Hypochondriasis (Health Anxiety) Reformulated: Similarities With Generalized Anxiety Disorder in A Routine Setting Single Case

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Abstract
Despite its long documented history, Hypochondriasis remains a ubiquitous enigma to virtually all health care disciplines and providers. Because such highly health anxious patients are not commonly open to psychological (i.e., non-medical) interpretations of their physiological symptoms, sufferers ironically often become fixed on a pathological quest for physical and/or medical explanations for their problems. This pervasive worry and worry-based behavioral pattern perpetuates and reifies the belief that one has- or will develop- a serious medical condition, and, due to its inappropriate fixity on perceived organic (versus psychic) etiology, leaves such excessive doubt largely untreated. Therefore, continual high medical utilization and costs persist, and profound human suffering goes largely unchecked and unmanaged. Collectively, this sad, but very real condition and circumstance is unacceptable. Fortunately, this oft recalcitrant and thorny phenomenon has recently been construed as perhaps being similar to another such condition, namely Generalized Anxiety Disorder. If this resemblance is more than a surface relationship, and the two phenomena are indeed related on a process level (i.e., the two share a common pathological core), then Hypochondriasis should be treatable via a specifically tailored treatment for Generalized Anxiety Disorder. Since Generalized Anxiety Disorder has been far more responsive to intervention than Hypochondriasis, as its central mechanism of pathology is now well understood (i.e., intolerance for uncertainty), it would be conceivable that Hypochondriasis is in actuality far more treatable than its current riddle-status would otherwise suggest. Indeed, if the two prove to be quite similar, if not outright forms of one another, then there would be far more hope that patients who suffer with this can be helped. This stirring hypothesis was tested by treating a particularly refractory, chronic and severe case of Hypochondriasis with a specialized Generalized Anxiety Disorder treatment package. "Joe" responded quite favorably to treatment, and obtained meaningful change according to Jacobson and Truax’s (1991) method for determining clinically significant- and reliable- change. In fact, according to their most restrictive standard, Joe could be labeled “Recovered” at the end of treatment, and at all 18-month follow-up points. His response, caveated around single case generalizability limitations, suggested that perhaps Hypochondriasis does indeed share a common etiological source, namely intolerance for uncertainty, with Generalized Anxiety Disorder; if so, then that would be highly explanatory of the strong results. Recommendations are given for conceptualizing pathological forms of health-related fears as just that, a concrete form of an anxiety disorder rather than a Somatoform condition. Suggestions for future research direction are provided.

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HYPOCHONDRIASIS (HEALTH ANXIETY) REFORMULATED: SIMILARITIES WITH GENERALIZED ANXIETY DISORDER IN A ROUTINE SETTING SINGLE CASE

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HEIDI J. MEEKE

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DOCTOR OF PSYCHOLOGY

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Abstract

Despite its long documented history, Hypochondriasis remains a ubiquitous enigma to virtually all health care disciplines and providers. Because such highly health anxious patients are not commonly open to psychological (i.e., non-medical) interpretations of their physiological symptoms, sufferers ironically often become fixed on a pathological quest for physical and/or medical explanations for their problems. This pervasive worry and worry-based behavioral pattern perpetuates and reifies the belief that one has- or will develop- a serious medical condition, and, due to its inappropriate fixity on perceived organic (versus psychic) etiology, leaves such excessive doubt largely untreated. Therefore, continual high medical utilization and costs persist, and profound human suffering goes largely unchecked and unmanaged.

Collectively, this sad, but very real condition and circumstance is unacceptable. Fortunately, this oft recalcitrant and thorny phenomenon has recently been construed as perhaps being similar to another such condition, namely Generalized Anxiety Disorder. If this resemblance is more than a surface relationship, and the two phenomena are indeed related on a process level (i.e., the two share a common pathological core), then Hypochondriasis should be treatable via a specifically tailored treatment for Generalized Anxiety Disorder. Since Generalized Anxiety Disorder has been far more responsive to intervention than Hypochondriasis, as its central mechanism of pathology is now well understood (i.e., intolerance for uncertainty), it would be conceivable that Hypochondriasis is in actuality far more treatable than its current riddle-status would otherwise suggest. Indeed, if the two prove to be quite similar, if not outright forms of one another, then there would be far more hope that patients who suffer with this can be helped. This stirring hypothesis was tested by treating a particularly refractory, chronic and severe case of Hypochondriasis with a specialized Generalized Anxiety Disorder treatment package. “Joe”
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Hypochondriasis (Health Anxiety) Reformulated: Similarities with Generalized Anxiety Disorder in a Single Case in a Routine Setting

I have a tumor in my head the size of a basketball. I can feel it when I blink.

- Woody Allen, Hannah and Her Sisters

**Statement of the problem**

Health Anxiety (HA), still formally known as Hypochondriasis (HS) in the *Diagnostic and Statistical Manual of Mental Disorders – Text Revision* (DSM-IV-TR; American Psychiatric Association, 2000), represents an enigmatic quandary for health care providers of various disciplines. Anxiety occurs as a normal response to the perception that a particular situation or stimulus is threatening or dangerous, if there is doubt in one’s ability to cope with and manage the perceived threat (Beck & Emery, 1985). Although it is not unusual for people to respond with mild and fleeting anxiety to illness, HA typically takes a different and often chronic, debilitating, and recalcitrant course (Hiller, Leibbrand, Rief, & Fichter, 2002). For those who suffer with clinically-elevated health fears, worries about having or contracting serious diseases or illnesses become ubiquitously problematic, as sufferers are commonly not open to psychological interpretations of physiological symptoms (Asmundson, Taylor, & Cox, 2001). Instead, medical and physiological explanations are erroneously and aggressively sought by health anxious patients because they have a difficult time believing or integrating the medical reality that there is typically nothing “wrong” per se, and that they are, instead, in fact medically healthy (Abramowitz & Braddock, 2008). Indeed, they often seem unable to accept that their somatic complaints are largely a function of overactive imaginations, or, in the case of actual symptoms,
a product of somatization and general stress. This may in part be due to the fact that people have
greater problems with processing information which is absent (called underweighting of negative
information), like negative medical findings; on the other hand, they normally have relatively
little trouble processing present information (called overvaluing of positive information), like
focusing on bodily complaints (Rassin, Muris, Franken, & van Straten, 2008). This irony leads
HA patients on pathological quests for explanations which better fit their presumed disease
etiology (Langois & Ladouceur, 2004; Walker, Vincent, Furer, Cox, & Kjernsited, 1999).

However, since there is actually nothing seriously wrong, but such patients keep seeking medical
answers nonetheless, this serious phenomenon goes largely untreated, psychologically.

De facto, such unfortunate and inappropriate quests leave these patients on a typically
chronic or unremitting course, marked by high medical utilization and often gross functional
impairment. The burden to healthcare systems, on providers who attempt to “help,” and the sheer
and unrelenting suffering endured is not only unacceptable, but is in actuality also (and more
importantly) unnecessary. To bring this point home, one needs only look more closely at the
associated medical costs, which are nine fold that of an average person (Smith, Monson, & Ray,
1986). While such an inflation might at first glance be acceptable, it quickly becomes not
suitable once it is understood that the major part of such high costs are almost exclusively (91%)
due to medical investigation (i.e., tests) and not due to psychiatric or psychotherapeutic treatment
(Rost, Kashner, & Smith, 1994). Additionally, inflated medical utilization rates are observed
when health care providers have difficulties accurately detecting the presence of anxiety
disorders (Fleet et al., 1996), which often, ironically, mimic various medical conditions.

Consequently, there are often a high number of medical tests administered to HA patients, as
providers often keep looking for that elusive “something” (i.e., a medical etiology of the
Hypochondriasis, which is in actuality not there. The economic burden of anxiety disorders in the United States alone is an estimated and staggering $42.3 billion dollars annually, which alarmingly represents almost one-third of the country’s total $148 billion mental health bill (Greenberg, Sitisky, & Kessler, 1999). Given that HA is common in medical practice (Clark, Salkovskis, Hackmann, Wells, Fennel, Ludgate, et al., 1998), representing 40% of patients on neurology wards and 26% of patients in family medicine (Rief, Hiller, & Margraf, 1998), accurately and quickly detection, triaging, and correctly treating HA is paramount (Deacon, Lickel, & Abramowitz, 2008; Roy-Byrne et al., 2002). After all, more than 22.84 billion of annual U.S. healthcare costs are associated with repeated (i.e., unnecessary) use of healthcare services by anxiety disordered people who seek relief from their symptoms that mimic physical illness, leading them to seek such services at three to five times that of non-anxious patients (Greenberg, Sitisky, & Kessler, 1999). Prevalence rates of HS among medical outpatients have been reported as ranging from 4.2 to 7.7% (Walker, Vincent, Furer, Cox, & Kjernsited, 1999), so statistics over the presence of the phenomenon varies. While only 4-5% of the general population exhibits somatoform symptoms, HA patients account for upwards of 50% of adult ambulatory health care costs (Lecci & Cohen, 2002), and unexplained medical symptoms account for up to 50% of all patient visits in primary care (Barsky & Borus, 1995). Unfortunately, the central pathological mechanism, or exact etiology of HA has not been well understood to date (Hiller, Leibbrand, Rief, & Fichter, 2002), limiting knowledge about central pathogenic and biological etiological processes, and as a result, appropriate care has not consistently been delivered to such patients, in a way that they are better able to accept psychological help in ameliorating their misguided fear. Indeed, substantial financial burdens generated by excessive and unnecessary medical utilization could be lessened by providing timely and empirically-supported treatment
(Barlow, 2002), but to date the prognosis has been considered relatively poor (Fallon, Klein, & Liebowitz, 1993).

