

## Personality Traits During Depression and After Clinical Recovery

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We investigated whether and how acute depressive symptoms affect the self-report of maladaptive personality traits. Sixty-eight acutely depressed patients underwent the Structured Interview for DSM–III Personality Disorder (SIDP) before and after pharmacological treatment, allowing us to determine whether self-reported maladaptive personality traits are different during depression and after successful clinical recovery. After the initial SIDP administration (during an episode of major depression), patients received desipramine treatment (dose range 150–300 mg/day) over a course of 4–5 weeks before readministration of the SIDP. For those who recovered from their depression ( $n = 39$ ), cluster III trait scores were significantly lower than those assessed at baseline, and there was a lower frequency of cluster III categorical diagnoses for a personality disorder after treatment than before treatment. Recovered patients also had significantly lower cluster I personality trait scores after treatment as compared with baseline ratings. For those who did not recover from their depression after treatment ( $n = 29$ ), cluster I trait scores were in fact higher than those measured at baseline, but there were no differences in categorical diagnoses before and after treatment. Cluster II personality traits and categorical diagnoses were not different between those who did and did not recover from their depression. Thus, depression may have a significant effect on the assessment of cluster I and cluster III personality traits. It is possible that cluster I and III 'personality traits' may be interwoven with depressive features and therefore subject to state influences, whereas cluster II personality traits may entail enduring, long-term characteristic modes of thinking, feeling, and behaving.

One of the major problems in the diagnostic assessment of a personality disorder is how to distinguish between an actual personality trait and the concomitants of an acute clinical state (i.e. anxiety or depression). Although DSM–III–R (American Psychiatric Association, 1987) notes that personality disorders are enduring, maladaptive modes of behaviour (i.e. traits), the task of determining maladaptive personality traits during an evaluation is difficult because patients are often in an acute crisis which itself may appear to be a personality disorder. Thus, the complex nature of help-seeking behaviour can confound psychiatric assessments, raising the question of whether or not a personality disorder actually exists. Indeed, studies by Hirschfeld (1986) and Reich *et al* (1987) suggest that patients, during periods of elevated psychopathology (i.e. depression or anxiety), may describe themselves as having more abnormal personality traits than during asymptomatic periods. Moreover, numerous studies (Perris, 1971; Liebowitz *et al*, 1979; Bech *et al*, 1980) have noted that subjects who recovered from a primary depressive illness had normal neuroticism scores on the Eysenck Neuroticism Scale.

The purpose of this study was to investigate how the *self-report* of personality traits may change in acute depressed patients before and after clinical treatment. Such a study may improve insights into

the influence of depressive symptoms on personality disorders.

### Method

All patients in this study were treated at the Foundation for Depression/Manic Depression (New York State Psychiatric Institute). A total of 68 patients (28 men and 40 women) participated in this evaluation. Their mean age (s.d.) was 39.54 (12.5) years. The average age for first onset of the depressive illness was 29.97 (10.1) years, and, in the two years before this study, the average length of time depressed was 10.93 (8.0) months. All patients met DSM–III criteria for major depression (American Psychiatric Association, 1980), and all had a minimum score of 18 (out of 21 items) on the Hamilton Rating Scale for Depression (Hamilton, 1967).

For assessment of the presence or absence of maladaptive personality traits, our depressed patients underwent the Structured Interview for DSM–III Personality Disorder (SIDP), which was developed by Pfohl *et al* (1984). The SIDP is based upon rateable criteria from the 11 DSM–III personality disorders. The number of rateable traits for each DSM–III personality disorder is as follows: three for schizoid and dependent; five for compulsive and avoidant; eight for schizotypal, narcissistic, borderline, and passive aggressive; ten for histrionic; 16 for paranoid; and 22 for antisocial. Each item is rated on a 0–2 point scale; however, items for the antisocial personality disorder are rated as

