

# Cognitive Behavior Therapy for Hypochondriasis

## A Randomized Controlled Trial

Arthur J. Barsky, MD

David K. Ahern, PhD

**H**YPOCHONDRIASIS IS DEFINED as a persistent fear or belief that one has a serious, undiagnosed medical illness. Occurring in as many as 5% of medical outpatients, it is a prevalent, disabling, and chronic condition that has generally been refractory to psychological and pharmacological treatment and costly for the health care system.<sup>1-6</sup> Until recently, there was no empirically validated treatment for the hypochondriacal patient's somatic symptoms, belief in the presence of an undiagnosed disease, health-related anxiety, and bodily preoccupation. Although limited by small samples, lack of randomization and control groups, the absence of long-term follow-up, limited generalizability, and the absence of validated outcome measures,<sup>7-12</sup> the existing treatment literature suggests a role for a variety of psychosocial interventions, although none has been conclusively validated. In the only large-scale, rigorous, randomized controlled trial to date, cognitive therapy and behavioral stress management were both more effective than a waiting list control condition.<sup>13</sup>

We have proposed that hypochondriasis can be understood as a self-perpetuating and self-validating disorder of cognition and bodily perception.<sup>14-16</sup> In this formulation, personally threatening life events prompt

**Context** Hypochondriasis is a chronic, distressing, and disabling condition that is prevalent in ambulatory medical practice. Until recently, no specific treatment has been clearly demonstrated to be effective.

**Objective** To assess the efficacy of a cognitive behavior therapy (CBT) for hypochondriasis.

**Design** A randomized, usual care control group design, conducted between September 1997 and November 2001. The individual primary care physician was the unit of randomization, and all patients clustered within each physician's practice were assigned to the experimental treatment (individual CBT and a consultation letter to the primary care physician) or to the control condition. Subjects were assessed immediately before and 6 and 12 months after the completion of treatment.

**Setting and Participants** Participants were 80 patients from primary care practices and 107 volunteers responding to public announcements, all of whom exceeded a predetermined cutoff score on a hypochondriasis self-report questionnaire on 2 successive occasions.

**Intervention** A scripted, 6-session, individual CBT intervention was compared with medical care as usual. The CBT was accompanied by a consultation letter sent to the patient's primary care physician.

**Main Outcome Measures** Hypochondriacal beliefs, fears, attitudes, and somatic symptoms; role function and impairment.

**Results** A total of 102 individuals were assigned to CBT and 85 were assigned to medical care as usual. The sociodemographic and clinical characteristics of the 2 groups were similar at baseline. Using an intent-to-treat analytic strategy, a consistent pattern of statistically and clinically significant treatment effects was found at both 6- and 12-month follow-up, adjusting for baseline covariates that included educational level, generalized psychiatric distress, and participant status (patient vs volunteer). At 12-month follow-up, CBT patients had significantly lower levels of hypochondriacal symptoms, beliefs, and attitudes ( $P < .001$ ) and health-related anxiety ( $P = .009$ ). They also had significantly less impairment of social role functioning ( $P = .05$ ) and intermediate activities of daily living ( $P < .001$ ). Hypochondriacal somatic symptoms were not improved significantly by treatment.

**Conclusion** This brief, individual CBT intervention, developed specifically to alter hypochondriacal thinking and restructure hypochondriacal beliefs, appears to have significant beneficial long-term effects on the symptoms of hypochondriasis.

JAMA. 2004;291:1464-1470

www.jama.com

**Author Affiliations:** Department of Psychiatry, Brigham and Women's Hospital and Harvard Medical School, Boston, Mass.

**Corresponding Author:** Arthur J. Barsky, MD, Department of Psychiatry, Brigham and Women's Hospital, 75 Francis St, Boston, MA 02115 (abarsky@partners.org).

predisposed individuals to suspect that they have become ill. This suspicion leads them to selectively attend to benign bodily sensations and health information that confirm their suspicion and to ignore disconfirmatory evidence. Benign bodily sensations are thereby amplified and misattributed to the putative disease, which further substantiates their disease convictions. We have developed a cognitive behavior therapy (CBT) that specifically targets the cognitive and behavioral amplifiers of benign bodily symptoms that propel this hypochondriacal cycle of disease conviction and symptom amplification. Patients are helped to correct faulty symptom attributions, restructure beliefs and expectations about health and disease, correct misunderstandings about the medical care process, modify maladaptive illness behaviors, and learn techniques of selective attention and distraction.

We hypothesized that the CBT intervention would alleviate the symptoms of hypochondriasis more effectively than medical care as usual.

## METHODS

### Study Design

A randomized, usual care control group design was used. To avoid a "contamination" effect, the individual primary care physician was the unit of randomization, and all patients clustered within each physician's practice were assigned to the experimental treatment (individual CBT and a consultation letter to the primary care physician) or to the control condition. Subjects were assessed immediately before and 6 and 12 months after the completion of treatment.

Subjects were recruited by screening with a self-report hypochondriasis questionnaire (composed of the Whiteley Index and Somatic Symptom Inventory), using a predetermined cutoff score selected to identify a sample, half of whom met *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)* criteria for hypochondriasis and half of whom had subthreshold (subdefinitional) hypochondriasis.

