The Construct Validity of Depressive Personality Disorder

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This study examined the construct validity of depressive personality disorder (DPD; American Psychiatric Association, 1994). Adult psychiatric outpatients ($N = 900$) underwent comprehensive Axis I and II evaluations and provided data on 4,768 of their 1st-degree relatives. Despite modest overlap, DPD was not redundant with any Axis I or II disorder. Participants with DPD exhibited more Axis I and Axis II comorbidity, and greater psychosocial dysfunction, than participants without DPD. Relatives of participants with DPD had higher rates of mood disorders, alcohol abuse, and antisocial personality. Results are consistent with findings of several other similar investigations. The authors argue that DPD is a valid construct and should be conceptualized as a personality disorder as opposed to a mood disorder.

Formulations of depressive personality have been the focus of clinical attention (Huprich, 1998, 2001; Phillips, Gunderson, Hirschfeld, & Smith, 1990; Shea & Hirschfeld, 1996) for the past 7 to 8 decades. Basic research on the rudimentary dimensions of personality has also examined very similar constructs such as neuroticism (Costa & McCrae, 1995; Jang, McCrae, Angleitner, Riemann, & Livesley, 1998). Interest among research-oriented clinicians reached a crescendo in the last 15 years as a debate has arisen concerning whether or not depressive personality disorder is a valid entity and, in turn, whether or not it should be included as an official diagnosis in the American psychiatric nomenclature (i.e., the next *Diagnostic and Statistical Manual of Mental Disorders; DSM–V*). Descriptions of depressive personality have varied over the years, but, in essence, the construct is succinctly described as an admixture of traits of gloominess, seriousness, self-reproach, negativity, and pessimism. Depressive personality is distinguished from both dysthymic disorder (DD) and major depression because its symptomatology does not include neurovegetative changes, nor is there an emphasis on the centrality of a mood disturbance.

Depressive personality disorder (DPD) was added to Appendix B of the *DSM–IV* (4th edition; American Psychiatric Association, 1994), in the section, "Criteria Sets and Axes for Further Study," and not the Axis II disorders because of concern about whether depressive personality could be distinguished from dysthymic disorder, major depression, and other personality disorders (Phillips et al., 1998; Widiger, 1999). Particular focus has been paid to whether or not DPD could be distinguished from DD. Empirical research on the construct of depressive personality disorder since 1990 has shown that although it overlaps with DD and major depression, it is nevertheless distinct (Hirschfeld & Holzer, 1994;

Klein, 1990; Klein & Miller, 1993; Klein & Shih, 1998; Phillips et al., 1998). Attempts to demonstrate the reliability of the construct have met with success, as criteria sets that have been used have shown moderate to good interrater reliability and internal consistency (Klein, 1990). Test–retest reliability has been moderately stable over 1 year (Phillips et al., 1998) to 2.5 years (Klein & Shih, 1998). Family history evidence shows a pattern of familial co-aggregation of DPD with mood disorders, which may mean that the disorders share common biogenetic etiological influences (Klein, 1990; Klein & Miller, 1993).

To date, however, little research has examined the reliability and validity of DPD using the criteria set forth by the *DSM–IV*. In fact, only two published studies have used the *DSM–IV* criteria set for DPD (Hirschfeld & Holzer, 1994; Ryder, Bagby, & Dion, 2001). Ryder et al.'s study was narrower in scope in that its primary focus was how DPD symptoms and DD symptoms would align themselves in a factor analysis. Hirschfeld and Holzer examined physical and psychosocial functioning in DPD, and comorbidity with Axis I depressive disorders. The purpose of this report from the Rhode Island Methods to Improve Diagnostic Assessment and Services (MIDAS) project was to examine the validity of DPD—diagnosed using *DSM–IV* criteria—using a large sample of 900 psychiatric outpatients and the psychiatric histories of 4,768 of their first-degree relatives.

The statistical analyses in the present report address several questions that are fundamental to establishing the validity of a psychiatric diagnosis (Robins & Guze, 1970). Rates of comorbidity between DPD and other Axis I disorders and personality disorders were calculated in an attempt to replicate previous work showing that DPD overlaps with, but is not entirely subsumed by, any other Axis I or Axis II disorder. Psychiatric morbidity and psychosocial functioning of subjects with and without DPD were determined to verify previous findings that the presence of DPD is associated with increased psychiatric morbidity and impaired psychosocial functioning. Finally, family history data were used in an attempt to see if there was evidence of a familial co-aggregation of DPD and Axis I mood disorders, which has been found by Klein and associates (Klein, 1990; Klein & Miller, 1993). A replication of Klein's findings is relevant to both the construct validity of

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DPD (Robins & Guze, 1970) and the controversial issue of whether or not DPD should be classified as a mood disorder or a personality disorder. In the Discussion section, the empirical evidence and rationale for conceptualizing DPD as a personality disorder (as opposed to a mood disorder), and the possible addition of DPD to Axis II, are addressed.

Method

Participants and Diagnostic Procedure

To date, 1,500 outpatients have been evaluated in the MIDAS project. The methodology was changed after 600 patients were assessed only for Axis I disorders, and the next 900 patients were assessed for both Axis I and Axis II disorders. The Rhode Island Hospital institutional review committee approved the research protocol, and all participants provided informed, written consent. Diagnostic raters included clinical psychologists and bachelor's-level research assistants who underwent 3 months of training. Throughout the study, ongoing supervision of the raters consisted of weekly diagnostic case conferences involving all members of the team. In addition, every case was presented to the project director (Mark Zimmerman). The methods of the study are described in further detail elsewhere (Zimmerman & Mattia, 1999).

Diagnoses and psychiatric symptoms. We used the January 1995 *DSM–IV* patient version of the *Structured Clinical Interview for DSM–IV Disorders* (*SCID–IV*; First, Spitzer, Williams, & Gibbon, 1995). Axis I disorders in partial remission and not otherwise specified diagnoses were not included. We incorporated most of the symptom items from the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) into the SCID; ratings on these items were used to calculate an extracted Hamilton Rating Scale for Depression (HRSD; Endicott, Cohen, Nee, Fleiss, & Sarantakos, 1981). In addition, the diagnostic interviewer rated the overall severity of depression on the Clinical Global Index (CGI; Guy, 1976), which ranges from 0 (*none*) to 6 (*extreme*). Severity of suicidal ideation was rated according to the SADS suicidality item, which rates suicidal tendencies on a scale ranging from 0 (*not at all*) to 6 (*very extreme*, e.g., suicide attempt with definite intent to die).

