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Cognitive and perceptual variables in hypochondriasis and health anxiety: A systematic review ☆

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Abstract

This review examined (a) whether hypochondriacal/health-anxious individuals hold distinct assumptions about health and illness, (b) if triggering these assumptions leads to increased hypochondriacal concerns, and (c) whether these individuals perceive their bodily sensations differently from others (i.e., experience greater somatosensory amplification). There was clear evidence that health anxiety is related dysfunctional health-related beliefs. Few studies have examined how hypochondriacal concerns are triggered, and inconsistent results emerged from those that have. Health anxiety is also associated with self-reported higher levels of somatosensory amplification. However, there was little evidence that individuals high in health anxiety are actually more accurate perceivers of the their own autonomic processes. Although the results generally supported the central tenets of the cognitive-behavioral model of hypochondriasis and health anxiety, further research will be necessary to determine whether these beliefs are specific to hypochondriasis and to identify any cognitive processes that may be unique to hypochondriasis.

Keywords: Hypochondriasis; Health Anxiety; Cognitive-behavioral; Meta-analysis

Although there has been a long history of motivational and psychodynamic theories of hypochondriasis (e.g., Fenichel, 1945; Lipsitt, 1973; Rosenfeld, 1958), most recent models of hypochondriasis and health anxiety[1] emphasize cognitive and perceptual factors. As Williams (2004) observed, "cognitive models have been the predominant recent approach to the understanding and treatment of health anxiety and hypochondriasis" (p. 632). Most notably, Salkoviskis and Warwick (1986, 2001; Warwick & Salkovskis, 1990) have developed a cognitive-behavioral model of hypochondriasis and health anxiety that is closely related to Clark's (1986) and Barlow's (2002) models of panic disorder. According to Salkoviskis and Warwick's model, dysfunctional assumptions and beliefs about (a) the prevalence and communicability of severe illnesses, (b) the meaning of bodily symptoms, and (c) the course and treatment of illnesses all serve as risk factors for hypochondriasis and health anxiety. Although these beliefs may remain latent, they can be activated by a variety of events, including reading about an illness, hearing about the illness of an acquaintance, illness-related media reports (e.g., news stories about avian flu, SARS, or AIDS), or by various

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[1] Throughout the manuscript, hypochondriasis refers specifically to the clinical syndrome, whereas health anxiety refers to full range of dysfunctional health concerns. However, hypochondriacal as an adjective may be used to refer to any excessive illness concerns.

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bodily alterations or sensations (e.g., a blemish, a headache). Once triggered, these beliefs result in automatic hypochondriacal thoughts that are specifically self-focused. As a result the person becomes increasingly anxious and hypervigilant for any sensations or signs that could be indicative of the disorder. Bodily sensations are then filtered through a confirmatory bias that exaggerates any evidence of illness. Barsky (1992, 2001; Barsky & Klerman, 1983) has proposed a similar approach to understanding hypochondriasis, but he places a greater emphasis on the premise that hypochondriacal individuals are more sensitive to and aware of bodily sensations and that this "somatosensory amplification" serves as a risk factor for hypochondriasis. Most recently, Taylor and Asmundson (2004) have integrated cognitive factors (including dysfunctional beliefs and selective memory), attentional factors, and somatosensory amplification into an integrated model of health anxiety.

To exemplify this cognitive-behavioral approach we consider "Ann." Ann may hold the dysfunctional beliefs that any unexplained bodily sensations must be indicative of a catastrophic illness, and if illnesses are not identified and treated immediately, they are likely to be fatal. If Ann sees a movie in which one of the characters complains about a headache and is later diagnosed with a brain tumor, the movie may activate these beliefs. Once such beliefs are activated, Ann is likely to be especially attentive to any indications that her head hurts. Because of a premorbid tendency toward somatosensory amplification, she may be especially sensitive to any pain sensations that others might not notice. When she feels her head ache, Ann is then likely to make catastrophic interpretations and discount more benign interpretations (e.g., tension). The anxiety resulting from these catastrophic interpretations may lead to behavioral and somatic changes that further confirm Ann's fears. For example, because of her worry and anxiety about being sick, Ann may have difficulty sleeping, and this sleeplessness may then result in additional headaches, supporting her belief that she has a brain tumor.

A number of studies have directly examined aspects of this model, and other health anxiety studies, although not direct tests, have also yielded relevant results. It is possible to dismantle the model and ask (a) whether there is evidence that health-anxious individuals hold dysfunctional assumptions about health and illness or think about health-related issues differently from how others think about these issues, (b) if triggering these assumptions leads to increased vigilance, concerns, and anxiety, and (c) if health-anxious individuals perceive their bodily sensations differently from others? The aims of this review are (a) to examine which aspects of the models have been subjected to empirical test, (b) to meta-analyze the results of these studies to determine the magnitude of the support for the model (for those aspects that have generated sufficient research) and (c) to examine moderators that may explain the variability among studies (if such variability exists). The results of this review may lead to refinements in the model and may guide future research by identifying aspects of the model that have been understudied.

