

Hoarding and Treatment Response in 38 Nondepressed Subjects With Obsessive-Compulsive Disorder

Donald W. Black, M.D.; Patrick Monahan, M.A.; Janelle Gable, R.N.;
Nancee Blum, M.S.W.; Gerard Clancy, M.D.; and Peggy Baker, M.D.

Objective: The authors studied factors associated with short-term treatment response in 38 nondepressed subjects with DSM-III-R obsessive-compulsive disorder (OCD).

Method: The subjects completed 12 weeks of treatment with paroxetine (N = 20), placebo (N = 8), or cognitive-behavioral therapy (N = 10). Clinician and self-rated measures were gathered at baseline, during treatment, and after treatment.

Results: Seventeen (45%) subjects had "much" or "very much" improvement and achieved at least a 40% decrease in their total Yale-Brown Obsessive Compulsive Scale (Y-BOCS) score. Responders had lower obsessive-compulsive scores on the Symptom Checklist 90-Revised, had a lower checking score on the Maudsley Obsessive-Compulsive Inventory, were less likely to have had prior drug therapy, and in general suffered more obsessive-compulsive symptoms. They were significantly less likely to have hoarding obsessions and corresponding compulsions. The latter finding was confirmed using multiple regression analysis.

Conclusion: Hoarding is an important symptom that predicts poor treatment response in patients with OCD.

(*J Clin Psychiatry* 1998;59:420-425)

Received Jan. 6, 1997; accepted Dec. 4, 1997. From the Department of Psychiatry, University of Iowa College of Medicine, Iowa City.

The authors acknowledge the financial support of SmithKline Beecham Pharmaceuticals.

Presented at the 150th annual meeting of the American Psychiatric Association, May 21, 1997, San Diego, Calif.

Reprint requests to: Donald W. Black, M.D., University of Iowa College of Medicine, Psychiatry Research - MEB, Iowa City, IA 52242-1000.

Effective treatments for obsessive-compulsive disorder (OCD) including serotonin reuptake inhibitor (SRI) antidepressants and behavioral therapy have been available for nearly a decade in the United States and, yet, research has pinpointed surprisingly few variables predictive of treatment response. The issue is important because up to 35% of compliant OCD patients respond poorly to

adequate medication trials.¹ Therefore, efforts to explain this phenomenon may eventually help to reduce the percentage of patients having an inadequate response.

There have been several recent efforts to assess response predictors. Alarcon et al.² evaluated 45 patients treated with clomipramine for a mean of over 18 months and identified greater illness severity, cleaning rituals (excluding handwashing), and a family history of OCD as significant predictors of poor response. Ravizza et al.³ reported that in a group of 53 patients treated with clomipramine or fluoxetine for 6 months, nonresponders had a younger age at onset and longer duration of illness, showed a higher frequency of compulsions and washing rituals, had a more chronic course, were more likely to have a concomitant schizotypal personality disorder, and had more prior hospitalizations. Jenike et al.⁴ had pointed out much earlier that schizotypal personality disorder specifically predicted poor treatment outcome. Axis II disorders in general (specifically avoidant, borderline, and schizotypal personality) were linked to poor response to clomipramine in a 10-week study.⁵ The presence of tics also predicts poor response to SRIs, although augmentation with antipsychotics may help.⁶ Depression has been cited by Foa⁷ and by Keijsers et al.⁸ as a predictor of poor response to behavior therapy, but other investigators⁹⁻¹³ have not found depression to interfere with response to medication. Several researchers^{9,10} have reported a complete absence of any response predictors in short-term medication trials, as well as in a 2-year follow-up of patients who had received SRIs,¹⁴ but in a reanalysis of the data from the Clomipramine Collaborative Study, age at onset and baseline depression were found to be associated with treatment response.¹⁵

In a study of children and adolescents with OCD, Leonard et al.¹⁶ showed that the National Institute of Mental Health Obsessive-Compulsive Scale score at 5 weeks, the presence of a tic disorder, a parental family history of psychiatric illness, and both severity and duration of illness were associated with a worse outcome at a mean follow-up interval of 3.4 years. Briefly, patients with more severe illness, tic disorders, a positive family history, and a longer duration of illness did less well. A nearly 16-year follow-up by a Danish investigator¹⁷ of 47 children and adolescents with OCD showed that only 1

factor—severity of OCD in childhood—predicted a poor outcome, defined as ongoing OCD in adulthood.