Global, social, and occupational functioning. The Global Assessment of Functioning scale (GAF; American Psychiatric Association, 1994) was used as a measure of overall symptomatic and functional impairment. Two SADS items were used to assess social functioning. Past social functioning was rated from 1 (*superior*) to 6 (*grossly inadequate*), as was current social functioning. The occupational functioning item on the SADS assesses time out of work in the last 5 years and was rated from 1 (*no missed work*) to 9 (*did not work*).

Axis II pathology. Personality disorders were assessed using the *Structured Interview for DSM–IV Personality* (SIDP; Pfohl, Blum, & Zimmerman, 1997). We had SIDP data for 900 of the initial pool of 1,500 outpatients, so results reported in this study are based on those 900 patients. The SIDP, a semistructured interview, enquires about the traits of the 10 "official" *DSM–IV* personality disorders (PDs) as well as three appendix PDs (depressive personality disorder, self-defeating personality disorder, and negativistic personality disorder). Following *DSM–IV*'s algorithm, participants were diagnosed with DPD if they were rated positive on five or more of the following seven traits:

1. Usual mood is dominated by dejection, gloominess, cheerlessness, joylessness, and unhappiness;

2. Self-concept centers around beliefs of inadequacy, worthlessness, and low self-esteem;

3. The person is critical, blaming, and derogatory toward the self;

4. The person broods and is given to worry;

5. The person is negativistic, critical, and judgmental toward others;

6. The person is pessimistic; and

7. The person is prone to feeling guilty or remorseful.

The SIDP is designed and administered to minimize the contaminating effects of episodic mood states (e.g., a depressive episode) on ratings of enduring personality traits. The interviewer is instructed to state explicitly, "I am most interested in what you are like when you are your usual self. If you are currently hospitalized or experiencing an illness, please try to remember what you are like when you are your usual self." This instruction is repeated several times throughout the interview. To deal with ambiguities surrounding whether or not a particular trait is present, the SIDP adheres to the "5-year rule," which states that personality features that have predominated for the greatest amount of time in the last 5 years should be rated as typical of the interviewee's long-term personality functioning.

Family history. The Family History—Research Diagnostic Criteria (FH–RDC; Andreasan, Endicott, Spitzer, & Winokur, 1977) were used to diagnose mood disorders, psychotic disorders, substance abuse disorders, anxiety disorders, and antisocial personality disorder in all first-degree relatives. Participants provided family history information.

Data Analysis

The *t* tests were performed to compare groups on continuously distributed variables. Categorical variables were compared by the chi-square statistic, with Yates's correction, or by Fisher's exact test if the expected value in any cell of a 2×2 table was less than 5. For the psychiatric family history data, morbid risks were calculated using age-corrected denominators based on Weinberg's abridged method (Stromgren, 1950). Thus, relatives over the age of risk for the particular illness were given a value of 1; those within the age of risk were given a value of 0.5, and those below it were given a value of 0. Limits for the ages of risk were 23 to 44 for depression, 16 to 29 for bipolar disorder, 16 to 57 for schizophrenia and schizoaffective disorder, 23 to 49 for unspecified psychosis, 18 to 31 for alcohol abuse/dependence, 17 to 25 for drug abuse/dependence, 23 to 43 for panic disorder, 24 to 42 for panic with agoraphobia, 10 to 30 for specific phobia, 8 to 19 for social phobia, 18 to 33 for obsessive–compulsive disorder (OCD), 17 to 36 for posttraumatic stress disorder (PTSD), and 14 to 36 for generalized anxiety disorder (GAD). Analyses of family history of a suicide attempt or of a history of any diagnosis were not age corrected. These ages of risk were determined on the basis of the distribution of ages of onset in the initial pool of 1,500 psychiatric outpatients from which the 900, who are the focus of this report, were drawn. The lower age limit represents the median age of onset in the age of onset distribution for that particular disorder. The upper age limit represents the 90th percentile in the distribution of ages of onset for that particular disorder.

Results

The 900 participants had a mean age of 37.1 years ($SD = 12.2$). Almost two thirds ($n = 556$) were women. Almost 40% ($n = 349$) were married, the majority (87.3%, $n = 786$) were White, and about one quarter (25.9%, $n = 233$) had a college degree.

Of the 900 outpatients, 198 (22.0%) met criteria for DPD. There was a higher proportion of women in the DPD group (68%) than in the non-DPD group (60%), $\chi^2(1, N = 198) = 3.74$, $p < .05$. Conversely, there was a higher proportion of men in the non-DPD group (39.9% vs. 32.3%). Members of the DPD group were less likely to be married at the time of the assessment (31.8% vs. 40.7%), $\chi^2(1, N = 198) = 5.18$, $p < .05$. There was no

difference between the groups on age, education, or racial composition.

Internal Consistency and Reliability

The internal consistency (Kuder–Richardson) of the seven-item scale for the sample of 900 outpatients was .77. Corrected item–total correlations for the seven criteria were as follows: .49 for usual mood dominated by dejection; .58 for self-concept that centers around inadequacy; .60 for self-critical; .49 for given to worry; .32 for critical and judgmental of others; .50 for pessimistic; .50 for guilty and remorseful. The following percentages of participants with DPD (i.e., item sensitivities) endorsed each item: 67% for usual mood dominated by dejection; 88% for self-concept centers around inadequacy; 97% for self-critical; 88% for given to worry; 57% for critical and judgmental of others; 86% for pessimistic; and 88% for guilty and remorseful.

To examine the interrater reliability of DPD, we used a paired-rater design in which an observer sat in on 28 interviews and independently rated each item and made independent diagnoses. The Pearson correlation for number of DPD symptoms was .93. The intraclass correlation for number of DPD symptoms was .97. Diagnostic concordance, as measured by kappa, was .52.

