A Double-Blind Placebo-Controlled Comparison of Phenelzine and Imipramine in the Treatment of Bulimia in Atypical Depressives

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Although antidepressants have been found to be superior to placebo in 12 of 14 studies, the relationship between improvement in the depressive diathesis and bulimia is unclear. In this study, the efficacy of placebo, imipramine, and phenelzine is examined in patients comorbid for atypical depression and bulimia. Greater improvement was observed for both depressive and bulimic symptoms with phenelzine than with either imipramine or placebo. Consistent with its poor antidepressant effects in atypical depression, imipramine seemed to have minimal efficacy for the bulimic symptoms of atypical depressives. These data suggest that the presence of bulimia does not alter the treatment response of atypically depressed patients. Furthermore, the data may suggest a link between depression and bulimia in atypical depressives. Demonstrating a statistical difference with a small sample suggests the effect size is robust, however conclusions are limited by a small sample size. © *1994 by John Wiley & Sons, Inc.*

A relationship between bulimia and depression has long been suspected because the two disorders frequently occur together. In a review of phenomenological studies con-

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ducted over the last 10 years, Hudson and Pope (1982) found that approximately one half of patients with bulimia had major depressive disorder. When past episodes were included up to three fourths of patients showed evidence of comorbidity. Familial association of bulimia and depressive illness also supports this hypothesis (Hudson, Pope, Jonas, Yurgelun-Todd, & Frankenburg, 1987). In contrast, Strober and Katz (1987) have argued against this hypothesis citing differences between the two disorders in symptom phenomenology and patterns of recovery, relapse, and chronicity.

In 12 of 14 double-blind studies, antidepressants demonstrated greater efficacy than placebo. Six studies found superior efficacy for tricyclic antidepressants (Agras, Dorian, Kirkley, Arnow, & Bachman, 1987; Kaplan, Garfinkel, & Garner, 1987; Pope, Hudson, Jonas, & Yurgelun-Todd, 1983; Hughes, Wells, Cunningham, & Ilstrup, 1986; Barlow, Blouin, Blouin, & Perez, 1988; Walsh, Hadigan, Devlin, Gladis, & Roose, 1991) and three for monoamine oxidase inhibitors (MAOI; Kennedy, Piran, & Garfinkel, 1988; Walsh et al., 1988; Price & Babai, 1987). In addition, both bupropion (Horne et al., 1988) and trazadone (Pope, Keck, McElroy, & Hudson, 1989) were found to be superior to placebo, whereas amitriptyline (Mitchell & Groat, 1984) and mianserin (Sabine, Yonace, Farrington, Barratt, & Wakeling, 1983) were not. Most recently, in a multicenter placebo-controlled trial comparing two dosages of fluoxetine, fluoxetine was significantly superior to placebo at 60 mg/day and modestly superior to placebo at 20 mg/day (FBNC Study Group, 1992). The effectiveness of antidepressants in treating bulimia also suggests an association between bulimia and depressive illness.

Only one study simultaneously examined the efficacy of two drugs and placebo (Walsh et al., 1988). In this crossover study of 10 nondepressed patients with bulimia, nomifensine and phenelzine were significantly better than placebo with little difference between the two drugs. Walsh (1989) appropriately indicates the limitations in interpreting data that rely on historic controls. Thus, available data cannot clarify the relative advantage of different classes of antidepressants in the treatment of bulimia associated with different types of depressive syndromes.

The relationship between improvement in depressive and bulimic symptoms in patients treated with antidepressants is obscure. Pope et al. (1983) attributed imipramine's efficacy in the treatment of bulimia to its antidepressant effect rather than a novel action. The onset of the antibulimic and antidepressant effect was similar. Furthermore, treatment failures with one class of antidepressants often exhibited improvement in both depression and bulimia with alternative antidepressants. In contrast, Hughes et al. (1986) studied a nondepressed bulimic sample (subjects met neither DSM-III [American Psychiatric Association, 1980] criteria nor Research Diagnostic Criteria for major depressive disorder). The improvement observed in this group may suggest a unique antibulimic effect of desipramine. Similarly, Walsh et al. (1988) have found that phenelzine is superior to placebo in reducing binge frequency in both depressed and nondepressed groups.