The precise cutoff score of 150 (score range, 52-258) was determined from previous studies<sup>3,17-19</sup> and was chosen because we believed that many subthreshold individuals were sufficiently hypochondriacal to be in need of treatment and would be able to benefit from it. Signed informed consent was obtained in accordance with the Brigham and Women's Hospital Human Research Committee requirements. Subjects meeting initial eligibility criteria and exceeding the screening cutoff completed the baseline research battery, which included a second administration of the screening questionnaire and a determination of their medical morbidity.

Physicians were randomized immediately following the baseline research interview by a staff member not connected to the research, using a random numbers table. Randomization was stratified by physician seniority (house staff or attending staff) and by practice volume (part-time or full-time for attending staff, primary care program or traditional internal medicine program for house staff). All research data were collected by research assistants who were blind to the patient's treatment status, and the study therapists and investigators had no foreknowledge of treatment assignment. The therapists had no role in data collection. Subjects were compensated for each research interview, but not for undergoing treatment.

### Subjects and Settings

Subjects were accrued from 2 sources: (1) successive patients attending primary care practices in 2 large, academic medical centers, and (2) volunteers responding to public announcements of a treatment study for "health anxiety and hypochondriasis."

The inclusion criteria were age older than 18 years, English fluency and literacy, having seen a primary care physician in the last 12 months, and exceeding the hypochondriasis screening cutoff score on both occasions. Exclusion criteria included major medical morbidity expected to worsen signifi-

cantly in the next 12 months; somatoform pain disorder; psychosis or suicide risk; and ongoing, symptom-contingent, disability determinations, workers' compensation proceedings, or litigation.

### Treatment Conditions and Therapists

Cognitive behavior therapy was administered individually in six 90-minute sessions at weekly intervals. Each session was tightly scripted (manual available from the authors) and devoted to 1 of 5 factors that cause patients to amplify somatic symptoms and misattribute them to serious disease: attention and bodily hypervigilance, beliefs about symptom etiology, circumstances and context, illness and sick role behaviors, and mood. Each session consisted of educational information about the symptom amplifiers, an illustrative exercise, and a discussion to personalize the material presented. The 3 study therapists had master's or doctoral degrees and prior CBT experience. Treatment sessions were held in offices in the department of psychiatry.

Because it was considered essential that the patients' ongoing medical management be coordinated with the CBT to alter their understanding of symptoms, disease, and medical care, a standardized consultation letter was sent to each patient's primary care physician. This letter (available from the authors) contained the following 5 practical suggestions for medical management that were designed to augment the individual therapy: (1) make improved coping with somatic symptoms rather than symptom elimination the goal of medical management; (2) uncouple access to the physician from symptom status by scheduling regular appointments; (3) provide only limited reassurance; (4) explain the patient's symptoms using the model of cognitive and perceptual symptom amplification; and (5) be conservative in medical diagnosis and treatment, within the bounds of appropriate medical practice.

Treatment fidelity was assessed by auditing audiotapes of randomly se-

lected therapy sessions from all 3 therapists; adherence to the CBT manual was excellent. Receipt of the consultation letter was acknowledged by 96.8% of the primary care physicians.

### Assessment

**Outcome Variables.** The primary outcome measure was the Whately Index, a widely used self-report questionnaire of hypochondriacal attitudes and beliefs, whose validity, reliability, and sensitivity to change have been demonstrated.<sup>20,21</sup> Secondary outcomes included health-related anxiety, assessed with the Health Anxiety Inventory, a 14-item, self-report questionnaire that is minimally influenced by the presence of major medical illness and has good validity, internal consistency, and reliability.<sup>22</sup> The frequency of hypochondriacal thoughts was assessed with the Hypochondriacal Cognitions Questionnaire, requiring the patient to rate how often each of 18 disease-related thoughts occurs. Hypochondriacal somatic symptoms were assessed with the Somatic Symptom Inventory.<sup>17,23</sup> The reliability, internal consistency, and convergent validity of this 13-item questionnaire have been demonstrated previously.<sup>17,18,23-26</sup> A clinical diagnosis of *DSM-IV* hypochondriasis was made by trained research assistants using the Structured Diagnostic Interview for Hypochondriasis, a highly structured interview shown in previous work to have concurrent, convergent, and discriminant validity.<sup>27-29</sup>

The tendency to amplify benign bodily sensation and experience it as noxious, unpleasant, and alarming was assessed with the Somatosensory Amplification Scale. This questionnaire has good reliability and validity and is sensitive to change as a result of attention training.<sup>14,30-33</sup>

Role impairment and functional status were determined with the Functional Status Questionnaire.<sup>34</sup> This valid and reliable self-report questionnaire was developed for use in ambulatory medical populations.<sup>34,35</sup> It includes subscales assessing intermediate activities of daily liv-

ing (eg, doing errands, working around the house) and social activities (eg, seeing friends, participating in community activities).