Relation to Axis I Disorders

Although dysthymic disorder was significantly more common in participants with versus without DPD, only a minority of the participants diagnosed with DPD were also diagnosed with dysthymic disorder (see Table 1). Looking at this another way, of the 75 participants with dysthymic disorder, almost half ($n = 36$, 48%) were also diagnosed with DPD. DPD was also significantly associated with a current diagnosis of major depressive disorder (MDD), although about 40% did not meet criteria for current major depression. Of the 403 participants diagnosed with current unipolar depression, 28.3% ($n = 114$) had comorbid DPD.

As seen in Table 1, compared with participants without DPD, participants with DPD had higher rates of current or lifetime anxiety disorders. Specifically, participants with DPD had higher rates of current or lifetime panic disorder with agoraphobia, specific phobia, social phobia, OCD, PTSD, and GAD. It is interesting that participants with DPD were less likely to ever be diagnosed with an adjustment disorder relative to participants without DPD. Although participants with DPD were not more likely to be diagnosed with a current comorbid eating disorder, participants with DPD were more likely to report a history of anorexia or bulimia.

Relation to Other Personality Disorders

Inspection of Table 2 shows that, compared with participants without DPD, participants diagnosed with DPD had significantly higher rates of all personality disorders except antisocial personality disorder (ASPD). DPD was most likely to co-occur with avoidant PD, obsessive–compulsive PD, and borderline PD. About one third (33.8%) of participants with DPD did not have an additional Axis II diagnosis.

DPD, Psychiatric Morbidity, and Psychosocial Functioning

Table 3 shows comparisons between participants with DPD and those without on measures of psychiatric morbidity and psycho-social functioning. On six of the seven measures of psychiatric morbidity and functioning, participants with DPD exhibited significant impairment.

Effect of DPD on Retrospective Course and Severity of Current Major Depression

In participants with current major depression, we examined whether DPD was associated with poorer course (retrospectively reported) and a more severe current episode. The rate of chronic MDD, defined by *DSM–IV* as an episode duration of at least 2 years (American Psychiatric Association, 1994), was significantly higher in depressed participants with comorbid DPD (see Table 4).

As seen in Table 4, participants with DPD were also significantly more likely to have had an earlier age of onset, and they exhibited significantly more severe depressive symptomatology as indexed by the CGI, extracted HRSD, and total number of *DSM–IV* depressive symptoms.

Family Psychiatric History

Family psychiatric history data (see Table 5) showed that first-degree relatives of participants with depressive personality were more likely to have a history of any psychiatric disorder. Specifically, DPD relatives were more likely to have a history of depressive disorder, bipolar disorder, alcohol use disorder, and ASPD.

Because the association between the probands' DPD and the relatives' depressive disorder in the family history findings could be explained by DPDs' overlap with depression (among probands), we did a follow-up in which we compared 1,405 relatives of participants without DPD with 236 relatives of participants with DPD but no lifetime history of major depression on psychiatric family history variables (see Table 6). By using this approach, we ensured that any increased risk of psychiatric disorders in relatives of the DPD group could not be attributed to the co-aggregation of depressive and other disorders in families of individuals with a lifetime history of comorbid depression.

Relatives of the DPD participants were at increased risk for history of any psychiatric disorder. Specifically, relatives of DPD probands had a significantly increased morbid risk for depressive disorder. Among relatives of DPD probands, trends were found for increased morbid risk for bipolar disorder and ASPD.

Differences Between Participants With Dysthymic Disorder and Depressive PD

To further elucidate the distinction between dysthymic disorder and depressive personality disorder, we compared participants diagnosed with current dysthymic disorder but not DPD (DD only; $n = 39$) to participants diagnosed with DPD but not dysthymic disorder (DPD only; $n = 162$). These two groups did not differ on demographic variables (age, gender, marital status, education, and race). Participants with DPD only were more likely to have a current diagnosis of MDD (57.4%, $n = 93$) than participants with dysthymic disorder (41%, $n = 16$), but this difference did not reach statistical significance ($p < .07$). The DD-only (69%) and DPD-only (72%) groups had similar rates of lifetime major depressive disorder. Compared with the DD-only group, the DPD-

Table 1
Current and Lifetime Axis I Comorbidity in Patients With and Without Depressive Personality Disorder (DPD)

Axis I disorder	Current			Lifetime		
	Without DPD ($n = 702$)	With DPD ($n = 198$)	χ^2	Without DPD ($n = 702$)	With DPD ($n = 198$)	$\chi^2(1)$
% MDD	41.2	57.6	16.81***	61.3	73.7	10.45***
n	289	114		430	146	
% dysthymic disorder	5.6	18.2	32.23****	5.4	18.2	33.37****
n	39	36		38	36	
% BPI	1.9	2.5	0.36	2.8	3.0	0.18
n	13	5		20	6	
% BPII	2.4	4.5	2.48	3.1	6.1	3.64
n	17	9		22	12	
% panic disorder	3.8	3.5	0.04	5.8	6.6	0.14
n	27	7		41	13	
% PDA	11.4	18.2	6.33*	15.8	22.7	5.15*
n	80	36		111	45	
% specific phobia	8.5	21.7	26.43****	10.0	22.2	20.95****
n	60	43		70	44	
% social phobia	20.9	54.0	83.43****	24.6	55.6	68.46****
n	147	107		173	110	
% OCD	5.8	13.5	15.59***	7.5	17.7	17.95***
n	38	27		53	35	
% PTSD	9.3	17.2	9.88**	17.5	26.3	7.53**
n	65	34		123	52	
% GAD	17.7	32.8	21.41****	17.8	33.8	23.65****
n	91	49		92	51	
% any anxiety disorder	49.1	79.3	56.91****	58.4	83.8	43.36****
n	345	157		410	166	
% adjustment disorder	7.1	0.5	12.65***	7.7	0.5	13.90***
n	50	1		54	1	
% drug/alcohol	13.5	12.1	0.27	47.0	51.0	0.99
n	95	24		330	101	
% any psychosis	1.7	2.0	0.01	2.1	2.0	0.01
n	12	4		15	4	
% any somatoform disorder	6.1	10.1	3.75	6.6	10.1	2.86
n	43	20		46	20	
% any eating disorder	1.1	1.5	0.18	3.4	7.6	6.44*
n	8	3		24	15	
% any impulse	4.8	3.0	1.12	10.1	12.1	0.66
n	34	6		71	24	