Phenelzine has been found more effective than both imipramine and placebo for patients with atypical depression according to Columbia University criteria (Liebowitz et al., 1988; Quitkin et al., 1990). Columbia criteria consists of reversed vegetative features including hyperphagia, hypersomnia, rejection sensitivity, and leaden paralysis. This selective responsivity to phenelzine distinguishes atypical depression from other subtypes of depression. A subset of these patients was identified as currently bulimic, or as having a history of bulimia. This sample of patients with a combination of disorders, and a unique pharmacological response to antidepressants, offers a novel group in which to examine the interaction of mood disorders, eating disorders, and different pharmacological agents.

This paper is a reanalysis. Depression outcome data have been previously published (Liebowitz et al., 1988; Quitkin et al., 1990). This is the first examination of the efficacy of medication on bulimia and its relationship to depression. In this paper we examine the following questions: (1) Will the superior efficacy of phenelzine for atypical depression also be true for the bulimic symptoms of atypical depressives? (2) What is the relationship of improvement in one set of symptoms to the other? Will improvement in bulimia be accompanied by improvement in depression?

To our knowledge, this is the first double-blind, placebo-controlled comparison of a tricyclic and an MAOI for the treatment of bulimia.

SUBJECTS AND METHODS

The study procedures, including dose of drugs, rating scales, biochemical measures, and reliability assessment were described in detail in the original study of atypical depression (Liebowitz et al., 1988). Briefly, the study consisted of a double-blind 6-week phase in which patients were randomized to either phenelzine, imipramine, or placebo, preceded by a 10-day single blind placebo period.

Of the 401 atypical depression patients included in this series of studies (Liebowitz et al., 1988; Quitkin et al., 1990) 20 patients were prospectively diagnosed as bulimic. Diagnoses were made according to DSM-III criteria by means of clinical interview. Four additional patients, admitted to the study prior to the 1980 publication of the DSM-III, were retrospectively judged to be bulimic through chart review by raters blind to outcome. Thus our sample consisted of 24 patients.

Standard ratings of depression were done prospectively and included the Clinical Global Impression (CGI), Hamilton Depressive Rating Scale (HAM-D, 21 items), and the Hopkins Symptoms Checklist (SCL-90). We specifically examined item 60 of the self-rated SCL-90: overeating—1 = none, 2 = a little bit, 3 = moderately, 4 = quite a bit, 5 = extremely. The Eating Attitudes Test (EAT), a widely used self-report questionnaire for assessing abnormal attitudes and behaviors associated with bulimia, was not included in the protocol of this study. However, in another patient sample simultaneously rated on the EAT and the SCL-90 item 60 at both the beginning and end of study, significant correlation was found between change in the two (B. T. Walsh, personal communication, 1991). Specifically, for change scores on the EAT and item 60, Pearson's $r = .56$, $df = 51$, $p = .00^{14}$. These data suggest that change in the SCL-90 item 60 approximates change on the EAT. The SCL-90 item 60 was assessed pre- and post-treatment by 23 of our 24 patients. Improvement on the SCL-90 item 60 was prospectively defined as a decrease of 2 or more points. We chose a 2-point change in this 5-point scale because we arbitrarily decided that a 2-point change was unlikely to be due to random fluctuation.

Doctor's weekly progress notes were retrospectively examined by raters blind to treatment. Binging and purging were only considered improved if the notes indicated at least a 50% reduction in the frequency of binging and purging episodes. These ratings were done conservatively; 10 of the 24 cases were considered to have insufficient detail to evaluate. Hence 14 patients were included in analyses of binging and purging, 23 in analyses of SCL-90 item 60 overeating (1 patient failed to complete the SCL-90), and 24 in analyses of depression.

Study completers received a minimum dose of at least 45 mg of phenelzine sulfate, or 150 mg of imipramine hydrochloride, or the equivalent number of placebo pills for a minimum of 28 consecutive days (out of 42 days). Patients rated 1 (very much improved)

or 2 (much improved) on the 7-point improvement scale of the CGI were considered responders with respect to their depression. Mood was the focus in evaluating change. Patients rated much improved or better had a marked amelioration in depressive symptoms.

