The Relationship of Personality Disorders to Treatment Outcome in Depressed Outpatients

Roger T. Mulder, M.B., Ch.B., Ph.D., F.R.A.N.Z.C.P.;
Peter R. Joyce, M.B., Ch.B., Ph.D., F.R.A.N.Z.C.P.; and
Suzanne E. Luty, M.B., Ch.B., F.R.A.N.Z.C.P.

Background: Many clinicians believe that depressed patients with comorbid personality disorder(s) may respond differently to standard treatments than patients with depression alone. Personality disorders appear to be common among patients with depression, suggesting potentially significant treatment implications for a large group of patients.

Method: Subjects with DSM-III-R major depression were recruited for a study looking at prediction of antidepressant response. All patients were assessed using the Structured Clinical Interviews for DSM-III-R Axis I and Axis II, as well as rated on the Hamilton Rating Scale for Depression and the Montgomery-Asberg Depression Rating Scale (MADRS). Patients were then randomly assigned to treatment with fluoxetine or nortriptyline and reassessed at 6 weeks. The major outcome measure was percentage reduction in MADRS scores.

Results: Of the 183 patients who completed the personality disorder assessment, 45% had at least 1 comorbid personality disorder. Subjects with comorbid personality disorders were slightly younger, more depressed at baseline, had poorer social adjustment, more general psychopathology, and more chronic depression. Despite these differences, the presence of a comorbid personality disorder did not adversely affect overall outcome at 6 weeks, but there was an interaction between having a comorbid personality disorder and drug type. The major effect was that patients with a cluster B personality disorder did relatively poorly on nortriptyline compared with fluoxetine treatment.

Conclusion: The finding that the presence of a comorbid personality disorder does not affect overall treatment response is similar to that reported by some recent studies. The finding that patients with cluster B personality disorders respond poorly to nortriptyline is also consistent with a small literature on borderline personality disorder.

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Corresponding author and reprints: Robert T. Mulder, Ph.D., Department of Psychological Medicine, Christchurch School of Medicine, P.O. Box 4345, Christchurch, New Zealand (e-mail: roger.mulder@chmeds.ac.nz).

The idea that personality pathology may influence response to treatment in depressed patients is not a new one. Both Kraepelin and Freud speculated that personality pathology affected depressed patients' response to treatment.[1] An extensive review of predictors of tricyclic antidepressant (TCA) response published in the 1970s concluded that patients with neurotic, hypochondriacal, or hysterical personality traits responded no better to TCAs than to placebo.[2] A number of studies in the 1960s and 1970s also reported that high neuroticism scores predicted worse treatment outcome for patients with depression.[3,4]

The introduction of a separate axis for personality disorder in DSM-III[5] allowed personality pathology to be conceptualized in a new way. A number of questionnaires and structured interviews have been designed to help clinicians and researchers to more reliably diagnose personality disorders in their patients. Studies using these instruments have generally reported that a high proportion of individuals with major depression also meet criteria for at least 1 Axis II personality disorder. Estimates range from 18% to 95%,[6] although prevalence rates usually fall between 35% and 65%.[7] The most commonly reported comorbid personality disorders are avoidant, borderline, and paranoid personality disorders. The meaning of these estimates and their reliability and validity have been extensively debated,[8,9] but the fact that such a large pro-

portion of depressed patients appear to have some personality pathology supports the view that there may be potentially significant treatment implications for this group of individuals.

Whether personality disorders influence treatment outcome is not clear. Many clinicians believe that the presence of a comorbid personality disorder in patients with depression adversely affects response to treatment. However, a recent systematic review by one of us (R.T.M.)[10] concluded that whether personality pathology significantly concerns outcome appeared to largely depend on study design. Studies that used structured interviews for personality disorders and randomly assigned patients to effective treatments for depression typically showed no difference in short-term outcome. Those that used nonstandardized personality disorder assessments and did not control for treatment usually reported that patients with comorbid personality pathology did worse on average.[10]

Using DSM Axis II to measure personality pathology has a number of significant methodological limitations that we have tried to address in this study. First, we attempted to get as reliable and valid a measure of personality disorder as possible by using a standardized clinician-rated structured interview for DSM-III-R Axis I and Axis II. There is some consensus that structured interviews are, at present, the most valid way to diagnose personality disorders.[8] Second, we attempted to reduce the effect of mood state as much as possible. Clinicians administering the Axis II interview were encouraged to exercise their clinical judgment to allow for the potential distortion produced by the dysphoric mental state. There is limited evidence that careful face-to-face interviews may reduce the effect of mood state on evaluation of personality disorders.[11,12]

Third, we examined the potential effect of confounders, which might independently influence treatment outcome. The major ones that have been reported to be associated with comorbid personality pathology are depression severity, chronicity, general psychopathology, and social functioning.[13,14] Finally, we looked for overall differences in response to the 2 drugs used in the study, as well as differential response in personality disorder clusters A (paranoid, schizoid, schizotypal), B (borderline, histrionic, narcissistic, antisocial), and C (avoidant, dependent, obsessive-compulsive).