Note. MDD = major depressive disorder; BPI = bipolar I disorder; BPII = bipolar II disorder; PDA = panic disorder with agoraphobia; OCD = obsessive–compulsive disorder; PTSD = posttraumatic stress disorder; GAD = generalized anxiety disorder; any anxiety disorder = any of the following: panic disorder, panic disorder with agoraphobia, specific phobia, social phobia, obsessive–compulsive disorder, posttraumatic stress disorder, generalized anxiety disorder; drug/alcohol = any drug or alcohol abuse or dependence; any psychosis = any of the following: schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic disorder; any somatoform disorder = any of the following: somatization disorder, undifferentiated somatoform disorder, conversion disorder, pain disorder, hypochondriasis, body dysmorphic disorder; any eating disorder = anorexia or bulimia; any impulse = any of the following: intermittent explosive disorder, pathological gambling, trichotillomania, kleptomania.
* $p < .05$.   ** $p < .01$.   *** $p < .001$.   **** $p < .0001$.

only group had significantly higher lifetime rates of specific phobia, social phobia, OCD, and GAD.

Participants with DPD only were twice as likely to be diagnosed with a personality disorder in addition to DPD, compared with participants with DD only (68%, $n = 110$ vs. 31%), $n = 12$; $\chi^2(1, N = 201) = 18.8$, $p < .0001$. The DPD-only group had higher rates of all personality disorders, but differences were only significant for schizoid, avoidant, dependent, and obsessive–compulsive personality disorders. In terms of psychiatric morbidity and psy-

chosocial functioning, participants with DPD were significantly more impaired with respect to the GAF (49.6 vs. 57.4), $t(199) = 4.47$, $p < .01$. Compared with the DD-only group, the DPD-only group was rated as being more suicidal at the time of the assessment ($p < .01$), and they were more likely to have had a previous suicide attempt (35.8% vs. 15.4%), $\chi^2(1, N = 201) = 6.03$, $p < .05$. Participants with DPD only were also more symptomatic on the HRSD ($p < .001$), CGI ($p < .05$), and number of depressive symptoms ($p < .01$) at the time of the assessment.

Table 2
Axis II Comorbidity in Patients With and Without Depressive Personality Disorder

Personality disorder	Without DPD ($n = 702$)	With DPD ($n = 198$)	$\chi^2(1)$
% paranoid	2.3	16.2	58.95****
n	16	32	
% schizoid	0.3	9.1	55.11****
n	2	18	
% schizotypal	0.3	7.1	40.73****
n	1	14	
% antisocial	3.0	1.5	1.30
n	21	3	
% borderline	6.1	21.7	44.71****
n	42	43	
% histrionic	1.1	4.5	9.67**
n	7	8	
% narcissistic	2.1	7.6	14.80****
n	15	15	
% avoidant	7.0	43.4	160.97****
n	49	86	
% dependent	0.7	8.1	36.80****
n	5	16	
% obsessive–compulsive	6.1	21.2	41.10****
n	43	42	
% self-defeating	1.1	14.1	67.99****
n	8	28	
% negativistic	1.6	14.6	62.21****
n	11	29	
% any Cluster A	2.8	20.7	77.95****
n	20	41	
% any Cluster B	10.1	26.8	36.06****
n	71	53	
% any Cluster C	13.0	54.0	151.86****
n	91	107	
% any PD	22.1	66.2	138.43****
n	155	131	

Note. Any Cluster A = any of the following: paranoid, schizoid, or schizotypal personality disorder (PD); any Cluster B = any of the following: antisocial, borderline, histrionic, or narcissistic PD; any Cluster C = any of the following: avoidant, dependent, or obsessive–compulsive PD.
* $p < .05$.   ** $p < .01$.   *** $p < .001$.   **** $p < .0001$.

Comparisons of 216 relatives of DD-only participants with 839 relatives of DPD-only participants revealed that the two groups of relatives had similar rates of mood disorders, suicide attempts, psychotic disorders, substance abuse disorders, anxiety disorders, and antisocial personality disorder.

Regression Analyses

Multiple regression analyses were conducted to see whether a diagnosis of DPD would add any unique information beyond that provided by the total number of Axis I diagnoses, the total number of Axis II diagnoses, and a diagnosis of DD when predicting measures of psychiatric morbidity. Total number of Axis I diagnoses per patient was chosen as an independent variable in the model because previous research conducted in our clinic showed it to be the best predictor of psychiatric morbidity and functioning (McDermut, Mattia, & Zimmerman, 2001). Consequently, we calculated a similar variable comprising the total number of Axis II symptoms. We conducted 10 regression analyses using the same

four independent variables each time. Diagnosis of DPD was always the last predictor variable entered into the regression model. The dependent variables for the separate regression equations were GAF, level of suicidality, number of hospitalizations, number of suicide attempts, past social functioning, current social functioning, months of missed work, age of depression onset, duration of current episode, and number of depressive episodes. We used the entire sample of participants ($n = 900$) for 7 of 10 analyses. However, only those participants with a diagnosis of current MDD ($n = 403$) were used for analyses of age of depression onset, duration of current depressive episode, and number of episodes, which included only those subjects with a current depressive episode. Dependent variables that were positively skewed were transformed either by square-root transformation (missed work and episode duration) or taking the inverse (suicidality, hospitalizations, suicide attempts, and number of episodes). Of interest is that diagnosis of DPD made a significant contribution over and above the three other independent variables for 6 out of 10 of our dependent variables (level of suicidality, suicide attempts, past social functioning, present social functioning, age of depression onset, and episode duration). Diagnosis of DD was a significant predictor in only one regression model (past social functioning).

Discussion

There is no "gold standard" for establishing the construct validity of a proposed psychiatric diagnosis (Klein, 1999). Beyond establishing the reliability of a diagnosis, the methodology developed by Robins and Guze (1970) is one of the most generally accepted sets of procedures for determining the construct validity of a diagnosis (Klein, 1999; Williams, 1999).