We recently had an opportunity to evaluate sociodemographic and clinical variables potentially predictive of short-term treatment response in a group of patients with OCD. The data were collected in the conduct of 2 treatment protocols, 1 involving medication (paroxetine or placebo), and the other involving cognitive-behavioral therapy. By pooling the data, we were able to look at a larger sample of subjects, all assessed in a similar manner using identical instruments.

METHOD

Subjects

Subjects with OCD were at least 16 years old and were recruited through physician referral and the news media. The subjects of this particular analysis were involved in either a randomized, double-blind study of 3 different doses of paroxetine (60 mg, 40 mg, 20 mg) versus placebo¹⁸ or an open trial of cognitive-behavioral therapy (CBT). The medication study involved a 2-week, single-blind placebo washout followed by 12 weeks of medication. The CBT study involved a 2-week observation period (or washout) followed by once-a-week therapy sessions for 12 weeks. The CBT was conducted by an experienced behavior therapist (N.B.). Written informed consent was obtained from the subjects after they were given a complete description of the study.

All subjects met DSM-III-R criteria¹⁹ for OCD and were in good physical health. The diagnosis was confirmed by using the Structured Clinical Interview for DSM-III-R (SCID),²⁰ which was also used to assess Axis I comorbidity. There was no systematic search for the presence of tics, Tourette's disorder, trichotillomania, or other disorders hypothesized to fall within an obsessive-compulsive spectrum not covered by the SCID. Patients meeting criteria for current major depressive disorder or any other primary Axis I disorder including substance abuse (past 6 months) were excluded. Persons who were pregnant, lactating, psychotic, suicidal, or demented or had significant medical illness were ineligible to participate in either protocol. Persons with a severe personality disorder that would have compromised their ability to participate were also excluded. Subjects were not allowed to undergo additional psychotherapy. Subjects taking psychotropic medication were asked to discontinue the medication 2 weeks before randomization in the medication study, or, in the case of fluoxetine, 30 days before randomization. In the case of CBT, subjects were asked to discontinue psychotropic medication 2 weeks before beginning therapy. Finally, subjects were required to have a Yale-Brown Obsessive Compulsive Scale (Y-BOCS)²¹ score of 16 or greater and a National Institute of Mental Health Obsessive-Compulsive Scale (NIMHOCS)²² score

of 7 or above, and could not have a Hamilton Rating Scale for Depression (HAM-D)²³ score over 16. The response on item 1 of the HAM-D could not exceed a score of 2.

Treatment Protocols

In the first protocol, medication consisting of 1 tablet of paroxetine (20 mg) or placebo was administered in a double-blind fashion. Subjects began taking 1 tablet per day for 7 days and then increased the number to 2 tablets for 7 days, and continued up to a total of 3 tablets per day. When side effects were reported, medication increases were slowed or the dosage was reduced. Study psychiatrists limited their interaction with subjects to discussion of clinical history, explanation of OCD and its symptoms, discussion of medication and its side effects, and general support.

Subjects assigned to receive CBT were all volunteers requesting nondrug therapy and had no prior experience with behavioral treatment. CBT is designed to assist patients in developing strategies to reduce and resist obsessions and compulsions.²⁴ It involves using cognitive therapy to address the errant estimation of danger (i.e., catastrophic thinking) and the exaggerated sense of personal responsibility often seen in OCD patients. This is combined with exposure to feared stimuli paired with response prevention. The therapy was begun by constructing a hierarchy of the patient's fears and avoidance behavior, each rated for severity and intensity. Patients were given a number of tasks to perform at home. All assignments were practiced in vivo, starting with the easiest. At each session, the patient's performance on tasks from the previous session was discussed, and the speed in working through the hierarchy was determined by the patient. Patients were also encouraged to accept a more realistic interpretation of their sense of danger and to lessen their inappropriate sense of responsibility.