Fortunately, more recent developments and ideas about this construct, and adaptations from novel models of Generalized Anxiety Disorder (GAD) have fared much better, both in terms of outcome and acceptability, offering much needed hope and direction to HA sufferers (e.g., Dugas, Gagnon, Ladouceur, & Freeson, 1998; Dugas, Ladouceur, Leger, Freeston, Langlois, Provencher, et al., 2003; Langlois & Ladouceur, 2004; Walker, Vincent, Furer, Cox, & Kjernisted, 1999; Wattar, Sorensen, Buemann, Birket-Smith, Salkovskis, Albertsen, et al., 2005).

**Literature review**

The term *Hypochondriasis* can be traced back to the early second century (Fischer-Homberger, 1970), and the term *hypochondria* reportedly dates back to approximately 350 BC (Asmundson, Taylor, & Cox, 2001), so the notion of what defines HA is not new per se. Lecci, Karoly, Ruehlman, and Lanyon (1996) interestingly reported that HS stems from the omnibus term *hysteria*, which in itself begins to shed some light to the multifaceted nature and complex picture that this phenomenon represents. To make matters worse, this particular combination of illness conviction, preoccupation, and fear (the overall cognitive pattern inherent to HS) has proven so highly resistant to intervention that it commonly results in a deterioration of doctor-patient relations (WHO, 1992). This often becomes particularly true after the practitioner does not confirm the hypochondriac’s convictions and concerns about his or her unverifiable “illness” (Lecci, Karoly, Ruehlman, & Lanyon, 1996). This is not necessarily a uni-directional issue either (i.e., patients rejecting doctors), as few practitioners even begin to attempt interventions because the phenomenon is typically defined and understood as a “treatment resistant” illness (Kellner,
So, once identified and labeled as so challenging, or sometimes even as “contrarian” and recalcitrant, healthcare professionals who otherwise work hard to help patients may withdraw support and help from sufferers; after all, there is no disease process (besides pathogenic thinking) and such patients are thought unresponsive to negative findings and medical reassurance anyways. Put simply, the average health care provider simply does not know how to “convince” such patients of their (good) health status differently than reporting their good health to the patient.

Hypochondriacal tendencies are presumed a combination of hard-wiring (i.e., heightened perceptual sensitivity or physiological reactivity) and modeling (i.e., social learning) that reinforce abnormal illness patterns (i.e., early exposure to medical settings, doctors, and/or information). Whatever the etiological reasons, health anxious individuals typically surround themselves with medically sensitive people (e.g., doctors, nurses, health care professionals, other hypochondriacs, and [surprisingly] sick people) as it serves to reinforce (maintain) one’s attention to illness (Lecci, Karoly, Ruehlman, & Lanyon, 1996). Most importantly, in arguing for an integral connection between HA and a predisposition to interpret events and stimuli in a negative fashion, Lecci, Karoly, Ruehlman, and Lanyon (1996) reported that hypochondriacs may have found their “niche” by gradually focusing their negative appraisals within the health domain (i.e., “specializing” their enduring predisposition to worry). Pennebaker and Watson (1991) reported that such a focusing process may be cultivated through differential reinforcement, underlying somatic conflicts, or because health is merely a convenient worry target. Nonetheless, this process serves to limit the scope of worry down to the central theme of health and well-being, and will make this focus virtually exclusive with time.
Rief, Hiller and Margraf (1998) reported that, as a group of patients, the phenomenon exhibited by HA patients is characterized by extremely high levels of psychological distress, perhaps accounting, to a degree, for why such patients seek services at unusually higher rates than conventional anxiety disordered patients. This heightened distress is also promoted by patients’ belief that good health is demonstrable of being relatively symptom free and, perhaps more pivotally, such patients often consider “symptoms to be equal to sickness” (p. 587). Indeed, they suggested that this style of rigid and absolute cognition may very well be an important, if not a central, part to the vicious circle that maintains somatic symptoms. Since catastrophic interpretations of body perceptions (e.g., bodily cues are always a sign of disease) can readily be understood as pathogenically problematic, it is not difficult to guess that hypochondriacally-fixed beliefs produce a very restrictive concept of health, in which it is can be far easier to be ill than well. Of course, this does not play out in the sheer health statistics about health problems in the world, vis-à-vis generally prevailing neutral or good health in the general populous. On the other hand, fear has not been well-known to necessarily conform to the laws of logic or reason, or to statistics for that matter, and this issue is certainly exacerbated in clinical anxiety conditions of all types. In this fashion, frequent but typically non-dangerous (i.e., benign) symptoms are readily interpreted as signs of disease, illness and impending doom, gloom or death amongst HA patients (Hitchcock & Matthews, 1992; Salkovskis, 1989). Ironically, Rief, Hiller and Margraf reported, HA patients do not take better precautions with their health—they simply worry more about it, which is how GAD patients treat real-life problems too (i.e., they worry a lot about issues, which technically could be fixed, but tend not to do anything actively to affect the “problem”). If anything, through a process of negative reinforcement, these patients often become avoidant of behaviors that produce physical sensations, so they commonly reduce
physical exercise and thereby fitness, which may actually serve to enhance the probability of body misperceptions (through increased physiological cues with decreased fitness and increased pathogenic thinking with increased stress and deficits in coping mechanisms).

Greeven, van Balkom, Visser, Merkelbach, van Rood, van Dyck, et al. (2007) reported that in light of the hypersensitivity accompanying HS it is surprising that pharmacological treatment is scarce and results ambiguous. Pharmacological treatment is commonly used to treat physiological and emotional reactivity, and since HA indeed loads on both factors it is surprising to learn that there is not more research apparently done with pharmacological interventions. However, Walker, Vincent, Furer, Cox, and Kjernisted (1999) suggested that this may occur because HA patients are sensation intolerant, and many medications commonly used to treat physiological and emotional reactivity (e.g., selective serotonin reuptake inhibitors [SSRIs]) can have, at least initially, negative side-effect profiles which amplify uncomfortable physical sensory information (e.g., stomach discomfort, headaches, blurry vision, heart palpitations). Without patients being willing to take pharmacological preparations, it would be next to impossible to conduct human subjects research with such substances, much less use them to treat patients who are likely to attribute fearful outcomes to benign physical symptoms, which could be experienced by even non-sensitive patients. Nonetheless, Greeven and colleagues still found that CBT was more effective than medications, which were still considered effective. Interestingly, paroxetine (Paxil) was not found more effective than placebo, lending additional support to the notion that perception, belief, and expectancy is influential in illness worry. Indeed, since there was no difference between drug and placebo, then it can be assumed that if the patient believed they would be better (and that produced a desirable effect) the power of thoughts alone is as potent as an active ingredient drug; taken together, this would translate to
thinking influencing illness perception and behavior (the very definition of this health focused condition).

Clark, Salkovskis, Hackmann, Wells, Fennell, Ludgate, et al. (1998) also reported that HS is as common as it is difficult to manage in medical practice, making it one of the more difficult conditions to treat, especially since there has been no commonly accepted, empirically validated treatment that seems consistently to work well for such patients. Nonetheless, they reported, it seems that a mixture of cognitive and behavioral techniques can modify patients’ beliefs and factors that maintain such beliefs. They proposed that any treatment for this vexing phenomenon ought necessarily to focus on helping patients realize that their problem is worrying about illness, rather than illness per se. Interestingly, they reported a broad mixture of techniques, such as: cognitive techniques (e.g., identifying and challenging patients’ evidence for their misinterpretations of symptoms and signs), behavioral experiments (e.g., inducing symptoms deliberately by focusing on various body parts or by dwelling on fearful thoughts), graded exposure (e.g., approach avoided illness-related situations), response prevention (e.g., stop bodily checking and reassurance seeking), and psychoeducation regarding the meaning of symptoms and of various medical interventions and opinions. While this approach seems plausible enough, and appears to cover the phenomenon well, the sheer breadth of interventions begs the question as to whether the true mechanism of HS is actually understood. By doing so much (i.e., using so many different interventions), it seems plausible that HS could be treated, as well as many other worry-related issues, because such a broad net is cast; however, it seems equally plausible that this approach does not, specifically, target a central core mechanism. Is this because such a mechanism continues to elude researchers, thereby requiring practitioners to use various broad techniques to try and capture the gist of what the problem is made up of? The
particular CBT approach they endorsed did appear to have some slippage, or deterioration, between their six- and 12-month follow-ups, reinforcing the distinct possibility that the phenomenon is not entirely understood. The evidence for this position can clearly be found in the sheer difficulty in its management. Without directly admitting it, Clark and colleagues did suggest that more research would be needed to understand which treatment components are more important, and specifically what the, “mechanism of their action” (p. 224) is. Roughly stated, how one treats HS is not understood because HS is not understood.

