

# Psychosocial Functioning and Depression: Distinguishing Among Antecedents, Concomitants, and Consequences

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In this article we attempt to distinguish empirically between psychosocial variables that are concomitants of depression, and variables that may serve as antecedents or sequelae of this disorder. We review studies that investigated the relationship between depression and any of six psychosocial variables after controlling for the effects of concurrent depression. The six variables examined are attributional style, dysfunctional attitudes, personality, social support, marital distress, and coping style. The review suggests that whereas there is little evidence in adults of a cognitive vulnerability to clinical depression, disturbances in interpersonal functioning may be antecedents or sequelae of this disorder. Specifically, marital distress and low social integration appear to be involved in the etiology of depression, and introversion and interpersonal dependency are identified as enduring abnormalities in the functioning of remitted depressives. We attempt to integrate what is known about the relationships among these latter variables, suggest ways in which they may influence the development of depression, and outline specific issues to be addressed in future research.

The identification of psychosocial factors that may cause depression has proven to be an arduous task. The difficulty of demonstrating causal relationships in naturalistic research has been compounded by an overreliance on cross-sectional methodology. Cross-sectional research has been successful in demonstrating differences between depressed and nondepressed individuals; that is, it has identified abnormalities in the functioning of depressed individuals that are present during depressive episodes. Many of these abnormalities, such as dysfunctional cognitions, distressed relationships, anaclitic personality types, and deficits in social behaviors, have been implicated in the etiology of depression by theorists of various orientations (e.g., Abramson, Seligman, & Teasdale, 1978; Beck, 1976; Brown & Harris, 1978; Hirschfeld, Klerman, Chodoff, Korchin, & Barrett, 1976; Lewinsohn, 1974). However, some of these problems in functioning may be symptoms, or concomitants, of depression that appear with the onset of a depressive episode and disappear with remission. Although they do co-occur with depression, such factors cannot be classified as causal because they do not precede the onset of symptoms.

It is clear, then, that prospective research is most appropriate for identifying variables that play an etiological role in depres-

sion. Unfortunately, the demonstration of a psychosocial variable's temporal antecedence to the initial onset of depression has proven extremely difficult (cf. Depue & Monroe, 1986). To obtain a truly premorbid sample, careful screening of the lifetime psychiatric history of each subject is necessary. Even without this rigorous subject selection procedure, the strategy of following an initially nondepressed group over time has seldom been adopted. Large samples and lengthy time lags are usually required to increase the probability that a sufficient number of subjects will become depressed during the course of the study. The prohibitive cost of such research likely accounts for its scarcity.

Although they are not as useful for making causal inferences as are studies of depressives' premorbid functioning, alternative designs do exist that provide valuable information concerning a variable's possible causal relationship with depression. One such strategy is the two-wave panel design, in which a psychosocial variable is used at one time to predict subjects' subsequent levels of depression. We discuss the nature and limitations of this design in greater detail later in this article and simply note here that research using this design provides information about the influence of a predictor variable on a *change* in depressive symptoms. The failure in most studies to evaluate the interaction between initial symptoms and the predictor variable, however, confounds attempts to link the predictor with the actual *onset* of depression. For example, a measure of cognitions may be a significant predictor of subsequent level of depression, but because subjects differ in their initial symptom levels, it is not clear whether cognitions are predicting the onset, exacerbation, or remission of depression in a group of subjects (cf. Hammen, Mayol, deMayo, & Marks, 1986). Nevertheless, positive results would suggest that an aspect of psychosocial functioning has an effect on the development or course of depressive symptoms and, as such, may have etiological significance.

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The two prospective designs discussed (i.e., the premorbid case-control and the two-wave panel design) are typically used to assess the main effect of a psychosocial variable on future depression. Three important theories of the etiology of depression, however, have suggested that a diathesis-stress model is a more appropriate heuristic framework for conceptualizing the development of depression than is a main effect model. Specifically, Beck's (1976) cognitive theory, the reformulated learned helplessness theory (Abramson et al., 1978), and psychodynamic theory (Hirschfeld et al., 1976) hypothesize that dysfunctional self-schemata, a self-deprecating attributional style, and anaclitic personality traits, respectively, are stable aspects of personal functioning that predispose individuals to become depressed under certain conditions. One testable hypothesis generated by each of these theories is that vulnerable individuals should score higher than nonvulnerable people on measures of the predisposing variable during both morbid and intermorbid periods. Remitted depressives constitute a group of individuals who have been vulnerable to depression in the past and who are statistically at increased risk for future depressive episodes (Beck, Rush, Shaw, & Emery, 1979; Stern & Mendels, 1980). Thus, within certain restrictions to be discussed later, the results of studies comparing remitted depressives and normal controls on these predisposing variables are appropriate for evaluating hypotheses derived from these etiological theories.