RESULTS

Twenty-one patients were female and 3 were male. Fifteen met Research Diagnostic Criteria for major depression, 1 for minor depression, and 13 for intermittent depression. Six patients completed the 6-week trial on imipramine, 8 on phenelzine, and 10 on placebo. The average doses at week six were 275 mg for the imipramine group and 75 mg for the phenelzine group. All but 3 of the patients where within 80–120% of ideal body weight. Three patients were 150% of ideal body weight; 2 of these were randomized to phenelzine and 1 was randomized to placebo. Demographic and clinical measures taken at randomization are summarized in Table 1. Analysis of variance at baseline indicated a difference on the SCL-90 item 60 overeating score ($F = 3.98$, $df = 2$, $p = .034$). The phenelzine group had the highest score on overeating. In addition a trend for a difference in age ($F = 2.95$, $df = 2$, $p = .074$) was found with the phenelzine group having the highest mean age. No other statistically significant differences were found.

Improvement in Depression

The proportion of responders using categorical judgment of depression on the CGI (improved = score of 1 or 2) were as follows: phenelzine 88% (7/8), imipramine 33% (2/6), and placebo 40% (4/10). Fisher's exact one-tailed tests revealed differences of trend

Table 1. Demographic and clinical measures at randomization

	Phenelzine ($N = 8$)	Imipramine ($N = 6$)	Placebo ($N = 10$)	Overall ($N = 24$)
Age*	37.1 ±7.5	32.2 ±7.2	29.7 ±4.7	32.8 ±6.9
Mean no. binge/week[a]	5.6 ±2.19	6.8 ±2.49	4.0 ±2.96	5.2 ±2.77
Proportion inducing vomiting	63% (5/8)	83% (5/6)	50% (5/10)	63% (15/24)
Proportion abusing laxatives	25% (2/8)	0%	20% (2/10)	17 (4/24)
HAM-D	16.1 ±3.9	13.0 ±2.3	15.7 ±3.9	15.2 ±3.7
SCL-90 summary	18.4 ±6.7	19.4 ±4.7	22.4 ±7.8	20.3 ±6.7
SCL-90 Depression subscale	2.88±.99	3.10±.88	3.41±.81	3.16±.88
SCL-90 item 60 overeating**	3.87±1.55	3.33±1.21	2.00±1.49	2.96±1.63
CGI severity	3.88±.83	4.00±.00	3.80±.63	3.88±.61
CGI improvement	3.75±.70	4.00±.00	4.00±.47	3.92±.50

Note. HAM-D = Hamilton Depressive Rating Scale; SCL-90 = Hopkins Symptoms Checklist; CGI = Clinical Global Impression.

*Analysis of variance, $F = 2.95$, $df = 2$, $p = .074$.

**Analysis of variance, $F = 3.98$, $df = 2$, $p = .034$.

[a]Binge frequency data available for: 5/6 patients on imipramine, 5/8 patients on phenelzine, and 7/10 patients on placebo.

significance for phenelzine versus imipramine ($p = .062$) and for phenelzine versus placebo ($p = .057$).

Analysis of covariance of week 6 HAM-D scores was performed using baseline measures as the covariate. A significant difference among the three drug groups was found ($F = 6.8$, $df = 2$, $p = .006$). The phenelzine group demonstrated approximately a 10-point improvement in the HAM-D at week 6 whereas the imipramine and placebo groups showed virtually no change (see Table 2). Analyses of covariance were also done on SCL-90 summary scores and SCL-90 depression subscale scores. The SCL-90 subscale scores range from 1–5, the summary score is a sum of the subscale scores. Significant differences among the three drug groups were found in the reduction of both the summary score ($F = 5.24$, $df = 2$, $p = .015$) and the depression subscale score ($F = 4.6$, $df = 2$, $p = .023$). The phenelzine group had the greatest improvement on the summary score (see Table 2). The relationship of depression subscale scores to treatment is similar. The phenelzine group showed clear improvement whereas the imipramine and placebo groups showed little change (see Table 2). Thus there was good agreement between doctor and patient self-ratings.