**Covariates.** Psychiatric comorbidity was assessed with the Hopkins Symptom Checklist-90.<sup>36,37</sup> This widely used, 90-item instrument has excellent psychometric properties and provides an overall measure of generalized psychiatric distress.<sup>37</sup> Participant status indicated whether the subject was a patient accrued in the clinic or a volunteer recruited from the community. Aggregate medical morbidity was assessed in 2 ways: the Duke Severity of Illness Scale<sup>38,39</sup> was used for patients accrued from primary care practices. This structured audit of the medical record was conducted by a research physician who was blind to treatment status. For study volunteers, their primary care physicians provided 2 ordinal ratings of aggregate medical morbidity.

### Data Analysis

An intent-to-treat approach was used in all analyses. The last-observation-carried-forward approach was used to impute scores for missing data values. Although the physician was the unit of randomization, only 11 physicians had more than 1 subject. Thus, any approach to estimating clustering as an adjustment for physician effects would likely lead to unstable estimates of the within-cluster correlation. Therefore, to address the potential effect of clustering on the results, we selected 1 subject at random from each of these 11 physicians for analysis, resulting in the exclusion of 24 subjects (n=163). These results did not differ in any respect from those obtained with the full sample (N=187), which was therefore used for all subsequent analyses. The 2 groups were compared 6 and 12 months after treatment on the primary and secondary outcome variables. General linear modeling was used as the primary analytic approach, in which a series of univariate and multivariate repeated measures analysis of covariance (ANCOVA and MANOVA) models were used. This approach permitted the modeling of

both individual and sets of outcome measures as a function of treatment effects (between subjects), time of assessment (within subjects), and covariates (educational level, generalized psychiatric distress, and participant status [patient vs volunteer]). Hence, the interaction effect of treatment  $\times$  time was of most interest. In cases where the overall MANOVA was significant, or a priori contrasts were planned, only the ANCOVA results are presented, using the Greenhouse-Geisser adjustment. Individual, planned contrasts were calculated for each outcome measure comparing baseline and 6-month, and baseline and 12-month assessments, respectively. Effect sizes and threshold values for clinical significance were also derived for each analysis. All tests of statistical significance were interpreted with a criterion of  $P < .05$ . Statistical analyses were performed using SPSS, release 12.0.1 for Windows (SPSS Inc, Chicago, Ill).

## RESULTS

### Subject Accrual

The study was conducted between September 1997 and November 2001. A total of 6307 individuals completed the screening questionnaire, of whom 776 (12.3%) exceeded the cutoff score (FIGURE). Two hundred nineteen individuals declined to participate, 156 proved to be ineligible, and 214 could not be reached subsequently, resulting in a total of 187 subjects (30.2% of those eligible) who participated. A random sample (n=191) of the 589 nonparticipants was compared with those who participated. The nonparticipants were older (46.4 vs 42.3 years,  $t_{376}=2.96$ ,  $P=.003$ ), had less education (19.1% completed college vs 29.4%,  $\chi^2_6=42.6$ ,  $P<.001$ ), and were more likely to be male (38.9% vs 23.5%,  $\chi^2_1=10.5$ ,  $P<.001$ ).

Of the 102 patients in the treatment arm, 63 (61.8%) attended all 6 sessions, 13 (12.7%) attended 4 or 5 sessions, 12 (11.8%) attended 1 to 3 sessions, and 14 (13.7%) attended none. Six-month follow-up was obtained for 85 (83.3%) of the 102 treatment pa-

tients and for 76 (89.4%) of the 85 control patients (87% in person and 13% by telephone). Twelve-month follow-up was obtained for 92 (90.2%) of treatment patients and 78 (91.8%) of control patients (84.7% in person and 15.3% by telephone). These attrition rates do not differ significantly between groups. Six-month follow-up was obtained for 92% of treatment completers (those attending  $\geq 4$  sessions) and 57.6% of treatment dropouts. Twelve-month follow-up was obtained for 94.7% of treatment completers and 76.9% of treatment dropouts.

### Treatment Groups at Baseline

The sociodemographic characteristics of the 2 treatment groups did not differ significantly (TABLE 1). They were predominantly women, middle-aged, and reported a history of hypochondriasis for approximately 11 years. Educational level and generalized psychiatric distress did not differ significantly in the 2 groups at baseline but were used as covariates in the analyses because of their established relationships to the outcome variables of interest. The treatment and control groups did not differ significantly in aggregate medical morbidity at baseline.

Eighty patients were recruited from primary care practices and 107 were volunteers. At baseline, volunteers were significantly more symptomatic and more disabled than patients on the major outcome measures of hypochondriacal symptoms ( $P < .001$ ), health-related anxiety ( $P < .001$ ), and intermediate activities of daily living ( $P < .001$ ). Repeated measures of ANCOVA were performed on the Whiteley Index modeled as a function of participant status (patients vs volunteers), treatment (CBT vs usual care) and assessment point (baseline, 6 months, and 12 months), and all 2- and 3-way interaction terms. The 3-way interaction was significant ( $F_{2,362} = 3.38$ ,  $P = .04$ ), indicating that volunteers had higher Whiteley Index scores at baseline and experienced a larger treatment effect (difference between CBT and usual care) at 12 months than the patients

( $F_{1,181} = 5.04$ ,  $P = .03$ ), but not at 6 months ( $F_{1,181} = 2.37$ ,  $P = .13$ ). Consequently, participant status (patient or volunteer) also was included as a covariate in the models.

