disorder, and health anxiety was not his main problem. These 4 patients were excluded and did not receive treatment. They met the criteria for the situation mentioned above, where post-randomisation was possible when participants were included and randomised by a mistake. It would have been preferable if the evaluation of eligible patients had been made before inclusion and randomisation, which might have been implemented if a pre-test had shown this problem.

Table 1: Exclusion and withdrawal rates for both treatment conditions.

	CBT (N=39)*	**SPP** (N=38)*	**Pearson X^2**	**P**
Post-randomisation exclusion	3 (7.7%)	2 (5.3%)	0.19	0.665
	CBT (N=36)	**SPP** (N=36)		
Completers	33(92%)	29(81%)	1.86	0.173
Drop-outs	1 (2,8%)	3 (8.3%)	1.06	0.303
No-acceptance	2 (5.6%)	4 (11.1%)	0.73	0.394

* 3 patients were not randomised after waiting list.
CBT, Cognitive Behavioural Therapy; SPP, Short-term Psychodynamic Therapy;

After post-randomisation exclusion 76 patients were included in the 3-group analysis. In the second analysis additional 4 patients were excluded after waiting list intervention. Three patients did not accept randomisation after waiting list intervention, while one patient had a waiting list improvement for health anxiety and did not meet the inclusion criteria after waiting list. Consequently, 72 patients were included in the 2-group analysis. For these 4 patients clinical circumstance evolved, so they did not receive re-randomisation and the secondary intervention. This circumstance is consistent with the third situation mentioned above. The patient with waiting list improvement was offered and completed treatment. No post-randomisation exclusion was caused by poor or excessively broad eligibility criteria. Six patients refused to accept the randomised treatment: two in the CBT group and 4 in SPP group. A total of 66 patients (83%) began treatment

of which 62 patients (78%) completed treatment. Four patients dropped out: 1 (3%) in the CBT group and 3 (10.7%) in the SPP group (See table 1). There was no significant difference between the 2 intervention groups for post-randomisation exclusion, non-acceptance of treatment, and dropout. We had missing data for most patients in dropout and no-acceptance group, but for a few of these patients data was collected at the end of treatment and at follow-up. Three patients, who completed treatment, were lost to follow-up. In total we had missing data for 10 patients (13.8%) at follow up. Different methods exist to handle missing data such as complete case analysis, mean substitution and more sophisticated methods based on statistical models such as multiple imputation and maximum likelihood methods (177). We chose to impute missing data instead of exclude observations, as randomisation do not protect against bias due to missing observation (178). The missing data were imputed as last observation carried forward. We therefore used information gathered after intervention was completed for some cases and information gathered before intervention for other cases, and assumed that these data correspond to data at follow up. This procedure contains the assumption that the responses from the last observation do not change after dropout (179).

Different treatment models for hypochondriasis

Different models of hypochondriasis have formed different specific psychotherapeutic strategies with the focus on behaviour, symptoms, and cognition. See table 2. These specific treatment models have resulted in a number of uncontrolled and controlled trials concerning the treatment of hypochondriasis. See table 3 (69;180-185). Kellner conceptualize (26) hypochondriasis as a psychophysical disorder, where patients do not comprehend the relationship between emotions and somatic symptoms. He introduces different cognitive and behavioural elements in his understanding of hypochondriasis such as selective perceptions and conditioned learning, and he developed a specific form of psychotherapy for hypochondriasis called explanatory psychotherapy (186) . The aim of explanatory psychotherapy is to persuade the patient that his belief is false, so it no longer reinforces psychopathology. It is essential that the patients gain the conviction that their symptoms are innocuous before therapeutic progress can be made. Explanatory therapy focuses on accurate information, clarification, and repetitions as means to explain psychological processes. The therapist must accept the patient unconditionally and it is important to empathize with the patients' problems. Suggestions coupled with accurate information about the patient's condition and examples from the patient's history should be employed to illustrate and emphasize the point.

Table 2 Different treatment models for hypochondriasis

	Model	**Intervention**
Explanatory psychotherapy	Eclectic model with aspects of Freud's actual neurosis, ego-psychological neurotic understanding and behavioural and cognitive feature. No comprehensive and clear model.	The hypochondrial patient does not comprehend the relationship between emotions and somatic symptoms. Explanatory therapies focus on accurate information, clarification, and repetitions as means to explain psychological processes. The aim of explanatory psychotherapy is to persuade the patient that his belief is false, so it no longer reinforce psychopathology
Amplification based cognitive behavioural therapy.	Hypochondriasis is a self-perpetuating and self-validating disorder of cognition and bodily perceptions. It constitutes a latent cognitive schema about health and disease and can be activated by current life stresses. The concept of bodily amplification has a central position in this model and intense and disturbing bodily sensations are the foundation for the misinterpretation of hypochondrial patients.	The therapy specifically targets the cognitive and behavioural amplification of benign bodily symptoms that propel the hypochondrial cycle of disease conviction and symptom amplification. The intervention consists of both cognitive and educational aspects with focus on 4 factors that amplify or attenuate somatic symptoms: attention and relaxation; cognition and symptom attribution; situational context; and dysphoric affect. The therapeutic relationship is more like that of a teacher or a student. Barsky further developed his group-based treatment to an individual cognitive behavioural therapy with six manualized sessions.
Cognitive-behavioural therapy with focus on misinterpretation and reassurance	The model is based on stimulus-response process: a particular type of stimuli or psychological reaction produces a characteristic physiological reaction. The two key elements of hypochondriasis model are preoccupation with bodily health out of proportion to existing justification and the pursuit of reassurance.	The therapist's target is to engage the patient sufficiently to be able to assess the problem collaboratively. The therapist attempts to arrive at a psychological formulation for treatment of the patient's problem. The treatment is directed at evaluating alternative, non-threatening explanations for the patients with health anxiety, where the explanation must provide the patient with a credible and normative account of his idea of disease. Behavioural experiments are being used. Treatment focus is the patient's health anxiety and reassurance behaviour.

Table 3 Uncontrolled and controlled studies of psychotherapeutic interventions for hypochondriasis.

Study	Design	Intervention	Randomisation	Outcome measures	Blinding	Participants flow
Kellner R.	Cohort study. 36 patients with hypochondrial neurosis, diagnostic criteria from DSM-III, no systematic diagnostic procedure. No systematic information of exclusion criteria. No control group.	Explanatory therapy. Detailed description of intervention.	None	No prescribed primary outcome measure. Both observer and self-rating outcome. No standardized, validated outcome measures were used.	None.	No information of number of patients evaluated for the trial. Inclusion period was many years, but not specified. No information of participants flow. No intention-to-treat principle. Two years follow-up.
Fava GA.	RCT. Randomised for treatment and waiting list. 20 patients satisfying the diagnostic criteria according to DSM-IV. Structured assessment procedure was used (SCID-I). Information about eligibility criteria. Randomised to explanatory therapy and waiting list. The patient on waiting list subsequently received explanatory therapy	Explanatory therapy. Detailed description of intervention.	Yes. No information about sequence generation or concealment	No prescribed primary outcome measure. Both observer and self-rating outcome. Standardized and validated outcome measures were used	The assessor was blinded to group assignment. No information about the success of blinding	No information of number of patients evaluated for the trial and inclusion period. There is information of participants flow, but no diagram. No information of intention-to-treat. Six month follow up for some patients but not under controlled conditions.
Avia MD	Controlled design. Allocated to treatment group and waiting list. No information about a possible randomisation procedure. 17 patients with a certain degree of hypochondrial symptoms. 8 patients meet all the criteria for hypochondriasis in DSM-III-R. Systematic diagnostic procedure specific for hypochondriasis was used (SDIH). Some patients in waiting list group received treatment subsequently.	Group treatment for hypochondriasis with focus on bodily amplification. Detailed description of intervention.	No information about a possible randomisation procedure.	No prescribed primary outcome measure. Only self-rating outcome. Standardized and validated outcome measures were used	None	No information of number of patients evaluated for the trial and inclusion period. Incomplete information about participants flow, no diagram. Six weeks follow up but not under controlled condition.
Barsky AJ	RCT. 187 patients allocated by cluster randomisation to short-term CBT or usual medical care. Patients consisted of both subthreshold hypochondriasis and hypochondriasis according to the criteria in DSM-IV. Systematic diagnostic procedure specific for hypochondriasis was used (SDIH) Information about eligibility criteria.	Individual treatment for hypochondriasis with focus on bodily amplification. Detailed description of intervention.	Cluster randomisation with GP as unit of randomisation. Random numbers table was used as sequence generation. Concealment strategy is described but is questionable	One prescribed primary outcome measure. Only self-rating outcome. Standardized and validated outcome measures were used. Effect sizes were calculated.	No assessor based outcome measure. Research assistant collecting data was blind to patients' treatment status.	Description of participants flow in a diagram with information of number of patients screened for eligibility. Data analyses used intention-to-treat and last-observation-carried-forward. 12 month follow up.
Warwick HM	RCT. 32 patients were randomly allocated to cognitive-behavioural treatment or waiting list. Patients on waiting list received treatment after 4 month. Patients fulfilled the criteria for hypochondriasis according to DSM-III-R. Patients were assessed using SCID-I. Information about eligibility criteria.	Cognitive-behavioural treatment with focus on misinterpretation of bodily sensations. Detailed description of intervention. Only one therapist	No information about randomisation procedure.	No prescribed primary outcome measure. Both observer and self-rating outcome. No standardized, validated outcome measures were used.	The assessor was blinded to group assignment. No information about the success of blinding.	Description of participants flow. Information of number of patients screened for eligibility. No intention-to-treat and last-observation-carried-forward. But only one patient was lost to follow-up. No follow up on waiting list group.
Clark DM	RCT. 48 patients were randomised to cognitive therapy, behavioural stress management or waiting list control group. Information about eligibility criteria. The patients meet the diagnostic criteria for hypochondriasis according to DSM-III-R, the patients were diagnosed using SCID-I	Cognitive-behavioural treatment with focus on misinterpretation of bodily sensations. Behavioural stress management. Detailed description of interventions. Several therapists.	No information about randomisation procedure.	No prescribed primary outcome measure. Both observer and self-rating outcome. Newly developed and standardized, validated outcome measures were used.	The assessor was blinded to group assignment. No information about the success of blinding	Information of inclusion period but not number of patients screened for eligibility. Description of participants flow. No intention-to-treat and last-observation-carried-forward, but only 2 patients dropped out, and data for one of these is included.
Visser S	RCT. 78 patients were randomised to cognitive therapy, exposure in vivo-response prevention, waiting list control. Information about eligibility criteria. The patients meet the diagnostic criteria for hypochondriasis according to DSM-IV. No information about systematic diagnostic procedure	Cognitive therapy and exposure in vivo-response prevention. Detailed description of interventions. Several therapists.	No information about randomisation procedure.	No prescribed primary outcome measure. Only self-rating outcome. Standardized and validated outcome measures were used.	No assessor based outcome measure. No information about blinding.	No information of inclusion period or number of patients screened for eligibility. Information about a high dropout rate (22%), but no diagram of patients flow. No intention-to-treat and last-observation-carried-forward.

Explanatory therapy uses few therapeutic components. It is simple, and does not require any specific therapeutic skills. It does not introduce any behavioural technique and is not based on a specific theoretical framework (187). The efficacy of explanatory psychotherapy was (188) examined in a uncontrolled design by Kellner in 36 patients, while Fava examined (189) explanatory psychotherapy for 20 hypochondrial patients in a randomised clinical trial. The findings indicate a positive treatment effect, but the study designs are problematic, and it is not possible to make any conclusive statements based on these findings.

According to Barsky (56) hypochondriasis is an chronic psychopathological condition defined as an unrealistic interpretation of bodily sensations leading to fear or belief of a serious disease. Hypochondriasis is characterized by an enduring attitude toward health, concern of the body, and belief of disease with a latent cognitive schema about health and disease, that can be activated by current life stresses (62). This cognitive schema is supported by confirmatory bias that leads hypochondrial individuals to attend selectively to information supporting the hypochondrial ideas and ignores disconfirmatory information. The model explains the refractoriness to medical reassurance in hypochondrial individuals (66). The concept of bodily amplification has a central position in this model, and intense and disturbing bodily sensations are the foundation for the misinterpretation, where hypochondrial patients amplify benign somatic sensations, and misattribute them as a serious disease. Barsky developed a cognitive treatment for hypochondriasis (68) with bodily amplification as a central target for the therapy. The treatment model consists of both cognitive and educational aspects with focus on 4 factors that amplify or attenuate somatic symptoms: attention and relaxation; cognition and symptom attribution; situational context; and dysphoric affect. The therapeutic approach is a group format of 6 to 8 patients who meet in 6 consecutive weeks. The educational nature is emphasized and the treatment is referred to as a course. The therapeutic relationship is more like that of a teacher or a student. Avia (190) used this therapeutic approach for 17 patients who were allocated to 3 groups, 2 groups receiving therapy with different therapists, and 1 group consisting of waiting-list control, who subsequently received treatment. There is no information about randomisation procedure. There is an incomplete follow up on some measures. There is no systematic evaluation of participants' flow and no information of intention-to-treat procedure and handling of missing data such as last-observation-carried-forward. There was a wide array of standardized and validated outcome measures but no identifiable primary

outcome measure. It is stated in the paper that treatment was effective for the target measures, but the trial design and unclear reporting leaves the findings inconclusive.

In a recently published large-scale randomised clinical trial with usual medical care (69) as control, Barsky further developed the group based treatment to an individual cognitive behavioural therapy. He states that the therapy specifically targets the cognitive and behavioural amplification of benign bodily symptoms that propel the hypochondrial cycle of disease conviction and symptom amplification. This trial represents an important development for the conduct of randomised trial for psychotherapy. There is a clear description of participants flow in a diagram with information of all patients screened for eligibility, which strengths the external validity. There is an identified primary outcome measure and intention-to-treat and last-observation carried forward for data analysis. The patients were recruited by screening with a self-report hypochondriasis questionnaire using a cut-off score and consisted of both sub-threshold hypochondriasis and hypochondriasis according to the criteria in DSM-IV. A total of 187 patients were randomised to brief, individual CBT intervention or usual medical care as control group. There was a 12-month follow up. Outcome measures consisted of questionnaires, so blinding was no beformed. Cluster randomisation was used with the primary care physician as the unit of randomisation. The allocation sequence was generated using a random numbers table. A staff member not connected to the trial took care of the randomisation, but concealment of the allocation sequence in this setting with cluster randomisation is not possible. In the trial a significant clinical treatment effect was found for the primary and all secondary outcome measures at 12-month follow up. It is an important finding, but questions were raised (191) about generalizability and the relationship between intervention and outcome. The design of the trial represents an improvement in comparison with earlier trials. The outcome measures are focused on symptoms related to hypochondriasis such as health anxiety and somatosensory amplification. There is no reported data about comorbide psychopathological measures of depression or general anxiety.

The cognitive behavioural understanding of hypochondriasis characterized by misinterpretation of benign bodily sensations was based upon experimentally validated principles of assessment and treatment. Methods and understanding were implemented from models of anxiety and obsessive disorders. This development was succeeded by controlled trials validating treatments based on these understandings. The importance (82) of psychological processes rather than diagnostic categories is

emphasised as a decisive treatment hypothesis. The key element in hypochondriasis according to this model is the tendency to perceive signs and symptoms as more dangerous than the really are. Misinterpretations of bodily sensations are a fundamental psychological mechanism in both hypochondriasis and panic attacks. But preoccupation of health in hypochondriasis is a key cognitive element, where the behavioural components as seeking reassurance from physicians and checking on bodily states serves as an important maintenance functions. There is a similarity to obsessive-compulsive disorder where avoidance behaviour in a similar way maintains the anxiety. The unrealistic fear of hypochondriasis persists not despite medical reassurance but because of repeated medical reassurance. The symptoms misinterpreted are not likely to be subject to direct amplification as in panic disorder and the feedback mechanism is more behavioural and longer term by maintaining focus on particularly parts of the body with repeated checking or physical manipulation. From a cognitive perspective, anxiety occurs when particular situation is experienced as a threat, and the patient's ability to cope with the situation is doubted. So the cognitive hypothesis of hypochondriasis propose that bodily sign are perceived as more dangerous, than they really are, and a particular illness is believed to be more probable than it really is. Once a critical incident has resulted in a particular assumption, this leads to confirmatory bias in the patients' thinking. When health anxiety has developed other mechanisms may be involved in maintenance of the problem.

The model of misinterpretation as a central psychological process in hypochondriasis has led to a well-defined treatment, which has been examined in case studies, uncontrolled trials and in two controlled trials (181;185;192). Warwick (185) conducted a study with 32 patients who met the DSM-IIIR criteria for hypochondriasis, who were randomised to cognitive-behavioural treatment (CBT) or waiting-list control. The CBT group was significantly improved compared with the waiting-list group. The improvement was maintained at 4-months follow-up. Clark (181) replicated and extended the trial by using several therapists and compared the cognitive treatment with behavioural stress management, which is seen as a credible alternative treatment. Forty-eight patients who met the DSM-IIIR diagnosis for hypochondriasis were included in the trial. Both treatments were significantly more effective than waiting list. The CBT group was significantly more effective than stress management on measures of hypochondriasis, but at the 12-month follow-up the two treatment groups did not differ significantly. Visser and Bowman (193;194) used the model of Warwick and Salkovskis to develop a specific cognitive therapy for hypochondriasis

in a randomised trial but gave no information about dissemination strategies. The result was ambiguous partly caused by the design of the study and insufficient reporting.