Walker, Vincent, Furer, Cox, and Kjernisted (1999) addressed the issue of acceptability of CBT amongst people diagnosed with HS. In their exploratory study, they investigated choices that health anxious participants would make between medication treatment or CBT. The participants of this investigation were presented with balanced descriptions of both medication and CBT for HS, including time commitments required as well as major advantages and disadvantages of each approach. Walker et al. found that people with HS consistently chose CBT. Participants also judged CBT to be more effective in both the short and long term than medications. Surprisingly, subjects rated CBT as more “acceptable,” with 74% choosing psychological over medication (4%). Another 48% reported they would only accept psychological treatment (i.e., medication was perceived as unequivocally “bad,” even when the treatments were described as equally effective). Although this response is compelling, it may be that the biological treatment approach (i.e., medications) fed directly into pathological fear about internal, somato-sensory experiences produced by negative side-effect profiles of medications. CBT is not typically associated with this type of physiological effect; as such, the “strong preference for psychological treatment,” (Walker et al., 1999, p. 255) might be tied to the pathological mechanism of the condition itself. Indeed, the most common reason for declining
participation was an unwillingness to take active medication, but a smaller number of potential participants also refused to take even a placebo, lending support to the notion that there is a strong fear of putting something into the body which might produce somato-sensory feedback (i.e., patients are phenomenologically unwilling to experience physiological messages, a sort of intolerance for uncertainty). Thus, the actual treatability of HS remains questionable. Given that medication treatment is far more broadly available, nationally and internationally, this strong preference adds another layer of challenge to adequately helping these patients. Walker and colleagues lamented, in the de facto reality that CBT is not broadly available, that increasing the availability of, “effective psychological treatments may decrease costs to the consumer and to health care systems” (p. 255). Within the reality that many somatically-focused patients show unusually high drop-out rates, an unfortunate double-bind is created, further adding to the complexity of the situation for these patients and the systems which attempt to help them.

Recognizing this dilemma, Hiller, Leibbrand, Rief, and Fichter (2002) carefully examined the predictors of course and outcome in HS after CBT, citing that little is actually known about the natural course of HS. Nonetheless, they too cite that, in a broad sense, illness behaviors such as “doctor shopping” and excess utilization of health care are conceptualized as maintenance factors (due to operant conditioning and negative reinforcement, presumably). While CBT is thought to be effective for patients suffering from unclear medical symptoms, Hiller and colleagues expressed concern that not all patients respond to treatment or reach clinically relevant improvements, further supporting the idea that some central mechanism of HS is still not yet well understood. If only 27% of patients obtain remission following inpatient CBT (i.e., a more restrictive treatment alternative, fraught with challenge to autonomy and independence in recovery and wellness promotion), then the outlook may indeed not even be that
good for the average patient who is unlikely to receive inpatient care. Additionally, they suggested that severity of the disorder is a clear negative prognostic indicator, suggesting that perhaps only patients with fleeting or milder forms of the phenomenon may be adequately helped by CBT. Indeed, better outcome seems to be related to less medical morbidity, less comorbid depression and anxiety, shorter duration, lower “neuroticism” (harkens back to the nebulous notion of “hysteria,” or that there is just “something” different about the non-responders), less combination of somatization, amplification of sensations, and disease attributions, and, finally, being employed and married. Fundamentally, it would appear that if one displays much of any symptom of HA, then one is most likely quite sick (i.e., disordered and dysfunctional).

Lecci and Cohen (2002) also points to the nebulous “neuroticism” as a risk factor for “reporting unfounded symptoms” (p. 147). While leaving the nature of this ambiguous concept undefined, they addressed how this general style is exemplified by a broad readiness to adopt illness beliefs and concerns, which is contrary to nonclinical population tendencies to believe instead they are healthy unless confronted with overwhelming evidence to the contrary. This strong HA bias towards believing they are unhealthy leads such patients to exhibit a selective attention to physical symptoms, often in conjunction with a negative affective experience. Attention has been proposed as one pathway to forming illness beliefs (Barsky & Klerman, 1983). Through this attending (i.e., “listening”) process, they are better able to detect somatic sensations, which in turn, for straight forward reasons, result in a clear bias toward perceiving various bodily symptoms. Perceptual biases therefore seem implicated in hypochondriacal thinking. Lecci and Cohen did report an interesting phenomenon, which may have important implications for how the condition is adequately treated. Namely, they found that the hypochondriacal preoccupation and fear did not emerge, unless illness concern was activated;
therefore, it may be relevant to know how to turn off illness concern, or to know how to prevent it from turning on to begin with (i.e., a better understanding of the process of activation is needed). It appears that “acute listening” may be part of the problem, because when patients do so, then illness concerns are activated and an illness-specific perseveration effect is observed. One plausible culprit in this HA phenomenon can thus be implicated as being attention, which can be an automated process (i.e., making deliberate intervention more complicated than if the behavior was voluntary). Because this monitoring process may be more automated, it then becomes potentially clearer why erroneous somatic beliefs can persist despite the openness of such beliefs to medical disconfirmation. Indeed, these tendencies can exist outside conscious awareness and control, so despite doctors’ (medical) reassurance this heightened somatically focused attention can create and exacerbate symptoms. This also fits previous research which illustrates that emotionally disordered people are prone to bias their attention toward threatening, emotionally relevant stimuli (MacLeod & Matthews, 1991).

Looper and Kirmayer (2002) also addressed this issue with needing a far better understanding of the mechanisms of symptom generation and amplification, as such normally cannot be directly observed but only inferred by patient history, illness behavior, and social circumstance. They reported that this lack of understanding is, in part, what accounts for why somatic complaints pose such a challenge for clinicians. They also reported that these patients, with unexplained medical complaints, experience unusually high levels of distress, which results in repeated requests for medical care. While physicians may at first attend to ruling out treatable medical conditions, and when none are found, it is not unusual to shift toward containment of excessive help-seeking behavior. This physician behavior, albeit understandable and medically supportable, contributes to an inevitable deterioration in the doctor-patient relationship, as
patients can sense that their care provider has lost interest in them or (worse) have come to view them as “vexatious,” and thereby may start shopping for another doctor. Sometimes this deterioration also occurs as the result of referring the patient to psychological and/or psychiatric treatment, as this may signal (not entirely incorrectly so) that the physician is calling into question the bodily reality of symptoms which patients are, by definition, convinced to be real and dangerous. Additionally, such patients may then also experience a certain level of stigma associated with having a psychiatric (versus medical) diagnosis (Kirmayer, 2000). Looper and Kirmayer suggested that one way to circumvent this particularly alienating and potentially stigmatizing scenario is to use behavioral medicine and symptom-focused CBT, as neither necessarily assumes a psychological etiology of the illness, thereby making these approaches “safer” to such patients. These approaches are presented as means of coping with physical problems, and can be additionally enhanced in the context of “shared-care” between mental health care providers and medical professionals. While this approach may assist in making treatment more palatable to prospective patients, it becomes nonetheless important to point out that “the size of the literature does not yet warrant a formal meta-analysis,” (p. 811) so it is not clear whether this common sense approach will actually ultimately be supported as improving treatment compliance and outcome. Looper and Kirmayer do make another interesting, if not diagnostically compelling argument; that is by definition, a diagnosis of HS requires that patients have received adequate reassurance, but that is not always the case. Therefore, they argue, any treatment of HS should begin with the provision of effective reassurance, something that is not without controversy but that at least some researchers agree with (Starcevic, 1991). There is even a CBT-esque treatment approach, called “Explanatory therapy,” (Kellner, 1982, 1983; Kellner & Sheffield, 1971) developed around this principle of information provision, but without long-term
follow it is difficult to tell what true effect this approach actually has. A last, but important, point they made was that the treatments they reviewed all used multiple treatment strategies, which does limit detection of differential treatment effects, and it adds to the notion that since a multitude of interventions are woven together it suggests that the exact nature (i.e., etiology, mechanisms of pathology, and change) is not yet completely understood.