1. Dysfunctional assumptions and cognitive processes

Central to Salkoviskis and Warwick's model is the proposition that health-anxious individuals hold dysfunctional medical and illness-related beliefs. Typically, researchers have examined this aspect of the model by providing respondents with ambiguous symptoms and asking them either to indicate which illness they would assume they had if they experienced the symptom or to estimate the likelihood that such symptoms are indicative of a catastrophic illness. For example, Hitchcock and Mathews (1992) found that college students higher in health anxiety, as measured by the Illness Attitudes Scale (IAS; Kellner, Abbott, Winslow, & Pathak, 1987), were more likely to interpret bodily sensations (e.g., "your chest feels tight and you find yourself sweating and flushed," p. 225) as indicative of catastrophic illness (e.g., having a heart attack) than were students who scored lower on the IAS. Marcus (1999) found that among college students, IAS scores positively correlated with estimates of the likelihood that ambiguous symptoms (e.g., headache) were indicative of serious illnesses (e.g., brain tumor). Patients diagnosed with hypochondriasis identify more physical symptoms as indicating that a person is "not healthy" than do medical patients who do not have hypochondriasis (Barsky, Coeytaux, Remy, Sarnie, & Cleary, 1993).

Although these studies, in which participants reported their beliefs and assumptions about symptoms and illnesses, bear most directly on the dysfunctional assumption component of the cognitive-behavioral model of health anxiety, studies that have used indirect methods to examine cognitive processes in health anxiety may also have some relevance to this aspect of the model. For example, Owens, Asmundson, Hadjistavropoulos, and Owens (2004) found that individuals higher in health anxiety displayed greater interference on a modified Stroop test to illness-related words; moreover, this interference was specific to illness-related words. Medical patients with hypochondriasis also recall more pain-related words (e.g., stinging, burning) than do other medical patients, and again, this memory bias is specific to pain (Pauli & Alpers, 2002).

2. Triggers

Our review yielded only three studies that examined Salkoviskis and Warwick's (1986) supposition that exposure to illness-related materials triggers increased hypochondriacal concerns or anxiety in health-anxious individuals. Lecci and Cohen (2002) reported two studies in which they primed college student participants by giving them a medical exam and informing them that their blood pressures were "dangerously high" (p. 149). In both studies, students in the experimental condition who reported high levels of somatosensory amplification displayed greater interference on a modified Stroop task for illness-related words, suggesting that the bogus medical feedback activated illness-related concerns. Furthermore, in both studies, students in the experimental condition who reported high levels of illness preoccupation on the Whitely Index (WI; Pilowsky, 1967) displayed greater Stroop interference for all words, perhaps resulting from increased arousal and anxiety. (Inconsistent with Owens et al. (2004), students who were high in health anxiety in the control condition – i.e., who were not primed – did not evidence Stroop interference for illness-related words in either of the two studies.)

Marcus (1999) attempted to trigger anxiety in college students using a scrambled sentences task that included illness-related words (e.g., cancer). Although IAS scores were positively related to state anxiety scores for students in the control condition, contrary to the cognitive-behavioral model, there was no relationship between IAS scores and anxiety in the priming condition. In other words, priming with illnesses-related words did *not* lead those students who were more health-anxious to become more anxious. Instead, it appears that the students lower in health anxiety became as anxious as the high health anxiety students. Marcus's (1999) priming task was more indirect and subtle than the one used by Lecci and Cohen (2002), but the inconsistent results could also be due to the very different dependent variables that were used in each study (i.e., Stroop interference versus self-reported state anxiety). With only three studies on which to draw, this aspect of the model was not examined meta-analytically.

3. Somatic perception and amplification

Studies of perception and amplification in health anxiety have generally used one of two methods. Numerous studies have examined the relationship between health anxiety and scores on the Somatosensory Amplification Scale (SAS; Barsky, Wyshak, & Klerman, 1990). The SAS is a 10-item self-report scale that assesses sensitivity to bodily sensations (e.g., "I can sometimes hear my pulse or my heartbeat throbbing in my ear" p. 327). Generally, SAS scores have been moderately positively correlated with self-reported health anxiety scores (Barsky & Wyshak, 1990) and medical patients with hypochondriasis score higher on the SAS than do other medical patients (Barsky et al., 1990).

Other studies have used in vivo procedures to examine whether health-anxious individuals are in fact more sensitive to physical sensations or more accurate in their perceptions of bodily processes. Women with high levels of health anxiety evidenced greater pain sensitivity on a cold pressor task (i.e., they rated the task as more unpleasant, withdrew their hands more quickly, and had a greater elevation in heart rate) than did women in a comparison group (Gramling, Clawson, & McDonald, 1996). However, despite higher SAS scores, patients with hypochondriasis were no more accurate on a heartbeat detection task than nonhypochondriacal patients were. On the contrary, there was a trend toward greater accuracy among the nonhypochondriacal patients (Barsky, Brener, Coeytaux, & Cleary, 1995). Similarly, Haenen, Schmidt, Schoenmakers, and van der Hout (1997) found that although patients with hypochondriasis had higher SAS scores, they were no more sensitive to tactile stimuli (assessed with a two two-point discrimination task) than was a control group of healthy adults. This pattern of results suggests that it is probably not the physical sensations per se, but people's misinterpretations of these sensations that is associated with health anxiety.