In sum, there is at present a paucity of research that clearly establishes the temporal antecedence of certain psychosocial abnormalities to the onset of depression. Nevertheless, by integrating the evidence provided by primarily longitudinal research of different types and, where appropriate, by using these data to evaluate specific hypotheses made by causal theories, it may be possible to begin now to clarify the relationships of select psychosocial variables with depression. The purpose of this article is to attempt to differentiate three classes of abnormalities that distinguish depressed from nondepressed adults: (a) those that precede and may play a causal role in the onset of depression (i.e., antecedents), (b) those that are observable only during a depressive episode (i.e., concomitants), and (c) those that persist beyond symptomatic recovery (i.e., consequences). In general, we adopt the position that variables that are observable only during a depressive episode are less likely to play a causal role in this disorder than are variables that either precede the disorder or persist following recovery. Nevertheless, we remain cognizant of the possibility of more complex time-lag causal patterns, in which some dysfunction that, along with a number of depressive symptoms, is caused by an environmental stressor might itself serve to activate or exacerbate still other symptoms of depression. Thus, although a variable might be observed only during a depressive episode (and thereby be relegated to concomitant status), it could in fact have played a causal role in activating other symptoms of depression. We will return to a more specific discussion of this issue in a later section.

Classification of variables as antecedents, concomitants, or consequences is useful for a number of reasons. First, expensive premorbid research could focus in the future on the more promising etiological variables, as opposed to those that appear to be simply concomitants or symptoms of depression. Second, the fact that remitted depressives are at increased risk for future

depression suggests that the enduring consequences of the disorder may also have etiological significance with respect to multiple episodes. Furthermore, the sequelae of depression may represent serious impairments in the functioning of recovered patients, impairments that may require specific interventions beyond those offered by relatively circumscribed symptom reduction approaches. Finally, the demonstration of major inconsistencies between empirical data and etiological theories regarding the stability and predictive power of predisposing variables would suggest the need to alter these theories accordingly. In short, an examination of our current ability to distinguish among the antecedents, concomitants, and consequences of depression may have implications for future theory, research, and treatment of this disorder.

This classification may be accomplished by comparing the results of cross-sectional research involving symptomatic probands with those of primarily longitudinal studies that meet the specific design criteria outlined in the next section. Despite repeated statements concerning the advantages of longitudinal studies (cf. Depue & Monroe, 1986; Monroe, 1983; Monroe & Steiner, 1986; Tennant, 1983), few systematic attempts have been made to organize this body of research as it pertains to depression. Furthermore, although recent reviews have discussed the relationships of individual psychosocial variables with depression or related psychological disorders (e.g., Akiskal, Hirschfeld, & Yerevanian, 1983; Cohen & Wills, 1985; Coyne, Kahn, & Gotlib, 1987; Gotlib & Colby, 1987; Sweeney, Anderson, & Bailey, 1986), much less consideration has been given to how these variables might interrelate and to how their interactions might affect the development or maintenance of depression. A second purpose of this article, therefore, is to integrate research on different aspects of the functioning of depressive individuals.

In this article we review research examining the relationship between depression and any of six specific psychosocial constructs: attributional style, dysfunctional attitudes, personality, social support, marital adjustment, and coping style. We chose to examine these variables for a number of reasons. First, there is relatively consistent evidence that while depressed individuals are symptomatic, they differ from nondepressed persons on each of these variables. Second, many of these constructs have been postulated to function as etiological factors in depression. However, results from studies that are not appropriate for evaluating hypotheses concerning unidirectional causality are often marshalled in support of these etiological formulations (cf. Coyne & Gotlib, 1983; Depue & Monroe, 1986; Monroe & Steiner, 1986). Research that is adequate for testing direct causal hypotheses about depression is scarce. The differentiation of symptoms or concomitants from more stable factors associated with depression, however, would provide preliminary evidence of the etiological nature of these theoretically important variables. Finally, there is now sufficient research involving these six variables to make the proposed comparison between cross-sectional studies and primarily longitudinal investigations that meet certain design criteria.<sup>1</sup> Because not all six areas

<sup>1</sup> Although level of social skill has been implicated in the etiology of depression (e.g., Lewinsohn, 1974), we did not include this variable in

of research have received equal attention, conclusions in some cases must be considered more tentative than conclusions in others.