Warwick (2004) takes this unstated truth (i.e., HS is not well understood yet), and suggested that a better way to understand it would be to think of it, in principle, as an anxiety disorder. Indeed, she asserted that HS may be more appropriately classified as an anxiety disorder (“severe HA”: Warwick & Salkovskis, 1990), which conceptually makes sense as the phenomenon “behaves” very much like anxiety disorders do, and particularly GAD. This suggestion is an important one, as there has been no empirically-supported treatment (EST) for HS, but there are many available for anxiety disorders of various sorts. If it indeed is true that HS could be conceptualized as an anxiety disorder, then it could seem plausible that HS would be more responsive to specifically anxiety-tailored protocols (Bolduc, Freeston, Mainguy, Marchand, & Todorov, 1999). This might be especially promising if a GAD-tailored approach was used, with an adjustment made for the “specialized focus” that HS would exhibit in comparison to GAD patients behavioral pattern (i.e., to worry about everything under the sun). In contrast to Looper and Kirmayer’s (2002) assertion that treatment should begin with adequate reassurance provision, Warwick argued, along the lines of how anxiety disorders are thought to function, that a reassurance provision functions as an operant reinforcer, and only leads to a longer-term increase in, or exacerbation of HA and instead actually maintains the problem. Additionally, Warwick argued, in order for a meaningful conceptualization to be possible (i.e., the driving force behind CBT treatment), the aim is to make a positive diagnosis of HA, rather
than making such a diagnosis on the exclusion of physical illness. The rationale behind this is to ensure that patients feel their concerns have been understood, and to acknowledge that the symptoms are indeed real and distressing, so that patients, ultimately, can be told what their problem is, as opposed to what they do not have (the standard medical practice).

Barsky and Ahern (2004) reported that although this often chronic condition has been predominantly recalcitrant and costly to the healthcare system, as there has been no empirically validated treatment to date, a relatively brief, individual cognitive-behavioral treatment intervention (specifically designed to alter hypochondriacal thinking and restructure hypochondriacal beliefs) appeared to have, “significant beneficial long-term effects on the symptoms of HS” (Barsky & Ahern, 2004, p. 1464). These researchers randomly assigned 102 individuals who met criteria for HA to either six 90-minute, weekly CBT sessions or to a usual care control group. Outcome assessments were conducted at baseline, post-treatment, and at 6 and 12 months after completion. The treatment was designed to modify factors that contribute to patients amplifying somatic symptoms and misattributing them to illness or disease, such as attention and bodily hypervigilance, beliefs about symptom etiology, circumstances and context, illness and sick role behavior, and mood. Alterations in hypochondriacal thinking and beliefs, and improvements in functioning remained evident at 6 months and were still persistent at 12 months. Given that the mean duration of illness in this study was 11 years, this promising outcome illustrated CBT as a plausible solution to this otherwise vexing phenomenon. One significant limitation of this study was that only 30% of eligible hypochondriacs agreed to enter the study. The authors posited that this interpretive caveat could be largely attributed to the notion that these individuals are convinced of the medical nature of their condition, leaving the
psychologically-oriented treatment rationale to seem nonsensical, which theoretically at least, could account for the low percentage of potential participants agreeing to be included.

Langlois and Ladouceur (2004) specifically examined the relationship between GAD and HS. They examined a specific adaptation of a protocol originally designed for GAD, and its application to HS. They too argued that HS is currently, but questionably, categorized with somatoform disorders, and would likely be more fitting if considered under anxiety disorders. Using conservative measures of change (i.e., clinical significance and reliable change: Jacobson & Truax, 1991) the researchers impressively found that all their subjects achieved high end-state functioning, lending strong support for the idea that HA is perhaps very similar to GAD. It additionally supports the idea that using strategies that are helpful in resolving other, more conventional forms of anxiety could be favorable in the treatment of HA. Their remarkable finding may indeed be the pivotal missing piece to comprehending why this phenomenon has been resistant, and how to better ameliorate the great suffering and costs associated with HA. Their approach specifically emphasized “intolerance for uncertainty” (i.e., an intolerance for not knowing the answer/outcome, or how to perform a particular task), which they proposed, “explained a significant and unique part of the variance of illness worry” (p 394). Their investigation illustrated that the general structure of illness intrusions closely resembled that of worry, and their treatment targeted intolerance for uncertainty about health. Thus further targeted examination of intolerance for uncertainty is warranted, as it might offer the best view yet of interventions that will help this otherwise seemingly intractable health worry condition.

Wattar, Sorensen, Buemann, Birket-Smith, Salkovskis, Albertsen, et al. (2005) tried to answer another interesting question about whether the effects of CBT for HS in routine clinical settings (as most published studies come from academic/research settings). The authors aimed to
eliminate suggestions that some of these preliminarily promising findings have only been artifacts of the “ivory tower” effect (i.e., efficacy research is often presumed to not be similar to effectiveness level research and findings, which is proposed to be a more legitimate view of how “real life” patients would respond). It was encouraging to see that Wattar et al. discovered that in a non-academic treatment clinic, hypochondriacal patients realized significant change (up to 90% improvement), and these results “compared well with those obtained in the previous trials” (p.166). Of a particular note, the treatment content in this study included aspects of intolerance for uncertainty; the central mechanism related to GAD that was mentioned above (Covin, Ouimet, Seeds, & Dozois, 2008; Dugas, Gagnon, Ladouceur, & Freeson, 1998; Dugas et al., 2003). This would suggest that focusing on this presumed central mechanism in HA, in routine settings with routine patients, offers an optimistic outcome for obtaining similar results to those found by Langlois and Ladouceur (2004) and Wattar and colleagues (2005).

Need, purpose and significance of the study

This case study examined this potential HA-GAD relationship in the single case of a refractory, severe, and chronic HA patient who was treated with a specifically GAD-tailored treatment protocol, in a routine setting, with the treatment focused on the understood central pathological mechanism of worry (i.e., intolerance for health-related uncertainties/worries). With a favorable outcome, the careful examination of the results of this treatment could lend further support for the assertion that the mechanisms of the two worry-based phenomena are so similar that they can arguably be conceptualized as identical; that is, they both load on intolerance for uncertainty (i.e., needing to know answers/solutions to worries), and treating this specific
intolerance will better provide acceptable and stable gains with an otherwise refractory and profoundly costly condition.

Given the range of newer literature which collectively and increasingly seem to support the idea that HA and GAD are related conditions, the primary task of this study was to conduct a careful analysis of a single and extreme case of HA, that was treated exclusively with the aforementioned GAD-tailored treatment, in a typical setting. This analysis would provide yet more support for the powerful assertion that, if the patient responded and recovered while treated in this manner, then the suspected connection between the two conditions, given the severity of the case and the exclusive nature of the treatment, would be further supported.

Patient response on GAD and HA instruments for measuring change have also not been compared, side by side, so a secondary task of this study was to examine the relationship between such instruments. Indeed, if the sets of instruments used for gauging the response in the two conditions exhibit similar reactivity to the treatment, further support could be offered for the notion of a common etiology, namely uncertainty.

Lastly, this study explored what recommendations could be made regarding the treatment of HA and what directions new research might take to better understand the presumed possible relationship between the two conditions, based on a hypothesized common etiological source of intolerance for uncertainty.

Thus, the purpose of this study was threefold: (1) to examine the responsivity of an extreme HA case treated with a GAD-tailored treatment protocol, (2) to examine the response patterns of instruments designed for GAD and HA, and explore whether the two sets respond similarly or dissimilarly, (3) to make recommendations for the continued treatment of HA, and
for future research into its relationship with GAD, and more broadly its connection to anxiety disorders and somatoform disorders.

**Method**

End state functioning (Jacobson & Truax, 1991) was elucidated by calculating both clinical significance and reliable change indexes, to further illustrate that using this adapted GAD model explains and treats HA effectively. Finding a very challenging HA patient fully “recovered” lends both theoretical and empirical support to the idea that HA is not as intractable as once thought. Indeed, by clearly explaining the presumed central pathological process within the HA phenomenon, better treatments can be developed and stronger outcomes can be achieved. In short, this case was evaluated through the methodological lens of an A-B design with follow-up. In this design, the target behavior (e.g., worry, intolerance for uncertainty, poor problem-solving) was precisely described and specified, and repeated measures given to the patient during the active and follow-up phases was evaluated against his baseline (A) performance on a range of psychometrically sound measures (e.g., Worry and Anxiety Questionnaire [WAQ], Intolerance for Uncertainty Scale [IUS]). In this way, the experimental phase (B) and the follow-up phase (6, 12, and 18-months following termination) illustrated change when compared directly against baseline observations, and “changes in the dependent measures can be attributed to the effects of treatment” (Barlow, Nock, & Hersen, 2009, p. 137). This methodology is often chosen in conventional treatment settings because of its few deleterious effects on patients, and is often the design choice for such standard settings as the Psychological Service Center (PSC). With the addition of the extensive follow-up period, especially considering the longevity and previously recalcitrant nature of the patient’s condition,
it would seem likely that by an obtained “recovery” status, the effective ingredient of change in this study could be reasonably attributed to treatment.