either absent (0) or present (1). We assigned a point score for each self-reported trait for a given personality disorder. A total personality disorder score was obtained by summing all trait items for a personality disorder. Furthermore, total personality scores were aggregated into three separate personality cluster scores, as defined by Pfohl *et al* (1984). That is, the cluster I score was the sum of all paranoid, schizoid, and schizotypal personality disorder items; the cluster II score was the sum of all histrionic, narcissistic, antisocial, and borderline personality disorder items; and the cluster III score was the sum of all avoidant, dependent, compulsive, and passive aggressive personality disorder items. Patients were also dichotomised according to whether or not they had a categorical diagnosis for each personality disorder and personality cluster. The clustering of personality traits into three main categories has not only been noted in both the DSM-III and DSM-III-R, but also has been supported by the work of Bell & Jackson (1992). Prior studies have also revealed personality clusters similar to those cited in the DSM-III and DSM-III-R criteria (Tyrer *et al*, 1979). It should be added that differences between the DSM-III and DSM-III-R personality disorders appear to be negligible, except for the schizoid and schizotypal personality disorders (Vaglum *et al*, 1989).

Kappa-coefficients of interrater agreement have been reported to be 0.70 or higher for most of the personality disorders (Stangl *et al*, 1985). In the current study, two raters evaluated each patient. With Pearson correlations, the interrater reliability for the two raters ranged from a low of  $r=0.76$  (for the compulsive and dependent personality disorders) to a high of  $r=0.95$  (for the paranoid personality disorder).

Patients received desipramine treatment over a period of 26–36 days (dose range was 150–300 mg/day). Those who recovered from their acute depressive episode had, by definition, a 50% reduction in their Hamilton score, a final score on the Clinical Global Impression (CGI) (Guy, 1976) scale of 2 or 1 (much or very much improved), and a final Hamilton score of 11 or less. After treatment, the SIDP was readministered to all patients. The pre- and post-treatment Hamilton ratings were performed blindly with respect to the pre- and post-treatment personality (SIDP) ratings. However, the rater performing the post-treatment SIDP rating may have had some indication of a patient's depressive status during the evaluation from appearance and interview.

The sample of depressed subjects mentioned here was identical to that cited in a previous report (Peselow *et al*, 1992). In Peselow *et al*'s study (1992), SIDP ratings at baseline (a consensus of rater, informant, and clinical record information) were explored in terms of their predictive validity for short- (4–5 weeks) and long-term (6 months) responses to desipramine treatment. Although confirmation of personality disorders was obtained in 43 of the 68 patients (63.2%) from informants, the remaining 25 patients were included in that study even though there was no external validation of these patients' characteristic modes of behaviour. The present study was concerned with personality traits during depression and after clinical recovery (regardless of the influence of these traits on treatment). In this study, we examined the patients' *self-report* of personality

disorders before and after treatment for all 68 patients. In the absence of informants for all patients, we felt that the patients' *self-report* provided a more consistent evaluation.

The overall approach to the data analysis was a mixed, two-factor analysis of variance (ANOVA). The between-subject factor was recovered/non-recovered status, and the within-subjects factor was treatment phase (before and after treatment). To interpret significant interactions ( $P<0.05$ ), we used simple main effects. Since the distributions for both the individual personality disorder scores and the three personality cluster scores were inherently skewed in a positive direction (subjects were not expected to display some maladaptive traits for *all* of the personality disorders or even for the personality clusters), the data were subjected to either square root or logarithmic transformations to obtain a normal distribution, an assumption for ANOVA. In some instances, transformations did not result in a fully normal distribution of the data. However, research has shown that ANOVA  $F$ -values are robustly insensitive to moderately skewed distributions (Lindquist, 1953). Since it was possible for subjects to show worsening of maladaptive personality traits after treatment for their depression, all subjects were retained in the ANOVA models, despite the presence of a transformed but still skewed data distribution. Accordingly, all reported  $F$ -values are those after data transformations, but the means of the untransformed data were reported for greater ease in interpreting the results.