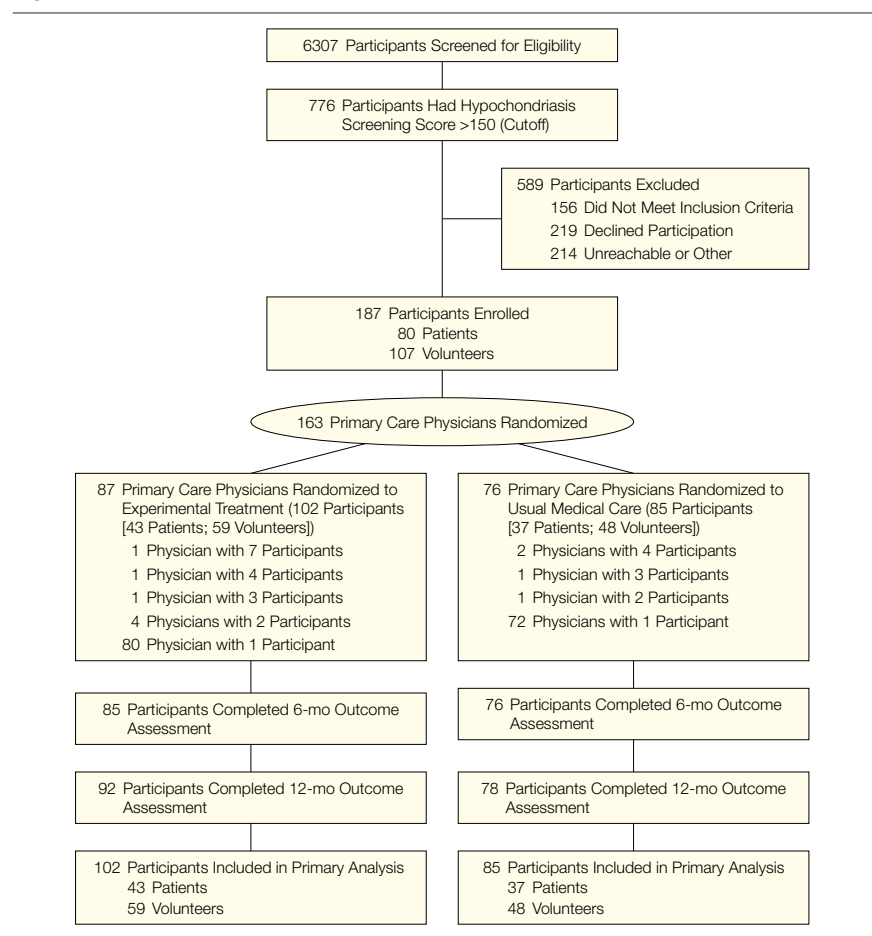
### Treatment Outcomes

The results for the Whiteley Index, the primary outcome measure, are shown in TABLE 2, using repeated measures ANCOVA. There was a statistically significant interaction effect for group (treatment vs control) by assessment time (baseline, 6-month, and 12-month follow-up). Post-hoc tests of within-subjects contrasts disclosed a statistically significant improvement in the treatment vs control group at both 6-month ( $F_{1,182} = 20.1$ ,  $P < .001$ ) and 12-month follow-up, compared with baseline ( $F_{1,182} = 14.2$ ,  $P < .001$ ). The treat-

ment effect size for the Whiteley Index was  $r = 0.31$  at 6 months and  $r = 0.27$  at 12 months.

The secondary outcome measures of hypochondriacal symptoms were analyzed in the same manner as the Whiteley Index. For hypochondriacal thought frequency, health anxiety, and somatosensory amplification, the interactions between group and assessment were statistically significant, indicating a significant treatment effect on these measures (see Table 2). Post-hoc tests of within-subjects contrasts for hypochondriacal thought frequency revealed a significantly greater improvement in the treatment vs control group at 6-month ( $F_{1,182} = 6.97$ ,  $P = .009$ ), and at 12-month follow-up ( $F_{1,182} = 5.64$ ,  $P = .02$ ). For health anxiety, post-hoc tests of within-subjects contrasts re-

**Figure.** Profile of Randomized Controlled Trial



vealed a significantly greater improvement in the treatment vs control group at both 6 months ( $F_{1,182}=6.73, P=.01$ ) and 12 months compared with baseline ( $F_{1,182}=7.01, P=.009$ ). For amplification, individual tests of within-subjects contrasts revealed a significantly greater improvement in the treatment vs control group at 6 months ( $F_{1,182}=8.47, P=.004$ ) and at 12 months

( $F_{1,182}=7.70, P=.006$ ). Hypochondriacal somatic symptoms were not significantly improved by treatment.