This article reports on the influences of comorbid DSM-III-R personality disorders on the 6-week outcome of a group of patients suffering from DSM-III-R major depression. The treatment was a randomized 6-week trial of fluoxetine versus nortriptyline.

METHOD

Subjects

The depressed patients for this study were recruited as part of the Christchurch Outcome of Depression Study, a study looking at predictors of antidepressant response.[15] For inclusion, the subjects' current principal diagnosis was DSM-III-R major depression, and the treating clinician considered that treatment with an antidepressant drug was appropriate. Apart from the use of oral contraceptive and an occasional hypnotic, patients were required to be free of all psychotropic drugs for a minimum of 2 weeks. Depressed patients were excluded only if alcohol and/or drug dependence was considered the current principal diagnosis, but current alcohol and/or drug dependence was allowed if the treating clinician judged major depression to be the current principal diagnosis. Patients were also excluded if they had a history of schizophrenia or bipolar I disorder, but patients with a history of hypomania (bipolar II) were included. Patients were required to be free of major medical illness (e.g., diabetes). For inclusion in this study, patients also had to have a minimum score of 14 on the 17-item Hamilton Rating Scale for Depression (HAM-D).[16] The study was approved by the local Ethics Committee.

Baseline Evaluation

After receiving an initial psychiatric evaluation and giving written informed consent, patients attended the Clinical Research Unit for a detailed baseline research evaluation. Patients were assessed by their treating psychiatrist or senior psychiatric resident utilizing the Structured Clinical Interview for DSM-III-R[17] for Axis I disorders (SCID) and the SCID-II[18] for Axis II disorders. Patients were also rated on the 17-item and 27-item HAM-D[16] and on the Montgomery-Asberg Depression Rating Scale (MADRS).[19]

Patients completed a series of self-report questionnaires including the Hopkins Symptom Checklist (SCL-90),[20] the Social Adjustment Scale (SAS),[21] and the SCID-Patient Questionnaire.[18] Chronic depression was defined as having major depression for more than half the time over the previous 5 years.

Reliability

Approximately every tenth SCID-II interview was videotaped and viewed by a different clinician who independently rated personality disorder symptoms. In addition, this subject was reinterviewed by another clinician who again independently rated personality disorder symptoms and diagnoses. The test-retest kappa for presence/absence of a personality disorder was 0.71.

Treatment

After the baseline evaluation, patients were randomly assigned to open treatment with either fluoxetine or nortriptyline. The randomization was undertaken by the research coordinator and research nurse, who told the treating psychiatrist which drug to utilize. Patients were then seen at least weekly for 20 to 60 minutes, depending on

Table 1. Baseline Characteristics of 183 Depressed Patients With No Personality Disorder, Any Personality Disorder, and Clusters A, B, or C Personality Disorders[a]

Characteristic	No Personality Disorder	Any Personality Disorder	Cluster A	Cluster B	Cluster C
Age, y	33.5 (11.4)	30.2 (10.9)†	31.4 (11.0)	27.7 (8.5)†	30.7 (11.0)
MADRS	29.7 (6.6)	32.6 (6.5)*	33.9 (6.6)*	31.7 (6.4)	33.2 (5.8)*
HAM-D	19.3 (4.7)	20.7 (4.0)†	20.8 (3.9)	19.8 (3.1)	21.1 (3.9)†
SAS	2.5 (0.44)	2.7 (0.40)*	2.8 (0.40)*	2.7 (0.38)	2.8 (0.39)*
SCL-90	11.5 (5.1)	14.1 (4.3)***	15.4 (3.9)***	14.7 (4.6)*	14.6 (4.0)***
Chronic depression[b]	3.8 (1.3)	4.5 (1.3)***	4.4 (1.4)	4.6 (1.1)*	4.6 (1.3)**

[a]All values shown as mean (SD). Significance levels quoted are the differences between each category and no personality disorder.
[b]The measure of chronic depression is the mean score from the Structured Clinical Interview for DSM-III-R (range, 1–6) for percent depressed in the last 5 years.
†$p < .05$, *$p < .01$, **$p < .001$, ***$p < .0001$.
Abbreviations: HAM-D = Hamilton Rating Scale for Depression, MADRS = Montgomery-Asberg Depression Rating Scale, SAS = Social Adjustment Scale, SCL-90 = Hopkins Symptom Checklist.

patient need. In the initial stages of treatment, emphasis was placed on ensuring a good trial with the antidepressant within the context of optimal clinical management. The patients received no formal psychotherapy but were seen regularly for education and support and to optimize compliance.