## Results

After 4–5 weeks of desipramine treatment, 39 patients recovered from their depression, whereas 29 patients did not. For those who recovered and those who did not, there was no difference in initial baseline depression as measured by baseline Hamilton scores (26.85 and 27.93, respectively;  $t=0.91$ , d.f. = 66,  $P=0.37$ ), Beck scores (28.80 and 30.86;  $t=1.29$ , d.f. = 66,  $P=0.20$ ), and CGI scores (4.44 and 4.52;  $t=0.34$ , d.f. = 66,  $P=0.74$ ). In addition, there was no significant difference ( $t=0.68$ , d.f. = 66,  $P=0.50$ ) in maximum mean dose of desipramine received between those who recovered (214.10 mg/day) and those who did not (223.28 mg/day). After desipramine treatment, Hamilton scores ranged from 0 to 9 for those who recovered (mean 5.39), as opposed to a range of 16–35 for those who did not (mean 23.90).

From the aforementioned ANOVA design, Table 1 lists the (untransformed) mean personality and cluster scores for recovered and non-recovered patients, both before and after treatment. For cluster I personality traits, the main effect for treatment phase was not significant, but the main effect for recovery group was significant ( $F=5.73$ , d.f. = 1,66,  $P<0.03$ ). However, the latter result was an artefact of the significant recovery group by treatment phase interaction ( $F=17.75$ , d.f. = 1,66,  $P<0.0001$ ). There was no difference between recovered patients and non-recovered patients at baseline, but recovered patients had significantly lower post-treatment scores than non-recovered patients ( $t=3.56$ , d.f. = 66,  $P<0.001$ ). Simple main effects for this interaction also showed that recovered patients had lower post-treatment scores than baseline scores ( $t=2.47$ , d.f. = 66,

Table 1  
Mean personality trait scores before and after treatment

Personality disorder	Patients who recovered from depression ( <i>n</i> = 39) Baseline/treatment	Patients who did not recover from depression ( <i>n</i> = 29) Baseline/treatment	Recovery group by treatment phase interaction: <i>F</i> (1,66) and probability	Simple main effects			
				R v. NR: Pre	R v. NR: Post	Pre v. Post: R	Pre v. Post: NR
Schizoid	0.74/0.46	1.00/1.69	14.61, <i>P</i> < 0.0001	NS	**	NS	***
Paranoid	1.05/0.62	1.76/2.31	10.60, <i>P</i> < 0.002	NS	****	*	*
Schizotypal	0.59/0.38	0.90/1.52	14.04, <i>P</i> < 0.0001	NS	**	NS	**
Cluster I total	2.38/1.46	3.62/5.52	17.75, <i>P</i> < 0.0001	NS	***	*	***
Histrionic	1.15/1.44	1.52/1.45	1.73, NS	NS	NS	NS	NS
Narcissistic	1.59/1.51	2.48/2.38	0.05, NS	NS	NS	NS	NS
Antisocial	1.21/1.23	1.10/1.14	0.05, NS	NS	NS	NS	NS
Borderline	1.44/1.64	2.14/2.38	0.10, NS	NS	NS	NS	NS
Cluster II total	5.38/5.59	7.28/7.14	0.44, NS	NS	NS	NS	NS
Dependent	0.87/0.44	1.31/1.48	4.35, <i>P</i> < 0.05	NS	**	**	NS
Avoidant	2.21/1.64	2.79/2.93	10.41, <i>P</i> < 0.002	NS	**	****	NS
Compulsive	1.72/1.36	1.55/1.83	9.04, <i>P</i> < 0.004	NS	NS	**	NS
Passive-aggressive	1.23/1.03	1.97/1.97	0.50, NS	NS	NS	NS	NS
Cluster III total	6.03/4.46	7.62/8.34	12.95, <i>P</i> < 0.001	NS	***	****	NS

R = recovered from depression; NR = did not recover from depression. Means are based on untransformed data, but statistics are based on transformed data. \**P* < 0.05; \*\**P* < 0.01; \*\*\**P* < 0.001; \*\*\*\**P* < 0.0001.