Robins and Guze (1970) have enumerated five phases of construct validation. These phases (which need not take place in any particular order) include the following: (1) clinical description, (2) laboratory studies, (3) delimitation from other disorders, (4) follow-up study, and (5) family study.

The purpose of clinical description (Phase 1) includes the following: identification of cardinal features of a putative diagnosis, core symptoms, associated symptoms, and other associated variables such as demographic features or precipitating factors.

Laboratory study (Phase 2) typically includes gathering data on biological markers for particular disorders.

Delimitation from other disorders (Phase 3) involves specifying exclusion criteria so that the index diagnostic category is homogeneous. Over the years, the purpose of this phase has been elaborated and clarified such that it is incumbent on researchers attempting to validate a psychiatric disorder to demonstrate that the disorder is distinct enough from other disorders so as not to be entirely subsumed by any other disorder.

Follow-up studies (Phase 4) are necessary to show that individuals with the same diagnosis have a similar course to one another.

Finally, the purpose of family studies (Phase 5) is to ascertain whether or not a putative disorder runs in families. The finding of an "increased prevalence of the same disorder among close relatives of the original patients strongly indicates that one is dealing with a valid entity" (p. 984). The family study method of validation has also been expanded over the past 3 decades such that the co-aggregation of near-neighbor disorders in relatives of individ-

Table 3

Psychiatric Morbidity and Impairment in Patients With and Without Depressive Personality Disorder (DPD)

Measure	Without DPD ($n = 702$)	With DPD ($n = 198$)	Statistic
GAF			
M	55.0	50.0	$t(898) = 5.85$****
SD	10.7	10.52	
SADS suicidality rating			
M	0.82	1.65	$t(898) = 7.29$****
SD	1.22	1.44	
Ever hospitalized			
%	21.5	27.8	$\chi^2(1) = 3.44$
n	151	55	
Ever attempted suicide			
%	18.8	33.3	$\chi^2(1) = 19.00$***
n	132	66	
Past social functioning rated poor			
%	3.6	14.6	$\chi^2(1) = 33.65$****
n	23	29	
Current social functioning rated poor			
%	9.0	18.7	$\chi^2(1) = 14.75$****
n	63	37	
Out of work at least 1 month in last 5 years			
%	28.1	39.9	$\chi^2(1) = 10.18$***
n	197	79	

Note. DPD = depressive personality disorder; GAF = Global Assessment of Functioning; SADS = Schedule for Affective Disorders and Schizophrenia.
*** $p < .001$.   **** $p < .0001$.

uals with the putative index disorder is often interpreted as validation of that putative disorder. More precisely, the index disorder and co-aggregating near-neighbor disorders would be considered members of a broader class of disorders that may share similar phenomenology and may have a common pathogenesis.

The present study focused on all phases except laboratory study and obtained results that support both the reliability and the validity of the depressive personality construct. Consistent with previous empirical research on DPD, the reliability of the DPD diagnosis in this study (kappa = .52) was satisfactory (Kaplan & Saccuzzo, 2001). Other studies have typically found even higher interrater reliability, with kappas ranging from .70 (Klein & Shih, 1998) to .82 (Klein & Miller, 1993). We obtained excellent reliability coefficients on dimensional ratings of number of DPD symptoms.

In terms of clinical description (Phase 1 of Robins & Guze's framework), our results show that the proposed symptoms have good internal consistency. In addition, consistent with previous research, our findings showed that, compared with outpatients without DPD, those with DPD had higher levels of depressive symptoms, were more suicidal at assessment, reported more past and present social dysfunction, and had poorer global functioning.

Delimitation from other disorders was made by ascertaining comorbidity rates between DPD and Axis I and Axis II disorders. Our findings support the common finding that although DPD overlaps with dysthymic disorder, major depression, and certain personality disorders (avoidant and borderline PDs in particular), the diagnosis of DPD is not subsumed by any other Axis I or II disorder. Longitudinal course, which falls under the rubric of follow-up studies (Phase 4) in Robins and Guze's framework, was

assessed retrospectively. This indirect assessment of course suggested that those with DPD had higher levels of past psychiatric morbidity and psychosocial impairment and that DPD in the presence of major depression was associated with an exacerbation of the depressive episode as reflected by earlier age of onset, higher rates of chronic depression, and greater symptomatology.

Our family history data were consistent with Klein's findings (Klein, 1990; Klein & Miller, 1993) that DPD shows a pattern of familial co-aggregation with unipolar depression. However, in contrast to Klein's (1990) findings, our data showed that morbid risk for depression among relatives of probands with DPD but not DD was equivalent to the morbid risk for depression among relatives of probands with DD but not DPD. We also obtained some data indicative of familial aggregation between DPD and bipolar disorder, consistent with Klein (1990), but this finding could not be replicated in a more stringent analysis that attempted to minimize the influence of comorbid lifetime major depression in probands.

Despite accumulating evidence supporting the validity of DPD, the validity of DPD has been challenged by several authors (McLean & Woody, 1995; Ryder & Bagby, 1999; Ryder et al., 2001). Ryder and colleagues (Ryder & Bagby, 1999; Ryder et al., 2001) have argued strenuously that an overlap of 50% is "an unacceptable level of comorbidity for two conditions that are also thought to share many theoretical similarities" (Ryder et al., 2001, p. 90). However, this argument is based on an arbitrary standard that lacks a cogent, underlying rationale, as there is no standard (objective or otherwise) regarding what constitutes an acceptable level of comorbidity.