The 2 treatment groups were compared on measures of functional status using MANCOVA as the omnibus test, with intermediate and social activities combined as dependent measures, again including baseline educational level, psychiatric comorbidity, and participant status as covariates. TABLE 3 indicates a statistically significant interaction effect for group by assessment time for intermediate activities ( $F_{2,364}=12.32, P<.001$ ), but no statistical significance for social activities ( $F_{2,364}=2.29, P=.10$ ). Post-hoc tests of within-subjects contrasts for intermediate activities of daily living revealed that there was a statistically significant improvement in the treatment vs control group at both 6 months ( $F_{1,182}=18.44, P<.001$ ) and at 12 months ( $F_{1,182}=18.30, P<.001$ ). The treatment effect size for intermediate activities at 12 months was  $r=0.30$ . For social activities, post-hoc tests of within-subjects contrasts revealed that there was a statistically significant improvement in the treatment vs control group at 12 months ( $F_{1,182}=3.74, P=.05$ ), but no significance at 6 months ( $F_{1,182}=3.06, P=.07$ ). The treatment effect size for social activities at 12 months was  $r=0.23$ .

No therapist effect was found, ie, when the patients treated by each of the therapists were compared, no significant differences were found for any of the outcome measures. Although the intervention did not include treatment for comorbid psychiatric disorder, subjects were free to obtain such treatment as they or their physicians saw fit. At 6-month follow-up, 20 CBT patients (19.6%) and 19 control patients (22.4%) had initiated a psychotropic medication or care with a mental health professional since inception. At 12 months, 6 CBT patients (5.9%) and 5 control patients (5.9%) had initiated a psychotropic medication or mental health care since their 6-month follow-up. These rates do not differ significantly between groups.

**Table 1.** Sociodemographic Characteristics

	Treatment Group, No. (%) (n = 102 [59 Volunteers, 43 Patients])	Control Group, No. (%) (n = 85 [48 Volunteers, 37 Patients])	P Value
Age, mean (SD), y	40.66 (12.60)	44.29 (13.75)	.06
Women	76 (74.5)	67 (78.8)	.49
Ethnicity			
White	72 (70.6)	63 (74.1)	.23
Black	17 (16.7)	11 (12.9)	
Hispanic	2 (2.0)	7 (8.2)	
Other	11 (10.7)	4 (4.8)	
Education			
Graduate/professional	29 (28.4)	17 (20.0)	.12
College graduate	34 (33.3)	21 (24.7)	
Some college	21 (20.7)	23 (27.0)	
High school graduate	11 (10.8)	19 (22.4)	
Grades 7-11	7 (6.8)	5 (5.9)	
Employed	70 (68.6)	53 (62.4)	.37
DSM-IV diagnosis of hypochondriasis	67 (65.7)	48 (56.6)	.20
Age of onset, mean (SD), y	30.54 (14.00)	32.65 (15.79)	.34

Abbreviation: DSM-IV indicates *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*.

**Table 2.** Hypochondriacal Symptoms (N = 187)

	Treatment Group, Mean (SE [95% CI])*	Control Group, Mean (SE [95% CI])*	P Value†
Whiteley Index (score range, 1-5)‡			
Baseline	3.58 (0.054 [3.47-3.68])	3.51 (0.060 [3.38-3.62])	<.001
6-mo follow-up	2.82 (0.075 [2.68-2.97])	3.21 (0.083 [3.05-3.38])	
12-mo follow-up	2.65 (0.084 [2.48-2.81])	30.02 (0.093 [2.85-3.21])	
Hypochondriacal thought frequency (score range, 1-5)‡			
Baseline	2.09 (0.066 [1.98-2.21])	2.29 (0.064 [2.16-2.41])	.008
6-mo follow-up	1.75 (0.057 [1.63-1.86])	2.14 (0.063 [2.01-2.26])	
12-mo follow-up	1.62 (0.056 [1.51-1.73])	2.02 (0.062 [1.90-2.14])	
Health anxiety (score range, 1-4)‡			
Baseline	2.68 (0.047 [2.58-2.77])	2.71 (0.051 [2.61-2.81])	.004
6-mo follow-up	2.29 (0.047 [2.20-2.38])	2.51 (0.051 [2.41-2.61])	
12-mo follow-up	2.20 (0.051 [2.10-2.30])	2.44 (0.056 [2.33-2.55])	
Somatosensory amplification (score range, 1-5)‡			
Baseline	3.25 (0.067 [3.12-3.38])	3.04 (0.073 [2.90-3.19])	.003
6-mo follow-up	2.92 (0.068 [2.79-3.06])	2.96 (0.074 [2.81-3.11])	
12-mo follow-up	2.82 (0.070 [2.68-2.96])	2.87 (0.077 [2.72-3.03])	
Hypochondriacal somatic symptoms (score range, 1-5)‡			
Baseline	2.73 (0.048 [2.63-2.82])	2.81 (0.053 [2.70-2.91])	.08
6-mo follow-up	2.16 (0.056 [2.05-2.27])	2.42 (0.061 [2.30-2.55])	
12-mo follow-up	2.00 (0.056 [1.88-2.11])	2.24 (0.061 [2.18-2.36])	

Abbreviation: CI, confidence interval.