However, to protect against an overly optimistic view of the treatment effect, existing literature findings were also incorporate, whether larger, randomized controlled trials (RCT) or smaller case series of HA patient outcomes. In this fashion, it was possible to indicate in which ways treatment of this particular patient was similar to existing treatment outcome studies, and it was possible to compare this patient’s outcome against various other outcome findings.

Lastly, progress and outcome data was evaluated for the construct of “sudden gains,” to assess whether there were any sudden dramatic changes in any between-sessions intervals. Some literature (e.g., Tang & DeRubeis, 1999; Zang, DeRubeis, Beberman, & Pham, 2005) suggests that the magnitude of these types of improvements is much larger than those of typical between-session symptom reductions. Interestingly, when present, these account for a surprisingly large portion of patients’ total symptom improvements; thus, this phenomenon has been connected with the notion of “therapeutic breakthroughs,” and these sessions may indeed be “critical” sessions. A required magnitude of a sudden gain has been proposed to be demonstrated if the progress measure change meets or exceeds a 25% change between a pre-gain and after-gain session. To date, this abrupt change phenomenon has only been demonstrated in major depression, with relative consistency, and only a few attempts has been made to study this in anxiety and related disorders (e.g., Present, Crits-Christoph, Connolly-Gibbons, Héaron, Ring-Kurtz, Worley et al., 2008; Hoffman, Schulz, Meuret, Moscovitch, & Suvak, 2006). Therefore, this HA patient’s progress data was also examined for evidence of sudden gains indicators. If present, it would have been the first documented demonstration of sudden gains in HA.
Measures:

In the interest of understanding how patients with health concerns respond on indices of more generalized anxiety, a variety of GAD measures were administered both as progress and outcome measures to this case study patient. Additionally, to gauge HA proper in this patient, a widely accepted HA measure was also administered. Each measure is described here.

The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec [1990]) is currently one of the most widely used and accepted questionnaires that measures a patient’s general tendency to worry excessively. It is a 16-item self-report questionnaire designed to measure intensity and excessiveness of worry without reference to specific worry content. In support of its broad acceptance, it is noteworthy to report that this instrument had already been translated by 2001 into Chinese, Dutch, French, German, Greek, Italian, Spanish, and Thai; presumably there are now further translations, but even these original translations back up the broad consensus that clinical worry is a common ailment. Psychometric normative properties are available (GAD patients – 67.66 [SD = 8.86]; non-anxious selected groups – 44.27 [SD = 11.44]) such that the calculations of clinical significance and reliable change indices are easily performed. The PSWQ has good and very good internal consistency (α ranges from .86 to .93 in clinical and college samples). Additionally, the PSWQ has adequate to good test-retest reliability (r), something which is relevant to the calculation of reliable change, and has been documented as .74 to .93 in college samples across 2 – 10 week periods. Collectively, the PSWQ can be asserted as a valid measure of excessive worry. It does appear most correlated with pathological worry, but is also moderately correlated with measures of anxiety and somewhat with depression. Nonetheless, the PSWQ appears to best capture clinically anxious patients who demonstrate excessive, non-constructive worry as their main feature. Lastly, but importantly, the
PSWQ has been found to be sensitive to change as scores are significantly reduced following CBT treatment for worry (Antony, Orsillo, & Roemer, 2001).

The Anxious Thoughts Inventory (AnTI; Wells, 1994) is a 22-item self-report measure that assesses worry along three dimensions, namely social worry, health worry, and meta-worry. The first two factors capture specific contents of worry while the third factor examines worry about uncontrollability of thoughts per se. This third factor has also been described as worry about worry, hence the term *meta*-worry. Psychometric normative information is available (clinical sample – 59 [SD = 12.8]; non-clinical sample – 37.6 [SD = 15.4]), and its test-retest reliability is reported (0.86), making calculations of clinical significance and reliable change possible.

The Worry and Anxiety Questionnaire (WAQ; Dugas, Freeston, et al., 2001) is an 11-item, self-administered measure originally designed as a screening tool for GAD. It covers the diagnostic criteria for GAD, such as “Do your worries seem excessive or exaggerated?” Careful examination of this instrument show that it can lead to false positives, but it does not produce false negatives. Stated differently, and showing its appeal perhaps, the WAQ will capture patients who demonstrate significant worry, and with some additional testing it can readily be clarified which may possibly be false positives. People who do not meet GAD criteria will not score in a fashion to mislead the WAQ. An especially attractive aspect of the WAQ is that it besides giving quantitative scores that can be tracked from one session to the next, for progress purposes, it provides qualitative information about what topics a person is focused on from administration to administration (and how those, in a rank order, may change over time). It has known psychometric normative (GAD – 35.8 [SD = 4.9]; non-GAD – 13.9 [SD = 12.5])
information and test-retest reliability \((r = 0.76)\), making the calculation of clinical significance and reliable change possible. It has good known-groups validity (Dugas, Freeston, et al., 2001).

The Intolerance for Uncertainty Scale (IUS; Freeston, Rheume, Letarte, Dugas, & Ladouceur, 1994) is a 27-item self-administered measure designed to assess the degree to which an individual has difficulty tolerating uncertainty, a factor which has been linked with GAD. While the scale was originally developed in French, and English version is also available. The scale measures various aspects of uncertainty, such as the emotional and behavioral consequences of being uncertain, how being uncertain reflects on one’s character, and expectations of controllability of the future. Psychometric normative information (clinical sample – 73.36 [SD = 19.07]; non-clinical sample – 54.38 [SD = 17.17]) and 5-week test-retest \((r = 0.74;\) English sample) is available, making meaningful change indices possible to calculate. Convergent validity correlated the IUS with other measures of worry \((r = .57\) and .63) and trait anxiety \((r = .57)\). When anxiety and depression is partialled out, worry still remain as a significant factor for clinical samples examined, so there is support for the specificity and sensitivity of the measure, as it assesses for worry as a construct. Indeed, the measure discriminates between GAD patients and non-anxious controls, supporting its sensitivity for pathological worry. IUS also appears sensitive to change, as scores are significantly reduced following CBT (Antony, Orsillo, & Roemer, 2001).

The Meta Cognitions Questionnaire (MCQ; Cartwright-Hatton & Wells, 1997) is a 65-item self-administered measure of beliefs about worry and intrusive thoughts. The measure can be broken down into five separate factors, namely *positive worry belief* (factor 1), *beliefs about controllability and danger* (factor 2), *beliefs about cognitive competence* (factor 3), *general negative beliefs* (factor 4), and *cognitive self-consciousness* (factor 5). Psychometric normative
information (individuals with GAD – 125 [SD = 23.6]; non-anxious controls – 105.7 [SD = 29.8]) and very good 5-week test-retest reliability ($r = 0.94$) among a university community sample is available. This scale correlates ($r = .26$ to $$.73$) with measures of trait anxiety, and the scale appears to distinguish well between individuals with GAD and other anxiety disorders (Antony, Orsillo, & Roemer, 2001).

The Why Worry Scale (WW; Freeston, Rheume, Letarte, Dugas & Ladouceur, 1994) is a 20-item, self-administered measure of reasons for why people engage in worry. Generally, this scale is examining positive attributions and justifications people make/give for why worrying, as a coping strategy, is good/positive. Subscales explore such ideas as worrying presumably protecting against negative emotions in the event of negative outcomes, that worrying even prevents negative outcomes, or that worrying represents a positive personality trait (i.e., it shows one cares). Like with the IUS, which also was developed in French Canada, the WW has French and English versions available. Psychometric normative information (clinical sample – 46.9 [SD = 22.5]; non-clinical sample – 32.8 [SD = 7.9]) and test-retest reliability ($r = 0.93$) is available. The WW demonstrates convergent validity via significant correlations with the PSWQ, and because it seemed to distinguish between groups diagnosed with GAD and other forms of anxiety or non-anxious controls. GAD diagnosed people also score higher on the WW than do those who do not meet criteria for GAD (Antony, Orsillo, & Roemer, 2001).

The Health Anxiety Questionnaire (HAQ; Lulock & Morley, 1996) was developed to identify individuals with highly anxious about health. It is a 21-item, self-administered measure that gauges the presence of important aspects of HA. Items cover such issues as fear of illness and death, worry about health, preoccupation and reassurance seeking behavior. Importantly, it also examines interference in the examinee’s life as such interference is almost defining of the
disorder itself. Psychometric normative information (clinical sample – 35.2 [SD = 13.4]; non-clinical sample – 8.6 [SD = 8.0]) and 6-week test-retest ($r = 0.95$) is available. The HAQ has adequate discriminative analysis, and it is able to separate different groups (lay group, nurses, medical out-patients, psychology out-patients). Additionally, HAQ still captured health-related fears and behaviors once such related constructs as depression is partialled out (Lulock & Morley, 1996).