4. Moderators

Because hypochondriacal concerns and health anxiety are believed to occur along a continuum (Costa & McCrae, 1985; Williams, 2004) and because Salkoviskis and Warwick developed their cognitive-behavioral model to encompass subclinical health anxiety, many of the studies we reviewed treated health anxiety as a continuous personality variable and sampled their participants from nonclinical populations (e.g., Aronson, Barrett, & Quigley, 2001; Marcus, 1999). Yet if health anxiety exists on a continuum, individuals drawn from clinical populations and diagnosed with hypochondriasis are likely to hold the most extreme dysfunctional beliefs about illness and to report the highest levels of somatosensory amplification. Therefore each study was coded for whether it used a clinical or

nonclinical sample and we hypothesized that sample type would be a moderator, with clinical samples yielding larger effect sizes.

Among those studies that examined dysfunctional assumptions and cognitive processes in health anxiety, we hypothesized that whether a study used direct methods to assess dysfunctional assumptions about illness or indirect methods to assess cognitive processes would also serve as a moderator in the meta-analysis, with larger effects sizes for those studies that used direct methods. In other words, we expected studies that examined the specific cognitions associated with health anxiety and hypochondriasis to yield larger effects than those that examined broader cognitive processes. Part of the rationale for this hypothesis was that dysfunctional assumptions about illness are central to the cognitive-behavioral model, whereas some of these process variables (e.g., Stroop interference, memory for pain words) are more peripheral to the model. Admittedly, shared method variance in the dysfunctional assumption studies (i.e., in these studies self-reported hypochondriacal attitudes are correlated with self-reported illness assumptions) may also contribute to the predicted larger effect sizes for studies that directly assessed assumptions and beliefs.

Among those studies that examined somatic perceptions, we expected moderate to large effect sizes when participants provided self-reports of somatic sensitivity (as in studies that correlated the SAS with a measure of health anxiety). In contrast, there was little reason to expect a significant average effect size when the participants' bodily perceptions were compared to an objective measure (e.g., heartbeat detection). Therefore, we expected studies that examined self-reported somatic sensitivity to yield larger effects than those that used in vivo measures of accuracy or sensitivity. Once again, part of the rationale for this hypothesis was simply that shared method variance could inflate the effect sizes in the former studies. Furthermore, this hypothesis is consistent with Barsky's (2001) narrative review of the somatosensory amplification literature. After finding that individuals high in health anxiety consistently score higher on the SAS but perform no better (and sometimes worse) than controls on objective measures of somatic perception, Barsky (2001) raised the intriguing possibility that "rather than being extremely sensitive detectors who are able to discern and discriminate individual, weak, or ambiguous bodily sensations, amplifiers and hypochondriacal patients are actually less able to distinguish these signals from background noise" (p. 242).

5. Method

5.1. Selection of studies

We used several strategies and searches to collect the studies. We first performed a PsycINFO search with the root "hypochondria∗", selecting all studies that examined some aspect of the cognitive-behavioral model. We then performed additional searches with the key words "somatosensory amplification" and "health anxiety." Additional PsycINFO searches of the names of authors who frequently publish in the area were also performed. We also searched PsycINFO and Google Scholar for publications that cited Salkovskis and Warwick's papers on hypochondriasis. These searches were supplemented with two Medline searches, one that paired "hypochondria∗" with "cognitive" and a second that paired "hypochondria*" with "amplification." The reference sections of the studies identified were also reviewed to expand the search.

To be included, a study had to be published in a peer-reviewed journal or book (all of the studies came from journal articles) and reported in English. Studies had to measure health anxiety or hypochondriasis either with a formal diagnostic interview or a valid self-report measure. They also had to include at least one dependent variable (DV) that measured cognitive or perceptual factors relevant to health anxiety. To be included, studies also had to report either a correlation between the health anxiety score and the DV, or a main or simple effect comparing a health-anxious or hypochondriacal group to a control group. For example, studies that only reported interaction terms without main effects and failed to provide sufficient information to compute an effect size were excluded (e.g., de Jong, Haenen, Schmidt, & Mayer, 1998; Durso, Reardon, Shore, & Delys, 1991). To avoid violating the assumption of independence, only one effect size per construct per sample was calculated.[2] When studies reported results from multiple indicators of a construct, the mean of these effect sizes was computed. Although studies that examined treatment outcomes for

[2] In three studies the researchers correlated measures of health anxiety with self-reported somatosensory amplification and also measured in vivo accuracy or sensitivity. To avoid violating the assumption of independence, the three correlations between the self-reported amplification and health anxiety were excluded from the meta-analytic analogue to the analysis of variance. These values were excluded because there were more studies that examined self-report than that actually provided in vivo measures of somatic accuracy or sensitivity.

therapies derived from the cognitive-behavioral model may provide indirect evidence for the model, treatment outcome studies were beyond the scope of this review (for a review, see Taylor & Asmundson, 2004). This search yielded a total of 46 studies.