As indicated in table 2 Barsky's conceptualization of the relationship between bodily sensations and hypochondriasis emphasise other aspects than Salkovskis and Clark's (71) cognitive model of health anxiety, which see misinterpretation of bodily perceptions as the central psychological mechanism. The two schools have different theoretically starting points. Barsky developed his cognitive understanding of hypochondriasis from the diagnostic psychiatric definition in DSM-III and DSM-III-R. He has from the beginning of his theoretically development stressed the importance of amplification of bodily sensations in the aetiology and maintenance of hypochondriasis, and he incorporated this concept in his cognitive model of hypochondriasis. The cognitive-behavioural model of Salkovskis and Clark developed from a more general cognitive understanding of psychology and psychopathology. The model introduces psychological processes conceptualized in other conditions such as panic disorder and obsessive-compulsive disorders and sees the misinterpretation of benign bodily signs as the central process in hypochondriasis. The model includes reassurance behaviour as maintenance factor, and the cognitive therapeutic interventions focus on patients' misinterpretation and reassurance behaviour, where the interventions based on Barsky's model have more educational aspects and the treatment is referred to as a course.

Both treatment models have been examined in randomised clinical trials. The results indicate that the treatments are effective, though the trials have problems in designs and reporting (see table 3). It is our impression that the treatment model of Salkovskis and Clark is the most coherent, so we used this treatment model for the Kathy trial.

The implementation of a cognitive-behavioural and psychodynamic treatment in a randomised clinical trial

A pivotal issue in designing a treatment outcome trial is ensuring that the treatment will be fair represented. An important issue, which emerged is how representative the treatment is and the implication of this finding (195). In our trial we used a cognitive-behavioural and psychodynamic model respectively as two treatment models for patients fulfilling the diagnostic criteria for hypochondriasis.

Cognitive-behavioural treatment for hypochondriasis

The cognitive-behavioural model proposes that an individual's actions are developed and maintained by basis of learning principles. Cognitive treatment focuses on the potential mediating role the cognition play in the development of psychological and behavioural difficulties. In the cognitive perspective, an individual interpretation or construct is a more important determinant of behaviour, than the stimulus itself or the consequence of a particular action. Cognitive theory conceptualises problematic behaviour as resulting from dysfunctional or distorted thinking. Treatment focuses more on cognition than behaviour. A reduction in dysfunctional distorted thinking mediates a decrease in symptomatic distress. Cognitive-behavioural treatment is an amalgamation of cognitive and behavioural procedures and interventions share an appreciation of both basic learning principles and the role cognitions play in human behaviour and affective experience. An important intervention is imaginal or in vivo exposure. The exposure intervention corrects erroneous association between stimuli, responses, and the meaning attached to them in an individual's emotional memory network. Correction of this mistaken evaluation requires both activation of the fear structure, and the presentation of corrective information inconsistent with the learned pathological association. Cognitive representations of stimuli, responses, and their representation interact with behavioural learning principles to evoke pathological levels of anxiety (196). This fundamental and general model for cognitive behavioural theory is consistent with the specific cognitive behavioural model for hypochondriasis or health anxiety developed by Salkovskis and Warwick(71). The misinterpretation of benign bodily sensations or information about health and disease is seen as the central psychological mechanism in maintenance of the pathological condition. The cognitive behavioural treatment presented in our trial is developed by the use of this model.

Psychodynamic treatment for hypochondriasis

In the psychodynamic model the unconscious is an essential concept. It constitutes elements, structures and processes, which are not available for the conscious part of the psyche, but have pervasive influence on the contents of the consciousness and the behaviour of the individual. The key features in the psychodynamic model include emphasis on the therapeutic relationship understood as transference, the patients' interpersonal interactions, recognition of patterns or themes in the patients' functioning, and the development of an understanding of unconscious fantasies (197). There is no comprehensive or unifying model for hypochondriasis in a

psychoanalytic understanding. It is part of a general theoretical understanding of the psyche and psychopathology. In a psychoanalytic understanding hypochondriasis could be seen as a neurotic symptom, which expresses or represents aspect of psychic conflict and unconscious conflicted wishes, or hypochondriasis could be seen as psychotic condition, which split off unthinkable aspects of the mind which is lodge somewhere in the soma as an alien possession or intrusion of the body.

Examination of treatment implementation and adherence

Our trial is focused on treatment outcome, but none of the treatments were manualized. A manual consists of written material to guide the therapist in procedures, technique, themes, therapeutic manoeuvres and action. It is an effort to operationalize the practice of doing therapy, and the purpose is to minimize variability in treatment delivery, and a manualization is seen as an explicit statement of how the therapy was implemented. Interpretation of treatment outcome is enhanced, when manuals are available. But a manual may lead to rigid application and cannot adequately capture the complex process of therapy. One of the problems with developing a manual of a psychodynamic technique is the loose relationship between psychodynamic theory and clinical practice. Psychodynamic therapists choose flexibility and autonomy and do not appreciate the constraints inherent in manualized treatment (164).

The cognitive-behavioural treatment (CBT) for hypochondriasis was implemented through a dissemination strategy (198). The therapists had no previous experience with treating patients with hypochondriasis, but were experienced with treating patients with anxiety and obsessive-compulsive disorders in a cognitive-behavioural model. They received a brief training course, which was subsequently supplemented by expert clinic and peer supervision. The short-term psychodynamic psychotherapy (SPP) was used a contrast and did not contain behavioural and cognitive interventions. We wanted to examine, whether a possible effect of CBT, were a result from the specific techniques that characterize the treatment, rather than from common factors. So we matched with an appropriate psychological treatment for non-specific therapeutic factors. We had to ensure that the treatments were proper implemented in the trial. As no manualization of the treatment interventions was available, we developed a method to assess, that the therapist performed treatment as designated and demonstrate adherence in the delivery of the specific treatment. We used the Delphi technique (199) to establish consensus for different features

consistent with respectively CBT and SPP. The Delphi technique is a consensus method to determine the extent of agreement of a clinical issue. The method uses a panel of experts who take parts in rounds to identify and clarify a common understanding of a therapeutic intervention. In our case the therapist in the Kathy trial determined items, which characterized the two interventions. These items were defined and described carefully and used as a rating instrument to distinguish interventions unique to each treatment. Therapists familiar with respectively cognitive-behavioural and psychodynamic therapy, who did not participate in the trial, used the rating instrument for transcripts of psychodynamic and cognitive-behavioural sessions. The experiences of these ratings provided a basis for changing the rating instrument. Two independent raters, who were experienced psychologists, used the instrument to distinguish between transcripts of different sessions from the Kathy trial. The raters were blind to the group assignment of the sessions. They each rated 12 CBT sessions and 12 SPP sessions.

The cognitive behavioural treatment (CBT) in the Kathy trial was a highly specific psychotherapeutic treatment focused on health anxiety, which did not depend on the productivity of the individual patient. Instead the patient should fulfil certain criteria according to the diagnostic definition of hypochondriasis. So the theoretically model for health anxiety provides a relatively unambiguously expression of the disorder, and the treatment techniques for the specific treatment did directly link to this understanding. The psychodynamic treatment was not a focal psychotherapeutic treatment but used more general aspects such as free associations, neutrality, and in limited degree interpretations. The therapeutic approach resembles the emphatic validation or interpretation proposed by Anna Ornstein (200), where empathy is understood as a particular listening position trying to maintain a contact with the patients inner life, where acceptance and understanding is the focus of the therapeutic interventions. This psychodynamic approach makes an examination of psychopathological experience unique to each patient possible. We used the conceptualisations of psychodynamic interventions by Greenson (201) and partly Gabbard (202) to describe and define the psychodynamic interventions in the Kathy trial, and modified the definitions according to the process described in connection with The Delphi technique. The final description and definition of the different psychodynamic interventions are shown in table 4. The description was originally in Danish but is translated and shortened. In table 5 the result of two raters evaluation are shown as mean numbers of interventions pr. session for all 24 rated sessions.

Table 4: Definition and description of psychotherapeutic interventions.

Cognitive-behavioural interventions	Behavioural experiments	Behavioural experiments conducted under a session.
	Direction of session activity	Introduction and completion of a certain agenda. Examination of questionnaires for health anxiety, global anxiety and depression. Feedback and evaluation are being used.
	Providing a theoretical model	A theoretical model are presented and discussed
	Cognitive discussions	Focus on health anxiety related cognitions and safety seeking and avoidant behaviour. Identification of alternative explanations for the patient's bodily sensations. Examination of proofs pro or against the patient's catastrophic interpretations. Challenge and change of health anxiety related cognitions towards a normalisation. To choose cognitive strategies and techniques for cognitive change, including behavioural experiments.
Psychodynamic interventions	Interpretation	An explanatory statement that links feelings, behaviour, thoughts and symptoms to its unconscious meaning or origin. Interpretation can focus on transference, the patient's past or present situation, resistance or fantasies. The purpose is to bring unconscious connections to consciousness.
	Clarification/confrontation	An understanding and empathic attitude, where parts of the patent's communication are selected and the patient are stimulated for more memories and experiences. The patient's statement can be rephrased or part of the statement extracted to give a more coherent picture. Focus is to enhance the patient's consciousness of vague and unclear ideas. The interventions does also cover a more confrontational attitude, where the purpose is reality-testing and to uncover therapeutic material and to test in which degree the patient will understand and accept this material.
	Confirmation	Confirm ideas, thoughts and information about a topic that the patient has introduced
Non-specific	Encourage to work	Elaborate on topic brought up by the patent
	Reassurance	Comfort the patient and make him stop worrying

The sessions are taken from different patients. There is a significant difference between the 2 psychotherapeutic approaches with cognitive and behavioural interventions connected to cognitive-behavioural sessions and the psychodynamic interventions connected to psychodynamic sessions. This result indicates that we use different psychotherapeutic approaches and with a certain degree of fidelity. The most common intervention for the cognitive behavioural sessions is cognitive discussion with a mean number of 30 pr. session and a behavioural experiment for each second session. The most common intervention for the psychodynamic session is clarification/confrontation with a mean number of 11 pr. session, and one interpretation pr. session. There is no direction of session activity or providing a theoretical model in the psychodynamic approach, and clarification/confrontation, confirmation and encourage to work are the most common interventions indicating that neutrality and free associations are the most important therapeutic principles. The content of health and disease were represented significantly more in CBT sessions than SPP and interpersonal issues significantly in SPP, while both shared non-interpersonal issues. These findings are consistent with the definition of the different interventions, and it is found that CBT is focused on health anxiety related cognitions.

Table 5: Mean number of intervention per session (total number of sessions:24)

Interventions	**CBT (SD)**	**SPP (SD)**	**P** (Mann-Whitney)
Behavioural experiments	0.4 (0.9)	0	0.180
Direction of session activity	6.8 (4.9)	0	<0.0001
Providing a theoretical model	7.0 (5.6)	0	<0.0001
Cognitive discussions	30.0 (7.4)	0	0.001
Interpretation	0	1.0 (0.9)	0.025
Clarification/confrontation	0.4 (0.9)	10.6 (4.0)	0.002
Confirmation	0	3.7 (1.8)	0.002
Encourage to work	0.8 (1.3)	3.0 (1.8)	0.023
Reassurance	0.6 (1.3)	0.2 (0.4)	0.926
Contents of interventions (%)	**CBT (SD)**	**SPP (SD)**	**P** (Mann-Whitney)
Health and disease (%)	12 (11)	48 (19)	0.04
Interpersonal issues (%)	63 (22)	21 (11)	0.09
Non-interpersonal issues (%)	25 (19)	32 (20)	0.463

CBT, Cognitive behavioural Therapy; SPP, Short term psychodynamic psychotherapy; SD, Standard deviation.

Outcome measures

We have chosen standardized outcome measures in the Kathy trial, which reflects different psychopathological manifestations. The reliance on symptom and behavioural change measures has

been seen as applicable to medical treatment programs, behavioural therapies and brief time-limited dynamic psychotherapies (203). The two therapeutic interventions in the Kathy trial are a cognitive-behavioural therapy and a short-term psychodynamic psychotherapy, which are consistent with this recommendation. But it is also stated that psychoanalytic psychotherapy works with more enduring personality reconstruction, and in this context it is better to use instruments, which reflect change in personality structure. The short-term dynamic psychotherapy uses therapeutic features from psychoanalytic psychotherapy, and a possible change in personality structure is not reflected in the same degree with behavioural and symptom measures. Millon Clinical Multiaxial Inventory-III (MCMI-III) (204) was developed to assess clinical personality styles both dimensionally and categorically, and personality disorders in accordance with both Millon's theory of personality and psychopathology and DSM-IV. We have used MCMI-III in the Kathy trial but the results have not yet been analysed.

The patients were well functioning with a high mean score on Global Assessment Functioning (GAF) (205). The GAF did not change significantly during treatment, and thus the substantial change in psychopathological outcome scores were not reflected in GAF. It has been seen as a global phenomena that symptoms and social function do not overlap substantially (195), and it is a limitation of the Kathy trial that other measures of behaviour are not available. The reassurance seeking behaviour of patients with heath anxiety is an important domain, which could be examined by collecting data on the patients' health care utilization pre and post treatment, which would reflect the impact of the reduced psychopathology on the patients' coping behaviour.

It is important that outcome measures pertain to processes that are considered critical to treatment and therapeutic change and that the outcome measures correlate with change. The impetus for seeking treatment usually is the presence of various symptoms. And the rationale is that the problem identified at the onset of a treatment is reduced after treatment. In the Kathy trial hypochondrial patients with a high level of health anxiety are referred to treatment to have their health anxiety reduced. We have chosen two variables as primary outcome measures. The Health Anxiety Inventory (HAI) is a newly developed questionnaire for hypochondriasis and health anxiety, which has shown to be a valid and reliable measure for health anxiety with a good internal consistency (151). It has good criterion validity and discriminates between patients diagnosed as suffering from hypochondriasis and patients suffering from anxiety disorders, and is not elevated in physically ill

patients. There is evidence that the scale is sensitive to change. HAI has been seen as an independent factor in hypochondriasis, consistent with current cognitive theories. Our findings in the enclosed paper with cross-sectional examination of 80 patients with hypochondriasis confirm by the use of multiple regression models, that HAI is independent of different measures of depression and anxiety. HAI consists of 18 items and responses are obtained on 4-point Likert scale. It is described as a continuous measurement of health anxiety that is sensitive to normal levels of health concern as well as clinical hypochondriasis. We have omitted the last 4 items in our analyses in the Kathy trial, as these items concentrate on negative consequence if a serious disease is present. We used the sum-score for the first 14 items as one of our two primary outcome measures.

Table 6. **Items in The Health Anxiety Inventory (HAI) in relationship with clinical aspects and diagnostic definition of hypochondriasis.**

Aspects of hypochondriasis	Items in HAI
Preoccupation	• Worry about health • Resisting thoughts of illness • Images of being ill • Difficulty taking mind off thoughts about health • Thinking you have a serious disease
Fear of disease	• Afraid of having a serious illness • Usually feeling a risk of having a disease
Bodily sensation/misinterpretation	• Bodily sensation change, makes you wonder what it means • Notice aches and pain • Hearing about an illness you have it • Awareness of bodily sensation or changes • Unexplained sensation, makes it difficult to think about other things
Reassurance	• Lasting relief if doctor tells nothing is wrong • What family/friends would say about your attitude to health

In table 6 the first 14 items of HAI are shown in relation with different clinical aspects of hypochondriasis, which is integrated in the diagnostic definitions. These items cover the different aspects of the diagnostic definition with emphasis on misinterpretation, which is a central part of the cognitive model for hypochondriasis. So HAI seems to be a reasonable measure of hypochondriasis

independent of other measures of psychopathological manifestations, a measure which reflects possible changes examined in the Kathy trial.
As the other primary outcome measure we chose Hamilton Anxiety Rating Scale (HAM-A)(171), which provides an overall measure of global anxiety. It is a widely used scale for measuring anxiety, and psychometric studies indicate good reliability with trained raters and a good measure of overall anxiety, which correlates with other measures of anxiety. It is sensitive to treatment (206). The scale is not recommended as a diagnostic instrument. In the Kathy trial HAM-A was used as an outcome measure of hypochondrial patients already diagnosed by the use of the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) (207).

HAM-A is an assessment-based measure, which allows for blinding of group assignment. HAM-A is not based on a cognitive model and reflects more global aspects of anxiety not primarily connected to health anxiety or a specific conceptualisation of hypochondriasis. The Kathy trial used four experienced psychologists to assess the patients. They received training in using the instruments, and the interrater agreement was measured and found reasonable. During 11 Hamilton training cases the mean level of agreement was 90.8% (5.3% [SD]).

The 2 primary outcome measures therefore have different targets related to anxiety. They have different formats, where HAI is a questionnaire, while HAM-A is a clinical-administrated rating scale. The difference in these two primary outcome measures reflects different aspects of anxiety and hypochondriasis, which allow for a more complex understanding of a possible change of psychopathology in these patients.

Statistical analyses

According to the null hypothesis we examine the probability of a given result, if there is no difference between randomised groups. The data analysis in the Kathy trial was divided into two parts: First, the three allocation groups (CBT, SPP, and waiting-list) were compared after intervention on primary and secondary outcome measures. Second, the two treatment groups (CBT, SPP), including the patients initially on waiting list, were compared after treatment and at 6 and 12 months follow-up on primary and secondary outcome measures.

We used a general linear model for the statistical data analysis. The linear model examines the relationship between variables to see if a systematic variation in a dependent variable results from a systematic variation in an independent variable. It is the ratio between random error in the dependent variable and the variation due to the independent variable, which determines if the linear model is a good fit. In the Kathy trial the statistical model proposes the intervention groups as the independent variables and the patients' psychopathology as the dependent variables, so the intervention groups determine the patients' psychopathology. The first analysis was a one factor ANOVA with the three groups as independent variables and the outcome variables as dependent variables. In case significant differences between the three groups were found, a post hoc test was included as a pair-wise comparison with a Turkey correction for type I error. The second analysis was a two factor, mixed design, repeated measure ANCOVA with outcome variables after treatment as dependent variables and outcome variables before treatment as covariates. The two treatment groups were between-subject factors, and time 0, 6, and 12 month after treatment was within-subject factors. For each analysis a 95% confidence interval was derived. All tests of statistical significance were interpreted with a criterion of $P< 0.05$.