The complexity of depressive phenomena, the difficulty of making causal inferences based on the results of naturalistic research, and the plethora of factors hypothesized to cause depression all militate against drawing firm conclusions concerning the etiological status of these variables. For these reasons, we have emphasized the role of theory in interpreting and integrating the results of the research selected for review. Although a lack of support for a given etiological theory would not necessarily eliminate a putative causal agent from the list of potential etiological variables, it would suggest the need for theoretical revisions that could explain the existing evidence. In the review sections that follow, we evaluate the fit of theory with data and, where necessary, consider explanations for a poor fit in our conclusions.

In the following section, we outline the design criteria we used to select the studies for this review. We then review the research on each of the six variables, beginning in each case with a brief presentation of relevant theory and an overview of the results of cross-sectional studies of that particular variable. We outline methodological issues relevant to the interpretation of the results and draw conclusions concerning the likely functional role played by each variable. Finally, we present a preliminary integration of the relationships among these variables, suggest ways in which they may influence the development of depression, and offer possible directions for future research.

### Selection of Research

We selected for inclusion those studies that examined the relationship between depression and at least one of the variables of interest after controlling for the effects of concurrent symptoms. This control could be accomplished in a number of ways. The most common method involves comparing depressive probands with normal controls both during the depressive episode and following recovery from the depression. In this way, the stability of the dysfunction apparent during the episode can be investigated, and any residual deficits exhibited by the remitted patients can be assessed. Similarly, we also included cross-sectional studies comparing groups of currently depressed, remitted depressive, and nondepressed subjects. Studies utilizing this design address the issue of whether specific differences between depressed and nondepressed subjects endure beyond symptomatic recovery.

We mentioned earlier that certain limitations on the kinds of inferences that can be made from research on depressives' postmorbid functioning must be recognized. Some variables, such as dysfunctional cognitive style or activity, have been identified by theories of depression as being stable abnormalities in probands' functioning that predispose to this disorder. These abnormalities, then, are predicted, at least implicitly, to be evident during intermorbid or postmorbid periods. Consequently, research evaluating postmorbid functioning would be expected

to demonstrate relevant differences between remitted depressives and nondepressed control subjects. The absence of such differences would call into question the goodness of fit of research to theory with respect to the hypothesized etiological status of the variable, unless an intervention specifically designed to alter the predisposing trait, such as cognitive therapy, had been utilized in treating the subjects' depression. In contrast, other abnormalities, such as marital distress, have not been hypothesized to be trait-like aspects of functioning among depressive probands. Thus, although results of research examining subjects' postmorbid functioning with respect to these variables would be relevant to questions concerning the sequelae of depression, inferences concerning the etiological significance of these variables could not be drawn. In the review section of this article, we present the predictions of individual theories and evaluate empirical results with respect to these predictions. In this way we highlight the validity of different aspects of the causal theories of depression and draw attention to the different implications that research of this kind may have across different areas.

The second most common method of controlling for concurrent symptoms involves a variation of the two-wave panel design in which the effects of individual differences in initial depressive symptoms are controlled statistically. The data in these studies are analyzed by predicting depression ( $Y$ ) at Time 2 ( $T_2$ ) from a variable ( $X$ ) measured earlier, at Time 1 ( $T_1$ ), after partialling out  $T_1$  symptoms ( $Z$ ) of depression. If the residualized psychosocial variable significantly predicts subsequent depression, it is suggested that a change in the severity of depressive symptoms is accounted for by this previous level of functioning. This design appears to meet the three criteria considered necessary for making causal inferences; that is,  $X$  and  $Y$  covary,  $X$  is a temporal antecedent of  $Y$ , and the relationship between  $X$  and  $Y$  is not accounted for by  $Z$  (i.e., third variable causality). As we noted earlier, however, the major limitation of this design is that the onset, increase, maintenance, and remission of symptoms are not differentiated; that is, the data analysis does not provide differential information about the relationship between the predictor variable and depression for subjects with different levels of initial symptoms.