If the patient was randomly assigned to nortriptyline, the initial dosing schedule was 25 mg for 1 night, 50 mg for 1 night, and then 75 mg. After 1 week, blood levels were obtained, and further dosage was adjusted based on clinical response, side effects, and blood levels. At 6 weeks, the mean nortriptyline dosage was 100 mg, and doses ranged from 50 to 175 mg.

For patients randomly assigned to fluoxetine, the initial dosage was 20 mg daily for 3 weeks, although this could be reduced to 10 mg to decrease side effects. At 3 weeks, the clinician was free to adjust dosage up to a maximum of 60 mg. At 6 weeks, the mean fluoxetine dosage was 27 mg, the most common dosage was 20 mg, and the range was 10 to 60 mg.

Outcome

At 3 and 6 weeks, patients were reassessed. This involved the clinician rating patients on the HAM-D and the MADRS and making a global clinical rating. In addition, patients completed the self-rating scales of the SCL-90 and the SAS.

Statistics

The analysis is a completer analysis. There was no significant difference between completers and noncompleters with regard to personality pathology. The percentage reduction in MADRS score was chosen a priori as the primary outcome measure since we consider it the most valid measure of change in mood in this clinical population.[22] All analyses were repeated utilizing the more commonly used HAM-D score.

Baseline parameters were compared between those with and without a personality disorder by using independent t tests. Where this revealed significant differences, these were further explored by comparing the no personality disorder group with each personality disorder cluster. A general linear model was used to explore the effects of personality disorder clusters on percentage reduction in MADRS score. Baseline variables that differentiated between the personality disorder and no personality disorder groups, gender, drug type, and the drug type–personality disorder cluster interaction were all included in this model. Where significant, the drug type–personality disorder cluster interaction was further analyzed in an additional hierarchical model that incorporated drug type–baseline variable interactions appropriate to the baseline differential between the personality disorder and no personality disorder groups.

RESULTS

Sample

One hundred ninety-five patients were enrolled in the study, and a total of 183 patients completed the personality disorder assessment. These were 76 males and 107 females with a mean (SD) age of 32.0 (16.3) years. The mean baseline MADRS score was 31.0 (6.7), and the mean baseline 17-item HAM-D score was 19.9 (4.4). Eighty-two patients (45%) were given a diagnosis of at least 1 comorbid personality disorder. The number and percentage of sample with individual personality disorders in descending order were as follows: avoidant, 43 (23.5%); borderline, 30 (16.4%); paranoid, 27 (14.8%); obsessive-compulsive, 16 (8.7%); dependent, 11 (6.0%); histrionic, 9 (4.9%); narcissistic, schizotypal, and antisocial, 6 (3.3%); and schizoid, 4 (2.2%). Of the 183 patients, 149 completed the 6-week treatment.

Table 1 shows baseline characteristics of patients with no personality disorder versus those with 1 or more personality disorders and those in clusters A, B, and C versus those with no personality disorder. Overall, depressed individuals with comorbid personality disorders are slightly younger, have more depressive symptoms at baseline as measured by both 17-item HAM-D and MADRS scores

Table 2. Effect of Baseline Variables, Personality Disorder, and Drug Type on Percentage Reduction in Montgomery-Asberg Depression Rating Scale Scores at 6 Weeks (N = 149)

Variable	F	df	p Value
Personality disorder	1.28	7	.262
Sex	0.44	1	.508
Age	0.32	1	.571
Chronic depression	6.76	1	.010
SAS	1.51	1	.221
SCL-90	0.09	1	.761
Drug type	9.64	1	.002
Personality disorder × drug type	2.59	1	.020

Abbreviations: SAS = Social Adjustment Scale, SCL-90 = Hopkins Symptom Checklist.

(although the HAM-D score is marginal), have worse social adjustment as measured by SAS scores, have more general psychopathology as measured by the SCL-90, and have more chronic depression. The last 2 factors are the most significant with over half a standard deviation difference between those with and without comorbid personality disorders.