To apply a general linear model at our data we need to be aware of the assumption that the variability of our data around the regression line is due to random error and should be more or less the same at each point of the regression line. To examine the homogeneity of variance we used Levene's test of homogeneity of variance on all analyses of variance used in the Kathy trial. The test was not significant in any of the 10 analyses. The second analysis in the Kathy trial was a repeated measure ANCOVA. We had to test for homogeneity of variance of the difference between patients, as we repeated the measure 3 times for all patients. By the use of Mauchley's test of sphericity it was found that sphericity assumption was violated for Beck Depression Inventory (BDI) and HAM-D, probably because depression was a carry-over-effect. We used the Greenhouse-Geisser correcting for the violation of the sphericity assumption in these 2 analyses.

The statistical result of the Kathy trial and the subsequently discussion is presented in the enclosed paper.

The mechanism of change

Fairburn examined (208) cognitive-behavioural psychotherapy versus interpersonal psychotherapy in an explanatory approach, and his analyses were based on patients who complete treatment. His

design is comparable with our design. Patients were selected from a diagnostic group based on symptomatic items, in the Fairburn trial patients with bulimia nervosa in our trial patients with hypochondriasis or health anxiety. The patients were randomised to two different psychotherapeutic treatments. The cognitive behavioural treatment focuses on the symptoms, which define the condition, while the psychodynamic treatment does not focus on the symptoms of the diagnostic entity. The psychodynamic intervention in Fairburn's is a manualized treatment with focus on the present interpersonal relationships. In the Kathy trial we used non-focal psychodynamic therapy characterized by keeping the patient observant on his own associations, thoughts, and feelings in the sessions. Both psychodynamic treatments do not explicit offer an explanation and intervention specific for the condition the patients are referred to treatment for. But Fairburn offers a specific focus in the psychodynamic treatment in the shape of present interpersonal relationship. We do not offer a general focus in the psychodynamic therapy in the Kathy trial, but use the principle of neutrality and free associations. It is Fairburn's hypothesis the two psychotherapeutic treatments must operate through different mediating mechanism. Cognitive behavioural therapy modifies patients' symptoms directly. The decrease in the level of general psychopathology and improved social functioning are likely to be secondary effects, while interpersonal therapy must operate differently. Fairburn and colleagues took great care to ensure that there was no discussion of symptoms under the sessions. The patients were making major positive changes in their relationship, and this interpersonal change resulted in the erosion of the eating disorder. The effect might take longer to be expressed because interpersonal therapy appears to operate indirectly, so the changes in the patients' relationship occur first and subsequently in patients' eating behaviour and attitudes.

A similar mechanism of change might be seen in the Kathy trial. But the SPP in the Kathy trial uses the principle of free association and neutrality, so the possible change works through the relationship in the sessions, as there are no specific focus on current interpersonal relationship and modification of the patients' behaviour according to this specific issue. To respect neutrality the SPP in the Kathy trial does not offer the patients a specific explanation or understanding of their anxiety and problems. In Fairburn's study the interpersonal therapy substitutes the cognitive-behavioural model with a treatment model for present interpersonal relationship. No specific model of the patients' problems is introduced in the psychodynamic therapy in the Kathy trial. The interventions are mainly clarification, confrontation and encouragement to express thoughts and

feelings with no general focus. The interventions are guided by the transference, but the transference is not the focus of the interventions. The psychotherapeutic setting creates a frame that demarcates the therapeutic setting from that of everyday life. The setting develops another level of relationship by the therapist action and the patients´ motivation for treatment and change. This relationship is non-specific and is shaped by the setting and the therapeutic intervention and can be experienced as a protection against the environment and the patient's own ruminations of health and disease, which can have persecutory aspects for the patient.

The psychodynamic treatment does not offer a tangible expression of reassurance, but the patient may understand the attitude and the relationship with the therapist as reassuringly with the possibility of integrating the relationship. Modell (209) proposed the term dependent transference for this therapeutic relationship, by this term indicating that the relationship is a transformation dependent of the therapeutic setting and the therapist's intervention. But the setting gives rise to another transference, which is particularized and idiosyncratic, and which is highly dependent of the patient and his idiosyncratic problems. Model denotes this category of transference for iconic or projective transference and it can be seen as an externalisation or projection of the patients' personal problems. The dependent transference is predominantly accommodative because it is a response to what is in the environment, while the projective transference is predominantly assimilative by imposition of an internal schema on the therapeutic setting. Both categories of transference are present in psychotherapy, but it is difficult to work with the iconic transference in short-term psychodynamic psychotherapy. You have to use a focal approach or intervene according to the patient's transference to protect the psychodynamic setting and facilitate the dependent transference, and simultaneously respect the principles of free associations and neutrality.

These principles of psychodynamic treatment are in conflict with the principles of a randomised controlled trial. Principles connected to a randomised trial such as randomisation, control group, and that the patient is referred to treatment, has an important influence on the patients' idea and expectations about treatment, and therefore the relationship the patient form in the beginning of psychotherapy. It is not possible to work with projective or iconographic aspects of transference in short-term psychotherapy. So in this understanding it is not possible to work with the patients' transference and resistance through interpretation. You have to facilitate and rely on the dependent transference as a curative factor or use a focal psychodynamic intervention. In respect to Fairburn's

iodel for change, the relationship between the therapist and the patient in a psychodynamic setting acilitates an integration of a therapeutic relationship through the dependent transference, which evelops a less treating experience of bodily sensations and ideas of health and disease, and ubsequently less anxiety and depression.

An examination of consecutive patients diagnosed with hypochondriasis.

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Abstract

Objective: To examine comorbidity in patients with hypochondriasis and evaluate hypochondriasis as a clinical diagnosis.

Method: In a cross-sectional design 415 consecutive patients referred to a liaison psychiatry clinic were examined and patients with a substantial level of health anxiety were diagnosed by the use of the Schedule for Clinical Assessment in Neuropsychiatry (SCAN). Questionnaires for psychopathology and personality disorder were applied. Comorbidity was examined by variance and regression analysis.

Result: Nineteen percent (n=80) were diagnosed with hypochondriasis. There was a high level of psychiatric comorbidity. Multiple regression analyses showed no significant relationships between a specific measure for hypochondriasis and anxiety, depression, and personality patterns but a significant relationship existed between hypochondriasis and personality disorders. The result indicates subgroups of hypochondrial patients with interpersonal problems and eccentric experiences.

Conclusion: The current diagnosis for hypochondriasis are clinical useful. There is a considerable psychiatric comorbidity independent of hypochondriasis consistent with the hypothesis that hypochondriasis is a primary condition.

Key words: hypochondriasis; health anxiety; comorbidity; diagnosis; personality disorder

Significant outcome:

- The current diagnostic criteria for hypochondriasis are clinical useful in a secondary health care clinic specialized in psychosomatic conditions.
- There is considerable comorbidity with other psychiatric disorders, but a specific measure of hypochondriasis was independent of comorbide disorders supporting the hypothesis of hypochondriasis as a primary condition.
- The result show subgroups of hypochondrial patients with interpersonal problems and eccentric experiences

Limitations:

- The sample is a selective group of patients who may not be a representation of hypochondrial patients in general.
- The cross-sectional design limits the possibility for examine causal relationship between comorbide conditions.
- Personality patterns and personality disorders were diagnosed solely by the use of a questionnaire.

Introduction

The first step in initiating appropriate treatment for a given disorder is establishing the correct diagnosis (210). Kenyon's critical attitude towards hypochondriasis as a nosological entity (211) was motivated in different and conflicting definitions of hypochondriasis, which existed at that time. With the conceptualisation and definition of hypochondriasis presented by the current diagnostic systems, a consensus of the understanding of hypochondriasis has developed (77). The current diagnostic definitions have facilitated empirical research in comorbidity of hypochondriasis and related disorders. The understanding of a primary hypochondriasis independent of other psychopathological conditions has been a controversial issue in psychiatry (212) and has caused the development of different conceptualisations of the disorder and empirical research to examine this issue (72). One position is the acceptance of primary hypochondriasis discussing comorbidity between hypochondriasis and other disorders such as anxiety. There is a high level of comorbidity between hypochondriasis and panic disorder (213), but findings indicate (109) that they are distinguishable clinical conditions. In a cognitive model (214), it has been proposed that both panic disorder and hypochondriasis develop in a vicious circle of catastrophic misinterpretation of bodily sensations. In panic disorder the misinterpretation is a sign of an immediate catastrophe, while in hypochondriasis it is seen as a future threat with development of reassurance-seeking and checking behaviour. A second position question hypochondriasis as a nosological entity; Noyes (105) stated that hypochondriasis is distinct from anxiety disorders but because of phenomenological similarities and extensive comorbidity should be classified among anxiety disorders. Other schools propose that hypochondriasis should be seen as part of the obsessive-compulsive spectrum (215), or as a modified form of somatization disorder, and Fink (103) proposes a new diagnostic entity with ruminations of illness as the most important item. It has been discussed if hypochondriasis is better conceptualized as a personality disorder (216) because of the long lasting patterns of dysfunctional beliefs and attitudes (137).

Only limited empirical research examines the relationship between hypochondriasis and different concepts of personality and personality disorder, but hypochondriasis has been found related to neuroticism (217), and in a recent study (218) a strong correlation was found between hypochondrial concerns and negative temperament as measured by the Schedule for Nonadaptive and Adaptive Personality (SNAP) (219). It was proposed that negative temperament represents trait-like vulnerability to hypochondriasis.

The examination of prevalence, distribution and characteristics of hypochondriasis has taken place in different settings, but predominantly in the primary health care sector or in in hospital setting such as clinics of general or specialized medicine. The present study took place in a liaison psychiatry clinic where all consecutive patients referred for treatment were evaluated. The study focus on 80 patients diagnosed with hypochondriasis according to the current diagnostic criteria. We examine the relationship between concurrent comorbide disorders including personality traits and personality pathology and a specific measure of hypochondriasis.

Methods

Setting and patients

The study is part of a randomised clinical trial, which examines the effect of a specific psychotherapeutic treatment for patients with hypochondriasis (The Kathy trial). The study was conducted in a liaison psychiatry clinic at Copenhagen University Hospital, Denmark. The clinic offers consultations and outpatient treatment to somatically ill patients with comorbide mental disorders or medically unexplained complaints, including health anxiety. Patients are referred from general practice and from primary and secondary health care settings. The clinic consists of a consultant, 3 staff specialists, and a nurse. All referred patients are evaluated and treated by a staff of specialists in psychiatry. The clinic cooperated with a clinical centre specialised in cognitive behavioural treatment for anxiety disorders, and in cooperation with this centre implemented and developed a specific treatment for patients suffering from hypochondriasis (220). A subgroup of patients with a substantial amount of health anxiety was evaluated for the randomsied trial, and a selected group fulfilling the diagnostic criteria for hypochondriasis, which consented to participate in the trial, was examined for different aspects of psychopathology and personality problems.

Data collection and assessment tools

Between August 2001 and January 2003 senior clinicians diagnosed all consecutive patients referred to the clinic using the criteria in ICD-10. On basis of referral notes and clinical findings a subgroup of patients with a substantial amount of health anxiety received a comprehensive psychiatric interview including the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) (99). The interviewer was a psychiatrist trained in the use of SCAN and certified at the WHO centre in Copenhagen. In order to examine the comorbidity between disorders, the hierarchical rule for somatoform, anxiety, obsessive-compulsive and depressive disorders was not applied. In the

interview we obtained socio-demographic and phenomenological data, and a number of assessment instruments measuring different aspects of psychopathology and personality were applied, among them a newly developed questionnaire for hypochondriasis and health anxiety, the Health Anxiety Inventory (HAI). HAI is an 18-item, self-report questionnaire that has shown to be a valid and reliable measure of health anxiety with a good internal consistency (151). To measure more general aspects of anxiety we used the Hamilton Anxiety Rating Scale (HAM-A) (171) and the Beck Anxiety Inventory (BAI) (221). To measure depression we used the Hamilton Rating Scale for Depression (HAM-D) (172) and the Beck Depression Inventory (BDI) (222). Global aspects of psychopathology were assed using SCL-90R (223) and the relationship between personality and psychopathology was measured by the use of MCMI-III (204), which was developed to assess clinical personality styles both dimensionally and categorically, and personality disorders in accordance with both Millon's theory of personality and psychopathology and DSM-IV (1). The patients' level of function was assessed using the Global Assessment Scale (GAS) (205). A team of experienced psychiatrists and psychologists reviewed two independent Danish translations of HAI. A professional translator retranslated the final version into English, which afterwards was accepted by the author. Validated versions of the remaining scales were available in Danish.

Table 1. Consecutive patients referred to a liaison psychiatric clinic

Diagnosis	Patients (N=415)	(%)
Depressive episode	92	22
Hypochondriasis	80	19
Somatoform disorder	77	19
Somatoform pain disorder	47	11
Anxiety disorders	39	9
Adjustment disorders	19	5
Dissociative disorders	17	5
Recurrent depression	9	2
Schizophrenia or schizotypical disorder	7	2
Personality Disorders	5	1
Delusional disorder	4	1
Organic mental disorder	4	1
Unspecified	15	4

Statistical method

The relationship between health anxiety and other continuous variables were examined by means of the Pearson correlation coefficients. The relationship between personality disorder and psychopathological measures was examined by a one-way ANOVA. Regression analyses and

multiple regression analyses were conducted to determine which psychopathological variables were most predictive of the continuous measure health anxiety inventory (HAI).

Results

The diagnoses of all 415 consecutively referred patients are shown in table 1. A total of 27% had a mood disorder, 51% a dissociative or somatoform disorder, and 9 % of the patients had an anxiety disorder. Three patients were diagnosed with schizophrenia; four patients with a schizotypical

Table 2: Demographic, clinical and social data of 80 patients fulfilling the diagnostic criteria for hypochondrias

Gender, %	
Male	39
Female	61
Age, years (SD)	37(11)
Marital Status, %	
Cohabitation/married	63
Divorced	22
Always single	15
Educational level, %	
Low	26
Intermediate	54
High	20
Working situation, %	
Employment	56
Student	18
Unemployment	4
On sickness benefit	8
Social security benefit	3
Disability benefit	11
Source of referral %	
GP	64
Psychiatric referral	15
Other	21
Previous psychiatric treatment, %	
None	63
Psychopharmacological treatment, %	
None	73
Antidepressant	21
Sedative	4
Antipsychotic medication	2
GAS (SD)	64(8.0)

disorder according to ICD-10, and three patients had a delusional disorder. Nineteen percent of the patients (n=80) fulfilled the clincal diagnosis for hypochondriasis according to ICD-10. Sociodemografic and clinical data for the 80 patients fulfilling the diagnostic criteria for hypochondriasis show (table 2) a reasonably high level of functioning with a mean GAS score of 64. A total of 63 % were married or in cohabitation, 75% had an intermediate or higher education, and 74% were employed or students. The patients had little previous experience with psychiatric

treatment and only few patients (27%) were in psychopharmacological treatment in spite of a high level of psychiatric comorbidity.

Hypochondriasis and psychiatric comorbidity

Table 3. Comorbide mental disorders in 80 patients diagnosed with hypochondriasis

Comorbide diagnoses	%
Generalised anxiety disorder	55
Undiff. somatoform disorder	54
Obsessive-compulsive disorder	31
Moderat depressive episode	29
Panic disorder	24
Somatization disorder	18

The comorbid diagnoses by use of SCAN in a non-hierarchical way show (table 3) a considerable comorbidity with anxiety, mood disorder, non-hypochondrial somatoform disorders and obsessive-compulsive disorder. This comorbidity is reflected in continuous measures for anxiety and depression (table 4).

Table 4. Mean score for psychopathological measures for patients fulfilling the diagnostic criteria for hypochondriasis

Measures	N*	Mean score (SD)
HAI (Health Anxiety Inventory)	80	27.7 (5.2)
BAI (Beck Anxiety Inventory)	79	20.8 (9.3)
BDI (Beck Depression Inventory)	78	14.8 (7.5)
HAM-D (Hamilton Rating Scale for Depression)	80	12.9 (4.8)
HAM-A (Hamilton Anxiety Rating Scale)	80	18.4 (7.0)

*Not all patients completed the questionnaires. SD, standard deviation.

The patients have a severe degree of anxiety according to BAI and HAM-A, but only a mild or moderate degree of depression according to BDI and HAM-D. The main score on SCL-90-R subscales and the high Global Severity Index (GSI) is consistent with the high degree of comorbidity in the structured diagnostic interview (table 5). Somatization and anxiety subscales

Table 5. Mean scores on SCL subscales ad well as Global Severity Index (GSI) for patients fulfilling diagnostic criteria for hypochondriasis.

Subscales	N*	Mean score (SD)
Somatization	69	1.60 (0.70)
Obsession/compulsion	71	1.34 (0.64)
Interpersonal sensitivity	73	1.02 (0.71)
Depression	72	1.55 (0.66)
Anxiety	71	1.84 (0.74)
Hostility	73	0,81 (0.50)
Paranoia	72	1.04 (0.85)
Phobic anxiety	73	0.53 (0.54)
Psychotisism	72	0.89 (0.50)
Additional items	71	1.44 (0.68)
GSI	71	1.27 (0.47)

*Not all 80 patients completed the questionnaire.

showed a high mean score compared with other psychiatric patients (223), while other subscales such as obsession/compulsion, phobic anxiety, paranoia, and psychotisism are lower.

Hypochondriasis and personality traits

To examine the relationship between hypochondriasis, personality trait, and personality disorder we assessed the patients with the MCMI-III questionnaire. No mean score item reached a level that indicates trait or disorder status. The highest score of clinical personality patterns were depressive, dependent and histrionic personality patterns, while severe personality pathology had a low mean score. With a cut-off score of 75 indicating a personality trait or personality disorder, 63 (85%) of the patients had one or more clinical important personality traits, while 10 (14%) patients had one or more personality disorders. There is a considerable comorbidity or concurrence between different personality traits and personality disorders, and only 11 (15%) patients had no presence of clinically important personality traits or disorder according to MCMI-III, while 37 patients (50 %) had two or more clinically significant personality traits or disorders.

