

Depressed Mood and Major Depressive Episodes: Differential Responsiveness to Psychosocial Experiences*

H. O. F. Veiel

Central Institute of Mental Health, J 5, P.B. 12 21 20, D-6800 Mannheim 1, Federal Republic of Germany

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Summary. In a sample of 64 remitted and discharged depressed in-patients the structure and quality of their social environment was examined in relation to the subsequent development of depressive symptoms. It is shown that mood-related symptoms were influenced by psychosocial factors, whereas vegetative and non-specific symptoms were not. Since the latter are rather more heavily weighted in the DSM-III criteria for Major Depressive Episode than in typical symptom sum scores, psychosocial variables did not predict DSM-III-defined clinical relapses. Possible implications for conceptualizing depressive disorders are discussed.

Key words: Depression – Social network – Social support – Relapse

Introduction

The important role of a supportive social environment in preventing the onset of unipolar depression now seems widely accepted (e.g. Alloway and Bebbington 1987; Brown and Harris 1978; Brown et al. 1986). Social support also seems to have a beneficial effect on the course of depressive disorders (Billings and Moos 1985), while negative social relationships increase the probability of relapses after remission from clinical depression (Hooley et al. 1986). However, not all positive, supportive relationships are beneficial. In a series of recent studies of depressed psychiatric in-patients, we (Veiel and Kühner 1990) showed that the quality and structure of social support networks encountered after remission and discharge from hospital affected the further course of the disorder in quite specific ways: a patient's general satisfaction with available support had a beneficial effect, whereas a large kin support network was associated with

a subsequent increase in depressive symptoms. (The size of the support network of friends and acquaintances on the other hand, although cross-sectionally correlated with indicators of depression, had no appreciable prospective effects.) Such a separation of effects was made possible by a differentiated assessment of supportive environments. The results, however, as well as those of Billings and Moos (1985), were obtained with continuous questionnaire scores as measures of depression, and this study examines whether the same relationships hold when diagnosable cases of depression are to be predicted.

Methods

Over-all-Design. This study is part of an ongoing major prospective research program on psychosocial predictors of the course of clinical depression after discharge from psychiatric in-patient treatment. Patients are interviewed 1 (T_1), 7 (T_2), and 25 months (T_3), after discharge, when depressive symptomatology, potential and actual social support factors, psychosocial needs, stressful life events, habitual coping tendencies and actual coping behaviours are assessed. The results reported in this paper are based on a subsample of 64 fully remitted patients, and they concern changes between T_1 and T_2 .

Instruments and Variables. At both T_1 and at T_2 the following instruments (among others) were used: the Inventory to Diagnose Depression (IDD; Zimmermann and Coryell 1987), the Present State Examination (PSE-9; Wing et al. 1982) (questions added to allow DSM-III diagnoses of a major depressive episode (MDE)), and the Mannheim Interview on Social Support (MISS; Veiel 1987, 1990). The IDD is a self-report questionnaire which also covers symptoms relevant for a DSM-III diagnosis of MDE and from which a symptom sum score was derived. Based on the expanded PSE two dichotomous variables were defined: "caseness" at T_1 (MDE/no MDE), which was used as a selection criterion (see below), and "relapse/no relapse", which was defined by the reappearance of depressive symptoms meeting MDE criteria at any time after T_1 (up to and including T_2). The MISS is a structured interview which maps a person's supportive environment. Three global parameters were derived from the MISS: (a) the patient's general satisfaction with available social support, averaged over 12

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prototypical support functions; (b) the size of the kin support network (excluding spouses but including in-laws); and (c) the size of the support network of friends and acquaintances. The variables were named "Satisfaction", "Kin Network Size", and "Non-kin Network Size", respectively.

Sample Selection. The sample was based on a cohort of 138 consecutively admitted 18 to 60-year-old patients of the Psychiatric Clinic of the Central Institute of Mental Health in Mannheim, FRG, who met the criteria for a DSM-III diagnosis of MDE or who scored 25 or higher on the IDD (corresponding to one standard deviation above the mean of normal samples). Patients with bipolar disorder, current or past schizophrenic spectrum symptoms, primary substance abuse, or organic brain damage were excluded. One hundred and eleven patients were interviewed at T₁, and 99 were followed-up at T₂. Of these, 35 patients who were unremitted at T₁ were excluded. Thus the final sample consisted of 64 discharged inpatients who had remitted from unipolar depression.