**The treatment**

The tailored CBT protocol used was a 2001 prepublication version of Dr. Michel Dugas’ Intolerance for Uncertainty (worry) focused treatment. This treatment protocol has since been published in 2007 (Dugas & Robinchaud, 2007). The original protocol was designed to systematically cover various problem domains of GAD, and was broken down into the following session topics: 1) orientation to GAD model and CBT generally; 2) recognition of different types of worry and relaxation training; 3) understanding maladaptive coping in response to two worry styles; 4) understanding intolerance for uncertainty and issues of control versus tolerance; 5) cognitive restructuring unhelpful beliefs about worry; 6) being more effective at problem-solving to address real life worries; 7) further problem-solving strategies; 8) reducing and stopping avoidance and neutralizing behaviors; 9) exposure – live versus in-imagination options; 10) reviewing all interventions and linking to GAD model; 11) mixed worries and how to address; 12) planning for future and generalization training; 13) relapse prevention. This treatment protocol was built around a theoretical model (Dugas, Gagnon, Ladouceur, & Freeston, 1998) which held that GAD has four main problem features, namely intolerance of uncertainty, positive beliefs about worry, negative problem orientation, and cognitive avoidance. The unique aspect of
this particular protocol is that unlike some previous treatment packages (which relied upon more
generic CBT treatment tools, such as relaxation, probability estimation, and decatastrophizing)
its focus was driven by the conceptualization of GAD as a syndrome driven by pervasive,
excessive, and uncontrollable worry. Therefore, worry was the primary treatment target of the
Dugas protocol. All of the treatment modules bear directly towards pathological worry, and not
some of the other commonly targeted GAD issues (e.g., physical tension). In fact, this protocol
argued that other side effect symptoms of worry, such as tension, would indirectly abate with
targeted treatment of worry as the driving force behind such concomitant symptoms. Dugas’
findings suggested that treating worry directly is a primary change agent in the reduction of
anything associated with worry, and as such the Dugas’ (Intolerance for Uncertainty; aka Worry)
protocol was thus focused.

The therapist

The therapist in this case was Johan Rosqvist, PsyD, who in 2003 was in his first year of
post-doctoral work under state-contract-required weekly supervision with Robin Shallcross,
PhD. She was also the referral source of this patient. The nature of the referral was such that Dr.
Shallcross had worked with the patient consistently for a couple of years, and had realized that
health-related symptom interference was not responding adequately under her care. She spoke
with the patient about this fact, portraying Dr. Rosqvist as an “expert” in the treatment of
anxiety, and ultimately recommended the patient seek specifically-tailored treatment for such
symptoms because of the proven track record of CBT with these types of problems (J. Rosqvist,
personal communication, June 17, 2011).
At the time of seeing the patient, Dr. Rosqvist had obtained a generalist PsyD degree from Pacific University’s School of Professional Psychology; however, part of this degree did include a pre-doctoral training experience at an American Psychological Association (APA) accredited internship training site that provided a specialized rotation experience in a formal anxiety disorders clinic, so he did have some specialized experience before treating this specific patient. Part of that specialized experience did include working more extensively with GAD patients and a just a couple of HA patients. While part of his training was indisputably specialized in nature, it becomes relevant and important to point out that Dr. Rosqvist diligently followed a published protocol, and by closely using this protocol (versus some particular “magic” that he could otherwise be presumed/asserted to possess) he ensured that replication by other therapists was, theoretically at least, quite possible. One of Dr. Rosqvist’s stated objectives in this deliberate approach to treating this patient was in fact to preserve the possibility of other providers also trying this novel application of an experimental protocol, should it prove successful (J. Rosqvist, personal communication, June 17, 2011).

Dr. Shallcross’ theoretical orientation was not CBT, but was instead Psychodynamic in nature, so outside of ensuring that Dr. Rosqvist appeared to follow his deliberate plan of action, and the progress data was indicating positive change with this intervention, she was not directly influencing the treatment direction or process per se. Instead, the progress made is more strongly attributable to the particular choice of treatment protocol and how well that fit this particular patient’s pathology (J. Rosqvist, personal communication, June 17, 2011).

**Operational definitions**

The following definitions are conceptualized and operationalized in this study:
(1) **HA:** Inappropriate or excessive health-related fears based on misperceptions of bodily sensations as dangerous/health threatening and indicative of a medical problem(s) which the sufferer perceives him- or herself as being incapable of coping with.

(2) **HS:** Severe HA or intense illness worry; essentially identical to HA, except HS is not thought to ever be fleeting or “deactivated;” HA is increasingly taking the place of HS to describe the phenomenon because there are a number of clinical disorders which have as their main features health-related fears and worries; whereas HA is more or less value neutral, HS has acquired pejorative connotations and is associated with malingering and an underlying character disorder (as is evident in its sources of hysteria and neuroticism).

(3) **GAD:** GAD is an anxiety disorder which is presumed to be driven by an intolerance for uncertainty, which is hallmarked by chronic worrying. In worrying, the GAD patient tends to have two types of worries, namely here-and-now, or real life (or Type 1) worries and future-oriented or imagined possible future (Type 2) worries. Type 1 is characterized by the notion that real-life solutions are actually available to the sufferer, but because of the pathological process of the phenomenon itself, no real life solutions are actually attempted in favor or chronic worry. Type 2 is characterized by feared possibilities which have not happened, but could possibly occur, but generally there are no current preventive strategies for such unknown and “as-of-yet-non-experienced” possibilities.

(4) **Intolerance for uncertainty:** Is thought to center around a perceived “need” to know the answer to some question, or the outcome to some circumstance. Pragmatically, it suggests that someone who is so intolerant cannot stand not knowing, which can be suggested to be equivalent having a need for control. Uncertainty then is essentially the antithesis of control. So, people who are intolerant of uncertainty are, in fact, intolerant of not having absolute control. Ironically, in
the real world such levels of control are not possible; indeed, such absolute control is theoretically not possible and probably not desirable actually.

(5) **Ambiguity**: Is similar (and perhaps related) to uncertainty, in that instead of not knowing an answer or an outcome, ambiguity means to not know how to do something or how to solve some problem. Intolerance of ambiguity, therefore, means not being able to stand not knowing how to do something to achieve some particular outcome (also a form of control).

(6) **Cognitive-Behavior Therapy**: CBT is based on the supposition that affect (emotions), thoughts (cognitions) and behaviors are interrelated in such a way that changing one necessarily changes the other(s). In this knowledge, CBT therapists set out to change feelings (often what patients want changed first) through the manipulation of, typically first, behaviors and the modification of unhelpful thinking.

**Summary**

This study carefully reviews the outcome of a serious case of HA that was treated with a GAD-tailored protocol. It will attempt to elucidate the generally- and increasingly suspected-relationship between HA and GAD. It will further attempt to examine the response pattern of HA, as it is measured both by instruments designed for HA and GAD. As these salient issues await further study, this case analysis will provide a systematic review of an often recalcitrant phenomenon which represents an enormous healthcare burden. The study will make recommendations for how to better understand (i.e., conceptualize) and treat this otherwise vexing and common problem.
Case Description

“Joe” (a pseudonym) presented as a 36-year-old, single (never married), Caucasian male. He was referred for “assistance with anxiety” after having undergone psychodynamic treatment for two years, without remarkable symptom relief. Joe’s clinical presentation was not an unusual one, except for its chronic and severe nature. At the time of referral, Joe reported living with two female roommates, working as a wine consultant for a local wine distributor. He had been working in the wine industry fulltime for 10 years when he presented for assistance with HA and concurrent panic attacks.

Joe reported the main concern as a distinct preoccupation, or worry about having- or contracting- a disease, or becoming sick with a major medical disorder. These fears centered around concerns about dying from medical issues related to cancer and tumors (e.g., colon, brain), heart attacks and heart disease, stroke or choking on foods. These fears always arose from misinterpreting bodily symptoms (e.g., stomach distress, hemorrhoids, headaches, heartburn, feeling tired). Not surprisingly, these preoccupations caused significant distress and significantly interfered with his personal and professional life (e.g., he was reluctant to exercise due to fear of causing a heart attack, leaving early from work due to feeling ill). Additionally, these fears of disease, and ultimately death, led to profound avoidance and other safety behaviors. For example, if he believed that he was experiencing a heart attack, most often due to misinterpreting the meaning of heartburn, he typically would go to an emergency room to get “checked out.” He also would frequently call doctors and various medical professionals for reassurance around his health. Ironically, but predictably, information which he obtained in this fashion never permanently relieved his worries and fears, and he was repeatedly faced with the same but worsening ruminations about his health status. Whatever relief he did experience from
reassurance seeking, checking, and other safety behaviors did not endure beyond a few days, or at most a week, leaving him to suffer for the 14 years preceding being introduced to cognitive-behavior therapy.