It was possible to divide the patients into 3 groups with a growing severity of personality disorder: A group with no clinical significant personality traits, a group with significant personality traits, and a group with a personality disorder according to the definition and criteria in MCMI-III. We conducted a one-way ANOVA with the 3 groups as the independent variable and different psychopathological measures and subscales from SCL-90-R as dependent variables (table 6). There is an increasing mean measure of psychopathological ratings for most measures with increasing

severity of personality disorder, but only three measures reach the level of statistical significance. There is a significant difference for BDI, but this difference is not replicated with HAM-D. No

Table 6 3-group analyses of mean psychopathological measures and SCL-90 subscales for different degree of personality disturbance in patients diagnosed with hypochondriasis.

Measures	No personality disorder (N=11)	Personality trait (N=53)	Personality disorder (n=10)	Grp. F(2,71)	P
Health anxiety inquiry (HAI)	25.6	28.0	30.2	1.98	0.146
Beck anxiety inventory (BAI)	21.2	20.2	24.1	0.71	0.498
Beck depression inventory (BDI)	10.2	15.4	18.4	3.83	0.026*
Hamilton Anxiety Rating Scale (HAM-A)	15.5	18.7	17.8	1.05	0.357
Hamilton Rating Scale for Depression (HAM-D)	11.0	13.2	12.7	1.05	0.357
Somatization	1.64	1.60	1.40	0.40	0.675
OCD	0.94	1.43	1.47	2.42	0.097
Sensitivity	0.60	1.03	1.59	5.15	0.008**
Hostility	0.70	0.80	1.02	1.09	0.343
Psychotisism	0.56	0.91	1.19	3.73	0.029*
Anxiety	1.68	1.85	2.02	0.49	0.623
Depression	1.22	1.62	1.85	2.51	0.089
Phobic anxiety	0.73	1.13	0.90	0.99	0.378
Paranoia	0.33	0.55	0.68	1.00	0.374
Global Severity Index (GSI)	1.06	1.30	1.42	1.41	0.252

difference was found for HAI, HAM-A, and BAI, indicating that the concept of personality disorder represented in MCMI-III has no significant impact on measures for anxiety, depression or health anxiety for patients fulfilling the diagnostic criteria for hypochondriasis. The overall measure for SCL-90-R, GSI, is not significantly different for the 3 groups, but the subscales interpersonal sensitivity and psychotisism reached the level of significance, indicating that different subscales of SCL-90-R correlate differently to clinical personality traits or disorders in this selected group of patients.

The relationship between psychopathology, personality, and hypochondriasis

A correlation matrix was generated for the sum score of HAI, BDI, and BAI and for HAI, HAM-A and HAM-D respectively (table 7). The Pearson correlation coefficients were significant for HAI and measures of anxiety and depression. A multiple regression analysis was performed to determine the proportion of variance of HAI accounted for by BAI and BDI. In the linear model HAI was the dependent variable and BDI and BAI predictor variables, the model predicted 6.5% (R^2) of the variance of HAI; this finding was not significant ($F(2,75) = 2.62$, $P=0.079$). A similar result is found with HAM-A and HAM-D as predictor variables and HAI as dependent variable. The linear

model predict 6.3% (R^2) of the variance of HAI and is not significant (F (2,77) = 2.61, P=0.080) different from random error.

Table 7 Intercorrelations (Pearson's product moment correlation) between HAI, BAI, and BDI

	HAI	P	BAI	P	BDI
HAI (Pearson Correlation)	1.00				
BAI	.213	.030	1.00		
BDI	.220	.027	.436	.000	1.00

Intercorrelations (Pearson's product moment correlation) between HAI, HAM-D, and HAM-A

	HAI	P	HAM-A	P	HAM-D
HAI (Pearson Correlation)	1.00				
HAM-A	.230	0.20	1.00		
HAM-D	.243	.015	.778	.000	1.00

HAI, Health Anxiety Inventory; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; HAM-D, Hamilton Rating Scale for Depression; HAM-A, Hamilton Anxiety Rating Scale

In a multiple regression model with HAI as the dependent variable and the subscales of SCL-90-R as predictor variables, this linear model can explain 13.1% (R^2) of the variance of HAI and is not significantly different from random error (F (10,56) = 0.845, P = 0.588). The sensitivity subscale had the highest correlation with HAI (r = 0.257, N=67, P=0.018) and in a linear regression model sensitivity as the only predictor variable can explain 6.6% of the variance of HAI, this model is significantly different from random error (F (1,65) = 4.593, P = 0.036).

A multiple regression model with HAI as the dependent variable and personality patterns of MCMI-III as predictor variable can explain 3.5% (adjusted R^2) of the variance in HAI. The model is not significant (F (14,59) = 1.190, P=0.307) different from random error, and no personality patterns are significant in this multiple regression model when controlled for simultaneously. A multiple regression model with HAI as dependent variable and the 3 personality disorders of MCMI-III as predictor variables explains 12.3% (R^2) of the variance, and this linear model is significantly different from random error (F (3,70) = 3.279, P = 0.026). In the multiple regression model with all 3 personality disorders only the borderline personality disorder is significant (t=2.356, P= 0.021) and the strongest predictor of HAI.

Discussion

In a secondary health care clinic, which specializes in patients with psychosomatic problems understood as comorbide mental disorders or medically unexplained complaints, including health anxiety, the most frequent disorders are depressive episodes, different kinds of somatoform disorders, and hypochondriasis. It has been discussed if the current diagnosis for hypochondriasis satisfies clinical and diagnostic validity requirements (103). In this examination one fifth of the patients (19%) fulfilled the diagnostic criteria for hypochondriasis and accepted a psychotherapeutic treatment specific for hypochondriasis. This indicates that the present diagnostic criteria serve a clinical purpose in this setting.

The strength of the study is the use standardised methods of psychopathological evaluation with the same rater, and that the setting is part of an ongoing clinic treating patients with medically unexplained complaint, where all consecutive patients were evaluated. This strengthens the external validity of the study. The weakness of the study is the cross-sectional design, the small number of patients, and selection bias.

By using SCAN in a non-hierarchical way, we found a considerable comorbidity between hypochondriasis, anxiety, mood disorders, somatoform disorders and obsessive-compulsive disorders. The question is whether this is the expression of comorbidity or if it is a conceptual problem. More than 50% of the patients fulfilling the criteria for hypochondriasis did also fulfil the criteria for generalized anxiety. It was not possible to differentiate between apprehension, motor tension and autonomic overactivity related to preoccupation with physical illness and preoccupation not related to physical illness. The non-specific distress of hypochondriasis can be represented as symptoms consistent with generalized anxiety, and it is not possible to differentiate sufficiently between these conditions with the current diagnostic criteria or the operationalisation in SCAN. Patients with hypochondriasis as their main problem may receive a diagnosis of general anxiety, which contain the risk of overlooking important aspects in treating the condition. There is varying comorbidity between hypochondriasis and general anxiety in different studies (78;224), but these findings have not led to any substantial conceptual discussion of the discrimination between hypochondriasis and general anxiety. In Barsky's (57) examination of psychiatric comorbidity in hypochondriasis using the definitions in DSM-III-R, he found that lifetime prevalence for generalized anxiety disorder was 71% and the current prevalence 24%. Starcevic (44) compared hypochondrial symptoms in patients with generalized anxiety disorder and panic disorder. Even though 30% with generalized anxiety had worries about health and illness, these patients were far

less likely to harbour hypochondrial fears and belief than patients with panic disorder. They experienced less somatic symptoms and their worries about health and disease did not have the intrusive quality and aspects of ruminations as patients with hypochondriasis.

A similar conceptual problem is seen with mood disorders, where 39% were diagnosed with moderate depressive episodes. Non-specific items such as fatigues, reduced concentration and disturbed sleep are not possible to differentiate from a general distress related to different psychopathological conditions. But the patients experienced depressed mood and loss of interest, which is often seen in relation with a worsening of the patients' hypochondriasis.

The causality of the psychopathological development is difficult to estimate with the current cross-sectional design. A specific treatment focused on aspects of hypochondriasis could illuminate this problem. In a recent paper Hiller (78) showed a high degree of comorbidity between 67 patients with hypochondriasis and major depression. This is consistent with our findings. But Hiller found much lower comorbidity with generalized anxiety and in the diagnostic procedure he must have been able to separate the worrying of hypochondriasis from the unspecific worrying of generalized anxiety.

We found a high comorbidity between hypochondriasis and somatization disorder. This is not consisting with Furer's finding (108) in which no patients who met the diagnostic criteria for hypochondriasis met the criteria for a somatization disorder. Fink (103) found a similar comorbidity between hypochondriasis and somatization as our study. In Furer's study the setting was an outpatient anxiety disorder clinic, while in the Fink study the setting was primary care. This indicates that different subgroups of hypochondrial patients are referred to different treatment facilities. The patients' in Furer's examinations did not seek treatment for their somatoform symptoms, and it was difficult to establish a significant impairment connected to the somatoform symptoms. Therefore they failed to meet the DSM-IV criteria for somatization disorder. Our patients are referred from general practice and general and specialized medical clinics. The patients seek treatment for somatic symptoms and may easier fulfil the criteria for somatization disorder. These patients therefore belong to a different subgroup than patients treated in an anxiety clinic. Different conclusions about the relationship between hypochondriasis and somatoform disorders have been made on the basis of these findings. Barsky (72) found the same degree of comorbidity between hypochondriasis and somatization disorder. He found a relatively weak correlation between somatization and hypochondrial symptoms consisting with our findings. According to Barsky (57) the distinction between hypochondriasis and somatization disorder is unclear, the

definitions arise from different conceptual traditions and could be different ways of describing the same clinical entity. Fink (103) proposes a new diagnosis with a mixture of items from somatoform disorder and hypochondriasis with the rumination of health and disease as the most important feature. This creates the risk of neglecting other features such as health anxiety, the relationship between bodily sensations and hypochondriasis, and the behaviour of patients' suffering from health anxiety.

Previous research suggested considerable overlap among the symptoms of panic disorder and hypochondriasis. Patients primarily diagnosed with hypochondriasis have a considerable comorbidity with panic disorder (43;107). This is consistent with our findings, where 24 % of patients with hypochondriasis fulfilled the criteria for panic disorder. The emotional, cognitive and behavioural reactions of panic patients are similar to those described for hypochondriacal disorder. The major difference is the episodic nature of the symptoms in panic disorder versus persisting complaints in hypochondriasis. Hypochondriasis and panic disorder are associated with distorted cognitive assessment of health status, which may be accompanied by a state of higher alertness and awareness of physiological processes (50). Patients with panic attacks tend to misinterpret autonomic symptoms which provide an obvious feedback mechanism in which anxiety may rapidly escalate. In hypochondrial problems, the symptoms misinterpreted a not likely to be subject to such direct amplification and the feedback mechanism is more behavioural and long term by maintaining focus on parts of the body with repeated checking or physical manipulation. The panic patient believes that the catastrophe is happening already, whereas the hypochondrial patient tends to believe that the symptoms indicate a more insidious course (83). The conclusion of this conceptual and empirical research is that some overlap between the two diagnoses exists, but the conditions are distinct and have discriminant validity (107). Our findings are consistent with this conclusion.

The significant correlation between HAI and the continuous measures of anxiety and depression was not confirmed in multiple regressions with HAI as the dependent variable. These models were not significantly different from random error, indicating that the high comorbidity with anxiety and depression was not substantially related to HAI. It has been shown (151) that HAI is able to discriminate patients with hypochondriasis from patients with anxiety disorders, and HAI has been seen as an independent factor in hypochondriasis, consistent with current cognitive theories. Our findings confirm the hypothesis of hypochondriasis as a distinct diagnostic entity. A similar pattern

is seen with SCL-90-R, in which a multiple regression model with HAI as the dependent variable and the subscales of SCL-90-R as simultaneous predictor variables is not significant.

The considerable psychopathology of the hypochondrial patients in this study had not to a substantial degree invalidated their occupational or family performances as reflected in a mean GAS score of 64. This finding is in contrast with earlier examinations of hypochondrial patients, where a high rate of psychiatric comorbidity have given a reduction in patients' level of social and occupational function in case-control designs (46;57). Our finding is probably related to selection bias. The patients who accepted to be referred to psychiatric or psychotherapeutic treatment must have had some psychological mindedness and motivation for change. The patients' were relatively young and with a high level of education. So it is reasonable to see these patients as part of a non-chronically spectrum of hypochondriasis, which might develop into a chronically course without appropriate treatment.

There is a considerable comorbidity with clinical important personality traits and personality disorders according to MCMI-III for this sample of hypochondrial patients. MCMI has been criticized of exhibiting high false-positive rates, and the results cannot be used to decide prevalence of personality disorder in hypochondrial patients. But the results indicate that personality trait or disorder has a substantial representation in this selected group of hypochondrial patients, and that more adjusted personality patterns are represented to a higher degree than personality disorders. The highest score of clinical personality patterns are depressive, dependent and histrionic personality patterns, while severe personality pathology has a low mean score. This finding indicates that the hypochondrial patients in this study belong to a group of patients with aspects of neuroticism or negative affectivity as important features. Barsky (57) compared a group of patients with DSM-R-III hypochondriasis with a random sample of outpatients, both groups taken from a general medical outpatients clinic. The patients with hypochondriasis had a 3 time higher personality disorder caseness measured with Diagnostic Interview Schedule and self-report questionnaires than the control group. Tyrer (137) found empirical support for a hypochondrial personality disorder which score high on scales measuring anxiety and dependence. In other studies (131) hypochondrial responses were related to neuroticism, low extraversion and negative affectivity (225) understood as a tendency to experience negative emotions and overreaction to stress. In this understanding hypochondriasis is correlated to or should be seen as personality disorder characterized with preoccupation and rigid belief about health. These results are consistent with our findings of a

considerable comorbidity or association with specific clinical important neurotic-like personality traits.

A multiple regression model with HAI as the dependent variable and personality patterns of MCMI-III as simultaneous predictor variables was not significant. This indicates that the substantial comorbidity with personality traits in this population of hypochondrial patients cannot explain the degree of health anxiety represented by HAI. If HAI can be seen as a representation of hypochondriasis, hypochondriasis is an independent in relation to the clinical personality patterns measured by MCMI-III and the relationship can be understood as an expression of comorbidity.

The result of a one-way ANOVA with three groups of patients with a growing severity of personality disorder as the independent variable and different psychopathological measures as dependent variables indicate, that the concept of personality disorder represented in MCMI-III has no significant impact on measures for anxiety, depression or health anxiety for patients fulfilling the diagnostic criteria for hypochondriasis. On the other hand the subscales of interpersonal sensitivity and psychotisism from SCL-90-R reach the level of statistical significance, meaning that different subscales of SCL-90-R correlate differently to clinical important personality traits or disorders in this selected group of patients. Interpersonal sensitivity consists of items belonging to different negative aspects of relating to other persons, and it has been proposed that hypochondrial patients with personality problems represent a subgroup with more mistrust of the interpersonal setting, where the mistrust is directed towards others (161). Noyes (52) found a high correlation with measures of unusual perceptions, feature seen among the schizoid personality disorder. This correlation point to a relationship with depersonalization (7;146) and may capture metaphysical and superstitious beliefs held by some hypochondrial persons (226). In our sample a significant higher score of the subscale psychotisism from SCL-90-R is found in the group with personality disorder compared to the group with no personality disorder. Psychotisism represents a continuous dimension of human experience from mild interpersonal alienation with an isolated, schizoid lifestyle to evidence of psychosis.

A multiple regression model with HAI as dependent variable and the 3 personality disorders as simultaneous predictor variables is significant and borderline personality disorder is significantly different from random error in this model. The borderline personality disorder expressed in MCMI-III predict to some degree health anxiety in this population of hypochondrial patients. Only 6 patients (7.5%) had a borderline personality disorder according to MCMI-III but some patterns consistent with the borderline construct had a more pervasive influence possibly related to an

unstable self-image and not the instability of interpersonal relationship, affects and impulsivity, which is not found in this population. The relationship between borderline personality disorder and health anxiety has not previously been described, and the generalizability of this finding is questionable. This is a highly selected group of patients and the measure of personality disorder was a questionnaire which validity has been discussed.

Our findings indicate that the current diagnostic criteria for hypochondriasis is clinically useful in a secondary health care clinic specialized in psychosomatic conditions. For 80 patients fulfilling the diagnostic criteria for hypochondriasis a considerable comorbidity with other mental disorders were found, but our measure of hypochondriasis was independent of these disorders suggesting that hypochondriasis is a primary condition and a distinct diagnostic entity. There was a high comorbidity with neurotic-like personality traits according to the findings of MCMI-III, and an association was found between severe personality traits and subscales in SCL-90-R, indicating subgroups of hypochondrial patients with interpersonal problems and eccentric experiences and personality disorders. Future trials should examine different psychotherapeutic strategies in relationship to these findings.

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Outcome of cognitive-behavioural treatment for health anxiety (hypochondriasis) in a routine clinical setting

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Running head: Treatment of health anxiety in clinical practice

Abstract

It has now been established in several randomized controlled trials that specialist cognitive-behavioural therapy (CBT) is an effective treatment for severe and persistent health anxiety (diagnostically, "Hypochondriasis"). It has not yet been established whether or not such results will generalize from academic research centers to routine clinical settings. The present study was designed to address the issue of generalization by evaluating the outcome of a consecutive series of patients meeting diagnostic criteria for hypochondriasis treated using CBT in a non-academic clinic in Copenhagen, Denmark. The delivery of the treatment was adapted to fit with the practice of the clinic, so that the later components of therapy were delivered in a group therapy setting. Therapists participated in a brief training course, which was subsequently supplemented by expert clinical and peer supervision. Patients received the same amount of treatment used in previous clinical trials. Results indicate that the degree of improvement obtained in this study was significant and compared well with those obtained in the previous trials. These results support the use of dissemination of new treatments using a specialist training model.