5.2. Coding and analysis

All studies were coded by two coders (various combinations of the first three authors) and coding disagreements were resolved through discussion and the inclusion of the third coder. The studies were coded for (a) sample size, (b) demographic variables, (c) whether the study used a clinical or nonclinical sample, and (d) the method for assessing health anxiety. Studies were also coded for whether they examined cognitive or perceptual variables. The cognitive studies were further coded for whether they examined (a) assumptions and beliefs or (b) cognitive processes. The perception studies were further coded for whether they examined (a) self-reported somatosensory amplification or (b) a laboratory measure of bodily perception. This coding system resulted in 21 studies that examined cognitive variables and 25 studies that examined perceptual variables (see Tables 1–4 for a summary of these studies).

Following Rosenthal and DiMatteo's (2001) recommendation, all of the effect sizes were converted to r values. The effect sizes were weighted by the sample size minus 3 (n-3). The results were pooled using Lipsey and Wilson's (2001) SPSS macros for meta-analysis. Because variable populations parameters are the rule and not the exception for most sets of studies (e.g., Field, 2003) and because the aim of the analyses was to make unconditional inferences (Hedges & Vevea, 1998) about the population of studies that could potentially be conducted to examine cognitive and perceptual variables in health anxiety, mean effects sizes were computed using a random effects model. This approach provides a more conservative test of statistical significance. Q statistics were computed to test for the homogeneity of the results. Additionally, we computed I^2 (Higgins & Thompson, 2002; Higgins, Thompson, Deeks, & Altman, 2003), which delineates the percentage of variance across studies that is not due to chance. When an analysis yielded significant heterogeneity, Hedges' (1982) meta-analytic analogue to the analysis of variance (again based on a random effects model) was used to test for moderators. The two primary moderator variables for the cognitive studies were not equally distributed across studies (i.e., more cognitive content studies used nonclinical samples (k=9) than clinical samples (k=7), whereas 4

Table 1
General information on the included cognitive content studies

	N	Health anxiety measure	Dependent variable	Effect size (r)
Clinical samples				
Barsky et al. (1993)	120	SCID	Health norms sorting task	.32
Ferguson et al., 2000 (study 4)	79	WI	Perceived control over the recurrence of infection	.24
Haenen, de Jong, Schmidt, Stevens, and Visser (2000)	40	SCID	Estimation of negative health outcomes	.43
Haenen et al. (1998)	54	SDIH	Misinterpretation of benign signs as signs of cancer	.41
Hadjistavropoulos et al. (2000)	81	IAS	Catastrophic interpretations of pain (from the CSQ)	.28
MacLeod, Haynes, and Sensky (1998)	32	IAS	Number of somatic explanations given for somatic symptoms	.66
Rief, Hiller, & Margraf, 1998 (sample 1)	124	WI	Catastrophic interpretations of bodily complaints	.51
Nonclinical samples				
Ferguson (1996)	58	WI	Knowledge of disease etiology	.26
Ferguson et al., 2000 (study 1)	150	WI	Appraisal of threats	.18
Ferguson et al., 2000 (study 2)	150	WI	Appraisal of threats	.19
Ferguson et al., 2000 (study 3)	154	WI	Perceived control over threats	.14
Hadjistavropoulos, Craig, and Hadjistavropoulos (1998)	192	IAS	Catastrophic interpretations of pain	.29
Hitchcock & Mathews, 1992 (study 1)	277	IAS	Probability that bodily sensations indicate catastrophic illness	.43
Marcus (1999)	158	IAS	Probability that symptoms indicate catastrophic illness	.24
Marcus & Church, 2003 (study 1)	133	IAS	Probability that symptoms indicate catastrophic illness	.19
Rief et al., 1998 (sample 2)	101	WI	Catastrophic interpretations of bodily complaints	.37

Note. IAS = Illness Attitude Scales; SCID = Structured Clinical Interview for DSM; SDIH = Structured Diagnostic Interview for Hypochondrias; WI = Whiteley Index.

Table 2
General information on the included cognitive process studies

	N	Health anxiety measure	Dependent variable	Effect size (r)
Clinical samples				
Brown, Kosslyn, Delamater, Fama, & Barsky, 1999 (sample 1)	40	DSM III-R diagnostic interview	Perception and memory of illness-related words	.14
Brown et al., 1999 (sample 2)	89	WI	Perception and memory of illness-related words	−.19
Pauli and Alpers (2002)	42	IDCL	Recall of pain words	.38
Smeets, de Jong, and Mayer (2000)	40	SCID	Use of safety and danger rules for illness-related threat	.15
Nonclinical sample				
Owens et al. (2004)	52	IAS	Stroop interference for illness-related words	.46

Note. IAS = Illness Attitude Scales; IDCL = International Diagnostic Check List; SCID = Structured Clinical Interview for DSM; WI = Whiteley Index.

of the 5 cognitive process studies used clinical samples). Therefore, separate analyses were conducted to compare (a) the studies that used clinical to those that used nonclinical samples to examine cognitive content and (b) the studies that used clinical samples to examine cognitive content to those that used clinical samples to examine cognitive processes.