Ironically, his preoccupation led to being highly avoidant of physical exercise, as exertion would invariably produce feared sensory information (e.g., an accelerated heart rate, chest pain, feeling hot and clammy). In his perceptions of threat and danger, he became a self-professed “couch potato” who actively sought to be predominantly inactive and to avoid activities that would raise sensory cues anywhere in his body; accordingly, he would always park as close to his work and shopping/errands was possible, he would take a downstairs/main floor level bedroom so he would not have to climb stairs in his apartment, and he would engage in various strategies to reduce exertion (e.g., walk very slowly if walking could not be avoided). Because his professional life centered on wine and entertaining with food, and he used emotional eating to soothe psychic fears, he subsequently became morbidly obese, ironically doubling his true chances of death. The obesity, in and of itself, contributed to an increase in sensory information, which forced Joe further into deeper dysfunctional patterns of avoidance and escape.

In response to these kinds of concerns, Joe became increasingly ruminative, self-focused, and avoidant. He would also extensively engage in checking behaviors and almost constantly looked for reassurance seeking. For example, whenever he was in Fred Meyer (a grocery store) he would always use the blood-pressure monitor to check his pressure before shopping, and if he felt his pressure was elevated he would choose to cancel shopping at the expense of obtaining no groceries. He would also typically check his blood-pressure upon finishing and gauge the remainder of his day depending upon whether he perceived that his pressure was too high. He
additionally sought prescriptions (e.g., Protonix and Zantac) and over-the-counter medications (e.g., Tums) to reduce sensations, especially for circumstances that he knew would be difficult for him (e.g., flying often produced uncomfortable physical experiences).

**Results**

At the end of 13 treatment sessions a planned termination occurred because the patient’s symptoms had dramatically improved. On all instruments administered during the progress of treatment, whether GAD or HA specific in nature, he had begun treatment in the clinical control sample range, passed the clinical threshold between clinical and non-clinical distributions, and finished in the non-clinical control sample range. See Table 1 below. Using guidelines outlined by Jacobson and Truax (1991), this allowed for the statistical label of “Recovered” to be used when describing Joe’s change during and following treatment. This label is an important description because it describes a particularly a significant amount and type of progress, and ultimately represents a qualitative change that, in terms of end-state functioning, ultimately makes Joe indistinguishable from non-clinical populations. This has clear implications for the veracity and sheer value of the particular treatment applied, and lends credence to any recommendations for additional research utilizing the particular approach applied in Joe’s case.
On the **PSWQ**, his score at intake (71) had at post-treatment (42) improved by 40.85%, at 6-months follow-up (30) by 57.75%, at 12-months follow-up (35) by 50.7%, and at 18-months follow-up (31) by 56.34% (each compared to baseline, respectively). On the **AnTI**, his score at intake (69) had at post-treatment (30) improved by 56.52%, at 6-months follow-up (33) by 52.17%, at 12-months follow-up (33) by 52.17%, and at 18-months follow-up (29) by 57.97% (each compared to baseline, respectively). On the **WAQ**, his score at intake (37) had at post-treatment (18) improved by 51.35%, at 6-months follow-up (20) by 45.95%, at 12-months follow-up (16) by 56.76%, and at 18-months follow-up (15) by 59.46% (each compared to baseline, respectively). On the **IUS**, his score at intake (103) had at post-treatment (54) improved by 47.57%, at 6-months follow-up (38) by 63.11%, at 12-months follow-up (27) by 73.79%, and at 18-months follow-up (25) by 75.73% (each compared to baseline, respectively). On the **MCQ**, his score at intake (180) had at post-treatment (87) improved by 51.67%, at 6-months follow-up (96) by 46.67%, at 12-months follow-up (83) by 53.89%, and at 18-months follow-up (72) by 60.00% (each compared to baseline, respectively). On the **WW**, his score at intake (70) had at post-treatment (25) improved by 64.29%, at 6-months follow-up (28) by 65.71%, at 12-months follow-up (23) by 68.17% (each compared to baseline, respectively).
follow-up (25) by 64.29%, and at 18-months follow-up (23) by 67.14% (each compared to baseline, respectively). On the HAQ, his score at intake (52) had at post-treatment (16) improved by 69.23%, at 6-months follow-up (17) by 67.31%, at 12-months follow-up (7) by 86.54%, and at 18-months follow-up (5) by 90.38% (each compared to baseline, respectively).

The threshold point “C” was calculated using Formula 1 below, taken from Jacobson and Truax’s (1991) descriptive article on how to calculate clinically significant (meaningful) change by using simple, readily available progress measure(s) psychometric information (i.e., non-clinical means and standard deviations, clinical means and standard deviations, and test-retest reliability). Generally speaking, this formula is used to assess whether scores obtained fall closer to a clinical or non-clinical comparison population samples; specifically, when scores exceed C then the patient functions more like that of the abnormal comparison population, and when scores fall below C then the patient functions more like a normal comparison sample.

Formula 1 (‘C’)

\[ C = \frac{M_{clinical} - M_{non-clinical}}{s_{non-clinical} + s_{clinical}} \]

Reliable Change (RC) was also calculated, using Jacobson and Truax’s (1991) described method, in an attempt to ensure that the degree of change observed in progress and outcome was unlikely to be attributable to chance. Test-retest reliability was used in these calculations as it is preferred for its protection against regression toward the mean effects. The formulas used for each RC calculation are demonstrated below in Formula 2. As specified by the cited method, when an RC calculation exceeded 1.96, then it was presumed to signal a robust change, attributable to direct effects of the specific treatment utilized rather than being due to chance, maturation, or other non-treatment effects. Jacobson and Truax (1991) reported that when an
observed change exceeds this value, then “it is unlikely that that the posttest score is not reflecting real change” (p. 14). RC in this regard provides a clear cut criterion for the measurement of improvement that is psychometrically sound, and allows the researcher to assert whether or not realized change reflect more than mere fluctuations of imprecise efforts to measure change over time.

\[
RC = \frac{x_{post} - x_{pre}}{\sqrt{2 \left( s_{clinical} \sqrt{1 - r_{test/retest}} \right)^2}}
\]

Following the trend of scores obtained on all measures passing C from clinical and ending in non-clinical, all RC calculations also proved to exceed the necessary 1.96-sized change difference to be deemed attributable to treatment effects rather than various chance variances. Specific RC values of each measure at each measurement point can be viewed below in Table 2. The sheer strength of results observed lends further credence to the notion that using a treatment protocol tailored to intolerance for uncertainty must have “hit on” a specific, if not central ingredient of a (if not the) pathological source code of HA.
Indeed, as was self-evident from the sheer strength of RC results obtained, the change realized by the patient was robust enough, across the board of data points obtained, that it can readily be asserted that observed change was undoubtedly due to direct treatment effects (i.e., RC values far exceed the minimal, yet conservative 1.96 value minimally needed in each case). This, from a practical stand-point, can eliminate any conservative concern that extraneous variance explained the effects achieved; in short, the treatment worked, and it worked very well.

Next, the data set was also closely scrutinized for evidence of instances of sudden change. None were found, but instead obtained data demonstrated gradual, smooth declines in values, indicating that no abrupt (i.e., $>25\%$) instances of change occurred. While it would have been interesting to examine any such precipitous change points more carefully, to seek understanding about which treatment component had been delivered just prior to such observed change (as this might lend insight into more central aspects of the overall treatment product in relevance to what incrementally better/faster produces change), this negative finding is in line with current literature reports about sudden gains in relation to anxiety disorders versus depressive disorders.

<table>
<thead>
<tr>
<th>Instrument</th>
<th>$R_{TEF}$</th>
<th>$S_E$</th>
<th>$S_{diff}$</th>
<th>Pre- to Post-RC</th>
<th>Pre- to 6-Mo RC</th>
<th>Pre- to 12-Mo RC</th>
<th>Pre- to 18-Mo RC</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSWQ</td>
<td>0.93</td>
<td>2.30</td>
<td>3.25</td>
<td>-8.92</td>
<td>-12.62</td>
<td>-11.08</td>
<td>-12.31</td>
</tr>
<tr>
<td>AnTI</td>
<td>0.86</td>
<td>4.74</td>
<td>6.70</td>
<td>-5.82</td>
<td>-5.37</td>
<td>-5.37</td>
<td>-5.97</td>
</tr>
<tr>
<td>WAQ</td>
<td>0.76</td>
<td>2.40</td>
<td>3.39</td>
<td>-5.60</td>
<td>-5.01</td>
<td>-6.20</td>
<td>-6.49</td>
</tr>
<tr>
<td>IUS</td>
<td>0.74</td>
<td>9.73</td>
<td>13.76</td>
<td>-3.56</td>
<td>-4.72</td>
<td>-5.52</td>
<td>-5.67</td>
</tr>
<tr>
<td>MCQ</td>
<td>0.94</td>
<td>5.66</td>
<td>8.00</td>
<td>-11.62</td>
<td>-10.50</td>
<td>-12.12</td>
<td>-13.50</td>
</tr>
<tr>
<td>WW</td>
<td>0.93</td>
<td>5.85</td>
<td>8.27</td>
<td>-5.44</td>
<td>-5.08</td>
<td>-5.44</td>
<td>-5.68</td>
</tr>
<tr>
<td>HAQ</td>
<td>0.95</td>
<td>8.70</td>
<td>4.17</td>
<td>-8.63</td>
<td>-8.39</td>
<td>-10.79</td>
<td>-11.27</td>
</tr>
</tbody>
</table>
For example, Hofmann and colleagues (2006) found that the sudden gains phenomenon, defined as “enduring reductions in symptom intensity from one session to the next” (p. 694), do not appear to occur in disorders outside of depression. He found that whatever instances of sudden gains observed in the treatment of anxiety do not behave predictably as it appears to do in the treatment of depression, neither does it predict better short- or long-term outcome, and it does not seem to support any particular treatment modality or treatment piece as most related with occurrences of sudden gains (p. 695). Lastly, he reported, sudden gains realized within the treatment of anxiety unfortunately seem to reverse too often to believe that observed instances of such change holds any significant meaning in the overall treatment approach to resolving anxiety phenomena. In the case of Joe, his observed changes seemed to fluctuate from session to session, but never exceeded the required 25% and it also illustrated occasional reversal of improvements (with an overall, obvious trend towards improving over multiple sessions on all measures).