1. Introduction

Severe and persistent health anxiety ("hypochondriasis") not only causes great suffering for the patient and those around them but is also costly in terms of health care provision. Patients very frequently seek reassurance from doctors and undergo many unnecessary and expensive medical investigations (Barsky & Klerman, 1983; Barsky, Wyshak, & Klerman, 1986). Although the incidence and prevalence of diagnostically-defined hypochondriasis is not known, it is clear that it is common in general medical (Barsky, Wyshak, Klerman, & Latham, 1990) and general practice clinics (Gureje, Ustun, & Simon, 1997). Until the development of cognitive-behavioural approaches, there was little evidence that any treatment helped patients suffering from severe and persistent health anxiety (Warwick & Salkovskis, 1990).

Cognitive-behavioural theories of health anxiety developed from the observation that, for some patients, medical investigation and reassurance had the effect of increasing health anxiety and the need for yet further reassurance (Salkovskis & Warwick, 1986). This work developed a not only into a cognitive behavioural theory of health anxiety derived from the principles of Beck's cognitive theory of anxiety (Beck 1976, 1985), but also a focused treatment approach (Salkovskis, Warwick and Deale, 2003).

The cognitive-behavioural theory described by Warwick and Salkovskis (1990) proposes that health anxiety arises from the misinterpretation of a range of stimuli which patients believe to indicate that they are seriously ill (or more seriously ill than they actually are). The focus of misinterpretations includes bodily variations (including but not confined to bodily sensations), information from health professionals and information that patients obtain from the media, internet and related sources. The misinterpretations persist as a result of psychological maintaining factors. These include selective attention (which results in a "confirmatory bias" with respect to stimuli perceived as being relevant to health); safety seeking behaviours (including, but not confined to, checking and reassurance seeking); changes in mood (including both anxiety and depression); and the magnification of feared bodily sensations by the anxiety arising from their misinterpretation in ways similar to that seen in panic disorder (Clark 1986). The similarities between health anxiety and both panic and obsessional problems (Salkovskis, 1996) have been noted. The main difference between panic and hypochondriasis appears to relate to the time course of misinterpretations. Panic patients usually regard the occurrence of bodily sensations as indicating that the catastrophe they fear is happening or about to happen, whilst hypochondriacal patients are more likely to believe that their illness is gradually developing, and will become serious over a longer period of time

(Salkovskis and Clark, 1993). This delayed time course of the feared catastrophe beliefs is also characteristic of obsessive-compulsive disorder, and has crucial implications for treatment (Salkovskis, 1996). In particular, it means that treatment strategies involving disconfirmation of the person's negative interpretations are unlikely to be successful. Treatment instead requires the individualized development of a credible account of how health anxiety and related maintaining processes can generate the problems the patient is experiencing, followed by explicit tests of this alternative, less threatening account of the person's problems.

This theoretical approach and the somatosensory amplification theory of Barsky have thus led to the development of well-defined cognitive-behavioural treatments for health anxiety. This type of treatment has now been shown to be effective in several randomised control trials. Cognitive-behavioural treatment has been found to be superior not only to treatment as usual and waiting list comparisons (Warwick, Clark, Cobb, & Salkovskis, 1996) but also to an equally credible psychological treatment with the same amount of therapist input (Clark et al., 1998). This latter finding indicates that the effect of cognitive-behavioural therapy is unlikely to be due to non-specific factors.

As with many recently developed evidence based treatments, evidence for efficacy has been obtained in research centres. The extent to which these treatments generalise to routine clinical settings is a crucial issue if they are to be disseminated more widely. The last few years has seen a growing recognition of the importance of generalisation and dissemination studies (Barlow, Levitt, & Bufka, 1999). There are different ways in which effective dissemination could be achieved. Traditionally, it has been suggested that there is a need to increase the number of trained psychotherapists, which is not only expensive but also slow and likely to achieve only minimal extra therapy capacity. The alternative (as investigated here) is both more rapid and cost effective, and involves a focussed specialist training approach in which the specifics of treatment for a particular problem are the subject of brief training and supervision.

Recently, good evidence has been found for this approach to dissemination of brief cognitive behavioural treatment for PTSD (Gillespie et al, 2002). In that study, brief training and subsequent supervision was offered to therapists working in the context of the British National Health Service. Treatment effects were comparable to those previously obtained in treatment trials. The study reported here seeks to establish the effectiveness of CBT for the treatment of severe and persistent health anxiety in a non-academic clinic setting.

2. Method

2.1 Overview

Patients who met the diagnostic criteria for hypochondriasis were recruited and offered treatment in a non-research treatment centre. They were treated by 6 therapists (all qualified as clinical psychologists, 3 with more than 10 years practice), none of whom had previously specialized in the treatment of hypochondriasis. Following training, these therapists saw patients as part of their normal busy caseload, receiving regular normal supervision within the clinic and specific specialist supervision every three months. Outcome measures and timings corresponded with those used in previous research trials.

2.2 Participants

The research group distributed a leaflet describing the project to General Practitioners in the greater Copenhagen area; this was intended to be offered to potential participants. Meetings were held to inform GPs about the project. In addition the trial was publicized in major newspapers. Forty-five referrals were received, and those which appeared to be suitable were assessed and diagnosed by experienced psychiatrists using ICD 10 criteria.

The selection criteria applied to referrals were: ICD 10 diagnosis of hypochondriasis (F45.2); in the age range 18 – 65; Danish as mother tongue and a score of more than 17 on the Danish translation of the short Health anxiety Inventory (main section). Patients were excluded if a medical illness explained their condition, if they had received CBT treatment previously, had a history of a psychotic disorder or currently had diagnosis of substance dependence. Patients were included if they participated in more than one treatment session; later dropout resulted in inclusion in "intention to treat" analyses.

Eighteen consecutive referrals meeting these criteria were recruited; one completed only one session of treatment and a second was withdrawn from the study when a psychotic condition was detected during therapy, giving a final study sample size of 16. Nine participants were referred through their GP, one through a psychiatric department in a general hospital, two from psychological clinics, and four self-referred. Eight had previous psychiatric treatment. The median age was 34, with a range of 21–52 years. Four of the clients were male and 12 were female. All were Danish citizens, of Caucasian origin. Fourteen were living with a long-term partner, 2 were single. Seven had children, nine did not. Four participants had university education, 8 had attended further education course (eg teacher training), and 3 were university students. One

had left school after primary school. Nine participants were in employment, 1 was self employed, 1 had a sheltered job, 3 were currently students, one was on maternity leave and 1 was a homemaker.

One patient was on a low dose of amitriptyline, one patient received paroxetine whilst a third patient received chlorprothixene at a low dose (as a tranquiliser rather than as an antipsychotic). Neither patient changed their medication during the period of treatment. Five participants had worries about having or developing cancer. Five had worries about degenerative illnesses of the nervous system and one participant was afraid of having a stroke. Five patients worries tended to change focus across different serious illnesses. Fifteen of the participants were not diagnosed with any known physical illness, while one was diabetic. One patient had a grandfather who was diagnosed with Huntington's Chorea, but had not herself been tested because of her worries.

According to the baseline diagnostic interview (WHO 1994), 6 participants had comorbid panic disorder (F41.0), 3 had OCD (F42.2), 3 had moderate depression (F32.1), and four had no comorbid psychiatric conditions. Six of the participants gave a history of health anxiety of greater than 15 years; in six participants it had been present for more than two years and 4 had had worries for more than six months and less than two years. Nine of the participants were not able to describe a clearly defined triggering event, 2 referred to stressful period of life leading up to the onset, and 5 participants were able to give a clear precipitating event that triggered the current episode: a stillborn child, a severe allergic reaction, the sudden death of a close family member, the sudden death of a relative to a close friend, and having had appendicitis.

2.3 Measures

The main outcome measures were Danish translations of standardized scales. These were the Short Health Anxiety Inventory (Salkovskis, Rimes, Warwick, & Clark, 2002) and the Beck Anxiety and Depression Inventories (Beck, Epstein, Brown, & Steer, 1988; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961).

2.5 Treatment

Setting and therapists

The treatment took place at the Cognitive Psychology Center (KPC), a private clinic that provides CBT based psychotherapy on an outpatient basis mainly for anxiety disorders. Six certified clinical psychologists were involved in delivering treatment; therapists' experience ranged from 2-25 years.

All had previous training in CBT, although none had previously specialized in the treatment of hypochondriasis or worked as research therapists.

Developing an integrated individualised and group treatment

It was intended to offer treatment of the type developed by Salkovskis and colleagues (Salkovskis & Warwick, 2001). However, the clinic usually offers group treatment programs for anxiety disorders such as panic disorder, agoraphobia, social phobia and OCD. On the basis of this experience it was therefore assumed that the treatment of health anxiety would be of similar nature and the treatment was to be modified to be implemented on a group basis.

Training in cognitive treatment of severe and persistent health anxiety was given in workshop format by PS, with follow up supervision groups on a peer basis and from PS. This process was used to modify the main treatment strategies for the group format. However, it rapidly became apparent that there were problems with implementing treatment in a group format. Group work with health anxiety patients seemed to differ from the treatment of other anxiety disorders in terms of the extent to which anxiety was increased by interactions within the group. The atmosphere in the initial group sessions was characterised by high levels of arousal and fear related to the cross comparison of symptoms and discussion about their causes. For example, a group member mentioned that she was afraid of having multiple sclerosis whereupon another group member promptly reacted to this statement by telling the therapist that by permitting a group member to mention multiple sclerosis, she was exposing the other group members to grave danger. The therapists found that much of their time was taken up with trying to deal with sensitivities and anxiety aroused by the discussion between patients. The therapists found that they were continuously attempting to control and reduce the level of anxiety and found it difficult to focus on treatment strategies.

In CBT group treatment of other anxiety disorders in this clinic, the specific cognitive model (involving key vicious circles) is initially presented as part of psychoeducational process, so that therapist and patients can jointly refer to this as therapy proceeds. The worries of the group members make up the foundation upon which the basic conceptualisation is elaborated and validated, and the interrelationship between symptoms, their meaning and emotional and behavioural reactions of the patients is then clarified. Usually this aspect of treatment is reassuring and de-arousing, as the patients are involved in clarifying the way their worries work. However, in the health anxiety group it appears that the other group member's worries become the focus of

mutually increased misinterpretation, worry and fear. These interactions also result in spiralling counter-productive attempts to be reassured which actually appeared to increase anxiety and the need for further reassurance.

The idea of initial group treatment was therefore abandoned in the third group, and the therapy instead started with individual sessions, during which the patient was helped to reach an individualised formulation of their problems and begin to test this out through discussion and behavioural experiments. It rapidly became clear that the individual therapy gave the therapists a much better chance of pacing the therapeutic progress and need of the individual participant.

An element of "Mindfulness" (Segal, Williams and Teasdale, 2002) was also included in the program as part of the group treatment, adapted for use with these patients. It was the experience of the participants that when they had come to accept the fact that a major part of their health anxiety was tied to their worries, thoughts, ideas as well as experience of their noticing physical changes in their bodies and they had abandoned their usual safety seeking behaviour they needed concrete strategies to be able to direct and control their attention and thereby control the direction of their thoughts. The main focus in the final stages of therapy was therefore directed at this and mindfulness proved to be a useful supplement to other commonly used CBT strategies directed towards attention control.

Content of treatment

Treatment was closely modelled on that used in the previous randomised controlled trial of cognitive-behavioural therapy (Clark et al., 1998), with the modifications described above to allow delivery of part of the treatment in a group format (see also Salkovskis, Warwick and Deale, 2003). Three booklets were integrated into the therapy process; these were given as homework between sessions at sessions 1-3. The booklets explain the nature of health anxiety in a gradual way, and suggest ways in which the person can apply the cognitive-behavioural strategies learned in the course of therapy. (These booklets were translated from the texts by Warwick and Salkovskis.)

Cognitive behavioural treatment involves helping the patient to develop and evaluate a personalised version of the cognitive model of health anxiety as an alternative, less threatening explanation of their problems (Salkovskis 1989; 1996). For example, the patient is helped to consider the possibility that their problems are better accounted for by the fear of cancer and their self-sustaining reactions to this fear rather than actually having cancer. This psychological explanation, which attempts to account for the patient's concerns must appear valid and credible. It

should not diverge from the patient's previous experience and with time should survive their future experiences, including behavioural experiments conducted as part of therapy. The patient is encouraged to discuss aspects of their problems that do not fit with the formulation. A crucial part of this re-attribution process is helping the patient to devise and carry out "behavioural experiments", in which they test out the alternative, cognitively based explanation in order to decide whether or not it is helpful to them. Overall, the health anxiety based explanation should lead the patient to re-interpret their innocuous symptoms and attribute them to a less threatening cause. It will also demonstrate that behaviours such as bodily checking and other maintaining factors serve to make their problems worse and should be terminated. Where appropriate, the patient is helped to re-engage in neglected areas of their life in order to help them disengage from their fears about health (for more detailed explanation of treatment, see (Salkovskis, 1989; Salkovskis, 1992; Salkovskis & Warwick, 1986; Salkovskis & Warwick, 2001; Salkovskis, Warwick and Deale 2003).

Procedure

Recruitment and assessment of patients was co-ordinated through Liaison Psychiatry Unit at Bispebjerg University Hospital, Copenhagen. Patients referred were offered assessment appointments at that centre, and suitability for inclusion determined at that point. Patients were then offered appointments at KPC; all patients offered appointments attended. Questionnaires (HAI, BDI, BAI) were administered to the participants at the initial session and at end of treatment. Follow up for group 1 was at 12 months post-treatment; for group 2 and 3 this was done at six months post-treatment.

Participation in both group and individual treatment sessions was better than 80%. Group one had one leader and one observer present. Group two and three had one leader. For reasons described above, the CBT treatment was offered in two different ways. Group 1 and 2 had similar treatment procedure. Group 3's procedure differed from these. Group 1 and 2 had 15 group sessions each with a duration of three hours including breaks of 15 min. per hour, that is 45 sessions of 45 min. duration. In addition Group 1 and 2 had 8 individual therapy sessions of one hour each. Group 1 and 2 initially received four group sessions over a period of two weeks. The remaining sessions followed on a weekly basis. After the first two weeks of group treatment the individual treatment was started and continued parallel to each other. Group 3 had 6 individual sessions. The first two sessions were 1½ hours and the remaining individual sessions lasted one hour. After the 6 sessions the CBT group treatment was initiated with 10 group sessions on a weekly basis. The CBT group

treatment had two foci, the first 6 sessions dealt exclusively with health anxiety and the remaining 4 sessions had focus on mindfulness training.

3. Results

3.1 Overview

Sixteen patients were treated in the study. On the main dependent variable (Health Anxiety Inventory), a significant reduction was found, and this reduction was sustained at follow up. Significant and sustained reductions were also noted in measures of clinical anxiety and depression.

3.2 Outcome measures

Health Anxiety Inventory: Repeated measures analysis of variance (treatment, post-treatment and follow up) corrected for serial dependence using the Greenhouse-Geisser procedure was carried out; this analysis indicated a significant overall effect of repeats ($F_{[2, 18.1]}$=74.0, $p<0.0001$). Bonferroni corrected paired t-tests indicated that this effect was accounted for by a difference between pre-treatment and post-treatment scores (t_p=9.5, $p<0.0001$), with the post-treatment to follow-up difference not being significant (t_p=1.1, $p>0.3$). Results are shown in Figure 1. The range of changes observed are shown in Figure 2, with the change expressed as a percentage of the initial score. This indicates that 9 out of 16 patients reduced health anxiety by more than 50%, and that the minimum decrease in health anxiety was 30%.

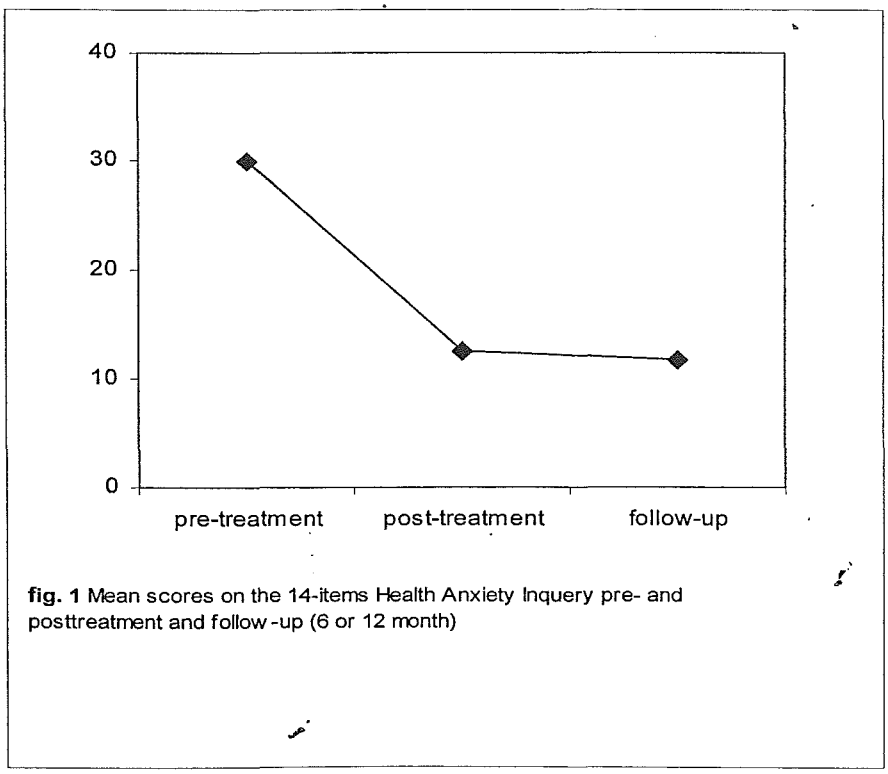

fig. 1 Mean scores on the 14-items Health Anxiety Inquery pre- and posttreatment and follow-up (6 or 12 month)

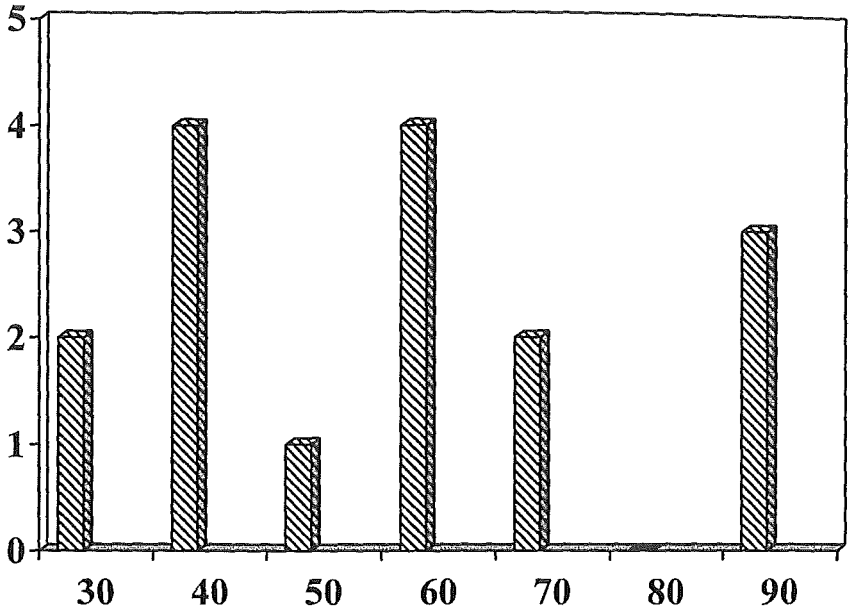

Figure 2: Percentage change in Health Anxiety Inventory at the end of treatment

Beck Depression and Anxiety Inventories: There were significant effects of repeats for both anxiety ($F_{[2, 18.1]}$=74.0, p<0.0001) and depression ($F_{[2,27]}$=9.9, p<0.001). Bonferroni corrected paired t-tests indicated that this effect was accounted for by a difference between pre-treatment and post-treatment scores (for the BAI: t_p=2.9, p<0.01; for the BDI: t_p=2.8, p<0.01); for the BAI there was a significant change from post-treatment to follow up (t_p=2.6, p<0.02); this comparison was not significant for the BDI when Bonferroni corrected (t_p=2.2, p=0.045). These results are shown in Figure 3.