Assessments

The Y-BOCS was used to assess the severity of obsessive-compulsive symptoms at each visit. The 10-item scale measures time involved, subjective distress, interference, the ability to resist, and success at resisting, each on a scale from 0 to 4, with 4 representing maximum severity. The NIMHOCS was also used at each visit to measure severity of obsessive-compulsive symptoms. Three Clinical Global Impressions (CGI)²⁵ ratings were made at each visit to assess severity of illness, global improvement, and overall therapeutic effect. The HAM-D was used at baseline and at termination to measure depressive symptoms. The Maudsley Obsessive-Compulsive Inventory (MOC)²⁶ was used to assess obsessionality at baseline. The Symptom Checklist 90-Revised (SCL-90-R)²⁷ was used to assess somatic and psychological symptoms at baseline. The Illness Behavior Questionnaire (IBQ)²⁸ was used to assess somatic concerns and hypochondriacal

behavior at baseline. The Structured Interview for DSM-III-R Personality Disorders (SIDP-R)²⁹ and the Personality Diagnostic Questionnaire-Revised (PDQ-R)³⁰ were both administered to gather information on Axis II disorders. The Family-History Research Diagnostic Criteria (FH-RDC)³¹ were used to assess family history of psychiatric illness. Each patient was assigned a Global Assessment of Functioning (GAF) score,¹⁹ which is used to measure overall functioning and is patterned after the Global Assessment Scale.³²

In addition to these clinical ratings, social, demographic, and illness data were gathered for each subject including age, gender, marital status, age at onset of OCD, occupation, education, prior treatment, and prior hospitalizations. Assessments were made by the project coordinator (J.G.) or a psychiatrist (D.W.B., G.C., or P.B.). Patients receiving CBT were rated by the project coordinator (J.G.). During the 2-week placebo washout (or observation), subjects had to maintain a Y-BOCS score of 16 or greater, and a NIMHOCS score of 7 or more.

Statistical Analysis

A positive response among the 38 completers was defined as achieving a CGI improvement score of 1 or 2 ("very much" or "much" improved) and at least a 40% decrease in the Y-BOCS total score at any visit of the study. Student *t* test was used to compare differences in continuous independent variables. Categorical independent variables were compared using the chi-square test, except when at least 25% of expected cell frequencies were less than 5. In that case, the 2-tailed Fisher exact test was used. Interactions among variables were assessed using logistic regression.

Variables and their interactions associated with a positive response at a *p* value less than .10 were entered into a stepwise logistic regression model. Variables were entered or removed from the model if their partial *p* values were less than .10. This procedure allowed us to derive a significant predictive model comprised only of variables that added a unique predictive effect. The partial *p* values in the regression table describe unique effects because the effect of each independent variable is adjusted for the confounding effects of all other predictors in the model. All calculations were performed with SPSS for Windows (version 8.0; SPSS, Inc., Chicago, Ill.).

The statistical comparisons in Tables 1 and 2 represent standard, preplanned comparisons (based on our clinical experience and the literature); we set the significance level at .05 for these tests. The comparisons of individual Y-BOCS symptoms (Figure 1) are based on clinical experience, but represent secondary hypotheses; we set the significance level at .01 for these tests. The comparison of individual personality items from the PDQ-R represents exploratory hypotheses. Because 134 comparisons are involved, a strict Bonferroni correction would be overly

Table 1. Sociodemographic and Illness Characteristics in 38 Nondepressed Subjects With Obsessive-Compulsive Disorder

Characteristic	Responders (N = 17)	Nonresponders (N = 21)
Age, y, mean (SD)	37.1 (13.9)	43.5 (17.1)
Female, N (%)	10 (58.8)	11 (52.4)
Ever married, N (%)	10 (58.8)	13 (61.9)
Age at onset of OCD, y, mean (SD)	15.6 (12.4)	13.7 (7.2)
Duration of illness, y, mean (SD)	21.4 (15.8)	30.2 (19.3)
Education level, 1–7, 7 = less than 9th grade, mean (SD)	2.4 (1.1)	2.6 (1.2)
Prior hospitalization for OCD, N (%)	0	2 (9.5)
Drug therapy for OCD prior to 3 mo ago, ^a N (%)	4 (23.5)	12 (57.1)
Drug therapy for OCD within last 3 mo, N (%)	4 (23.5)	5 (23.8)
Current Axis I comorbidity, N (%)		
Panic disorder	1 (5.9)	0
Generalized anxiety disorder	1 (5.9)	0
Simple phobia	2 (11.8)	3 (14.3)
Trichotillomania	1 (5.9)	0
Chronic motor tic disorder	1 (5.9)	0
Any Axis I disorder	5 (29.4)	4 (19.0)

^a $\chi^2 = 4.35$, *df* = 1, *p* = .037

conservative since the personality items are not independent, but are rather highly related items from the same instrument. We used an approximate Bonferroni correction in which an alpha of .05 is divided by 10, yielding a significance level of .005.