*P* < 0.02); however, non-recovered patients surprisingly had higher scores after treatment than before treatment (*t* = 3.43, d.f. = 66, *P* < 0.001). For cluster III personality traits, significant main effects were found for both treatment phase (*F* = 6.74, d.f. = 1,66, *P* < 0.02), and for recovery group (*F* = 7.19, d.f. = 1,66, *P* < 0.01). The recovery group by treatment phase interaction also was significant (*F* = 12.95, d.f. = 1,66, *P* < 0.001). Simple main effects revealed that there was no difference between recovered patients and non-recovered patients at baseline, but that recovered patients had significantly lower post-treatment scores than non-recovered patients (*t* = 3.38, d.f. = 1,66, *P* < 0.001). In addition, simple main effects for this interaction showed

that patients who recovered from their depression had lower post-treatment scores than baseline scores (*t* = 4.74, d.f. = 66, *P* < 0.0001), but that pre- and post-treatment scores were not significantly different for patients who did not recover from their depression. For cluster II personality traits, the overall ANOVA model was not significant because the main effects for recovery group (*F* = 0.99, d.f. = 1,66, *P* = 0.32), and treatment phase (*F* = 1.52, d.f. = 1,66, *P* = 0.22) were not significant, as was the recovery group by treatment phase interaction (*F* = 0.06, d.f. = 1,66, *P* = 0.82).

Table 1 also lists the results for the individual personality disorders that make up each of the three personality clusters. From cluster I, the paranoid, schizoid, and schizotypal

Table 2  
Categorical personality disorder before and after treatment

	No personality disorder either before or after treatment	Personality disorder both before and after treatment	Personality disorder before treatment; no personality disorder after treatment	No personality disorder before pretreatment; personality disorder after treatment
<i>Cluster I</i>				
Recovered from depression	33	3	3	0
Did not recover from depression	19	6	0	4
<i>Cluster II</i>				
Recovered from depression	30	4	2	3
Did not recover from depression	20	7	2	0
<i>Cluster III*</i>				
Recovered from depression	23	4	10	2
Did not recover from depression	15	12	0	2

\* $\chi^2 = 4.42$ , 1 d.f., *P* = 0.035 with Yates' correction. Probability based on multi  $2 \times 2 \times 2$  contingency table with 1 d.f. comparing patients whose categorical diagnosis of personality disorder remained stable (either had no personality disorder at both evaluation points or had personality disorder at both points - columns 1 and 2) with those whose diagnosis of personality disorder changed between the two points (columns 3 and 4).

personality disorders all showed a similarly significant recovery group by treatment phase interaction. From cluster III, the dependent, avoidant, and compulsive personality disorders also displayed a similarly significant recovery group by treatment phase interaction pattern; however, analyses for the passive-aggressive personality disorder were not significant.

Table 2 shows the frequency of categorical diagnoses for personality disorders before and after treatment. Overall, 29 of the 68 patients (42.6%) met criteria for one or more DSM-III personality disorders during the depressed phase of the illness, a result essentially equivalent to the 42.1% frequency found in Zimmerman *et al's* study (1991), which used the SIDP. Of the 29 patients, 15 recovered subsequently, and 14 did not ( $\chi^2(1, n = 68) = 0.66, P = 0.42$ ).

Table 2 shows how patients were grouped into one of four categories, depending on whether or not they had a personality disorder before and after treatment (i.e. no personality disorder before and after treatment, personality disorder *both* before and after treatment, personality disorder before treatment and no personality disorder after treatment, and no personality disorder before treatment but personality disorder after treatment). On the basis of a categorical diagnosis for personality disorders, the presence of a cluster III personality disorder at baseline was associated with significant personality trait improvement in those subjects who recovered from their depression. Of the 14 patients who had a cluster III personality trait disorder at baseline (before recovery), 10 were found to have no cluster III personality disorder after successful treatment for their depression. For the 12 non-recovered patients who had a cluster III personality disorder at baseline, all 12 exhibited a cluster III personality disorder after treatment (while still depressed). With a  $2 \times 2 \chi^2$  design (those patients who had stable diagnosis before and after treatment and those whose diagnosis changed), the results were statistically significant ( $\chi^2(1, n = 68) = 4.42, P = 0.035$ , with Yates' correction).