Analyses and Results

Linear regression analyses with the IDD-derived, continuous symptom sum score at T₂ as the dependent variable, and logistic regression analyses with PSE-derived relapse as the dichotomous criterion were performed in parallel. In both analyses, the following variables which had shown substantial predictive power on their own were forced into the prediction equation: the patients' sex, the IDD sum score at T₁, and the sum of all PSE items at T₁ on which the MDE diagnosis was based. All three support variables were then entered into the regression equations and, by a backward-stepping procedure, subsequently removed if their regression coefficient had an associated chance probability of $P \geq 0.10$. Thus, the power of the social support variables to predict T₂ depression was evaluated *after* the effects of the patients' sex and of the T₁ symptom level had been removed. Table 1 shows the results.

In order to make the results of the two analyses comparable, the net effects of the predictor variables are represented as partial correlations with the respective criteria. For the regression analyses, straightforward partial product-moment correlations are used. For the logistic regression, the chi-square associated with each predictor variable has been transformed into a partial phi correlation coefficient. The social support variables significantly predicted general depressive symptom levels at T₂ (first column of Table 1). As expected, satisfaction was negatively, and the size of the kin network was positively correlated with the T₂ symptom score. Clinical relapses, however, were *not* predicted by the support variables. This discrepancy in predictive power could have been caused by any of the following four factors: (a) the difference between self-reported (IDD) and interviewer-rated (PSE) symptomatology; (b) the emphasis on severe levels of depression in the relapse criterion (MDE), as opposed to the broad range of symptom levels reflected in the sum score; (c) the difference between a symptom count (IDD sum score) and a non-summative diagnostic algorithm (PSE-MDE); (d) the emphasis on different *kinds* of symptoms.

In order to assess (a), the linear regression analysis was repeated with a sum score of the PSE depression

Table 1. Social support variables as predictors of symptom sum scores and of clinical relapses

Predictors	Outcome	
	Symptom sum score at T ₂ (IDD) Partial <i>r</i> (lin. regression)	Relapse (MDE) after T ₁ (PSE) Partial Phi (log. regression)
IDD sum score T ₁	0.11	0.01
PSE sum score T ₁	0.34	0.24
Sex (m = 1; f = 2)	0.19	0.33
Kin network size (T ₁)	0.33**	(0.08)
Nonkin network size (T ₁)	(-0.06)	(-0.18)
Satisfaction (T ₁)	-0.32*	(-0.01)

* $P < 0.05$; ** $P < 0.01$

Figures represent partial correlations between the respective predictors and outcomes after all other predictors with an associated $P < 0.10$ (backward stepping) are accounted for. Figures in brackets show the net effects of the other predictors when entered in an additional step.

IDD: Inventory to Diagnose Depression

PSE: Present State Examination

Table 2. Prediction of symptom sum scores based on interviews and of relapses defined by a questionnaire cut-off

Predictors	Outcome	
	PSE-based symptom sum score at T ₂ Partial <i>r</i> (lin. regression)	Relapse defined by IDD sum scores > 25 Partial Phi (log. regression)
IDD sum score T ₁	0.06	0.25
PSE sum score T ₁	0.31	0.21
Sex (m = 1; f = 2)	0.20	0.40
Kin network size (T ₁)	0.31*	0.31*
Nonkin network size (T ₁)	(-0.15)	(0.03)
(Satisfaction (T ₁))	-0.21	-0.33**

* $P < 0.05$; ** $P < 0.01$

Figures represent partial correlations between the respective predictors and outcomes after all other predictors with an associated $P < 0.10$ (backward stepping) are accounted for. Figures in brackets show the net effects of the other predictors when entered in an additional step.

IDD: Inventory to Diagnose Depression

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items (interviewer-based) at T₂ as the dependent variable. To assess (b), the IDD sum score was dichotomized at 25 (the selection criterion at T₁). In this way, relapse was defined by means of a high symptom total instead of the diagnostic algorithm of DSM-III. The logistic regression was repeated with so-defined relapse as the outcome criterion. Table 2 shows the results of these two analyses.