RESULTS

There were few differences between responders and nonresponders (Table 1). Only 1 illness characteristic, "drug therapy for OCD prior to 3 months ago," was significantly associated with a good response; that is, responders were less likely to have had past drug therapy. Nonresponders had a younger age at onset and had been ill longer, but these differences were not significant. The 2 groups did not significantly differ in their prevalence of current Axis I disorders, as assessed with the SCID.

Table 2 shows that subjects receiving paroxetine or CBT were more likely to respond (50% and 60%, respectively) than those receiving placebo (12.5%), although this trend was not statistically significant. A positive response was associated with several baseline measures of illness severity. Although there was no significant difference in the mean Y-BOCS score between responders and nonresponders, responders were less symptomatic on their baseline NIMHOCS and MOC doubting/conscientious scores (*p* < .10), and were significantly less symptomatic on the MOC checking score. Response groups did not differ on the other more general baseline illness measures such as HAM-D or GAF scores.

Responders had lower (i.e., less severe) scores on the obsessive-compulsive subscale of the SCL-R-90. Responders scored marginally better than nonresponders at baseline on the SCL-90-R Positive Symptom Distress In-

Table 2. Treatment Group Assignment and Baseline Illness Measures in 38 Nondepressed Subjects With Obsessive-Compulsive Disorder*

Group and Measure	Responders (N = 17)	Nonresponders (N = 21)
Treatment group, N (row %)		
Paroxetine (N = 20)	10 (50.0)	10 (50.0)
Cognitive-behavioral therapy (N = 10)	6 (60.0)	4 (40.0)
Placebo (N = 8)	1 (12.5)	7 (87.5)
Obsessive-compulsive measures, mean (SD)		
Y-BOCS total score	25.9 (2.7)	27.3 (3.8)
NIMHOCS score†	8.9 (1.3)	9.6 (1.2)
Maudsley Obsessive-Compulsive Inventory, mean (SD)		
Total score	16.5 (5.8)	19.7 (6.4)
Checking score‡	4.4 (1.9)	5.8 (2.0)
Washing score	3.8 (3.3)	4.4 (2.9)
Slowness/repetition score	3.4 (1.8)	3.4 (1.2)
Doubting/conscientious score§	4.2 (1.8)	5.3 (1.6)
Ruminations Score	0.6 (0.7)	1.1 (0.8)
Clinical Global Impressions scale, severity score	4.8 (0.8)	5.0 (0.7)
Personality Disorders, N (%)		
Any PDQ-R disorder ^a	8 (47.1)	8 (44.4)
Any SIDP-R disorder ^b	3 (21.4)	2 (18.2)
Hamilton Rating Scale for Depression score, mean (SD)	5.6 (2.0)	5.8 (2.9)
Global Assessment of Functioning score, mean (SD)	55.8 (5.2)	52.2 (7.5)
SCL-90-R, ^c mean (SD)		
Symptom Dimension Scores		
Somatization	0.5 (0.4)	0.5 (0.4)
Obsessive-compulsive	1.6 (0.6)	2.1 (0.7)
Interpersonal sensitivity	1.0 (0.7)	1.0 (0.7)
Depression	1.2 (0.5)	1.4 (0.9)
Anxiety	1.0 (0.5)	0.9 (0.5)
Hostility	0.8 (0.7)	0.6 (0.6)
Phobic anxiety	0.4 (0.5)	0.3 (0.4)
Paranoid ideation	0.8 (0.9)	0.6 (0.6)
Psychoticism	0.5 (0.4)	0.7 (0.6)
Global Severity Index (GSI)	0.9 (0.4)	1.0 (0.5)
Positive Symptom Distress Index (PSDI)¶	1.6 (0.3)	1.8 (0.5)
Positive Symptom Total (PST)	49.8 (16.6)	46.4 (13.8)

*Abbreviations: Personality Diagnostic Questionnaire-Revised = PDQ-R; Structured Interview for DSM-III-R Personality Disorders = SIDP-R; Symptom Checklist 90-Revised = SCL-90-R.