Since most patients (51 of 68, or 75%) did not meet criteria for a categorical cluster I diagnosis either before or after treatment, the number of patients in the four dichotomous groups was too small to obtain statistical significance. However, it is interesting to note that three of six recovered patients had a cluster I personality before treatment (when depressed), but not after treatment. On the other hand, all six non-recovered patients had a cluster I personality disorder both before and after treatment (while still depressed). In addition, four non-recovered patients who did not have a cluster I personality disorder before treatment were found to have one after treatment (while still depressed). For the cluster II group, there were no statistically significant findings nor any predominant patterns or trends.

### Discussion

There has been much research in Europe on how personality inventory scores change as a function of depression level. For instance, both Coppen & Metcalfe (1965) and Garside *et al* (1970) reported a

significant decrease (or improvement) in Maudsley Personality Inventory neuroticism scores after successful recovery from depressive illness. Using the Eysenck Personality Inventory (EPI), Kendell & DiScipio (1968) found a significant decrease in neuroticism scores and a significant increase in extroversion scores in patients who recovered from their depression. In this latter study, it is noteworthy that some patients, when completing the EPI during their depression, were given additional instructions to answer the questions according to how they felt and behaved when not depressed. Neuroticism and extroversion scores of depressed patients who received such instructions were lower and higher, respectively, than those of depressed patients who were given no additional instructions. Moreover, for those who received the additional instructions, there were no significant differences in neuroticism and extroversion scores from baseline to after treatment. However, it is clear that a depressed mood increases the accessibility of negative cognitions (Martin *et al*, 1983), and, in some studies, the use of instructions, like that mentioned above, does not yield reliable information (Hirschfield *et al*, 1983).

To our knowledge, there have been only three studies that have specifically examined personality traits/disorders before and after pharmacological treatment for depression. Using the Personality Disorder Examination and the Structured Clinical Interview for DSM-III Personality Disorders, O'Boyle & Self (1990) found that the depressive state did not affect categorical diagnoses of personality disorders, but that higher-dimensional (maladaptive) traits were noted in the depressed state (in a sample of 17 patients), a result that was significant for the borderline and compulsive personality disorders. Gartner *et al* (1989) administered the Personality Disorder Inventory upon hospital admission and then just before discharge on 35 patients who had eating disorders (anorexia, bulimia, and anorexia and bulimia). These researchers found that the mean axis II criteria upon discharge was significantly lower than those during admission (with an average 30% drop in depression, and 40% decrease in anxiety from baseline to discharge); however, categorical diagnosis of personality disorders was unchanged from admission (55.5%) to discharge (57.1%). Using the Personality Disorders Examination, Loranger *et al* (1991) noted that in 84 patients, 45 of whom had primary depressive symptoms, there was a trend toward fewer maladaptive traits at follow-up (i.e. significantly fewer paranoid, schizotypal, compulsive, histrionic, dependent, narcissistic, avoidant, borderline, passive-aggressive, and masochistic traits) when subjects had a considerable reduction

in their depressive symptoms (i.e. an average baseline Hamilton score of 11.1 and an average end-point score of 7.0). However, depressive states did not affect categorical diagnoses for a personality disorder before and after treatment.

Two other studies (Mavissakalian & Hamann, 1987; Joffe & Regan, 1989) examined the effect of depression on personality traits. Mavissakalian & Hamann (1987) evaluated patients who had agoraphobia with the Personality Diagnostic Questionnaire. After four months of medication and behaviour therapy, these researchers found that both responders and, to a lesser degree, non-responders had a significant decrease in 16 axis II traits common to agoraphobic subjects. Using the Millon Clinical Multiaxial Inventory, Joffe & Regan (1989) evaluated 42 depressed patients treated with antidepressants over a 12-week course. At baseline, virtually all patients (41 of 42) had one or more personality disorder diagnoses. After treatment, 30% of patients who responded favourably to antidepressants (7 of 23) had no personality disorder, in contrast to the 11% rate (2 of 19) for those who did not respond favourably to antidepressants ( $P = NS$ ).