Improvement in Bulimia

Hierarchical stepwise regression analysis of SCL-90 item 60 scores at week 6 was done using baseline scores as a covariate (see Table 3. Baseline item 60 scores did not predict outcome at week 6 (for all pairs of drug comparisons $R^2 = .01$, $p = .6$). Thus the fact that the phenelzine group had a higher score at baseline did not appear relevant in determining treatment response. An overall drug difference was found ($R^2 = .039$, $p = .008$) and contrasts between pairs of treatments showed differences for phenelzine versus imipramine ($R^2 = .81$, $p = .001$) and phenelzine versus placebo ($R^2 = .32$, $p = .025$). The phenelzine group showed improvement from a mean score at randomization of 3.87 to 1.12 at week 6. The imipramine and placebo groups showed minimal change with no difference between the two (see Table 3 and Figure 1). Because there was a trend for an age difference among drug groups, age was entered into the regression equation as a covariate. Although age did not significantly contribute to the differences

Table 2. Mean scores for HAM-D summary, SCL-90 item 60, SCL-90 summary, and SCL-90 depression subscale at randomization and week 6 by drug

	Phenelzine ($N = 8$)		Imipramine ($N = 6$)		Placebo ($N = 10$)	
	Randomized	Week 6	Randomized	Week 6	Randomized	Week 6
HAM-D summary*	16.13±3.9	6.62±2.6	13.00±2.3	13.67±4.8	15.70±3.95	13.80±8.4
SCL-90 item 60 overeating[a]	3.87±1.55	1.12±.35	3.33±1.21	3.00±.63	2.00±1.49	2.44±1.51 ($N = 9$)
SCL-90 summary**	18.41±6.7	13.10±4.0	19.36±4.7	17.92±4.2	22.38±7.8	22.59±8.4 ($N = 9$)
SCL-90 Depression subscale***	2.88±.99	1.73±.57	3.10±.88	2.71±.71	3.41±.81	3.26±1.28 ($N = 9$)

Note. HAM-D = Hamilton Depressive Rating Scale; SCL-90 = Hopkins Symptoms Checklist.
*Analysis of covariance, $F = 6.8$, $df = 2$, $p = .006$.
**Analysis of covariance, $F = 5.24$, $df = 2$, $p = .015$.
***Analysis of covariance, $F = 4.6$, $df = 2$, $p = .023$.
[a]Hierarchical stepwise regression, see Table 3.

in item 60 scores among drug groups, these differences were less distinct than when age was not covaried. The differences among drug groups were generally similar to those reported in Table 3, specifically for imipramine versus placebo R^2 = .12, R^2 change = .11, F change = 1.43, p = .25, for phenelzine versus placebo R^2 = .33, R^2 change = .21, F change = 4.16, p = .06, and for phenelzine versus imipramine R^2 = .82, R^2 change = .70, F change = 38.11, p = .0001. There were no significant interactions between drug and age. To simplify, we have chosen to present the data without the correction for age; those analyses are available upon request.

With respect to improvement in binging and purging, data were available for 14 of the 24 patients. Comparisons between the group for which data were available and the group for which data were missing showed that the group with missing data had higher baseline measures on the SCL-90 depression subscale (t = 2.19, df = 22, p = .039). However, analysis of variance revealed no significant difference between the two groups in their decrease in SCL-90 depression subscale scores from baseline to week 6 (F = 0.885, df = 1, p = .36). No other differences were found in measures taken at baseline or at week 6. Of the 14 patients with binging and purging data, 5 of 5 phenelzine-treated patients evidenced improvement in binging and purging whereas 0/3 imipramine treated, and 3/6 placebo-treated patients showed improvement. Phenelzine was significantly better than imipramine in reducing the frequency of binging and purging (Fisher's exact p = .02) and showed a trend toward superiority over placebo (Fisher's exact p = .10). With only 3 patients in the imipramine group no firm conclusions are warranted. In general, a small sample dictates a conservative interpretation. However, this is an encouraging trend.