† $t = 1.70$, $df = 36$, $p = .098$.

‡ $t = 2.11$, $df = 35$, $p = .042$.

§ $t = 1.86$, $df = 34$, $p = .071$.

|| $t = 2.32$, $df = 36$, $p = .03$.

¶ $t = 1.86$, $df = 36$, $p = .07$.

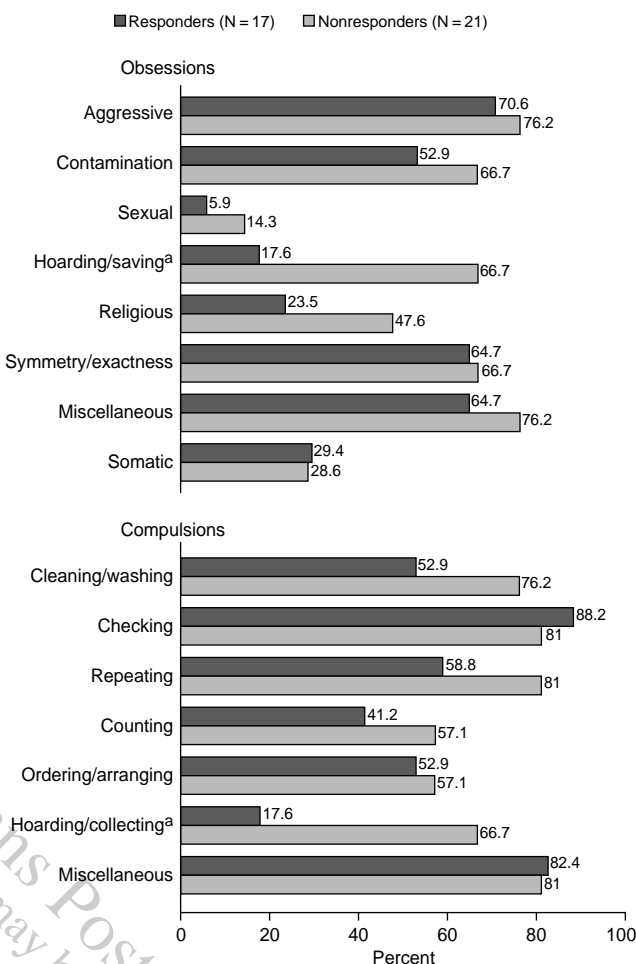
^aN = 18 for nonresponders.

^bResponders, N = 14; nonresponders, N = 11.

^cHigher scores = worse; GSI = 90-item grand total/90; PST = number of positive (non-zero) responses; PSDI = 90-item grand total/PST.

dex (PSDI; $t = 1.86$, $df = 36$, $p = .07$). This index yields the average item score of only the items with non-zero responses. There were no differences between the groups for individual IBQ items (data not shown). The presence of a personality disorder was not related to response, although 1 PDQ-R item separated responders from nonresponders.

The strongest finding was the relationship between response and hoarding/saving obsessions and hoarding/collecting compulsions (Figure 1). Responders were sig-

Figure 1. Baseline Y-BOCS Symptom Checklist

^a $\chi^2 = 9.13$, $df = 1$, $p = .003$.

nificantly less likely to have these symptoms than nonresponders (17.6% versus 66.7% for both symptoms). No other obsession or compulsion predicted poor treatment response. Inspection of the figure shows that nonresponders had more obsessive-compulsive symptoms than responders in 12 of 15 categories (goodness-of-fit test, $\chi^2 = 5.4$, $df = 1$, $p = .02$).

Several differences were statistically significant within a particular treatment cell. Age, not a significant predictor in the overall analysis, significantly predicted response within the CBT cell ($t = 2.52$, $df = 8$, $p = .04$), where responders were on the average 20 years younger than nonresponders (mean = 28.2 versus 49.3 years, respectively). Paroxetine responders (mean = 41.7 years) were only an average of 6 years younger than paroxetine nonresponders (mean = 47.4), a nonsignificant difference. A poor score on the IBQ irritability subscale significantly predicted good response only within the cognitive therapy cell (mean = 2.7 for responders, 0.5 for nonresponders, $t = -2.35$, $df = 8$, $p = .05$).