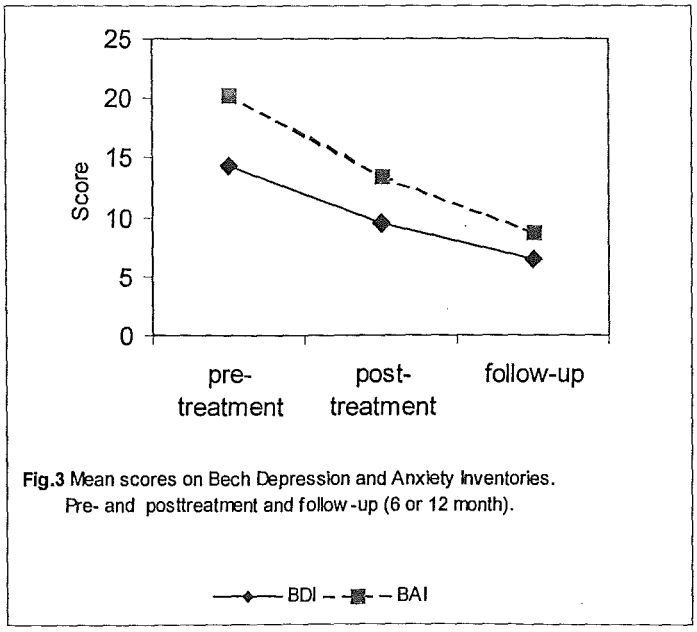

Fig.3 Mean scores on Bech Depression and Anxiety Inventories. Pre- and posttreatment and follow-up (6 or 12 month).

4. Discussion

This study provided preliminary evidence that cognitive-behavioural treatment can be delivered effectively in a normal clinical setting on the basis of relatively small amounts of specific training and supervision. The size of changes observed at the end of treatment was substantial and likely to be clinically significant (in the range 30-90% improvement). The changes were also fully maintained at follow up. Improvement was also seen in more generic measures of depression and anxiety, and there was some evidence that further improvement in anxiety occurred during the follow up period.

This study is important because it is the first to show the apparently successful transfer of CBT for severe and persistent health anxiety from an the academic research clinic to a routine clinical setting. There are, however, some factors which limit the conclusions which can be drawn. By definition, this generalization study was carried out in a center which did not previously specialize in the treatment of Hypochondriasis, which meant that patients had to be actively recruited from other sources, including the Liaison Psychiatry Unit of the Hospital and direct advertisement. This may have resulted in recruitment of an atypical sample; the demographic variables are consistent with this. For example, the sample includes an unusually high proportion of patients with high educational attainment and in employment. However, there was evidence of considerable comorbidity, and the clinical impression was that there was a relatively high proportion of "complex cases". Future dissemination research in this field needs to focus primarily on a setting in which patients with severe and persistent Hypochondriasis are already being routinely referred. An important feature of this study was the need to adapt treatment strategies to fit the normal working practice of the clinic; that is, the use of group treatment format. The present study indicated that this adaptation is not necessarily a straightforward enterprise.

The outcome of the present study is similar to that obtained in previous treatment trials of health anxiety (Avia, Ruiz, Olivares, Crespo, & et al., 1996; Clark et al., 1998; Warwick, D. M. Clark, A. M. Cobb, & P. M. Salkovskis, 1996), and the degree of change in the main outcome measure is very similar to that previously noted (Salkovskis et al., 2002).

There is now evidence that cognitive-behavioural treatments developed in academic research settings generalize well to "normal" clinical settings; this has previously been shown in CBT for Post Traumatic Stress Disorder (Gillespie et al, 2002). The present finding is the latest demonstration of generalization of specific CBT treatments from the settings in which they were developed; having demonstrated this, there are a range of further questions which need to be

addressed. For example, we do not know how the pre-existing levels of expertise of therapists trained in the specific treatment affect results. The optimum amount (and modality) of training and supervision is not known. If CBT is to be effectively disseminated, then it is important that effective training be carried out on a "snowball" basis, where those trained in effective treatment then go on to train others, and so on. Subsequent studies need to evaluate the effectiveness of second and third generation trainers. In dissemination studies conducted so far, including the present one, training and supervision has been provided by clinicians who have been closely involved with the initial development of the treatment itself.

The study described here has been used to develop a more comprehensive randomized controlled trial of CBT compared to generic counseling and waitlist. The results of that larger study will allow firmer conclusions to be drawn not only about the effectiveness of the treatment, but also the extent to which the treatment effects are due to specific and non-specific factors. It has previously been shown (Clark et al., 1998) that CBT for health anxiety is more effective than a stress-management based package; however, that treatment included the engagement components of CBT delivered by expert CBT trained therapists. The RCT presently under way has avoided this problem.

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A randomized clinical trial of cognitive behavioural therapy versus short-term psychodynamic psychotherapy versus no intervention for patients with hypochondriasis

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Background: Severe and persistent health anxiety (hypochondriasis) is common in the clinic and the community. Cognitive behavioural therapy (CBT) has been found to be effective in previous trials.

Aims: To compare both CBT and Short-term Psychodynamic Psychotherapy (SPP) to a waiting list control and to each other.

Methods: Eighty patients were randomised to CBT, SPP, or waitlist and were assessed on measures of health anxiety and general psychopathology before and after a six month treatment period. Waitlist patients were subsequently offered one of the two active treatments on the basis of re-randomisation, and assessed on the same measures post treatment. All patients were assessed at six and twelve month follow up.

Results: Those patients who received CBT did significantly better on all measures compared with the waitlist control group and on anxiety measures compared with SPP. The SPP group did not significantly differ compared with the waitlist group regarding any of the outcome measures. Similar differences were observed between CBT and SSP during follow up, although some of the significant differences between groups were lost at follow up.

Conclusion: Cognitive behavioural treatment is beneficial in the treatment of hypochondriasis and these effects are not due to non-specific factors such as participant's expectations and the relation between the therapist and the participant.

Introduction

Hypochondriasis is common (227) and costly (228). Cognitive behavioural therapy (CBT) is more effective than an equally credible stress management package. The effects are thus not due to non-specific factors (181), but the comparison treatment did not reflect current clinical practice and it incorporated CBT elements to improve engagement. CBT tends to be relatively costly, requiring 16 hours of one-to-one therapy. A briefer (six hour) treatment has been found to be effective(69), but possibly at the cost of smaller effect sizes. In a previous study examining the generalizability (see discussion) of treatment to a non-research setting, Wattar and colleagues (220) combined individualised and group CBT; time required was reduced without apparently compromising effect size. Aims of the present randomised trial were to evaluate the modified CBT programme compared with waitlist control and a wellconducted short term psychodynamic therapy.

Methods

Settings and participants

All patients consecutively referred to a liaison psychiatry unit in Copenhagen between August 2001 and January 2003 were evaluated for inclusion in the trial. Inclusion criteria were: 1) age between 18 and 65 years, 2) fluency in the Danish language, 3) meeting ICD-10 research criteria for hypochondriasis, (4) experiencing significant levels of health anxiety, as indicated by a score of more than 17 on the health anxiety inventory (HAI); this was intended to exclude patient with body dysmorphic disorder (151). Exclusion criteria were: 1) presence of a current psychotic condition, 2) current substance dependence, 3) the presence of another medical or psychiatric condition requiring immediate treatment, 4) psychopharmacological treatment initiated or increased during the 6 weeks prior to the assessment, and 5) previous adequate cognitive behavioural treatment.

The psychiatric interviews included the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) (207). The interviewer was an experienced psychiatrist trained in the use of SCAN and certified at the WHO Centre in Copenhagen. In order to examine the prevalence of psychiatric comorbidity in the sample, the hierarchical rules for somatoform, anxiety, obsessive-compulsive, and depressive disorders were not applied for diagnoses identified in the interview and reported in this paper.

As a specific treatment for hypochondriasis had not previously been available in Denmark and there therefore were concerns about referral rates, general information about the trial was published on radio and in newspapers; public meetings were held, and a leaflet describing the trial was distributed to potential sources of referrals. However, in order to be assessed, potential participants were required to obtain a referral from their general practitioner, a medical consultant, or a psychiatrist. Advertisements were not used. A third of those referred had come forward as a result of the publicity; the remainders were routine referrals to the Liaison Psychiatry Unit. This unit employs 3 senior psychiatrists. The first author led the team which managed the trial and oversaw data collection; he also conducted the initial assessment interviews.

The short-term psychodynamic psychotherapy (SPP) was conducted at the Liaison Psychiatry Unit . The cognitive-behavioural treatment (CBT) was conducted at the Cognitive Psychology Centre (KPC): a private clinic, which provides CBT-based psychotherapy for outpatients, mainly anxiety disorders. The Danish healthcare system is free of charge, and the study was supported by grants from The Danish Ministry of Social Affairs.

Objectives

The aims of the trial were to offer treatment for patients suffering from hypochondriasis, where the main feature was disease conviction and anxiety about health. Specifically, 1) to evaluate the effectiveness of properly conducted SPP and CBT relative to a waitlist (of the same duration as the treatment) and 2) to compare directly the longterm effectiveness of the two treatments. It was predicted that CBT would reduce hypochondriacal symptoms significantly more than SPP, but that both would produce similar changes in general psychopathology. The trial was also designed to be a controlled evaluation of the generalisability of CBT in health anxiety to as setting outside the one in which it was developed, building upon a previous open trial (220).

Design

Patients with severe health anxiety fulfilling diagnostic criteria for hypochondriasis according to ICD-10 (95) and other trial criteria were randomised to CBT, SPP, or a waiting list of the same duration as treatment (six months). To increase the power of the between treatment comparison, waitlist patients who met diagnostic criteria at six months were then randomly allocated to one of the two active treatments. Patients were followed up for a year after the end of treatment.

1 (1:2:1)

Randomization

The randomised allocation sequence was computer-generated in permuted blocks of 8. The block sizes were concealed until the end of the trial. The randomization was stratified according to gender and level of depression at base line with a cut-off of 12 on the HAM-D. Concealment of allocation from the initial assessor was ensured by a procedures involving centralised telephone randomisation at the Copenhagen Trial Unit (CTU). Patients were assigned initially to one of the three groups/ After 6 months, those on waiting list were re-randomised to either CBT or SPP using the same concealment strategy, but here the computer generated block size was 4/ / and allocation ratio 1:1.

Interventions

Cognitive behavioural therapy: The cognitive behavioural treatment developed by Salkovskis and Warwick (82;83) was used, with adaptations for the specific setting. After eight individual sessions, patients joined a health anxiety group (ranging in size from 5 to 9); this group continued with the CBT program, with the addition of brief mindfulness training. Treatment was delivered by six experienced therapists, all qualified clinical psychologists and certified CBT therapists. The therapists had no previous experience of treating patients with hypochondriasis. They received an initial two day training workshop with a follow up session to consolidate their training. The treatment consisted of 16 sessions delivered in individual and group format and lasted up to 6 months. Professor Paul Salkovskis visited every few months to supervise treatment on a peer basis. The training and supervision was established and implemented in a previously reported pilot study (220), and finished before this trial started.

Details of the treatment are described elsewhere (229). In summary, the treatment involved therapists working with the patients to develop a "shared understanding" of their health anxiety. This required their identification of an idiosyncratic and personalised version of the cognitive model of health anxiety. Therapy emphasised that idea that this was a less threatening explanation of their problems (e.g. "Maybe it is not that my problem is that I have cancer, but my problem might be that I am unduly worried about and preoccupied with the idea that I might have cancer. These worries make me react in counter-productive ways, which make things worse and increase my worry"). As a crucial part of this reattribution process, the therapist not only engaged the patients in discussion of their problem but also helped them to design and carry out behavioural experiments to test the alternative cognitive account of their problems. When the patients started the group treatment (at

session 8), they were already able to derive and evaluate such an individualised alternative formulation of their problems, and were encouraged to support each other in testing their beliefs and making changes in the way in which they dealt with their health anxiety and other aspects of their life. Therapists sought to foster a collaborative therapeutic relationship, but did not make psychodynamic interpretations, clarification, or confrontations,

Short-term psychodynamic psychotherapy: Short-term psychodynamic psychotherapy (SPP) consisted of 16 weekly sessions. The therapist was an experienced psychiatrist trained in psychoanalytic psychotherapy with more than 7 years of training and supervision.
SPP is based on the understanding that the unconscious constitutes elements, which are not available for the conscious part of the psyche, but have pervasive influence on the contents of the consciousness and the behaviour of the individual. The key therapeutic features are the therapeutic relationship, the patients' interpersonal interactions, recognition of patterns or themes in the patients' functioning, and the development of an understanding of unconscious fantasies (197). There is no general model for hypochondriasis in a psychodynamic understanding and no specific interventions. The treatment emphasizes psychodynamic principles such as free associations and neutrality, and avoids explicit and active challenge of patients' belief about health and disease and in this way differentiates psychodynamic clarification and confrontation from cognitive discussion on health anxiety. In a limited degree interpretations are used. The therapeutic approach was based on the emphatic validation proposed by Anna Ornstein (200), where empathy is understood as a particular listening position trying to maintain a contact with the patients inner life, where acceptance and understanding is the focus of the therapeutic interventions and with no explicit challenge of the patients' belief about health and disease. We used the conceptualisations of psychodynamic interventions by Greenson (201) and partly Gabbard (202) to describe and define the psychodynamic interventions such as interpretations, confrontations/clarifications, confirmation, and encourage to work.

Waiting list: Patients in the waiting list group were asked to keep in touch with their GP, who had been informed of the trial in writing. In Denmark, no specific treatment was available for patients with hypochondriasis, so patients in the waiting list group could be regarded as receiving "treatment as usual". After 6 months, the patients on the waiting list were contacted and re-evaluated for inclusion and exclusion criteria and, if they still met criteria, re-randomised to CBT or SPP.

Table 1: Mean number of intervention per session (total number of sessions:24)

Interventions	**CBT (SD)**	**SPP (SD)**	**P** (Mann-Whitney)
Behavioural experiments	0.4 (0.9)	0	0.180
Direction of session activity	6.8 (4.9)	0	<0.0001
Providing a theoretical model	7.0 (5.6)	0	<0.0001
Cognitive discussions	30.0 (7.4)	0	0.001
Interpretation	0	1.0 (0.9)	0.025
Clarification/confrontation	0.4 (0.9)	10.6 (4.0)	0.002
Confirmation	0	3.7 (1.8)	0.002
Encourage to work	0.8 (1.3)	3.0 (1.8)	0.023
Reassurance	0.6 (1.3)	0.2 (0.4)	0.926
Contents of interventions (%)	**CBT (SD)**	**SPP (SD)**	**P** (Mann-Whitney)
Health and disease (%)	12 (11)	48 (19)	0.04
Interpersonal issues (%)	63 (22)	21 (11)	0.09
Non-interpersonal issues (%)	25 (19)	32 (20)	0.463

CBT, Cognitive behavioural Therapy; SPP, Short term psychodynamic psychotherapy; SD, Standard deviation.

Therapy distinctness: A modified Delphi technique (230) was used to reach a consensus on the extent of specific and non-specific components of treatment. This consensus was operationalized in a rating instrument identifying the presence or absence of components unique to and shared by the two models of treatment. A list of definitions is available from the authors. At random, we chose 24 audiotaped sessions 12 from CBT and 12 from SPP. The sessions were rated independently to evaluate the therapists' adherence to the treatment protocol. Assessors were blind to the origin of the tape. Each intervention was classified according to items seen in table 1, where the distribution of different types of intervention between CBT and SPP is shown. The statistically difference between the interventions of the two models was tested using a nonparametric independent samples Mann-Whitney U-test. Direction of session activity, providing a theoretical model in session, and cognitive discussions are components specific for CBT, which were only found in the CBT sessions, while interpretation, clarification/confrontation and confirmation were found only in SPP session. So it was possible to differentiate between psychodynamic interventions such as clarification/confrontation and cognitive discussions. The content of health and disease were represented significantly more in CBT sessions than SPP and interpersonal issues significantly in SPP, while both shared non-interpersonal issues. These findings are consistent with the definition of the different interventions, and it is found that CBT is focused on health anxiety related cognitions. The results clearly indicate good adherence to the specific treatment models in both types of psychotherapy.