\*Adjusted for baseline educational level, psychiatric comorbidity, and participant status.

†P value based on Greenhouse-Geisser adjustment for the interaction between group and assessment from analysis of variance (ANOVA) model covarying for educational level, psychiatric comorbidity, and participant status.

‡Higher scores indicate more severe symptoms.

**COMMENT**

This 6-session CBT, specifically targeting the cognitive and perceptual mechanisms thought to underlie hypochondriasis, appears to significantly improve a range of hypochondriacal symptoms, beliefs, and attitudes. These effects are evident at 6-month follow-up and persist at 12 months. The effects on role functioning are not consistently significant at 6 months but emerge at 12 months, the primary end point. These treatment effects are seen using an intent-to-treat analysis, after adjusting for psychiatric comorbidity, sociodemographic characteristics, and participant status (patient vs volunteer) at baseline. The findings are compatible with the only other major trial reported to date and expand on it by having a control group available for comparison at long-term follow-up.<sup>13</sup>

Though the magnitude of the treatment effect is modest, it is important to remember that hypochondriasis generally has been considered a refractory and chronic disorder (the mean duration of illness was 11 years in this study) for which there has been no empirically validated treatment. In addition, this CBT was brief (only 6 sessions) and included no follow-up “booster” sessions. Finally, patients were not treated in the study for comorbid psychiatric disorder, and the continued presence of these disorders likely moderated the treatment effect.

The study has several limitations. First, many eligible patients did not participate, limiting the generalizability of the findings. Those who did consent to participate might have been more receptive to a psychosocial approach and hence benefited more from it than those who did not consent. On the other hand, the study participants might be less severely hypochondriacal and more responsive to their medical physician's ministrations and/or more likely to improve spontaneously, which would tend to reduce the treatment effect. In addition, 25% of the subjects attended less than 4 treatment sessions, suggesting that future efforts must be directed toward reducing treatment dropout. This

**Table 3.** Functional Status and Role Impairment (N = 187)

	Treatment Group, Mean (SE [95% CI])*	Control Group, Mean (SE [95% CI])*	P Value†
Intermediate activities of daily living (0-100)‡			
Baseline	74.92 (2.01 [70.89-78.96])	74.99 (2.25 [70.57-79.43])	<.001
6-mo follow-up	82.54 (2.11 [78.39-86.71])	70.61 (2.31 [66.05-75.18])	
12-mo follow-up	83.42 (2.03 [79.43-87.42])	72.23 (2.22 [67.85-76.62])	
Social activities (0-100)‡			
Baseline	81.41 (2.35 [76.78-86.05])	81.61 (2.58 [76.52-86.69])	.10
6-mo follow-up	86.02 (2.20 [81.69-90.35])	80.55 (2.41 [75.80-85.30])	
12-mo follow-up	86.11 (2.34 [81.50-90.72])	79.26 (2.56 [74.21-84.32])	

\*Adjusted for baseline educational level, psychiatric comorbidity, and participant status.

†P value based on Greenhouse-Geisser adjustment for the interaction between group and assessment from analysis of variance (ANOVA) model covarying for educational level, psychiatric comorbidity, and participant status.

‡0-100 (100 = maximal function).

is not unusual for such studies however; Kashner et al,<sup>40</sup> for example, found that 56% of somatization disorder patients randomized to group therapy failed to attend a single session.

Second, we lacked an “attention” control, ie, a generic psychosocial intervention providing nonspecific attention, support, concern, and positive expectation. This limits our ability to attribute the treatment effect to the specific cognitive and behavioral strategies of the intervention. However, the fact that the cognitive processes thought to underlie the disorder (eg, hypochondriacal cognitions, health beliefs, amplification) improved with treatment suggests that the treatment had a specific effect.

Third, considerable improvement occurred in the control group. This was likely due to the inadvertent inclusion of patients with transient hypochondriasis, probably because the 2 screening measures were too close to each other in time (approximately 3 weeks). Additionally, regression to the mean and the supportive effect of being enrolled in a longitudinal study contributed to the high rate of spontaneous improvement. A Hawthorne effect may also have occurred whereby control physicians, having learned of the study, made a greater effort to help their hypochondriacal patients.

Finally, study subjects came from 2 different sources. Participant status, however, was included as a covariate in all analyses, and the fact that these 2 groups differed at baseline confers some measure of generalizability on the findings.

Hypochondriacal attitudes and concerns improved more than somatic symptoms did. This finding, although it might seem counterintuitive, was actually expected: the treatment was intended to improve coping with symptoms rather than curing them outright (“care rather than cure”). This had both an empirical and a conceptual basis. Empirically, clinical experience and intervention trials for a variety of functional somatic syndromes suggest that the patients who do best are those who learn to compensate for, rather than attempting to eliminate, their somatic distress. Conceptually, hypochondriacal somatic symptoms cannot simply be stripped away with symptomatic treatment because they exist for underlying psychological and interpersonal reasons. This suggests that a realistic goal in treating hypochondriasis is amelioration of distressing fears and beliefs and improved coping, rather than the elimination of somatic symptoms per se.