Table 3. Stepwise Logistic Regression in 38 Subjects With OCD*

Independent Variable	Wald χ^2	df	p	Adjusted Odds Ratio
Hoarding/saving obsessions	6.81	1	.009	4.0
Interaction: Treatment by "any affective disorder"				
Paroxetine versus CBT	7.08	1	.008	.05
Placebo versus CBT	4.65	1	.03	.04

*Model $\chi^2 = 19.7$, $df = 3$, $p = .0002$, $N = 38$.

Abbreviation: CBT = cognitive-behavioral therapy.

Hoarding/saving obsessions were a significant predictor in the stepwise logistic regression model. In addition, the interaction between "family history of any affective disorder" (major depressive disorder or bipolar disorder) and treatment cell added significant prediction to the model (Table 3).

DISCUSSION

The study produced several interesting findings. Hoarding, an important and complex phenomenon involving obsessions and compulsions, appears relatively treatment resistant. Hoarders constitute a large subgroup of OCD patients and are characterized by their need to save, store, and collect items not generally saved by most people and their inability to discard items.³³ These behaviors were true of our hoarders as well—houses chockablock with goods and often only narrow paths to navigate through the hoarded items. These patients typically dread "garbage day" when refuse is collected, because of their severe anxiety associated with the act of discarding goods. In fact, what separates hoarding from other obsessive-compulsive symptoms may be that hoarding is usually not preceded by obsessional cognitions and anxiety, which occur only when the behavior is prevented.³⁴ Anecdotally, hoarding has long been considered a predictor of poor response (J. Schwartz, M.D., oral communication, 1996), but until now there have been no systematically collected data to verify this belief. In fact, no study assessing outcome predictors has specifically looked at hoarding, with one exception.² In that study, cleaning but not hoarding rituals were associated with poor response; hoarding obsessions were not looked at separately. Perhaps one aspect that separates hoarding from more typical obsessive-compulsive symptoms is the relative lack of insight hoarders display. When asked why she saved used food wrappers and tin cans, one of our patients said she was "recycling." Yet, despite the patient's claim, nothing was actually recycled, since she never got rid of anything.

A recent study³⁵ using factor analysis concluded that symptoms of OCD fell into 4 dimensions: obsessions and checking, symmetry and ordering, cleanliness and washing, and hoarding. This suggests that OCD may be multidimensional and perhaps etiologically heterogeneous.

Thus, hoarding may comprise a biologically and phenomenologically distinct subset of OCD, which may in part explain our findings.

The data also showed that treatment-resistant patients have more severe illness, although the measurable differences were relatively small. Several measures of severity, including subscales of the MOC and SCL-90-R, differed, but not the more commonly used scales, such as the Y-BOCS and NIMHOCS. Nonresponders also tended to have more symptoms, as is shown in Table 2. The fact that nonresponders were more likely to have had previous drug trials suggests they may have a more severe and, hence, treatment-resistant illness. All of these findings are generally consistent with reports from other groups^{2,15,16} and are not surprising since severity is often associated with poor outcome for many Axis I and II disorders. A family history of depression or a mood disorder appeared related to good response to paroxetine but not CBT. This may mean that a familial predisposition for mood disorder is associated with response to somatic therapies. Axis II disorders were of little use in predicting outcome, but severe personality disorder was one reason for exclusion. One PDQ-R item was associated with response: "I believe that my brain is not working properly." Responders were less likely to endorse this item.

There were no other significant response predictors including age (except within the CBT cell), gender, age at illness onset, presence of comorbid disorders, or other specific obsessive-compulsive symptoms. The relative lack of predictors remains a frustrating aspect of OCD treatment because many patients respond poorly to treatment, and attempts to explain this phenomenon have generally been futile.³⁶ Depression has been identified as a predictor of poor response to behavioral therapy,^{7,8} but we were unable to directly address this issue since depressed subjects were excluded from our treatment protocols.