Outcome measures

Two primary outcome measures were included to test for changes in specific and general psychopathology. The Health Anxiety Inventory (HAI) is an 18-item, self-report questionnaire with good reliability, validity and internal consistency (151); and the Hamilton Anxiety Rating Scale (HAM-A) (171). Secondary outcome measures included Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), Hamilton Rating Scale for Depression (HAM-D).

Experienced psychiatrists and psychologists reviewed two independent translations of the HAI, and a professional translator back-translated the final version into English; this back translation was accepted by the author. Validated versions in Danish of the remaining scales were available. Four experienced psychologists assessed the patients with HAM-A and HAM-D. They received training in using the instruments, and were blinded for the group assignment. The interrater agreement was measured. During 11 Hamilton training cases the mean level of agreement was 90.8% (5.3%[SD]).

Assessor blinding

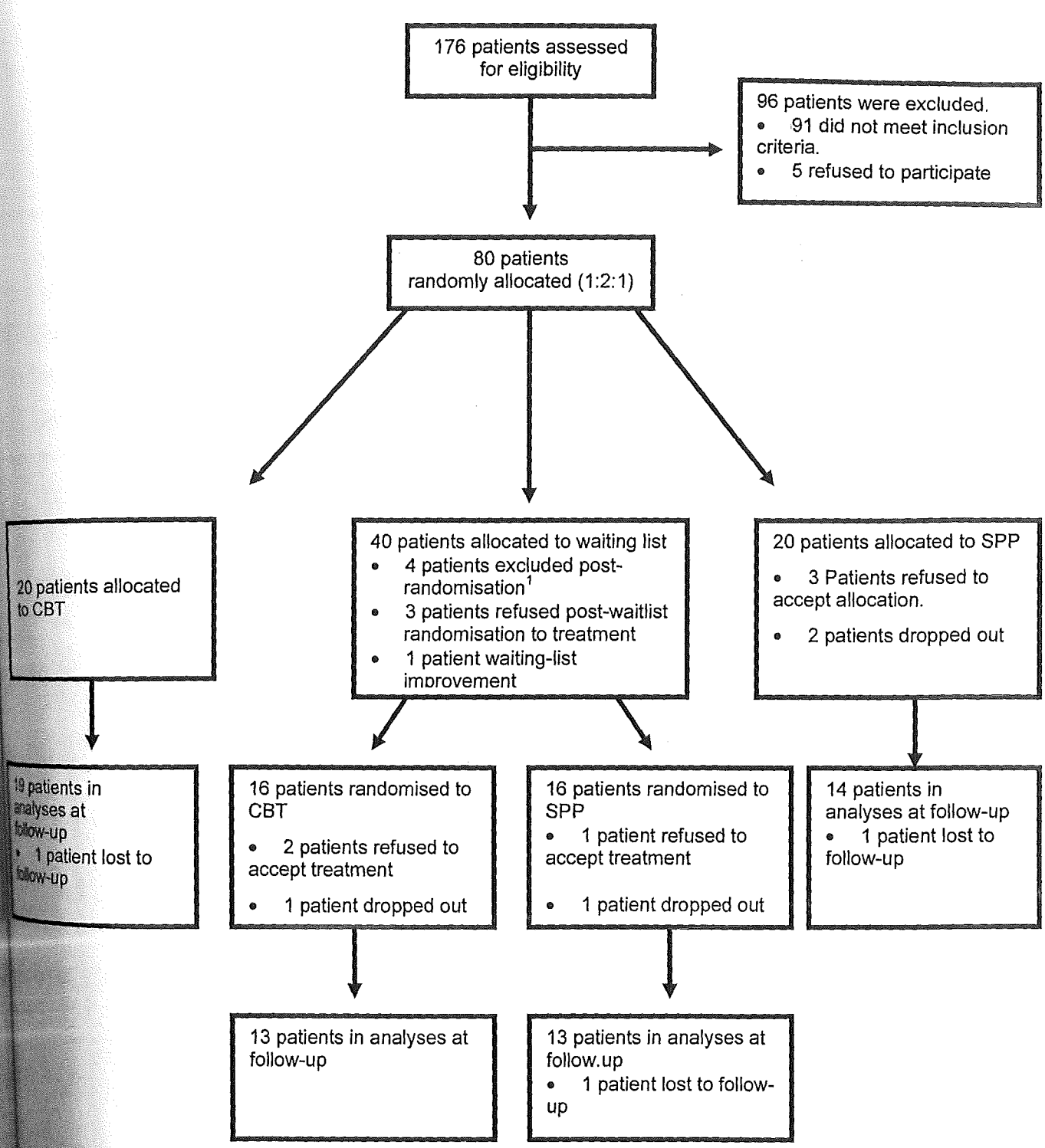
Due to the character of the intervention, it was not possible to blind the patients and therapists to the group allocation, but the raters assessing the outcome were blinded to group assignment for the duration of the study. The blinding was evaluated. The persons involved in encoding the data were blinded to the group allocation.

Statistical methods

We used a modified intention-to-treat analysis with post-randomisation exclusion unrelated to non-compliance, withdrawal or losses to follow up (176). Missing data were imputed as last-observation-carried forward. The data analysis was divided into two parts: First, the three allocation groups (CBT, SPP, and waiting-list) were compared after intervention on primary and secondary outcome variables. Second, the two treatment groups (CBT, SPP), including the patients initially on waiting list subsequently allocated to the active treatments were compared after treatment and at 6 and 12 months follow-up on primary and secondary outcome variables.

The first analysis was a one factor ANOVA with outcome variables as dependent variable and the three groups as independent variables. In case significant differences between the three groups were

Figur 1: Flow of participants through the randomized trial



[1]Two patients had previous received CBT, 2 patients were diagnosed wrongly at inclusion

found, a post hoc test was included as a pair-wise comparison with a Tukey correction for type I error. The second analysis was a two factor, mixed design, repeated measure ANCOVA with outcome variables after treatment as dependent variables and outcome variables before treatment as covariates. The two treatment groups were between-subject factors and time 0, 6 and 12 month after treatment was within-subject factors. For each analysis a 95% confidence interval was derived. Pearson's chi-squared analysis was used for baseline data, refusal rate, withdrawal rate and success rate. All tests of statistical significance were interpreted with an alpha level set to 0.05. From the previous study (220) it was expected that CBT treatment would reduce health anxiety expressed as the mean on HAI with 12 (SD=7). With a type II error level of 11%, an estimated mean difference between intervention groups of 5 on HAI would be detected as a significant difference (p= 0.05, two-sided) with 39 patients in each intervention group. It was therefore decided to include 20 patients in the CBT group, 20 patients in the SPP group and 40 patients on a waiting list, subsequently randomized and allocated to either CBT or SPP. No interim analyses were planned during the trial.

Results

The participant flow is shown in fig 1. A total of 176 patients were assessed for eligibility, 91 patients did not meet inclusion criteria. Eighty-five patients fulfilled the inclusion criteria, but 5 patients declined to participate, so eighty patients were randomised. All patients included in the trial were analysed by an adjudication committee blinded to intervention.

Table 2: Exclusion and withdrawal rates for both treatment conditions.

	CBT (N=39)*	**SPP** (N=38)*	**Pearson X^2**	**P**
Post-randomisation exclusion	3 (7.7%)	2 (5.3%)	0.19	0.665
	CBT (N=36)	**SPP** (N=36)		
Completers	33(92%)	29(81%)	1.86	0.173
Drop-outs	1 (2,8%)	3 (8.3%)	1.06	0.303
No-acceptance	2 (5.6%)	4 (11.1%)	0.73	0.394

* 3 patients were not randomised after waiting list.
CBT, Cognitive Behavioural Therapy; SPP, Short-term Psychodynamic Therapy;

The exclusion and withdrawal rates are shown in table 2. Four patients had initially been inappropriately included. Two had previously received CBT, and 2 were not diagnosed correctly: one patient was psychotic the other had a severe personality disorder such that health anxiety was

Table 3: Demographic, clinical and social baseline data of intention to treat study sample (3 group comparison)

	CBT (N=20)	SPP (N=20)	WLG (N=36)	Total (N=76)*
Gender, %				
Male	35	35	39	37
Female	65	65	61	63
Age, years (SD)	38(12)	34(7)	37(12)	37(11)
Marital Status, %				
Cohabitation/married	55	65	67	63
Divorced	30	20	19	22
Always single	15	15	14	15
Educational level, %				
Low	25	35	19	25
Intermediate	55	30	70	55
High	20	35	11	20
Working situation, %				
Employment	55	50	61	56
Student	20	25	14	18
Unemployment	10	5	0	4
On sickness benefit	10	15	3	8
Social security benefit	0	5	3	3
Disability benefit	5	0	19	11
Source of referral				
GP	70	55	75	68
Psychiatric referral	10	20	17	16
Other	20	25	8	16
Previous psychiatric treatment, %				
None	70	60	64	65
Psychopharmacological treatment, %				
None	80	75	69	74
Antidepressant	15	20	25	21
Sedative	0	5	6	4
Antipsychotic medication	5	0	0	1
Somatic disease, %				
No	75	85	69	72
Yes	25	15	31	28
Mean GAF (SD)	68(4.9)	66(6.3)	66(7.7)	67(6.7)
Mean HAI (SD)	28.8(5.8)	29.3(4.3)	26.1(5.0)	27.6(5.2)
Mean HAM-A (SD)	17.1(6.0)	18.1(6.7)	18.9(7.4)	18.2(6.8)
Psychiatric Comorbidity, %				
Panic disorder	30	30	44	37
Moderate depression	30	15	33	28
Somatoform disorder	25	0	22	17
Obsessive-compulsive disorder	20	35	28	27

* 4 patients were excluded post randomisation

GP, General Practise; GAF, Global Assessment of Functioning; HAI, Health Anxiety Inventory; HAM-A, Hamilton Rating-scale for Anxiety; CBT, Cognitive Behavioural Therapy; SPP, Short-term Psychodynamic Therapy; WLG, waiting-list Group

not his main problem. These patients were excluded and did not receive treatment in the trial. Thus, 76 patients were remained in the 3-group analysis.

In the second analysis additional 4 patients did not continue after waiting list period and assessment. Three of these refused randomisation after the waiting list, whilst one patient improved on the waiting list to the point that she no longer met the diagnostic inclusion criterion. Consequently, 72 patients were included in the 2-group analysis. Six patients refused to accept the proposed treatment: two in the CBT group and 4 in SPP group. A total of 66 patients (83%) began treatment of which 62 patients (78%) completed treatment. Four patients dropped out during the course of treatment itself: 1 (3%) in CBT group and 3 (10.7%) in SPP group.

Baseline data

The socio-demographic and clinical baseline data of the 76 patients are presented in table 3. There were no major differences between the three groups.

If the hierarchical rules for SCAN were disregarded, there was evidence of substantial comorbidity. This is consistent with previous findings (57). The patients were relatively young; and had a high level of function with a mean GAF (SD) = 66 (7.0). More than two thirds had no previous experience with psychiatric treatment. A fifth of the patients received psychopharmacological treatment, predominantly antidepressants. One patient had been on antipsychotic medication for more than 10 years. There was no information about psychotic episodes, and the patient was not psychotic during the trial.

Outcome measures

The intention to treat (ITT) analysis detected significant group effects, with statistically significant main effects of group for all primary and secondary outcome measures. These were the measure of health anxiety, HAI ($F_{[2,72]}$= 17.6, p<0.0001), Hamilton Anxiety Rating ($F_{[2,72]}$= 7.6, p=0.001) , Beck Anxiety Inventory ($F_{[2,72]}$= 7.3, p=0.001), Hamilton Depression Rating ($F_{[2,72]}$= 9.0, p<0.0001) and the Beck Depression Inventory ($F_{[2,72]}$= 8.6, p<0.0001). Table 4 shows the means and the results of an ANOVA which was used to derive Tukey HSD multiple comparisons. These results indicate that those who received CBT were significantly more improved than the waiting list comparison group on both primary and all secondary outcome measures. There was no significant difference

Table 4: 3-group analyses with mean measures by group pre and post treatment

Intention-to treat	CBT N=20	SPP N=20	WL N=36	Grp F(2,73)	P	WL-CBT (95%CI)	P	WL-SPP (95%CI)	P	SPP-CBT (95%CI)	P
Health Anxiety Inventory (HAI)											
Pretreatment	28.8 (5.8)	29.3 (4.3)	26.1 (5.0)	14.9	<0.0001	9.6 (5.3 to 13.9)	<0.0001	1.2 (-3.1 to 5.5)	0.785	8.4 (3.5 to 13.3)	<0.0001
Posttreatment	15.2 (6.8)	23.6 (5.8)	24.8 (6.7)								
Hamilton rating Scale for Anxiety (HAM-A)											
Pretreatment	17.1 (6.0)	18.1 (6.7)	18.9 (7.3)	7.9	0.001	9.2 (3.6 to 14.7)	<0.0001	3.5 (-2.1 to 9.0)	0.299	5.7 (-0.6 to 12.0)	0.081
Posttreatment	11.4 (7.9)	17.1 (8.9)	20.5 (9.0)								
Beck Anxiety Inventory (BAI)											
Pretreatment	22.4 (11.1)	18.2 (7.7)	21.1 (9.6)+	5.4*	0.007	8.4 (2.1 to 14.7)	0.006	5.1 (-0.1 to 115)	0.135	3.3 (-3.9 to 10.4)	0.524
Posttreatment	10.5 (9.6)	13.7 (7.2)	18.8 (10.4)+								
Beck Depression Inventory (BDI)											
Pretreatment	15.0 (8.8)	16.3 (7.9)	14.0 (6.9)	4.8	0.011	6.5 (1.3 to 11.7)	0.011	0.6 (-3.8 to 5.0)	0.963	5.9 (-0.1 to 11.9)	0.053
Posttreatment	5.9 (7.0)	11.8 (7.6)	12.4 (8.2)								
Hamilton rating Scale for Depression (HAM-D)											
Pretreatment	12.5 (4.3)	13.2 (3.8)	12.7 (4.5)	7.6	0.001	6.7 (2.5 to 10.7)	0.001	1.4 (-2.7 to 5.5)	0.696	5.2 (0.6 to 9.9)	0.025
Posttreatment	8.9 (5.4)	14.1 (5.7)	15.4 (6.7)								

+=35 *= F (2,72); CI, confidence interval; CBT, cognitive behavioural treatment; SPP, short-term psychodynamic psychotherapy; WL, Waiting-list; Grp, group

between SPP and waiting list for any outcome measures. The comparison between the SPP and CBT treatment groups for this analysis showed that the CBT group were significant better in terms of the HAI and HAM-D but not on the HAM-A or BAI, with the comparison on the BDI showing a trend for CBT to do better than SPP (p=0.053).

Tabel 5: 2-group analyses with means for main measures by group at 0, 6 and 12-month follow-up

	CBT	SPP	Grp.diff	Grp.diff	Time	Time	Grp x time	Grp x time
Intention to treat	**N=36**	**N=36**	**F(1,69)**	**P**	**F(1,138)**	**P**	**F(2,138)**	**P**
Health Anxiety Inventory (HAI)								
0 mdr. Follow-up	15.3 (6.3)	21.6 (7.0)						
6 mdr. Follow-up	15.4 (8.4)	21.7 (7.2)	12.34	0.001*	0.05	0.952	3.171	0.045
12 mdr. follow-up	17.7 (8.2)	20.6(8.8)						
Hamilton rating Scale for Anxiety (HAM-A)								
0 mdr. Follow-up	12.6 (7.9)	16.0(8.8)						
6 mdr. Follow-up	10.5 (8.4)	15.3(10.2)	3.06	0.085	0.04	0.995	3.169	0.045
12 mdr. follow-up	12.4 (9.5)	12.6 (9.3)						
Beck Anxiety Inventory (BAI)								
0 mdr. Follow-up	10.6 (8.9) +	14.4(10.2)						
6 mdr. Follow-up	10.7 (9.6) +	15.3 (10.4)	4.44†	0.039*	0.150††	0.861	0.139††	0.870
12 mdr. follow-up	11.1 (9.8) +	15.0(12.1)						
Beck Depression Inventory (BDI)								
0 mdr. Follow-up	7.5 (7.3) +	10.2 (7.9) +						
6 mdr. Follow-up	10.2 (8.7) +	11.6 (7.9) +	4.71‡	0.033*	1.072‡‡	0.345	1.09‡‡	0.341
12 mdr. follow-up	8.4 (8.3) +	12.2 (10.9) +						
Hamilton rating Scale for Depression (HAM-D)								
0 mdr. Follow-up	10.3 (6.7)	13.4 (7.0)						
6 mdr. Follow-up	8.6 (7.3)	11.8 (7.9)	4.02	0.049*	0.100	0.887	1.19	0.306
12 mdr. follow-up	10.1 (7.5)	11.5 (8.4)						

+N=35 †F(1,68) ‡F(1,67) ††F(2,136) ‡‡F(2,134)

CBT, cognitive behavioural treatment; SPP, short term psychodynamic psychotherapy; GRP, group; Diff, difference

For the two group comparison treatment effects (table 5) were present as a significant main effects of group for the HAI, BAI, HAM-D and BDI, with a trend for the HAM-A (p=0.085). For the HAI and the HAM-A the main treatment effect was modified by a significant group X time interaction. These interactions were examined further using one way ANOVA for the change scores relative to pre-treatment levels. For the HAI, the difference in change scores was significant for the end of treatment ($F_{[1,70]}$=14.2, p<0.0001), the six month follow up ($F_{[1,70]}$=10.8, p=0.002), but not for 12

month follow up ($F_{[1,70]}$=1.9, p=0.17). The same analysis of change scores for the HAM-A indicated a trend for a difference at the end of treatment ($F_{[1,70]}$=3.2, p=0.076), a significant difference at six months ($F_{[1,70]}$=4.5, p=0.038) and none at 12 month follow up ($F_{[1,70]}$=0.08, p=0.93).