We are unable to partial out the variance in treatment effect between the CBT and the physician consultation letter. That the latter may have been beneficial is suggested by 2 studies with somatization disorder patients in which a psychiatric consultation letter alone resulted in lower health care costs along with either improved or stable physical functioning.<sup>41-43</sup> This points to the critical importance of seamlessly integrating the psychosocial care of these patients with their medical care.

The treatment offered in this study was not attractive to many hypochon-

driacal patients, and only 30% of those eligible entered the trial. Hypochondriacal individuals are by definition convinced of the medical nature of their condition and therefore psychosocial treatment seems nonsensical to them. Although a major problem, this should not detract from the fact that those patients who did undergo treatment benefited from it. And since hypochondriasis is a prevalent problem in ambulatory medical practice,<sup>6,44</sup> this fraction of hypochondriacal patients still represents a sizeable population. The

treatment must be made more attractive in the future by seamlessly integrating it into the primary care process and conducting it in the medical setting (as our treatment was not). The treatment effect could also be strengthened by increasing the number of sessions to 8 and by adding booster sessions.

**Author Contributions:** Dr Barsky, as principal investigator, had full access to all of the data in this study and takes responsibility for the integrity of the data and accuracy of the data analysis.  
**Study concept and design:** Barsky, Ahern.  
**Acquisition of data:** Barsky, Ahern.

**Analysis and interpretation of data:** Barsky, Ahern.  
**Drafting of the manuscript:** Barsky, Ahern.  
**Critical revision of the manuscript for important intellectual content:** Barsky, Ahern.  
**Statistical expertise:** Ahern.  
**Obtained funding:** Barsky, Ahern.  
**Administrative, technical, or material support:** Barsky.  
**Supervision:** Barsky.  
**Funding/Support:** This investigation was supported by research grant MH40487 from the National Institute of Mental Health.  
**Role of the Sponsor:** The sponsor played no role in the data collection and analysis, manuscript preparation, or authorization for publication.  
**Acknowledgment:** We wish to acknowledge the enormous contributions made by Duff Bailey, MD, Heli M. Peekna, PhD, and Daniel T. Beck, MSW; and by the research assistants Susan L. Clancy, Beth A. Delamater, Jeanne M. Fama, Shera D. Gruen, Elizabeth Liu, and Lindsay B. Renner.