Compared with studies mentioned above, our study found greater changes in both the individual personality traits and the categorical diagnoses. This discrepancy may be explained by the greater levels of initial depression and the greater changes in Hamilton scores (for those who recovered) in our study relative to those seen in two of the three aforementioned studies (i.e. Gartner *et al*, 1989; Loranger *et al*, 1991); that is, the recovery group in our study went from an initial mean Hamilton score of 26.85 to 5.39, quite unlike those reported by Gartner *et al* (1989) (9.56 to 6.28) and Loranger *et al* (1991) (11.1 to 7.0). O'Boyle & Self (1990), however, did report a substantial change in Hamilton scores (23.5 to 7.9).

As noted by Zimmerman *et al* (1986, 1991), depression can bias responses to self-report personality inventories (usually toward higher ratings of personality traits/disorders), even when patients are asked to describe their usual normal state when not depressed (Reich *et al*, 1987; Hirschfeld *et al*, 1983). Although personality assessment during asymptomatic periods may reduce such biasing effects of depression, it does not take into account the possibility that remitted depressive states may lead to alterations in personality in a way that is qualitatively different from baseline levels (Akiskal *et al*, 1983). Because the experience of depression can affect self-image, personality, emotional expressions, and behaviour, a truly prospective study examining the effects of personality on treatment response and

outcome in depression would have to identify a patient's personality traits before the onset of a depressive episode. In such a study by Hirschfeld *et al* (1989) on older patients, it was discovered that the first onset of a depressive episode was associated with decreased emotional strength, increased interpersonal dependency, and increased thoughtfulness.

Informants have been used to assess personality traits or disorders during periods of depression (Pfohl *et al*, 1984). Although informants may diminish the biasing effect that depression can have on the self-report of personality traits, it is possible that family members may hold different perceptions of a patient's personality during depressed and non-depressed periods (Farmer & Nelson-Gray, 1990).

In this study, successful recovery from depression was associated with diminished cluster I and cluster III personality traits, a finding that, while in need of replication, warrants some discussion. The 'personality traits' that showed a reduction after clinical recovery, such as the emotional coldness and aloofness of the schizoid personality, the decreased self-confidence of the dependent personality, and the social withdrawal of the avoidant personality (among others), may have been associated more with symptoms of depression than with the enduring, long-term characteristic patterns of a person. A possible reason for finding no difference in cluster II personality traits after clinical recovery is that such traits, which include the inappropriate and intense anger of the borderline personality, the grandiose sense of uniqueness of the narcissistic personality, and the vain and demanding features of the histrionic personality (to name a few), may be more associated with enduring maladaptive patterns of thinking and relating, which are the core with respect to personality disorders (DSM-III-R, 1987), unlike the features associated with cluster I and cluster III personality traits which may be associated with clinical symptoms. The number of subjects who had prominent cluster II personality traits was fairly small, diminishing the power to detect a significant difference, but those who recovered from their depression had slightly more borderline and overall cluster II traits. This finding is counterintuitive to the hypothesised relationship between depression and cluster II (particularly borderline) traits (see Joffe & Regan, 1989).

Other explanations of the findings presented here are possible. For instance, it is possible that the difference in cluster I and cluster III personality traits during depression and after clinical recovery may have been due to the inability of raters to distinguish between, for example, depressive withdrawal and schizoid features, such as aloofness and coldness. As noted by Farmer & Nelson-Gray (1990), better

research approaches are needed to distinguish between transient symptoms and deeply ingrained patterns of thinking, feeling and behaving, because various personality assessment instruments do not adequately make this distinction (Reich, 1987).

In summary, we found that cluster I and cluster III personality traits, as well as a cluster III categorical diagnosis, during a depressive episode tended to decrease after successful clinical recovery from depression. In contrast, cluster II personality traits and a cluster I and II categorical diagnosis were unchanged in this sample, regardless of recovery status from depression. Future studies are needed to verify these findings, but it is clear that more refined methods and assessment tools are needed to distinguish more clearly the confounding effects that depression may have on personality traits and disorders.

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