Table 2 summarizes the effect of the selected baseline variables, including the presence or absence of personality disorder and drug type, on percentage reduction in MADRS scores at 6 weeks. The baseline MADRS is not included since using the percentage reduction largely adjusts for this variable. There are 4 findings. First, the presence or absence of personality disorder does not significantly affect outcome. Second, baseline variables, with the exception of chronicity of mood disorder in the past 5 years, do not affect outcome. Third, drug type affects outcome; at 6 weeks, patients were significantly better on fluoxetine treatment. Fourth, there was an interaction between having a comorbid personality disorder and drug type. The results were the same when using percentage reduction of HAM-D as the outcome measure.

Table 3 further explores the interaction between drug type and personality disorder. Baseline mood chronicity remains a significant predictor of the percentage reduction in MADRS scores, but drug type does not. The drug type–personality disorder interaction is largely explained by cluster B comorbidity. Since depressed patients with a cluster B personality disorder were younger, had more chronic depression, and had more general psychopathology (based on SCL-90 scores) as shown in Table 2, these variables were also tested for interactions in the model. Age and SCL-90 total score were significantly related to drug type. The cluster B drug interaction remained significant.

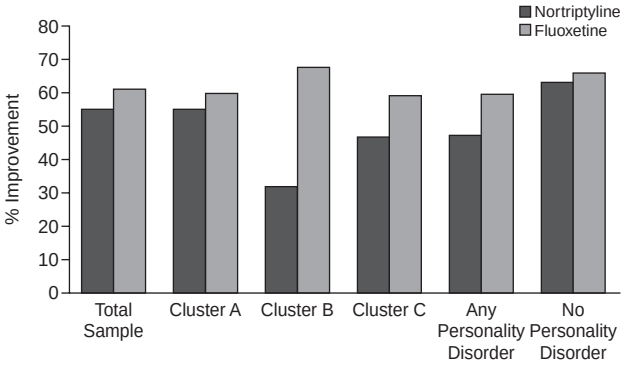
Figure 1 shows the mean percentage improvement on treatment with each drug within the total sample and by personality disorder and personality disorder clusters. The results are consistent with Tables 2 and 3. There is an overall trend for depressed patients to do worse with nortriptyline than with fluoxetine. This trend is influenced by

Table 3. Effect of Baseline Variables, Personality Disorder Clusters, and Drug Type on Percentage Reduction in Montgomery-Asberg Depression Rating Scale Scores at 6 Weeks (N = 149)

Variable	F	df	p Value
Sex	0.07	1	.79
Age	0.90	1	.34
SAS	2.28	1	.13
SCL-90	0.09	1	.76
Chronic depression	5.73	1	.02
Drug type	1.68	1	.20
Drug × cluster A	0.16	1	.69
Drug × cluster B	7.73	1	.006
Drug × cluster C	0.09	1	.77
Drug × age	4.33	1	.04
Drug × SAS	1.14	1	.29
Drug × SCL-90	4.44	1	.04
Drug × mood 5 y	0.09	1	.77

Abbreviations: SAS = Social Adjustment Scale, SCL-90 = Hopkins Symptom Checklist.

Figure 1. Mean Percentage Improvement in 149 Depressed Patients Based on Personality Disorder, Cluster, and Drug Type



the presence or absence of comorbid personality disorders. The percentage response to nortriptyline and fluoxetine in patients without a personality disorder is very similar, while in patients with a personality disorder, nortriptyline produces a lower percentage response. This effect is largely explained by the poorer response of patients with a cluster B personality disorder to nortriptyline. Although there also seems to be an effect in patients with cluster C personality disorders, this is mainly due to their comorbidity with cluster B personality disorders. In Table 3, which allows for interactions, there is no suggestion of a distinct cluster C personality disorder effect ($F = 0.09$, $p = .77$).

DISCUSSION

The results from this study suggest that the presence of a comorbid personality disorder in depressed patients does not affect overall response to antidepressant drugs at

6 weeks. This lack of association is despite the fact that the presence of a comorbid personality disorder is associated with somewhat more chronic and severe depression, poorer social adjustment, and more general psychopathology. There is an interaction between drug type, comorbid personality disorder, and outcome. While there is a trend for all patients to do worse with nortriptyline compared with fluoxetine, the difference is significant in depressed patients with a comorbid cluster B personality disorder, for which their mean percentage response on nortriptyline is only 30%. This poorer response to nortriptyline remains significant after adjustment for a variety of demographic and psychopathologic features associated with cluster B personality disorders.