## REFERENCES

- Kellner R. *Somatization and Hypochondriasis*. New York, NY: Praeger; 1986.
- Noyes R, Kathol RG, Fisher MM, Phillips BM, Suelzer M, Woodman CL. One-year follow-up of medical outpatients with hypochondriasis. *Psychosomatics*. 1994;35:533-545.
- Barsky AJ, Fama JM, Bailey ED, Ahern DK. A prospective 4-5 year study of DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1998;55:737-744.
- Lipsitt DR, Starcevic V. *Hypochondriasis: Modern Perspectives on an Ancient Malady*. New York, NY: Oxford University Press; 2001.
- Kellner R, Abbott P, Pathak D, Winslow WW, Umland BE. Hypochondriacal beliefs and attitudes in family practice and psychiatric patients. *Int J Psychiatry Med*. 1983;13:127-139.
- Barsky AJ, Wyshak G, Klerman GL, Latham KS. The prevalence of hypochondriasis in medical outpatients. *Soc Psychiatry Psychiatr Epidemiol*. 1990;25:89-94.
- Warwick HM, Clark DM, Cobb AM, Salkovskis PM. A controlled trial of cognitive behavioural treatment of hypochondriasis. *Br J Psychiatry*. 1996;169:189-195.
- Avia MD, Ruiz A, Olivares ME, et al. The meaning of psychological symptoms: effectiveness of a group intervention with hypochondriacal patients. *Behav Res Ther*. 1996;34:23-31.
- Warwick HMC, Marks IM. Behavioural treatment of illness phobia and hypochondriasis: a pilot study of 17 cases. *Br J Psychiatry*. 1988;152:239-241.
- Stern R, Fernandez M. Group cognitive and behavioral treatment for hypochondriasis. *BMJ*. 1991;303:1229-1231.
- Papageorgiou C, Wells A. Effects of attention training in hypochondriasis: a brief case series. *Psychol Med*. 1998;28:193-200.
- Bouman TK, Visser S. Cognitive and behavioural treatment of hypochondriasis. *Psychother Psychosom*. 1998;67:214-221.
- Clark DM, Salkovskis PM, Hackmann A, et al. Two psychological treatments for hypochondriasis. *Br J Psychiatry*. 1998;173:218-225.
- Barsky AJ, Wyshak G. Hypochondriasis and somatosensory amplification. *Br J Psychiatry*. 1990;157:404-409.
- Barsky AJ. Somatosensory amplification and hypochondriasis. In: Lipsitt DR, Starcevic V, ed. *Hypochondriasis: Modern Perspectives on an Ancient Malady*. New York, NY: Oxford University Press; 2001: 223-248.
- Barsky AJ. A 37-year-old man with multiple somatic complaints. *JAMA*. 1997;278:673-679.
- Barsky AJ, Wyshak G, Klerman GL. Transient hypochondriasis. *Arch Gen Psychiatry*. 1990;47:746-752.
- Barsky AJ, Wyshak G, Klerman GL. Hypochondriasis: an evaluation of the DSM-III criteria in medical outpatients. *Arch Gen Psychiatry*. 1986;43:493-500.
- Barsky AJ, Cleary PD, Sarnie MK, Klerman GL. The course of transient hypochondriasis. *Am J Psychiatry*. 1993;150:484-488.
- Pilowsky I. Dimensions of hypochondriasis. *Br J Psychiatry*. 1967;113:89-93.
- Pilowsky I. A general classification of abnormal illness behaviours. *Br J Med Psychol*. 1978;51:131-137.
- Salkovskis PM, Rimes KA, Warwick HMC, Clark DM. The Health Anxiety Inventory: development and validation of scales for the measurement of health anxiety and hypochondriasis. *Psychol Med*. 2002;32:843-853.
- Weinstein MC, Berwick DM, Goldman PA, Murphy JM, Barsky AJ. A comparison of three psychiatric screening tests using receiver operating characteristic (ROC) analysis. *Med Care*. 1989;27:593-607.
- Barsky AJ, Wyshak G, Latham KS, Klerman GL. Hypochondriacal patients, their physicians, and their medical care. *J Gen Intern Med*. 1991;6:413-419.
- Kroenke K, Spitzer RL, deGruy FV, Swindle R. A symptom checklist to screen for somatoform disorders in primary care. *Psychosomatics*. 1998;39:263-272.
- Barsky AJ, Wyshak G, Klerman GL. Medical and psychiatric determinants of outpatient medical utilization. *Med Care*. 1986;24:548-560.
- Noyes R, Happel RL, Yagla SJ. Correlates of hypochondriasis in a nonclinical population. *Psychosomatics*. 1999;40:461-469.
- Noyes R, Kathol R, Fisher M, Phillip B, Suelzer M, Holt C. The validity of DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1993;50:961-970.
- Barsky AJ, Cleary PD, Spitzer RL, Williams JBW, Wyshak G, Klerman GL. A structured diagnostic interview for hypochondriasis: a proposed criterion standard. *J Nerv Ment Dis*. 1992;180:20-27.
- Speckens AEM, Spinhoven P, Sloekers PPA, Bolk JH, van Hemert AM. A validation study of the Whitley Index, the Illness Attitude Scales, and the Somatosensory Amplification Scale in general medical and general practice patients. *J Psychosom Res*. 1996;40:95-104.
- Gramling SE, Clawson EP, McDonald MK. Perceptual and cognitive abnormality model of hypochondriasis: amplification and physiological reactivity in women. *Psychosom Med*. 1996;58:423-431.
- Wise TN, Mann LS. The relationship between somatosensory amplification, alexithymia, and neuroticism. *J Psychosom Res*. 1994;38:515-521.
- Barsky AJ, Wyshak G, Latham KS, Klerman GL. The Somatosensory Amplification Scale and its relationship to hypochondriasis. *J Psychiatr Res*. 1990;24:323-334.
- Jette AM, Davies AR, Cleary PD, et al. The Functional Status Questionnaire: reliability and validity when used in primary care. *J Gen Intern Med*. 1986;1:143-149.
- Cleary PD, Jette AJ. Reliability and validity of the Functional Status Questionnaire. *Qual Life Res*. 2000;9:747-753.
- Derogatis LR. *The SCL-90-R: Administration, Scoring, and Procedures Manual II*. Towson, Md: Clinical Psychometric Research; 1983.
- Derogatis LR. *SCL-90-R: Administration, Scoring, and Procedures Manual*. 3rd ed. Minneapolis, Minn: National Computer Systems Inc; 1994.
- Parkerson GR Jr, Michener JL, Wu LR, et al. Associations among family support, family stress, and personal functional health status. *J Clin Epidemiol*. 1989;42:217-229.
- Parkerson GP, Broadhead WE, Chin-Kit JT. Quality of life and functional health of primary care patients. *J Clin Epidemiol*. 1992;45:1303-1313.
- Kashner TM, Rost K, Cohen B, Anderson M, Smith GR. Enhancing the health of somatization disorder patients: effectiveness of short-term group therapy. *Psychosomatics*. 1995;36:462-470.
- Rost K, Kashner TM, Smith GR Jr. Effectiveness of psychiatric intervention with somatization disorder patients: improved outcomes at reduced costs. *Gen Hosp Psychiatry*. 1994;16:381-387.
- Smith GR, Rost K, Kashner TM. A trial of the effect of a standardized psychiatric consultation on health outcomes and costs in somatizing patients. *Arch Gen Psychiatry*. 1995;52:238-243.
- Smith GR Jr, Monson RA, Ray DC. Psychiatric consultation in somatization disorder: a randomized controlled study. *N Engl J Med*. 1986;314:1407-1413.
- Escobar JI, Gara M, Waitzkin H, Cohen Silver R, Holman A, Compton W. DSM-IV hypochondriasis in primary care. *Gen Hosp Psychiatry*. 1998;20:155-159.