Discussion

Hypochondriasis has been considered a chronic disorder refractory to treatment (231), but more recent studies have established the effectiveness of cognitive-behavioural treatments specifically focussed on health anxiety and disease conviction (69;181;185).

Key findings

We compared a well-defined treatment (CBT) for hypochondriasis with short-term psychodynamic psychotherapy (SPP) and a waiting-list group. The three group comparison at the end of active treatment indicated that patients receiving CBT improved compared with waitlist patients on all measures, and compared with the SPP group on health anxiety and depression measures, but not on anxiety measures. The SPP group did not show significant improvements relative to the waitlist group on any measures. The follow up analysis showed that CBT did significantly better overall on all measures except the HAM-A, where only a trend was evident (p=0.085). However, significant interactions suggested that the difference in health anxiety was reduced at the final follow up point. Note that a similar reduction in treatment effects at the longer-term follow up has previously been noted in CBT for health anxiety (181).

Comparison with other trials

The model of misinterpretation as a central psychological process in hypochondriasis has led to a well-defined cognitive behavioural treatment (229), which has been examined in case studies, uncontrolled trials, and in two controlled trials (181;185;192). Barsky developed a similar understanding of hypochondriasis as a self-perpetuating disorder of cognition and bodily perception with focus on the cognitive and behavioural amplification of benign bodily symptoms (232). A treatment model based on this understanding has been examined empirically in 2 controlled designs. One study included only few patients (233) with waiting-list group as control, while another large-scale randomized controlled trial had usual medical care (69) as control. A significant clinical treatment effect was found, but questions were raised about generalizability and the relationship between intervention and outcome. The present trial is the second trial to compare

CBT for health anxiety with a highly credible alternative treatment. In the previous study, Applied Stress Management (ASM, an individualised package of applied relaxation and behavioural strategies emphasising stress as the likely cause of health anxiety) was used as the comparison condition. ASM was found to be more effective than a waiting list control group but not as effective as misinterpretation focussed CBT. Note, however, that at the last follow up point differences between the treatment groups was beginning to be lost. However, the researchers (on the basis of pilot work) incorporated a range of CBT based engagement strategies in the early stages of ASM to ensure low drop out rates, thus diluting the distinctness of the comparison.

For the present trial, a dissemination strategy with training and supervision was established and implemented as part of a published pilot study finished before this trial started (220). SPP was used to control for as many non-specific factors as possible without incorporating any CBT strategies. The treatment effect of CBT in the present study was comparable with previous large-scale controlled trials (69;181).

Limitations

Sampling in this study was both from normal clinical sources and solicited then filtered by clinicians; thus, the sample may not be representative of either health anxiety patients in the community or the clinic. There were some indications that the sample was not severely disabled (e.g. in terms of marital and employment status). A few patients eligible for the treatment refused randomisation and there were also some dropouts relatively evenly spread between the randomised groups.

The identification of primary hypochondriasis as the patients' main problem was initially a clinical judgment. However, an adjudication committee also oversaw this judgment; there were disagreements in only two of 80 cases. Many patients had mild to moderate depressive episodes according to ICD-10 criteria and scored low on both BDI and HAM-D. Some patients had recurrent depression, but the hypochondrial symptoms were found to be independent of the depressive episodes. The hypochondriacal patients had a relatively high comorbidity for anxiety disorders, somatisation disorder and OCD, but in all instances the hypochondriasis was the main complaint.

We chose a modified form of intention-to-treat in our analyses in which patients were excluded after randomisation, if they had been inappropriately randomised into the trial; this decision required a unanimous decision of the adjudication committee, who were blind to randomisation. Erroneous inclusion was, in each instance, detected before active treatment started and was therefore unrelated to treatment response. Patients, who dropped-out after the waiting list, were only included in waiting list group for analysis purposes, as was the single patient who no longer met diagnostic criteria at the end of the waiting list. This patient received treatment in any case, but was excluded from post waiting list follow up analyses. Patients who dropped out or were lost to follow-up were all included in the analyses. An additional intention-to-treat analysis without post randomisation exclusion did not differ substantially from the findings presented (data not shown).

The trial was not double blind, as this is not possible in psychotherapy trials. As the purpose was to measure the efficacy of a treatment, it was problematic, that it was not possible to blind the patient and the therapist. We were not able to examine the bias this may have caused in the present trial, as we have no systematic information about the patients' expectation to the allocated treatment. The observerbased outcome measures were conducted by blinded independent assessors. The patients were instructed not to tell the assessor what treatment they received.The assessors were asked to guess the treatment the patient had received and were able to guess treatment/waitlist allocation significantly better than would be expected by chance ($X^2 = 25.1$, $p<0.0001$). The blinding was therefore compromised, though the assessors often were in doubt. In this respect the trial can be regarded as un-blinded. The reason for the compromised blinding was probably that the patients revealed the treatment unwillingly by the manner they answered the questions concerning different symptoms.

The cognitive behavioural treatment in this trial was modified in format terms by the use of group treatment in the later stages of treatment and in terms of content by the addition of mindfulness techniques. Nevertheless, it was established in supervision that the treatment was very similar to CBT as originally devised for health anxiety (229). In terms of SPP, there are a number of issues. A single therapist conducted all therapy in this condition. In research terms it would have been preferable to have had several therapists but this was not possible for practical reasons. The therapist conducting SPP was both committed to and experienced in psychodynamic treatment, and had been trained according to principles of psychoanalytic psychotherapy (234). Unfortunately,

there is no consensus as to what constitutes appropriate short term psychodynamic treatment for hypochondriasis, so a pragmatic decision was made based on a high quality version of the treatment most likely to be offered by psychodynamically oriented psychotherapists in the University Hospital.

Concluding remarks

We investigated cognitive-behavioural and a psychodynamic treatment for patients with hypochondriasis and found a benefical effect of the cognitive-behavioural treatment, which was still evident at follow-up on most measures. The psychodynamic treatment was not found to have any specific effects in the present study, but it is possible that the effectiveness of this treatment may have been under-estimated as it may not have been delivered in an optimal way (e.g. treatment was too short and not focused on hypochondriasis). However, the failure to show improvement relative to a waitlist raises concerns in this respect, particularly given the effectiveness of CBT as an alternative treatment. In terms of CBT, this is the second study in which the differences found at post-treatment and short term follow up were less apparent at longer term follow up. The reduced level of difference at longer term follow up are less due to any substantial fallback of improvement in CBT and more due to a blurring of differences at that later stage. This is, of course, a common finding in psychotherapy studies as life events, occurrence of actual health threats and other psychological factors impact on the participants in all groups followed up, resulting in initially large differences to be diluted. Given the chronic nature of health anxiety we should perhaps consider the possibility of offering maintenance sessions, for example on a three or six monthly basis during the immediate follow up period.

CONCLUSION

This Ph.D. thesis consists of two parts. In the first part I discuss the concept of primary hypochondriasis from a theoretical standpoint and examine the hypothesis of primary hypochondriasis in cross-sectional study, where health anxiety is found to be independent of other psychopathological measures. The second part is a randomised trial examining the effect of a specific psychotherapeutic treatment for hypochondriasis. The result of this trial is highly significant and a confirmation of findings from previous trials that a specific cognitive-behavioural treatment is benefical for patients suffering from hypochondriasis. These results strengthen hypochondriasis or health anxiety as a useful clinical concept. Further trials are recommended to examine the long-term effect of the treatment and with less selected participants. The psychodynamic treatment did not have a specific effect, but a short-term treatment demand a focal approach, which was not available. A psychodynamic model using attachment patterns as a framework is under development and could be useful for future trials (51;52;235).

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DANISH SUMMARY

Denne ph.d. afhandling udspringer af en undersøgelse af 80 patienter som opfylder de diagnostiske kriterier for en hypokonder tilstand, og som i en randomiseret klinisk forsøg modtog enten kognitiv adfærdsterapi, kort tids psykodynamisk psykoterapi eller ingen behandling (The Kathy Trial).

Afhandlingen består af to teoretiske kapitler og tre artikler. Det første kapitel "The concept of hypochondriasis" forbinder "The Kathy Trial" med den aktuelle forståelse af hypokondri. Det er en oversigt over hypokondris udvikling som et psykopatologisk begreb herunder diskussionen om hypokondri som en gyldig diagnostisk enhed. Fokus er den nuværende forståelse og er en undersøgelse af hvorledes forskellige aspekter repræsenteres i spørgeskemaer og bedømmelsesbaseret instrumenter, samt en undersøgelse af komorbiditet mellem hypokondri og beslægtede tilstande. Kapitlet indeholder beskrivelser af forskellige modeller for hypokondri begrebsliggjort som mønstre af adfærd og kognition, hvor det undersøges hvorledes disse modeller har påvirket de aktuelle diagnostiske definitioner og muliggjort fokuseret psykoterapeutisk behandling baseret på disse modeller. Herunder en beskrivelse af en kognitiv-adfærdsterapeutisk model for hypokondri med fokus på mistolkningen af kropslige sansninger. Denne model og tilhørende behandling undersøges klinisk i denne afhandling. Det andet kapitel "Randomised clinical trial as a method of evaluation of psychotherapy to hypochondriasis" omhandler generelle aspekter af det randomsierede kliniske forsøg som metode til at evaluere psykoterapi, specielt psykodynamisk orienteret psykoterapi. Udgangspunktet er "The Kathy Trial".

De 3 artikler omhandler forskellige dele af forsøget. Den første artikel "An examination of consecutive patients diagnosed with hypochondriasis" er en tværsnitsundersøgelse af patienter diagnosticeret med hypokondri med fokus på den psykiatriske komorbiditet. Formålet er at evaluere hypokondri som en klinisk diagnose og teste hypotesen om hypokondri som en primær psykopatologisk tilstand. Den anden artikel "Outcome of a cognitive-Behavioural treatment for health anxiety (hypochondriasis) in a routine clinical setting" fokusere på spredning af en specifik psykoterapeutisk behandling for patienter diagnosticeret med hypokondri fra en videnskabelig, akademisk afdeling til en klinisk enhed. Den tredje artikel beskriver, analyserer og tester hypotesen for det randomiserede kliniske forsøg.

I en tværsnitsundersøgelse af 415 konsekutive patienter henvist til en liaisonpsykiatrisk enhed opfyldte 80 patienter (19 %) de diagnostiske kriterier for en hypokonder tilstand og ingen eksklusionskriterier for den randomiserede kliniske forsøg. Patienterne havde en høj psykiatrisk komorbiditet, specielt angst, somatisering og depression. Ved multiple regressions analyser fandt man ingen signifikant relation mellem et specifikt mål for hypokondri (HAI), angst, depression eller personlighedstræk. Dette fund støtter den hypotese, at hypokondri er en primær psykopatologisk tilstand og ikke blot en dimension ved andre psykiatriske tilstande. Begrænsninger i studiet var blandt andet at patientgruppen var selekteret og muligvis ikke en rimelig repræsentation af hypokondri i befolkningen, at undersøgelsen omhandlede et lille patientantal og at der var tvivl omkring specificiteten af det benyttede mål for hypokondri.

En tværsnitsundersøgelse begrænser muligheden for at undersøge den kausale relation mellem komorbide psykiatriske tilstande, så det følgende ukontrollerede pilotstudie gav muligheden for prospektive observationer. Vi undersøgte om en specifik kognitiv-adfærdsterapi (CBT) for hypokondri kunne generaliseres fra det forskningscenter, hvor behandlingen var udviklet, til en klinisk behandlingsenhed. I den kognitiv-adfærdsterapi søger terapeut og patient at udvikle en fælles forståelse for sygdomsangst, hvilket kræver identifikation af patientens idiosynkratiske kognitive model for sygdom og sundhed og den medfølgende angst. Terapien stimulerer en mindre truende forklaring på patientens problemer og gennem den kognitive diskussion, herunder adfærdsmæssige øvelser, testes alternative kognitive forklaringer. Behandlingen blev tilpasset behandlings enhedens praksis, så en del af behandlingen foregik i en gruppeterapeutisk ramme, og mindfullness blev inkluderet i behandlingsprogrammet. Seksten patienter blev behandlet i pilotstudiet, hvor forandringen observeret ved afslutningen af behandlingen var substantiel og statistisk og klinisk signifikant. Forandringen blev opretholdt ved follow up. Resultatet tyder på en succesfuld spredning af denne specifikke behandling, men visse faktorer begrænser konklusionen. Studiet var ikke randomiseret, så forandringen kunne være udtryk for "regression toward the mean". Patienterne havde et højt niveau af psykiatrisk komorbiditet, så det er ikke klart, alene på baggrund af disse fund, om den signifikante forbedring er specifik for hypokondri eller om andre psykopatologiske manifestationer er involveret. Antallet af patienter var meget lille, og rekrutteringsproceduren har muligvis medført en atypisk patientpopulation med et højt funktionsniveau, hvilket delvist kompromitterer konklusionen.

Erfaringerne fra pilot studiet blev brugt i udviklingen af et randomiseret klinisk forsøg (RCT) for at sikre konklusionen vedrørende behandlingens effektivitet. Resultatet af pilotstudiet blev brugt i en styrkeberegning og estimat af gruppestørrelser. Konsekutive patienter henvist til et liaisonpsykiatrisk ambulatorium blev undersøgt. I alt 176 patienter blev vurderet, hvoraf 80 patienter opfyldte de diagnostiske kriterier for en hypokonder tilstand og ingen eksklusionskriterier. Disse 80 patienter blev randomiseret til enten den specifikke kognitiv-adfærdsterapi (CBT) udviklet til hypokondri, en kort tids psykodynamisk psykoterapi (SPP) eller venteliste (ingen behandling). Patienterne på venteliste blev efterfølgende randomiseret til de to aktive behandlingsformer for at styrke sammenligningen mellem grupperne som modtog aktiv behandling. Alle randomiserede patienter blev vurderet efter behandlingens afslutning og ved 6 og 12 måneders follow up. To primære effekt mål blev defineret inden forsøget blev påbegyndt, som vi fandt repræsenterede forandringer i en specifik og generel psykopatologi. Vi benyttede Health Anxiety Inventory (HAI) for sygdomsangst og Hamiltons angstskala (HAM-A) for generel angst. Ved Hamilton rating blev bedømmerne blindet for den gruppe patienterne var allokeret til.

Den psykodynamiske korttids behandling (SPP) benyttede psykoterapeutiske principper som neutralitet og frie associationer og undgik eksplicit at udfordre patienternes ideer om sygdom, som var forbeholdt CBT. Fokus var den terapeutiske relation, patienternes interpersonelle relation, genkendelse af mønstre og temaer i patientens funktionsmåde og udviklingen af en forståelse for ubevidste fantasier. Vi udviklede et rating system som identificerede komponenter der enten adskilte eller var fælles for de to behandlingsformer. Vi benyttede dette rating system til at kontrollerede, hvor nøjagtig de to behandlingsformer blev efterlevet i forsøget.

Nulhypotesen var todelt. For det første at der ingen forskel var mellem de 3 interventionsgrupper efter behandlingen, for det andet at der ikke var forskel ved follow up mellem de to aktive behandlingsgrupper. Vi benyttede en modificeret intention-to-treat analyse med post-randomisation eksklusion uafhængig af non-kompliance, udtræden af forsøget og manglende opfølgen. Vi benyttede forskellige typer variansanalyser til at teste nulhypotesen. For manglende cases benyttede vi last-observation-carried forward. I alt 62 patienter (68 %) gennemførte behandlingen. De patienter som modtog CBT klarede sig signifikant bedre på alle mål sammenlignet med ventelistegruppen, og på sygdomsangst- og depressionsmål sammenlignet med SPP gruppen, men ikke på angstmål. SPP gruppen viste ingen signifikant forbedring i forhold til venteliste. Ved follow

up analyse klarede CBT sig signifikant bedre undtagen for HAM-A, men en signifikant interaktion antydede at forskellen for begge primære angstmål blev reduceret ved den afsluttende follow up ved 12 måneder. Behandlingseffekten for CBT var sammenlignelig med tidligere større kontrollerede undersøgelser, mens den psykodynamiske behandling ikke viste sig at have nogen specifik effekt i dette studie.

Forskellige spørgsmål rejses i afhandlingen omkring design af det randomiserede kliniske forsøg og disse fund. Et spørgsmål er forsøgets eksterne validitet og om patienterne på rimelig vis repræsenterer hypokondre patienter i befolkningen. Et andet spørgsmålet er om de to behandlingsformer er implementeret i relevant grad i forsøget. Vi benyttede en specifik disseminations strategi for CBT, mens SPP blev opfattet som en tilgængelig, troværdig og veludført terapi, hvor det i den terapeutiske relation ikke blev brugt komponenter fra CBT. Det forstærkede det unikke i de to behandlinger og styrkede sammenligningen. Vi benyttede en ikke-fokuseret psykodynamiske behandling, men fastholdt generelle kliniske principper for psykodynamisk behandling. Det er muligt at behandlingens effektivitet undervurderes, da behandlingen var for kort til ikke-fokuseret psykodynamisk behandling, og psykoterapeutisk behandling derfor ikke var rimeligt implementeret i forsøget.

Men resultatet af forsøget er statistisk signifikant og en bekræftelse fra tidligere forsøg, at en specifik kognitiv-adfærds terapi med fokus på mistolkninger af kropslige sansninger er gavnlig for patienter med hypokondri. Resultatet styrker hypokonder tilstand eller sygdoms angst som et brugbart klinisk begreb. Yderligere undersøgelser anbefales med fokus på langtidseffekten af behandlingen med en gruppe mindre selekterede patienter. Samtidig bør nye effekt mål inddrages som omhandler patienternes funktionsniveau og forbrug af sundhedsydelser. Den psykodynamiske behandling havde ingen specifik effekt, men en korttids behandling kræver et specifikt fokus, som ikke var tilgængeligt. En psykodynamisk model som benytter tilknytningsmønstre som sit grundlag er under udvikling og kunne være brugbart for fremtidige undersøgelser.