6. Results

6.1. Cognitive components of hypochondriasis

The mean *r* across the 21 studies that examined cognitive variables in health anxiety was .28 (95% CI = .21–.35; $Z = 7.77$, $p < .001$). However these studies were highly heterogeneous ($Q = 52.09$, $p < .001$, $I^2 = 61.60\%$). Across the 16 studies that examined cognitive contents the mean *r* was .30 (95% CI = .24–.37; $Z = 9.32$, $p < .001$). Although there was less heterogeneity when the analysis was limited to these 16 studies ($I^2 = 46.10\%$), there was still significant

Table 3
General information on studies that examined the relationship between hypochondriasis and somatosensory amplification

	N	Health anxiety measure	Effect size (r)
Clinical samples			
Barsky et al. (1990)	116	DSM III-R diagnostic interview	.53
Barsky et al. (1995)	118	Diagnostic interview	.56
Barsky and Wyshak (1989)	177	WI	.56
Brown et al., 1999 (sample 1)	40	DSM III-R diagnostic interview	.63
Brown et al., 1999 (sample 2)	89	WI	.50
Fabbri, Kapur, Wells, and Creed, (2001)	65	HAQ	.28
Haenen et al. (1997)	54	SCID	.52
Rief et al. (1998) (sample 1)	124	WI	.46[a]
Sayar, Kirmayer, and Taillefer (2003)	100	WI	.31
Nonclinical samples			
Aronson et al., 2001 (study 1)	81	IWS	.23
Aronson et al., 2001 (study 2)	131	IWS	.37
Ferguson et al., 2000 (study 2)	150	WI	.35
Ferguson et al., 2000 (study 3)	154	WI	.48
Gramling et al. (1996)	30	IAS	.49
Rief et al., 1998 (sample 2)	101	WI	.14[a]
Vervaeke, Bouman, and Valmaggia (1999)	57	GIAS	.43[b]

Note. Unless otherwise indicated, somatosensory amplification was assessed with the Somatosensory Amplification Scale. HAQ = Health Anxiety Questionnaire; GIAS = Groningen Illness Attitude Scale; IAS = Illness Attitude Scales; IWS = Illness Worry Scale; SCID = Structured Clinical Interview for DSM; WI = Whiteley Index.

[a] Somatosensory amplification was assessed with the Autonomic Sensations of the Cognitions About Body and Health Scale.

[b] Somatosensory amplification was assessed with the Body Consciousness Questionnaire.

Table 4
General information on studies that examined the relationship between hypochondriasis and in vivo measures of somatosensory accuracy or sensitivity

	N	Health anxiety measure	Dependent variable	Effect size (*r*)
Clinical samples				
Barsky et al. (1995)	120	Diagnostic interview	Awareness of resting heartbeat	−.08
Haenen et al. (1997)	54	SCID	Tactile Sensitivity (2-point discrimination)	−.11
Lautenbacher, Pauli, Zaudig, and Birbaumer (1998)	28	IAS	Heat pain thresholds	.09
Tyrer, Lee, and Alexander (1980)	60	Physician diagnosis	Correlation between subjective and objective heartrate	.19
Nonclinical samples				
Gramling et al. (1996)	30	IAS	Pain sensitivity (cold pressor task)	.33
Hadjistavropoulos et al. (1998)	192	IAS	Pain sensitivity (cold pressor task)	.22
Hanback and Revelle (1978)	43	WI and MMPI H_s	Auditory sensitivity and visual sensitivity on a two-flash fusion task	.22
Pauli, Schwenzer, Brody, Rau, and Birbaumer (1993)	28	IAS	Heat pain thresholds	.03
Steptoe and Noll (1997)	40	IAS	Estimates of sweat gland activity	−.36

Note. IAS = Illness Attitude Scales; MMPI H_s = Minnesota Multiphasic Personality Inventory – Hypochondriasis Scale; SCID = Structured Clinical Interview for DSM; WI = Whiteley Index.

heterogeneity (Q=27.83, p=.02). An analogue of the one-way ANOVA with sample type as the moderator indicated that much of this heterogeneity could be accounted for by whether the study used a clinical or nonclinical sample (Q_B(1)=4.52, p<.05; Q_W(14)=12.95, *ns*). Although both types of samples yielded significant effect sizes, the 7 cognitive content studies that used a clinical sample yielded a significantly larger effect size (r=.39; 95% CI=.29–.50; Z=7.57, p<.001) than the 9 studies that used nonclinical samples (r=.26; 95% CI=.19–.33; Z=7.07, p<.001).