The presence of an Axis II disorder—particularly schizotypal personality—is probably the best-studied response predictor.^{4,5} As only 3 of our subjects were identified by the PDQ-R as having schizotypal personality, this issue could not be formally addressed. Likewise, Tourette's and other tic disorders are believed to complicate treatment, yet only 1 subject had multiple motor tics, and he responded well to paroxetine.⁶ Ravizza et al.³ recently identified an "episodic" form of OCD as having a relatively good response to treatment defined as involving symptom-free intervals of 2 months or longer. Because all of our subjects had a chronic form of illness (i.e., none reported symptom-free intervals lasting 2 months or more), we were unable to directly test their finding.

There are several problems that complicate the interpretation of our results. The relatively small sample and the even smaller treatment cells limit our statistical power to detect differences between groups. Nonetheless, hoarding obsessions and compulsions strongly predicted poor

response in the overall sample, and this trend was similarly strong within both the paroxetine and CBT cells. Another concern is that subjects from 2 independent studies were combined. However, the studies were conducted in parallel and the same selection criteria and assessments were used. Combining the data allowed us to look at a larger sample of patients, boosting statistical power. Also, the large number of comparisons could have led to chance findings. Finally, assessing response predictors based on response at week 12 may appear arbitrary, but most treatment studies for OCD have lasted 10 to 12 weeks,⁹⁻¹³ and, for most patients, 12 weeks is considered an adequate trial.¹ Likewise, a study of CBT showed that response occurred relatively early for most subjects.³⁷

Drug names: clomipramine (Anafranil), fluoxetine (Prozac), paroxetine (Paxil).