Table 4
Severity of Depression in Depressed Patients Without and With Depressive Personality Disorder (DPD)

Measure	MDD without DPD ($n = 289$)	MDD with DPD ($n = 114$)	Statistic
Recurrent MDD			
%	59.2	56.1	$\chi^2(1) = 0.31$
n	171	64	
Chronic MDD			
%	29.1	59.8	$\chi^2(1) = 32.55$****
n	84	68	
Psychotic MDD			
%	5.5	3.5	$\chi^2(1) = 0.71$
n	16	4	
Melancholic MDD			
%	65.1	55.3	$\chi^2(1) = 3.34$
n	101	51	
CGI			
M	3.03	3.27	$t(401) = 3.22$***
SD	0.69	0.69	
No. of depressive symptoms			
M	5.7	6.2	$t(401) = 3.79$***
SD	1.40	1.31	
Hamilton			
M	16.6	19.5	$t(401) = 4.25$****
SD	5.1	5.5	
Age of depression onset (years)			
M	26.5	20.7	$t(401) = 4.25$****
SD	12.4	11.5	
Duration current episode (weeks)			
M	136.6	394.2	$t(401) = 5.87$****
SD	285.8	591.2	
No. of MDEs			
M	3.39	4.73	$t(393) = 1.52$
SD	6.96	9.98	

Note. MDD = major depressive disorder; CGI = Clinical Global Index; Hamilton = Hamilton Rating Scale for Depression; MDEs = major depression episodes.
*** $p < .001$. **** $p < .0001$.

Several additional counterarguments can be made. First, the levels of comorbidity reported are probably overestimates of the true amount of overlap in the population at large. Klein (1999) has contended that the amount of comorbidity, usually expressed as a proportion, does not take into account the fact that some overlap can result simply from chance. Klein noted that expressing comorbidity, in the form of kappa to correct for chance, yields comorbidity rates that are much more modest (with a median of .25 across several studies). Another reason that the amount of overlap is probably inflated can be explained by Berkson's fallacy (Berkson, 1946). Berkson's fallacy deals with the notion that studies of clinical populations can result in exaggerated estimates of comorbidity because the likelihood of seeking treatment is greater for persons with more than one disorder, compared with persons with only one disorder.

Second, the amount of overlap seen between DPD and DD (i.e., around 50%) is by no means strikingly discrepant from levels of overlap seen between other pairs of Axis II and Axis I disorders. Comorbidity of 50% is also by no means uncommon among pairs of Axis I or pairs of Axis II disorders.

Excluding pairs of disorders hypothesized to have a spectrum relationship (i.e., avoidant personality disorder and social pho-bia; schizotypal personality disorder and schizophrenia; borderline personality and major depression; and antisocial personality disorder and substance use disorders; Widiger & Shea, 1991), there are several pairs of Axis I–Axis II disorder pairs that have comorbidity rates in the neighborhood of 50%. For example, the majority of reports on depressed outpatients suggest that avoidant and dependent personality disorders both occur at rates of about 25% to 65% (Dolen-Sewell, Krueger, & Shea, 2001). The so-called spectrum pairs co-occur at such high rates (in one study, 83% of individuals with borderline personality disorder had comorbid major depression; Zanarini et al., 1998) that a debate has arisen concerning whether or not each of these Axis I–Axis II pairs should be considered manifestations of the same underlying pathology (Gunderson & Phillips, 1991; Widiger & Shea, 1991).

In terms of Axis I disorders that have consistently high rates of comorbidity, perhaps the most obvious example is the high rate of major depression in individuals with dysthymic disorder. Markowitz (1995) reviewed studies that measured Axis I comorbidity in dysthymic disorder and found that, on average, 60% of dysthymics also meet criteria for major depressive disorder. Major depressive disorder has been reported in 50%–90% of patients with panic

Table 5

Morbid Risks for Psychiatric Disorders in First-Degree Relatives of Psychiatric Outpatients With (n = 1,043) and Without (n = 3,725) Depressive Personality Disorder (DPD)

FH–RDC Axis I disorder	Probands without DPD[a]		Probands with DPD[b]		χ^2
	Relatives at risk	Morbid risk (%)	Relatives at risk	Morbid risk (%)	
Depression	2,430.0	18.9	658.5	22.3	39.21****
Bipolar	3,076.0	1.2	842.5	2.3	4.31*
Past suicide attempt	3,725.0	1.7	1043.0	2.3	0.80
Schizophrenia	2,445.0	0.4	660.0	0.2	0.38
Schizoaffective	2,445.0	0.3	660.0	0.0	1.07
Unspecified psychosis	2,158.0	0.3	571.0	0.3	0.01
Alcohol abuse/dependence	2,901.5	12.2	796.0	16.0	5.49*
Drug abuse/dependence	3,046.0	5.4	832.0	5.8	0.05
Panic disorder	2,421.0	2.4	658.0	3.8	2.79
Panic disorder with agoraphobia	2,501.0	1.7	682.0	1.9	0.03
Specific phobia	3,067.5	0.7	848.5	0.7	0.02
Social phobia	3,307.0	0.4	768.0	1.3	0.84
Obsessive–compulsive disorder	2,813.0	0.9	768.0	1.1	0.00
Posttraumatic stress disorder	2,896.0	1.7	794.5	2.1	0.37
Generalized anxiety disorder	2,789.0	2.9	771.5	4.0	1.95
Antisocial personality disorder	2,763.0	1.7	761.5	3.5	8.51**
Any Axis I disorder	3,725.0	25.42	1,043.0	31.0	6.98**

Note. Family history data were missing for 9 non-DPD participants and 3 DPD participants. FH–RDC = Family History—Research Diagnostic Criteria.
* $p < .05$.   ** $p < .01$.   **** $p < .0001$.
[a] $n = 693$.   [b] $n = 195$.

disorder, and 35%–70% of patients with social phobia (Dubovsky & Buzan, 1999).

In terms of comorbidity among Axis II disorders, studies show that comorbidity is the rule rather than the exception (Oldham et al., 1992; Pfohl, Coryell, Zimmerman, & Stangl, 1986; Zimmerman & Coryell, 1989). Any number of pairs of Axis II disorders could be adduced as evidence of rates of comorbidity approaching 50% or greater. One example is that approximately 70% of patients with borderline personality disorder have histrionic personality disorder. In a review of seven studies reporting Axis II comorbidity in schizotypal personality disorder, Siever, Bernstein, and Silverman (1995) noted that the average rate of comorbidity of borderline personality disorder was 58%, and the average for avoidant personality disorder was 53%. Numerous other reviews report comorbidity rates among pairs of Axis II disorders in the area of 50% (Livesley, 1995).