The mean r across the 11 studies that used clinical samples to examine cognitive variables was .30 (95% CI=.16–.44; Z=4.15, p<.001). Once again there was significant heterogeneity across these studies (Q=35.57, p<.001, I^2=71.89%). An analogue of the one-way ANOVA indicated that much of this heterogeniety could be accounted for by whether the study examined cognitive contents (i.e., beliefs or assumptions about illness) or processes (e.g., memory for illness-related words) (Q_B(1)=8.81, p<.01; Q_W(9)=10.75, *ns*). The 7 cognitive content studies that used a clinical sample yielded a significantly larger mean effect size (r=.39) than the 4 cognitive process studies that used clinical samples (r=.08; CI=−.10–.25; Z=.86, *ns*). The one study that examined cognitive processes in a nonclinical sample (Owens et al., 2004) yielded a very large effect size (r=.46) that was inconsistent both with other studies that examined cognitive processes and with those that used nonclinical samples. It was also inconsistent with the other study that had used Stroop interference as its DV (Lecci & Cohen, 2002).

Overall, there appears to be clear and consistent evidence that individuals high in health anxiety hold dysfunctional beliefs and assumptions about illness-related constructs and that this relationship is stronger when clinical samples are studied than when nonclinical samples are used. On the other hand, the few studies that examined cognitive processes yielded highly variable results and the overall effect size for the process studies that used clinical samples was not significant.

6.2. Somatic perception and amplification

The first analysis examined the overall effect size for the 16 studies that assessed the relationship between health anxiety and scores on self-report measures of somatosensory amplification (usually the SAS). The overall relationship was large (r=.42; 95% CI=.36–.49; Z=12.66, p<.001). However these studies were marginally heterogeneous (Q=24.54, p<.06, I^2=38.87%). Once again, an analogue of the one-way ANOVA with sample type as the moderator indicated that much of this heterogeneity could be accounted for by whether the study used a clinical or nonclinical sample (Q_B(1)=5.51, p<.02; Q_W(14)=13.96, *ns*). The 9 SAS studies that used a clinical sample yielded a significantly larger effect size (r=.49; 95% CI=.41–.56; Z=12.55, p<.001) than the 7 studies that used nonclinical samples (r=.35; 95% CI=.26–.433; Z=8.00, p<.001).

In contrast to these very robust effect sizes, the 9 studies that actually measured accuracy of bodily perception or pain sensitivity yielded a small nonsignificant overall effect size (r=.06; 95% CI=−.08–.20; Z =.84, *ns*). These studies were also highly heterogeneous (Q=18.68, p<.02, I^2=55.88%), most likely due to the variety of dependent measures that were used. Therefore, although individuals higher in health anxiety clearly self-report higher levels of somatosensory amplification, there is little evidence that they are actually more accurate in their bodily perceptions or more sensitive to physical sensations.

Not surprisingly, an analogue of the one-way ANOVA revealed that the 13 studies that solely examined the correlations between health anxiety and self-reported amplification yielded significantly larger effects sizes than did the 9 studies that measured in vivo accuracy or sensitivity ($Q_B(1)$=21.80, p<.001; $Q_W(20)$=20.46, *ns*). Furthermore, it is worth examining the three studies (Barsky et al., 1995; Gramling et al., 1996; Haenen et al., 1997) in which participants completed the SAS and also participated in in vivo procedures to measure their somatosensory accuracy and sensitivity. All three studies yielded large correlations between health anxiety and the SAS (.49–.56). However, whereas Gramling et al. found a relation between health anxiety and pain sensitivity (N =30; r=.33), Barsky et al. found no relation between hypochondriasis and heart rate detection (N=*120;* r=−.08) and Haenen found no relationship between hypochondriasis and tactile sensitivity (N=54; r=−.11).

7. Discussion

The aim of these meta-analyses was to examine the empirical support for the cognitive/perceptual models of hypochondriasis/health anxiety that have been proposed by Salkoviskis and Warwick (1986, 2001) and Barsky (1992, 2001). Whereas some aspects of these models have received strong empirical support, for other aspects there has either been insufficient research or the extant research has yielded inconsistent results.

7.1. Cognitive variables in health anxiety

Overall, there was consistent and robust evidence that hypochondriacal and health-anxious individuals have different beliefs and assumptions about health and illness than do those with low levels of health anxiety. Despite a wide variety of methodologies and dependent variables, virtually all of the studies that examined beliefs and assumptions in health anxiety yielded moderate to large effect sizes. Thus, for example, individuals higher in health anxiety have a tendency to make catastrophic interpretations of bodily sensations and symptoms (e.g., Hadjistavropoulos et al., 1998; Rief et al., 1998), to assume that in order to be healthy one must be without any physical symptoms (Barsky et al., 1993), and to believe that they have less control over the recurrence of illness (Ferguson et al., 2000). Furthermore, although the clinical samples yielded larger effect sizes than the nonclinical samples, there was evidence for these hypochondriacal beliefs and assumptions regardless of whether the participants were sampled from clinical populations and diagnosed with hypochondriasis or whether they were college students who scored higher on a self-report measure of health anxiety.