In line with relying on an A-B design with follow-up design, the experimental phase results obtained throughout the treatment and follow-up were examined directly against the baseline data. Following Barlow, Nock, and Hersen’s (2009) position that “changes in the dependent measures can be attributed to the effects of treatment,” (p. 137) it can be reported here, especially given the robustness of findings (i.e., Recovery) during treatment and for 18-months following the cessation of treatment, that observed effects can be directly attributed to the treatment. This holds significant relevance in this case because the treatment delivered was not a conventional HS protocol per se, but was instead something that has instead been developed to treat problems of intolerance of uncertainty.
Discussion

HA is arguably a virtually ubiquitous experience, and certainly for some it readily becomes an experience that is as extreme as it is distressing and debilitating. In fact, HS has long been considered a severe and recalcitrant phenomenon, and it has commonly proven too much for clinicians of all versions to effectively treat. This unfortunate, albeit now perhaps historical, reality has left many health anxious persons un- or under-treated, which in real-life terms translate to unnecessary suffering and continued burdensomeness to all sorts of systems (e.g., familial, social, occupational, healthcare, and cultural).

No matter how the construct of cost is examined, the toll of uncontrolled health-related fears has been far too high, and leaving this condition alone within systems to function as it always has/does grows incrementally more unacceptable. Arguably, a better understanding of the phenomenon itself has been needed for many years, and better methods for resolving it have been seemingly lacking. Fortunately (if there is such a thing when human suffering is discussed plainly), the intensity and impact of the condition has led many theorists, researchers, and clinicians to seek better answers as to what exactly might mediates and perhaps better moderate the condition. Improvements are being sought.

Conceptualizing HA as essentially a not too distant cousin of GAD, perhaps with just an inordinate focus on one particular topic (i.e., pathological fragility of health) versus many, may begin to offer hope to an otherwise seemingly- if not actually- treatment-resistant condition. Indeed, severe health concerns do “overlap with- and have much comorbidity with anxiety disorders” (Asmundson et al., 2001, p. 370). While such patients commonly end up undergoing psychotherapy treatment after frustrating care by non-psychology healthcare providers (typically for nominal effects which leave patients with troubling residual symptoms, and at subsequent
risk for relapse), the case of Joe readily demonstrated that formulating the condition as a form of GAD (e.g., GAD, Somatic Type) and using newly developed methods tailored to his condition quickly produced substantive gains that lasted far beyond the cessation of formal treatment. This represents a positive type of outcome, in that the (unusual) strength of response was great, and Joe appeared to show no immediate or subsequent signs of relapse or even slippage which is so common to these patients. The robustness results suggested that there seems to be something quite important to be gained by re-formulating a previously virtually untreatable condition into a specific form of a well-understood and very treatable anxiety disorder. Interestingly, and this seems relevant for thinking about severe forms of health concerns as a potential anxiety disorder rather than as a somatoform disorder, “the central features of HS include fears and morbid preoccupations,” (Asmundson et al., 2001, p. 371), just like most of the anxiety disorders do, most notably in similar terms to GAD, Obsessive-Compulsive Disorder (OCD), and Panic Disorder (PD). Ultimately, this case offers new hope, and it illustrates that meaningful change can be had in a relatively short amount of time, even for significantly impaired cases, such as Joe’s.

While this one case did not support the occurrence of sudden gains in HA, the construct is interesting enough that further study of its relation to health-related fears may be warranted. In fact, HA has conceptual overlap with depressive conditions (i.e., hopelessness, helplessness, sadness, etc.) that it is conceivable that over many more subjects such a connection could be discovered. On the other hand, since HA (for the purposes of this examination) is being conceptualized as an anxiety disorder, it is probably useful to acknowledge the general lack of support for sudden change within anxiety. While practitioners would likely be thrilled to know what would induce those naturally occurring “ah ha” moments in HA patients, the reality may
instead be that the abrupt change following such moments are really not possible, but instead slow and steady is the course back to mental health from HA.

One of the obvious limitations regarding recommending future research is one inherent to all single case studies, namely inadequate generalizability. With only this one case demonstrating such an effect, it becomes difficult to assert much beyond the immediate findings (i.e., Joe responded well); however, it would at least support the idea of the novel application of an existing treatment protocol to other cases to see if similar outcomes amongst more health anxious people can be replicated. If a case series could be established as essentially following suit to Joe’s realized progress and outcome, then greater assertions about broader generalizability of findings could be made. Current literature about pathological levels of health-related fears collectively seem to point to a troubling question, namely which treatment approach is best. Unfortunately, there appears to be few definitive and clear answers to this question, and it appears that more answers, through the production of meaningful and interesting data, may be the only way to a better answer which interventions are “better” (read as produces more effective/high functioning end-states) and why.

However, the case of Joe could be considered a “micro pilot study” in its own right, as it deliberately considered a seemingly plausible hypothesis about a suspected root mechanism of pathological health concerns, set out to systematically test that guess, and the robustness of response to the question of the case (i.e., could HS be thought of as fundamentally a related condition to GAD, in how it appears and responds). It draws credible attention to a distinct possibility that an otherwise vexing condition may actually be far more treatable than previously thought.
In fact, it may be that approaching this single case as a Local Clinical Scientist (Stricker & Trieweiler, 2006) may turn out to be the key to unlocking this commonly perplexing and challenging presentation. The value to deliberately treating all patients as case studies, and deliberately seeking to understand how conditions work and could be resolved cannot be underscored enough. Praxis is best understood as “reflection and action upon the world in order to transform it” (ref). There is a cyclical process of learning inherent in deliberately treating pathologies to (1) see how such respond, to (2) influence change in treatments, and to (3) reduce and (aspirationally) eliminate human suffering represent the praxis of clinical psychology. This deliberate approach to understanding and resolving human ailments, and importantly, was what allowed Joe to regain a life and perspective. By critically thinking like a scientist about a single patient, and about the knowledge base behind his presenting concern and similar conditions, and being creative about how to bend otherwise potentially pedantic rules of protocols, a flexible novel application of an existing intervention met with great success. By bridging the gap between science and practice, in just a single case, a new source of hope for the future of treating health-related fears was created. Implications for the contribution of deliberately approaching psychotherapy are self-evident, and the value of living is enhanced.

It is high time that a bridge is finally laid across the virtual chasm still largely remaining between science and practice, to finally bring scientific practice and scientific attitudes into everyday clinical practice. Science and practice are, in fact, not distinct endeavors, but much could be done by all vested parties to close this unacceptable lab-couch gap. Patients like Joe deserve the kind of clinical mindset that promoted the novel application of something which conceptually should help, as the kinds of struggle, strife, and suffering he had been enduring for the decade immediately preceding treatment was in actuality unnecessary. Notably, this single
case study illustrates that ingenuity, creativity, and an innate desire to better understand the human condition in meaningful ways are inherent to integrating science into practice. Future research should take the following aims, (1) systematically apply the GAD protocol to a larger pool of health anxious patients, to further discover whether intolerance for uncertainty can be more strongly asserted as a central mechanism in HS, (2) evaluate further whether pathological HA belongs better in its current location (i.e., Somatoform Disorders), or whether it should be proposed for relocating to Anxiety Disorders, and (3) further study the systematic application of efficacy-derived protocols in effectiveness settings, to evaluate further whether the everyday application of established actions of change can in fact (as asserted by much efficacy literature) produce meaningful alleviation of suffering contrary to popular practitioner beliefs. It is time for clinical psychology to take its proper place in the healthcare field. It is time for the real clinical psychology, namely scientific clinical psychology, to stand up (McFall, 1991).
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