REFERENCES

- Rasmussen SA, Eisen JL, Pato MT. Current issues in the pharmacologic management of obsessive compulsive disorder. *J Clin Psychiatry* 1993;54 (suppl 6):4-9
- Alarcon RD, Libb JW, Spitzer D. A predictive study of obsessive-compulsive disorder response to clomipramine. *J Clin Psychopharmacol* 1993;13: 210-213
- Ravizza L, Barzega G, Bellino S, et al. Predictors of drug treatment response in obsessive-compulsive disorder. *J Clin Psychiatry* 1995;56: 368-373
- Jenike MA, Baer L, Minichiello WE, et al. Concomitant obsessive-compulsive disorder and schizotypal personality disorder. *Am J Psychiatry* 1986;143:530-532
- Baer L, Jenike MA, Black DW, et al. Effect of axis II diagnosis on treatment outcome with clomipramine in 54 patients with obsessive-compulsive disorder. *Arch Gen Psychiatry* 1992;49:862-866
- McDougle CJ, Goodman WK, Leckman JF, et al. Haloperidol addition in fluvoxamine-refractory obsessive-compulsive disorder: a double-blind, placebo controlled study in patients with and without tics. *Arch Gen Psychiatry* 1994;51:302-308
- Foa EB. Failure in treating obsessive-compulsives. *Behav Res Ther* 1979; 17:169-176
- Keijsers GPJ, Hoogdvin CAL, Schaap CPDR. Predictors of treatment outcome in the behavioral treatment of obsessive-compulsive disorder. *Br J Psychiatry* 1994;165:781-786
- DeVaugh J, Katz R, Landau P, et al. Clinical predictors of treatment response in obsessive-compulsive disorder: exploratory analyses from multicenter trials of clomipramine. *Psychopharmacol Bull* 1990;26:54-59
- Jenike MA, Buttolph L, Baer L, et al. Open trial of fluoxetine in obsessive-compulsive disorder. *Am J Psychiatry* 1989;146:909-911
- Goodman WK, Price LH, Rasmussen SA, et al. Efficacy of fluvoxamine in obsessive-compulsive disorder: a double-blind comparison with placebo. *Arch Gen Psychiatry* 1989;46:36-44
- Perse TL, Greist JH, Jefferson JW, et al. Fluvoxamine treatment of obsessive-compulsive disorder. *Am J Psychiatry* 1987;144:1543-1548
- Tollefson GD, Rampey AH, Potvin JH, et al. A multicenter investigation of fixed-dose fluoxetine in the treatment of obsessive-compulsive disorder. *Arch Gen Psychiatry* 1994;51:559-567
- Orloff LM, Battle MA, Baer L, et al. Long-term follow-up of 85 patients with obsessive-compulsive disorder. *Am J Psychiatry* 1994;151:441-442
- Ackerman DL, Greenleaf S, Bystritsky A, et al. Predictors of treatment response in obsessive-compulsive disorder: multivariate analyses from a multicenter trial of clomipramine. *J Clin Psychopharmacol* 1994;14: 247-254
- Leonard HL, Swedo SE, Lenane MC, et al. A 2- to 7-year follow-up study of 54 obsessive-compulsive children and adolescents. *Arch Gen Psychiatry* 1993;50:429-439
- Thomsen PH. Obsessive-compulsive disorder in children and adolescents: predictors in childhood for long-term phenomenological course. *Acta Psychiatr Scand* 1995;92:255-259
- Wheadon DE, Bushnell WD, Steiner M. A fixed-dose comparison of 20, 40, or 60 mg paroxetine to placebo in the treatment of obsessive-compulsive disorder. Presented at the 32nd annual meeting of the American College of Neuropsychopharmacology; 1993; Honolulu, Hawaii
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*. Washington, DC: American Psychiatric Association; 1987
- Spitzer RL, Williams JBW, Gibbon M. *Structured Clinical Interview for DSM-III-R (SCID)*. New York, NY: Biometric Research, New York State Psychiatric Institute; 1989
- Goodman WK, Price LR, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale, I: development, use, and reliability. *Arch Gen Psychiatry* 1989;46:1006-1011
- Insel TR, Murphy DL, Cohen RM, et al. Obsessive-compulsive disorder: a double-blind trial of clomipramine and clorgyline. *Arch Gen Psychiatry* 1983;40:605-612
- Hamilton M. Developing a rating scale for primary depressive illness. *Br J Soc Psychol* 1967;6:278-296
- Foa EB, Wilson R. *How to Overcome Your Obsessions and Compulsions*. New York, NY: Bantam Books; 1991
- Guy W. *ECDEU Assessment Manual for Psychopharmacology*. US Dept Health, Education, and Welfare publication (ADM) 76-338. Rockville, Md: National Institute of Mental Health; 1976:218-222
- Hodgson RJ, Rachman S. Obsessive-compulsive complaints. *Behav Res Ther* 1977;15:389-395
- Derogatis LR. *Symptom Checklist-90 (Revised): Administration, Scoring, and Procedures Manual, I*. Baltimore, Md: Clinical Psychometric Research; 1977
- Pilowsky I, Spence ND. *Manual for the Illness Behavior Questionnaire*. 2nd ed. Adelaide, Australia: Department of Psychiatry, University of Adelaide; 1983
- Pfohl B, Zimmerman M, Blum N. *Structured Interview for DSM-III-R Personality Disorders*. Iowa City, Iowa: Department of Psychiatry, University of Iowa; 1987
- Hyler SE, Reider RO, Spitzer RL. *PDQ-R: Personality Diagnostic Questionnaire-Revised*. New York, NY: New York State Psychiatric Institute; 1987
- Andreasen NC, Endicott J, Spitzer RL, et al. The family history method using diagnostic criteria: reliability and validity. *Arch Gen Psychiatry* 1977;34:1229-1235
- Endicott J, Spitzer RL, Fleiss JL, et al. The Global Assessment Scale: a procedure for measuring overall severity of psychiatric disturbance. *Arch Gen Psychiatry* 1976;33:766-771
- Rasmussen S, Eisen JL. Phenomenology of OCD: clinical subtypes, heterogeneity, and co-existence. In: Zohar J, Insel T, Rasmussen S, eds. *The Psychobiology of Obsessive-Compulsive Disorder*. New York, NY: Springer Publishing; 1991:13-43
- Miguel EC, Baer L, Coffey BJ, et al. Phenomenological differences of repetitive behaviors in obsessive-compulsive disorder and Tourette's syndrome. *Br J Psychiatry* 1997;170:140-145
- Leckman JF, Grice DE, Boardman J, et al. Symptoms of obsessive-compulsive disorder. *Am J Psychiatry* 1997;154:911-917
- Jenike MA. Management of patients with treatment-resistant obsessive-compulsive disorder. In: Pato MT, Zohar J, eds. *Current Treatments of Obsessive-Compulsive Disorder*. Washington, DC: American Psychiatric Press; 1991:135-155
- Emmelkamp PMG, Visser S, Hoekstra RJ. Cognitive therapy versus exposure in vivo in the treatment of obsessive-compulsive disorder. *Cognitive Ther Res* 1988;12:103-114