Association Between Improvement in Bulimia and Improvement in Depression

Change scores from randomization to week 6 for the HAM-D and the SCL-90 item 60 were positively correlated for the overall sample (Pearson's r = .48, $p < .05$). Because item 60 overeating is correlated with the EAT, this suggests an association between

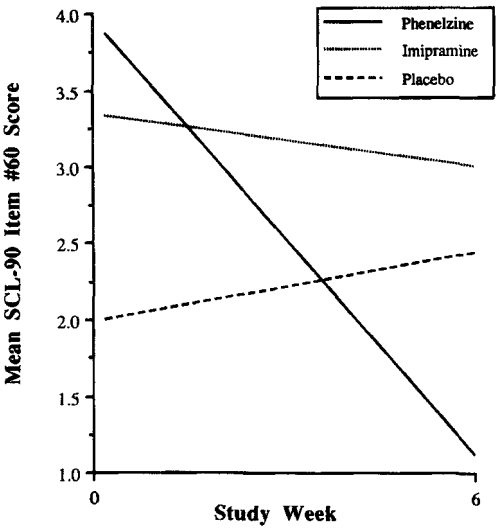

Figure 1. Mean Hopkins Symptoms Checklist item 60 Overeating by treatment.

Table 3. Hierarchical stepwise regression analysis of the SCL-90 item 60 overeating

	Overall Drug Difference		Phenelzine vs. Placebo		Phenelzine vs. Imipramine		Imipramine vs. Placebo	
	Baseline	Drug Difference	Baseline	Drug Difference	Baseline	Drug Difference	Baseline	Drug Difference
R^2	0.01	0.40	0.01	0.32	0.01	0.81	0.01	0.11
R^2 Change	0.01	0.39	0.01	0.31	0.01	0.80	0.01	0.10
F Change	0.28	6.16	0.20	6.33	0.16	46.63	0.18	1.31
Significance of F Change	0.60	0.008	0.66	0.025	0.69	0.001	0.68	0.27
B at last step	−0.09	—	−0.89	−1.62	−0.89	−2.23	−0.09	0.87

Table 4. Improvement in depression at the end of 6 weeks by improvement in bulimia at the end of 6 weeks

Response at 6 Weeks	Depression Improved	Depression Unimproved
Bulimia improved	7	2
Bulimia unimproved	5	9
Total	12	11

Note. Fisher's exact test, $p = .06$.

change in depression and change in bulimia. Using categorical judgments decreases the power to detect statistical differences but enhances the ability to clinically interpret change. Depression was considered improved if the CGI change score was much improved or very much improved and overeating was considered improved if there was a 2-point reduction in the SCL-90 item 60 score. The relationship of improvement on the SCL-90 item 60 and improvement in depression is shown in Table 4. Again, a relationship between improvement in depression and overeating is suggested (Fisher's exact one tailed test, $p = .06$).

DISCUSSION

In these atypical depressives with concurrent bulimia, there seemed to be greater improvement in both disorders with phenelzine than with either imipramine or placebo. That bulimia responds better to phenelzine than either imipramine or placebo is suggested by both the prospective SCL-90 item 60 overeating and our retrospective ratings of improvement in binging and purging. The superiority of phenelzine to imipramine is consistent with treatment outcome of atypically depressed patients in general (Liebowitz et al., 1988; Quitkin et al., 1990). This suggests that the co-occurrence of bulimia does not alter the treatment response of the atypically depressed patient to antidepressants.

Our data also suggest that in patients with selective response to MAOIs for depression, bulimia has an increased chance of improving as well. This finding is consistent with the hypothesis that the efficacy of antidepressants for bulimia is due to an antidepressant effect of the drug.

These findings may suggest a link between depression and bulimia in this population of atypical depressives. Demonstrating statistical difference with a small sample is consistent with a robust effect size. Despite our small sample size, an association between improvement in depression and improvement in bulimia seems plausible. Changes in Ham-D and SCL-90 item 60 overeating scores were positively correlated. Frequencies of improvement in bulimia as measured by both the SCL-90 item 60 and decreased binging and purging were found to be associated with improvement on measures of depression. Improvement in bulimia was most often accompanied by euthymia. The nature of this association, however, is difficult to discern.

This report is limited by a small sample. Also, it should be noted that even though the SCL-90 item 60 has been found to be correlated with the EAT, reductions in binging and purging are generally used to determine outcome in drug trials for bulimia. Our conclusions would be strengthened by corroborating measures of change in bu-

limia. It should also be noted that this sample is not entirely typical of other bulimic samples. The sample included 3 males, the mean age was somewhat older than is usually reported, and 3 subjects were considerably overweight. A large sample stratified by bulimia and type of depression with multiple treatments, including crossover of nonresponders, would further elucidate the relationship of these disorders.

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