The characteristics of depressed patients with and without comorbid personality disorders have been compared in a number of studies. The methodology varies widely, and the results are inconsistent. In general, depressed patients with comorbid personality disorders have been reported to have an earlier onset of depression,[14] more severe depression, higher rates of atypical features, and more comorbid dysthymia,[6,23,24] but results are conflicting (e.g., Skodol et al.[6] reported no difference in the age at onset of depression or atypical features). The present study supports the idea that depressed patients with personality disorders have a more chronic depression and are generally more unwell. These differences are relatively modest, but they are significant and appear to be most pronounced in patients with a cluster C personality disorder.

One important finding is that the presence of a comorbid personality disorder did not affect overall treatment response. As we discussed in the introduction, the belief that the presence of a comorbid personality disorder adversely affects response to treatment is widely held by clinicians despite limited evidence. While overall the studies using DSM Axis II criteria suggest a worse outcome,[1] the largest, best conducted trials (i.e., those using structured interviews and controlling treatment) are more equivocal. Of the 7 studies that we could find that used structured interviews and controlled treatment, 4 reported no difference in response,[25–28] 2 reported worse outcome,[29,30] and 1 reported mixed results.[31] The present study is the second largest to systematically address the question of whether comorbid personality disorders adversely affect short-term treatment outcome, and our results support the view that in a sample of moderately depressed outpatients they do not. The largest sample (N = 623), which consisted of chronically depressed patients, reported the same finding.[28]

A second important finding is that having a comorbid personality disorder may affect response to specific treatments. Differential response has been poorly studied, but there is evidence that depressed patients with personality disorders may respond differently to treatments than those without personality disorders[32–34] and that patients with certain personality disorders may respond preferentially to specific treatments. Our finding is that patients with cluster B personality disorders respond poorly to nortriptyline, but not to fluoxetine. Our finding is consistent with an emerging literature suggesting that behaviors associated with cluster B personality disorders—most studies have focused on borderline personality disorders—may respond poorly to TCAs but moderately well to selective serotonin reuptake inhibitors (SSRIs).[35–37] It is also consistent with the observation by Fava et al.[38] that depressed patients with borderline personality disorder responded better to fluoxetine than those with other personality disorders or no personality disorder.

Although our results are consistent with these studies, methodological shortcomings mean they must be regarded as provisional. The most significant problem is that the study was not double-blind nor placebo-controlled. The reason for the former was that the study was intended to be a long-term study; we wished to maximize compliance, and the drugs were so different with regard to dose and side effects that we felt that true double-blindness would not be possible. Placebo-controlled studies for depression are very difficult to justify when effective treatments are available.

Another potential problem is the relatively short duration of the study. Although the length of treatment is similar to most antidepressant studies, it may argued that 6 weeks is too early to study the effects of personality pathology on response. Nonetheless, a robust differential response was found. It is hard to justify continuing with an unsuccessful drug treatment for depression beyond 6 weeks, making the study of differential responses beyond this time difficult. It is possible that the long-term treatment outcome is adversely affected by baseline personality pathology; we are collecting data on this and will report the results in the future.

Finally, there are the general problems common to all studies that have looked at personality disorders in a clinical setting. While structured interviews are considered "state of the art," significant concerns about their validity, particularly in depressed patients, still exist. Using informant and longitudinal data might strengthen the validity of the diagnoses, but clinicians often need to base treatment decisions on a more limited assessment. It should be kept in mind that using structured interviews is no guarantee of unbiased independent assessment of personality disorders.

In addition, the validity of personality disorder diagnoses themselves remains open to question.[9] Ideally, the validity of instruments diagnosing personality disorders should be examined through the predicted correlates of the personality construct. Unfortunately, it has not yet been demonstrated that the operational criteria in DSM are themselves valid. We therefore face the difficult task of validating diagnoses as well as instruments. For clinicians,

however, whether the measured construct is actually a valid measure of personality disorder is of limited concern. If it predicts something about treatment response, then it may be useful, regardless of what it is actually measuring.

In summary, our study is generally consistent with a growing literature on the relationship of personality disorders to major depression. Personality disorders, as currently conceptualized, seem to be common in patients with major depression, particularly avoidant and borderline personality disorder. Overall, the presence of a personality disorder has little effect on short-term treatment outcome. Differences reported in some studies may partly be the result of worse treatment that those with comorbid personality disorders sometimes receive in noncontrolled studies. The presence of personality disorder may be important when choosing treatments for depressed patients. Our study supports a small number of trials suggesting that tricyclics may be less effective than SSRIs in patients who have comorbid cluster B personality disorders.

Drug names: fluoxetine (Prozac and others), nortriptyline (Aventyl and others).

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