These findings provide strong support for the cornerstone of the cognitive-behavioral model of hypochondriasis and health anxiety. However, they also raise a question about the line between description and explanation. Inaccurate beliefs that one has, or is in danger of developing, a serious illness is part of the Diagnostic and Statistical Manual of Mental Disorders (4th ed.) definition of hypochondriasis. So a failure to have found that health-anxious individuals have catastrophic thoughts about physical symptoms or that people with hypochondriasis overestimate the frequency of serious illnesses would have come as a surprise. In other words, some of the dependent variables in these studies come very close to overlapping with the diagnostic criteria for hypochondriasis or with items from the scales that were used to assess health anxiety. For example, a number of studies that examined catastrophic interpretations of bodily symptoms (e.g., Hitchcock & Mathews, 1992; Marcus, 1999; Marcus & Church, 2003) assessed health anxiety using the IAS, which has items inquiring whether respondents are afraid that they may have cancer or heart disease Thus, individuals who interpret tightness in their chest as a likely indicator that they are having a heart attack (Hitchcock and Mathews) or who think that a person with chest pain is likely to be having a heart attack (Marcus) are also more likely to report that they are afraid that they might have heart disease. Although the IV and DVs in these studies are not identical, there is certainly some conceptual overlap between them. Still, these findings suggest that researchers and clinicians should attend to the health-related beliefs of health-anxious individuals.

On the other hand, less direct attempts to identify cognitive process in health anxiety cannot be faulted for criterion overlap between their independent and dependent variables. Furthermore, such studies have the potential for identifying underlying processes that may serve as risk factors for health anxiety and hypochondriasis. However, these studies yielded much less consistent results and an overall effect size that was much smaller than the cognitive content studies. For example, although Pauli and Alpers (2002) found strong evidence that hypochondriacal patients were more likely to recall pain words than controls, the two studies by Brown et al. (1999) that examined recall of health-related words yielded inconsistent results. Similarly, although the largest effect size among the cognitive process studies was Owens et al.'s (2004) finding of Stroop interference for illness-related words, Lecci and Cohen (2002) only found this effect when they first induced illness-concern in their participants by telling them that their blood pressure was too high. In the control condition, which was analogous to the procedure of Owens et al., there was no Stroop interference for illness-related words.[3] Some of the positive findings in this area suggest that future research examining cognitive processes in health anxiety may prove fruitful, but there is not yet sufficient evidence to conclude that health-anxious individuals process illness-related materials differently from nonhypochondriacal individuals, even if the contents of their health-related beliefs do differ.

7.2. Triggers

The diathesis-stress component of the cognitive behavioral model of health anxiety, which proposes that various external or internal events can trigger hypochondriacal concerns in at-risk individuals (i.e., those who hold dysfunctional assumptions about health and illness) has been the least studied component of the model. With only three published studies, it was not appropriate to perform a meta-analysis on this component. Worse, the results of these studies were not consistent. Lecci and Cohen (2002) were able to trigger a Stroop interference effect for health-related words by getting health-anxious students to believe that they were at risk of serious illness, whereas Marcus' (1999) attempt to trigger increased anxiety in health-anxious students using a scrambled sentence task failed. Obviously, further research in this area is warranted, not only because it is a component of the model, but also because it has broader implications for how hypochondriasis is conceptualized and defined. Hypochondriasis is portrayed in the cognitive behavioral model as an episodic disorder with discrete events, much like panic disorder or major depressive disorder. However, if these hypochondriacal concerns are not so much latent contents that are triggered by events, but are instead chronic concerns, then hypochondriasis may be better conceptualized as being similar to generalized anxiety disorder or dysthymia. Karoly and Lecci's (1993) finding that college women with high health-anxiety scores report more health-related goals than women with lower scores is consistent with the possibility that health anxiety, and perhaps hypochondriasis, involves an ongoing state of mind and not a series of discreet events.

7.3. Health anxiety and somatosensory sensitivity

The results generally confirmed the conclusions that Barsky (2001) reached in his narrative review. Individuals diagnosed with hypochondriasis and/or those who score high on self-report health anxiety measures also report high levels of somatosensory amplification on the SAS and related measures: Hypochondriacal individuals believe that they are especially aware of and sensitive to their bodily processes. However, when researchers have attempted to assess somatosensory sensitivity and awareness in vivo, the results have been mixed. This heterogeneity is likely explained by the variety of ways in which amplification and sensitivity has been operationalized. First, sensitivity and accuracy may be two separate constructs. An individual may be more sensitive to pain or pressure and yet be no more accurate at guessing her heart rate or sweat gland activity. Second, different aspects of sensitivity (or accuracy) may not be related to health anxiety in the same way. For example, although there was no a priori reason to expect health-anxious individuals to be more sensitive to cold than to heat, the two studies (Gramling et al., 1996; Hadjistavropoulos et al., 1998) that measured pain sensitivity using a cold pressor task both yielded moderately large effect sizes, whereas the two studies that assessed heat pain thresholds (Lautenbacher et al., 1998; Pauli et al., 1993) did not find a relationship between health anxiety and pain sensitivity.