In both analyses, the effect of the social support variables was present to the same extent as with the IDD sum score. This means that the effect of the social sup-

Table 3. Predicting depressed mood vs. vegetative and non-specific symptoms

Predictors	Outcome	
	"Mood" symptom score at T2 (IDD) Partial <i>r</i> (lin. regression)	"Vegetative" symptom score at T2 (IDD) Partial <i>r</i> (lin. regression)
IDD sum score T1	0.03 (−0.20)	0.23 (0.31)
PSE sum score T1	0.38 (0.33)	0.20 (−0.11)
Sex (m = 1; f = 2)	0.14 (−0.10)	0.29 (0.26)
Kin network size (T1)	0.33** (0.26*)	0.21 (−0.04)
Satisfaction (T1)	−0.34** (−0.29*)	−0.18 (0.09)

* $P < 0.05$; ** $P < 0.01$

Figures represent partial correlations between the respective predictors and outcomes after all other predictors with an associated $P < 0.10$ (backward stepping) are accounted for. Figures in brackets show the effects of the predictors when the "purified" mood and vegetative scores are used as dependent variables, i.e. when the respective other variable is also forced into the prediction equation (see text).

IDD: Inventory to Diagnose Depression

PSE: Present State Examination

"Mood score": Sum of all mood-related items of the IDD

"Vegetative score": Sum of the remaining IDD items (vegetative and non-specific)

port variables on symptomatic changes was not restricted to low-to-medium symptom levels, nor was it specific to self-reported symptoms. In other words, it was not due to scaling or assessment artefacts. In order to decide between the remaining two possibilities (diagnostic algorithm vs. symptom count, and emphasis on different kinds of symptoms), the IDD sum score was split into a sum score of mood-related items (12) and a sum score of the remaining items (10), comprising mainly vegetative (appetite, sleep etc.) and non-specific (e.g. irritability) symptoms. The split was made roughly in accordance with the distinctions obtained by Goldberg et al. (1987). Table 3 shows the results when the "mood" sum score and the "vegetative" sum score were predicted separately. Compared with the total symptom score, the support variables predicted the "mood" score even better, but the correlations were lower for the "vegetative" score. In a second set of analyses, the "vegetative" and "mood" scores at T₂ were predicted after removing their common variance, i.e. by forcing the "vegetative" score at T₂ into the prediction equation for the "mood" score at T₂, and vice versa. In Table 3, the figures in brackets represent the effects of the support variables on the "purified" scores; while significant for the "mood" score, they are practically zero for the "vegetative" score. (It is also interesting to note that the associations of the other predictor variables – T₁ symptom scores and sex – with mood and with vegetative symptoms had opposite signs.) Thus the effect shown in the left column of Table 1 must be attributed to the mood component of the IDD sum score, and the failure to predict clinical relapses is because mood-related symptoms are weighted much more heavily in symptom sum scores than in MDE diagnoses.

Discussion

Among the various facets of depression the mood component seems to be primarily responsive to psychosocial influences. This finding is consistent with the view held by many clinicians that clinical or "major" depression is qualitatively as well as quantitatively different from "normal" variations of mood. Two recent prospective studies also found different predictors for interview-based clinical diagnoses of depression and for symptom scores (Lewinsohn et al. 1988; O'Hara et al. 1984). Neither study, however, has examined the details of this difference. The authors – as indeed most clinical authors – have apparently assumed a kind of hierarchical relationship between depressed mood and "real", severe, or major depression, where the former appears as a precursor as well as a symptom of the latter. Lewinsohn et al. (1988), for example, postulated that negative affect mediates the effect of cognitive vulnerability factors on major depression.

Such a hierarchical organization seems only plausible, however, if one accepts the notion of a homogeneous entity "depression", which is reflected in a clinical diagnosis. Given the symptomatic and nosological diversity of depressive disorders, however, a non-hierarchical conceptualization is perhaps more adequate. The complex psychological and physiological changes associated with depressive disorders may be organized in clusters reflecting the activity of distinct, relatively autonomous, and only moderately correlated psycho-physiological systems, each with its specific set of associated triggers and vulnerability factors. One such subsystem, whose activation is experienced by the individual himself or herself and by his or her environment as depressed mood and anhedonia, may be particularly responsive to (negative and positive) social experiences. Other sub-systems are conceivable, in particular one primarily responsive to stress experiences and involving arousal symptoms associated with changes in the adrenal hormonal system (such as tension, irritability, sleeplessness, and, eventually, exhaustion and inactivity – cf. Selye 1974). While the notion of autonomous subsystems with possibly different degrees of specificity for depression is intriguing and carries some potential for clarifying such issues as "double depression" (Keller and Shapiro 1982) and the overlap of anxiety and depressive disorders, it is still speculative. Research with a variety of other predictors (stress, physiological parameters) is needed, and partly under way, to obtain a clearer view.

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