The above line of argument says, in essence, that the degree of overlap between DPD and dysthymia is not uncommon and not unacceptable to framers of the psychiatric nomenclature. Another line of reasoning rests on the notion that, regardless of degree of comorbidity, if a diagnosis provides useful information about individuals with a particular diagnosis—whether it be related to course, prognosis, or treatment response—then the diagnosis has empirically demonstrated validity. The regression analyses in the current study were conducted to address that issue. The regression analyses revealed that the presence versus absence of the DPD diagnosis accounted for unique variance in several measures of severity and impairment above and beyond the variance accounted for by the number of Axis I diagnoses, the number of Axis II diagnoses, and the presence versus absence of dysthymic disorder. In fact, diagnosis of dysthymic disorder accounted for additional

variability in only 1 of 10 regression analyses. Thus, from an empirical–quantitative standpoint, DPD has explanatory value in the statistical prediction of important clinically relevant variables.

The findings of this study are relevant to two more wide-reaching issues discussed below. Assuming DPD is a valid construct, the issue of whether DPD is a personality disorder or a mood disorder remains a matter of considerable debate. An extension of this first issue is whether DPD should be placed on Axis I or Axis II if it were to be included in the *DSM–V*. Regarding DPD's axial placement, the following discussion is very pragmatic and focuses strictly on placement as opposed to addressing the many complex nosological issues germane to the axial system used by the *DSM–IV*. For a more elaborate discussion of nosological issues relevant to the Axis I–Axis II distinction and other relevant nosological debates, readers are referred to Akiskal, Hirschfeld, and Yerevanian (1983), Klein and Riso (1993), Widiger (1989), and Widiger and Shea (1991).

With respect to the issue of mood versus personality disorder, the family history results in the present study (which are consistent with previous research) show a pattern of familial co-aggregation of DPD and unipolar depression, with rates of co-aggregation similar to those seen among dysthymic disorder and unipolar depression (cf. Klein 1990). The co-aggregation is often interpreted as evidence of a spectrum model of depressive disorders in which depressive personality disorder lies at the milder end of the spectrum of severity, dysthymic disorder in the middle, and some forms of chronic or highly recurrent major depressive disorders (Klein, 1999; Klein & Vocisano, 1999) at the most severe end. This model has two important implications. The first implication is that DPD is a least severe variant on the spectrum of chronic depressions. The second implication is that the difference between

Table 6

Morbid Risks for Psychiatric Disorders in First-Degree Relatives of Non-DPD Probands With No Lifetime History of MDD (n = 1,405) to First-Degree Relatives of DPD Probands Without Lifetime MDD (n = 236)

FH–RDC Axis I disorder	Probands without lifetime MDD and without DPD[a]		Probands without lifetime MDD but with DPD[b]		χ^2
	Relatives at risk	Morbid risk (%)	Relatives at risk	Morbid risk (%)	
Depression	903.0	13.7	150.5	21.3	8.38**
Bipolar	1,144.0	1.0	188.5	2.7	2.59†
Past suicide attempt	1,405.0	1.2	236.0	1.7	0.09
Schizophrenia	918.0	0.5	151.0	0.7	0.03
Schizoaffective	918.0	0.1	151.0	0.0	0.16
Unspecified psychosis	809.0	0.5	130.5	1.5	0.62
Alcohol abuse/dependence	1,082.0	8.6	179.5	12.3	2.03
Drug abuse/dependence	1,134.0	4.0	186.5	6.4	1.78
Panic disorder	904.5	2.1	150.5	3.3	0.40
Panic disorder with agoraphobia	932.0	2.0	158.5	1.3	0.03
Specific phobia	1,142.5	0.5	191.0	1.6	1.34
Social phobia	1,230.0	0.3	205.5	1.0	0.56
Obsessive–compulsive disorder	1,043.5	0.7	175.0	1.1	0.04
Posttraumatic stress disorder	1,079.5	1.1	179.0	0.6	0.08
Generalized anxiety disorder	1,036.5	1.9	178.0	2.2	0.08
Antisocial personality disorder	1,027.5	2.1	174.5	4.6	2.70†
Any Axis I disorder	1,405.0	19.6	236.0	29.7	11.79***

Note. Family History data were missing for 3 non-DPD participants. Non-DPD and DPD = without and with depressive personality disorder; MDD = major depressive disorder; FH–RDC = Family History—Research Diagnostic Criteria.
[a] $n = 269$. [b] $n = 52$.
† $p < .07$ (marginally significant).
** $p < .01$.   *** $p < .001$.

depressive personality, dysthymic disorder, and chronic major depression is a matter of degree, as opposed to kind. Researchers and theorists from Kraeplin (1921) to Akiskal (1989) and Klein (1999) have postulated, in substantial part on the basis of family history evidence, that depressive personality is a genetically based temperamental foundation predisposing individuals to dysthymic disorder and major depression.

There are two notable problems with the spectrum model. First, our data are incongruent with the notion that DPD is less severe than dysthymic disorder (DD). In fact, when we compared participants with DPD but not DD with participants with DD but not DPD, the pattern of results clearly showed greater psychiatric morbidity and poorer psychosocial functioning in the DPD-only participants compared with those with DD only. However, the DPD-only group had higher rates of several comorbid anxiety disorders and personality disorders, which could account for more severe levels of impairment. Another problem with a spectrum model, which postulates that a depressive temperament is the predisposing starting point, is the implication that as more severe variants of depression evolve out of the temperamental foundation, the individual will meet criteria for the most severe disorder plus every disorder lower on the spectrum of severity. However, as already noted emphatically, DD and major depression overlap with, but do not always occur in the presence of DPD, an established finding which is inconsistent with a spectrum model.

Although our data strengthen the growing body of literature showing a familial (possibly biogenetic) link between Axis I mood

disorders and DPD, this finding also does not support a spectrum model. In fact, there are numerous instances in the literature of the familial co-aggregation of phenomenologically distinguishable disorders. The most obvious example is the familial co-aggregation of unipolar depression and anxiety disorders (Kendler, Heath, Martin, & Eaves, 1987). In their study, Kendler and colleagues presented data suggesting that depression and anxiety, two phenomenologically distinct classes of disorders, share the same genetic liability but manifest themselves differently as a consequence of environmental influences. Another example is that of antisocial personality disorder and somatization disorder (Cloninger, Reich, & Guze, 1975). Cloninger et al. presented research demonstrating that antisocial personality disorder and somatization disorder have the same genetic liability but manifest themselves differently depending on gender.