[3] Although the Lecci and Cohen (2002) study did not provide the necessary data for inclusion in the meta-analysis, the figures in the article indicate either near zero or negative effect sizes for Stroop interference for health related words in the control condition.

Although these studies leave open the possibility that health-anxious individuals may be more sensitive to some aversive stimuli, examination of Table 4 suggests that there is little evidence of a positive relationship between health anxiety and the ability to perceive autonomic processes accurately. Although this pattern of findings suggests that the concept of somatosensory amplification may need to be modified (at least as it is applied to health anxiety), it is certainly consistent with a cognitive approach to health anxiety. Health-anxious individuals' possibly greater sensitivity to certain discomforts may lead them to ascribe more serious connotations to benign but unpleasant sensations that might not be noticed by others. Furthermore, because they mistakenly believe that their perceptions of their own autonomic processes are especially accurate, they may be less likely to be dissuaded from their concerns that something is wrong.

7.4. Specificity and continuity

The results also bear on issues that were not the primary foci of this paper, namely the specificity of this cognitive behavioral model to health anxiety, and whether hypochondriasis is a discrete condition or whether it exists along a continuum. Because very few studies partialed out other relevant constructs such as state anxiety or worry (e.g., Marcus & Church, 2003) or compared a hypochondriacal group to another group with an anxiety or somatoform disorder (e.g., MacLeod et al., 1998; Pauli & Alpers, 2002), these few effect sizes were not included in the meta-analyses. Instead, when zero-order correlations were provided, those values were included in the meta-analyses, and when there were multiple comparison groups, the effect size from the comparison between the hypochondriacal group and the nonclinical group was used. Therefore, although the results of these analyses suggest that hypochondriacal individuals have different beliefs and assumptions about health and illness and that they report higher levels of subjective somatosensory amplification, they do not indicate whether these results are unique to hypochondriasis or whether they are also found in related conditions. Marcus and Church (2003) found a significant relationship between health anxiety scores and estimates of the probability that ambiguous symptoms are indicative of catastrophic illnesses, even after partialing out negative affect, trait anxiety, worry, and other related constructs. Similarly, MacLeod et al. found that compared to anxious patients who were not hypochondriacal, patients who were both anxious and hypochondriacal gave more somatic explanations for common bodily sensations.

Despite this promising evidence of specificity, given the high levels of comorbidity between hypochondriasis and related anxiety disorders (Noyes, 1999) and the conceptual overlap between the cognitive behavioral model of hypochondriasis and similar models of panic disorder, further research examining the specificity of this model to hypochondriasis is warranted. For example, Salkovskis and Clark (1993) have proposed that the difference between hypochondriasis and panic disorder is that hypochondriacal individuals are primarily concerned with the significance and long-term consequences of their symptoms, whereas individuals with panic disorder mainly focus on the perceived immediate consequences of their anxiety symptoms. Consistent with Salkovskis and Clark's suggestion, hypochondriacal patients scored significantly higher than panic disorder patients on the IAS question "Are you afraid that you might have cancer?", but the two groups did not differ on the question about fearing heart disease (Hiller, Leibbrand, Rief, & Fichter, 2005).

A related issue is whether hypochondriasis is better understood as a dimensional construct or as a discrete diagnostic entity. The cognitive behavioral model (Salkovskis & Warwick, 2001) presupposes that subclinical health anxiety and hypochondriasis exist on a continuum, and there is some evidence to support a dimensional structure (e.g., Barsky, Wyshak, & Klerman, 1986; Kirmayer, Robbins, & Paris, 1994). Furthermore, the results of these meta-analyses appear to be consistent with a dimensional hypothesis. On average, clinical samples yielded larger effect sizes than nonclinical samples, but even studies that used nonclinical samples to examine cognitive contents and self-reported somatosensory amplification produced average effect sizes that were significant. Still, a definitive answer to the question of whether hypochondriasis exists as a category or on a continuum awaits taxometric analyses (Waller & Meehl, 1998) specifically designed to address the question of latent structure.

7.5. Conclusions

Compared to cognitive models of anxiety disorders like panic disorder, the empirical study of health anxiety and hypochondriasis is in its infancy. However, the results of these meta-analyses suggest that this model holds

considerable promise. There is ample evidence that health-anxious individuals have different beliefs about health and illness compared to those low in health anxiety. At this point, future research that simply compares the beliefs of those with high health anxiety to a comparison group may not add much to extant literature. Instead, studies that identify beliefs that differentiate health-anxious individuals from those with other anxiety and mood disorders (especially panic disorder), and studies that identify underlying cognitive processes in health anxiety, may help further develop this cognitive behavioral model.

Similarly, the relationship between health anxiety and self-reported somatosensory amplification is clearly well-established. Yet, questions remain about whether health-anxious individuals are more sensitive to bodily sensations, even if their perceptions of autonomic processes are not more accurate. Finally, the idea that various triggers can elicit hypochondriacal concerns in individuals who hold latent health-related beliefs is one of the most intriguing but least studied aspects of this cognitive behavioral model.

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