Although the family history data in the present report could be interpreted as evidence of a spectrum relationship between DPD and Axis I mood disorders, research examining biological markers of depression may shed light on whether or not DPD is a mild variant of a mood disorder. For example, if individuals with DPD and those with unipolar mood disorders evince similar patterns of sleep EEG abnormalities or cortisol nonsuppression in response to dexamethasone administration, then the spectrum model of DPD and Axis I mood disorders would garner more direct support.

The classification of DPD as a mood disorder or personality disorder is also dependent on the definitions of mood disorders and personality disorders of the existing official psychiatric nomencla-

ture. In considering these definitions, bear in mind that the framers of the *DSM–IV* acknowledge the imperfections of a categorical system of classification: "In *DSM–IV*, there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries" (American Psychiatric Association, 1994, p. xxii). Widiger (1989) has elaborated and clarified the problems of a categorical system specifically as it relates to the distinction between personality and affective disorders. Among other things, Widiger (1989) has noted that the official psychiatric nomenclature gives little guidance regarding what constitutes a mood disorder versus a personality disorder.

However, three aspects of the *DSM–IV*'s definitions of mood and personality disorders are relevant to whether DPD should be considered a mood or personality disorder: (a) the centrality of mood disturbance; (b) the level at which the associated features are conceptualized; and (c) the types of features that constitute the polythetic criteria sets. The *DSM–IV* defines mood disorders simply as "disorders that have a disturbance in mood as the predominant feature" (American Psychiatric Association, 1994, p. 317). By this very definition, despite the connotations of its name, DPD does not fall under the rubric of a mood disorder as defined by the *DSM–IV*. Although the criteria set does contain one mood-related item, it is neither necessary nor sufficient to make a diagnosis of DPD. In our sample, about one third of participants who meet criteria for DPD did not endorse the mood-related criterion. Of those who did endorse the mood-related item, 67% endorsed six or seven criteria and therefore would have met criteria for DPD without having endorsed the mood item.[1] In contrast, by definition, both dysthymic disorder and major depression require the presence of mood disturbance.

Second, features of the Axis I depressive disorders are conceptualized at the level of symptoms, whereas DPD is conceptualized at the level of enduring traits (Klein, 1999). This distinction is described in the *DSM–IV*'s definition of what constitutes personality: "Personality traits are enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts" (American Psychiatric Association, 1994, p. 630). In assessing personality traits in the current study, the SIDP repeatedly redirects interviewees away from their current symptoms to answer questions about their characteristic ways of thinking, feeling, and behaving when they are their "usual selves."

Third, despite some overlap, the symptoms of unipolar depressive disorders differ from the types of traits associated with DPD. Axis I unipolar depressive disorders (DD and MDD) consist of symptoms related to emotion, cognition, self-concept, and somatic functioning. Depressive personality disorder, on the other hand, is characterized primarily by traits reflecting "excessive negative, pessimistic beliefs about oneself and other people" (Hirschfeld & Holzer, 1994) and, in some cases, a primarily gloomy or cheerless mood as well.

Numerous proposals about how the psychiatric nomenclature should handle DPD have been proffered and debated extensively. These recommendations have included eliminating dysthymic disorder, reconstituting the symptom criteria for DD and/or DPD, including DPD as a subset of DD, and converting to a dimensional model of mood disorders or a dimensional model of all personality disorders (Klein, 1999; Phillips & Gunderson, 1999; Ryder & Bagby, 1999; Widiger, 1999). In the context of the *DSM–IV*

system, in which DPD would be defined as a personality disorder, another proposal that should be given serious consideration, based on its parsimony and pragmatism, is simply to add DPD as another personality disorder on Axis II.

Several limitations suggest cautious interpretation of our findings. First, our sample is not a random sample of psychiatric outpatients and thus cannot be assumed to generalize to all psychiatric outpatients. Second, the same rater conducted the Axis I and II assessments and the FH–RDC. However, the biases due to nonblind assessments must be counterbalanced against the potentially more valid ratings made by diagnosticians who have a more complete overview of the patients' histories. Third, family psychiatric histories were obtained using the family history method rather than a family study method. The family history method tends to underdiagnose psychopathology in relatives (Andreasen, Rice, Endicott, Reich, & Coryell, 1994), although it is unclear how this may contribute to the significant differences between groups. A related issue is how to interpret the higher rate of ASPD in relatives of DPD probands. The main methodological impediment is that ASPD is the only personality disorder assessed by the FH–RDC; therefore, the presence of DPD may be associated with increased rates of all personality disorders in relatives as opposed to being associated only with high rates of ASPD in particular. Fourth, the degree to which current mood state influenced ratings of DPD is uncertain. Mood state has been shown to result in inflated reports of negative personality traits (Hirschfeld et al., 1983). However, given that instructional set can dampen this effect (Hirschfeld et al., 1983), we believe that our use of the SIDP may have minimized the overreporting of negative personality traits.

Despite these limitations, the results of the current study add to a growing body of literature supporting the construct validity of DPD. Depressive personality disorder fills a gap in the psychiatric nomenclature by identifying many individuals with depressive traits for whom a diagnosis of dysthymic disorder or major depressive disorder is inappropriate. In lieu of more convincing neurobiological evidence, and in accord with the *DSM–IV*'s conceptualizations of mood and personality disorders, DPD should be classified as a personality disorder. Finally, assuming that the *DSM–V* retains its current definitions of mood and personality disorders, we submit that if DPD is adopted as an official diagnosis, it should be placed on Axis II.

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[1] This is relevant information, because one way to reduce comorbidity between diagnoses is to delete overlapping criteria (Widiger & Shea, 1991). As the diagnosis of DD is primarily determined by the presence of chronic depressed mood (Klein, 1999), eliminating the mood-related item from the DPD criteria set might be one approach. In the current sample, deleting the mood item would have reduced the number of participants diagnosed with DPD by 22%.

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