In some of the studies we will review, additional information was provided that increases the interpretability of the results obtained using this design. For example, if it is assumed that there is not a significant interaction between  $T_1$  depression and the predictor variable, then evidence of a significant increase or decrease in the mean level of symptoms from  $T_1$  to  $T_2$  might suggest that the predictor variable influences the development of, or recovery from, depression, respectively (e.g., O'Hara, Rehm, & Campbell, 1982). Alternatively, separate analyses involving the data for subsamples defined on the basis of initial symptom level may suggest the presence of the aforementioned interaction (e.g., Monroe, Bromet, Connell, & Steiner, 1986). Without a direct evaluation of the interaction, however, such interpretations must remain tentative. Unfortunately, we found no research that did assess this interaction.<sup>2</sup>

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the present review because we could find no research that met the methodological criteria for inclusion.

<sup>2</sup> There is a strategy for data analysis that might be used to examine whether the relationship between a predictor variable and depression is moderated by subjects' initial level of depression across the entire range

One final question concerning the two-wave panel design is whether it is appropriate for evaluating the predictions of the diathesis-stress models of depression (Abramson et al., 1978; Beck, 1976; Hirschfeld et al., 1976). These models suggest that depression is the result of an interaction between a relatively stable vulnerability factor and psychosocial stress. Tests of the full models, therefore, would include an assessment of the effect of this interaction on subsequent depression. Because psychodynamic theory has not been investigated in research using a two-wave panel design, this issue is relevant only for research assessing depressogenic cognitions. We found only two studies that examined the interaction of cognitions and psychosocial stress in a variation of the two-wave panel design (P. A. Barnett & Gotlib, in press; Metalsky, Halberstadt, & Abramson, 1987). Nevertheless, there are two reasons why cognitions alone might be expected to predict future depression. First, a significant main effect for cognitions has been found in cross-sectional research involving both subjects with mild dysphoria and subjects with more severe affective disturbance (e.g., Gotlib, 1984; Sweeney, et al., 1986; A. N. Weissman & Beck, 1978). Second, as Coyne and Gotlib (1986) pointed out, given a reasonably large sample, a main effect for cognitions would be masked by the interaction of cognitions and stress only if cognitions and stress were highly negatively correlated. In the absence of this improbable correlation, the two-wave panel design in which initial symptoms are statistically controlled does appear to be a useful empirical tool for investigating certain aspects of the diathesis-stress models of depression.

A third method of controlling concurrent symptoms also involves following initially nondepressed subjects over time. Those subjects who subsequently become depressed are compared with those who do not on relevant aspects of functioning measured at T1. This approach involves a loss of information about individual differences in initial symptoms: All subjects who score below a certain cutoff on a self-report inventory of symptoms or who do not meet diagnostic criteria for depression, regardless of their range of scores on a symptom intensity inventory, are considered to be symptomatically equivalent at T1 (i.e., nondepressed). Offsetting this disadvantage, however, is an increase in the specificity of inferences that may be drawn from the results of this design. The criterion in this design is group membership (i.e., depressed or nondepressed), so that only an increase in symptoms is predicted, as opposed to the more general criterion of a change in symptoms. This "premorbid" design comes closest to an appropriate method for studying the etiology of depression. As mentioned earlier, few such studies have been conducted: Only four are reviewed in this arti-

cle (Hammen, Marks, deMayo, & Mayol, 1985; Lewinsohn, Steinmetz, Larson, & Franklin, 1981; O'Hara, 1986; Phifer & Murrell, 1986).

Two additional procedures for separating symptomatic or concomitant dysfunction from more enduring disturbances associated with depression have been criticized as unsound. First, retrospective interviews have been used in an attempt to establish the temporal antecedence of social conditions to the onset of depression (e.g., Brown & Harris, 1978). Retrospective assessment, however, is not only inappropriate for measuring certain variables, such as cognitions, but, moreover, is vulnerable to the effects of selective recall. As Tennant (1983) argued, respondents may be biased in their accounts of previous adjustment, particularly if they are currently depressed (see Gotlib, Mount, Cordy, & Wiffen, 1988, for a more detailed discussion of this issue). A second technique considered at one time to be appropriate for evaluating causal hypotheses involves the use of cross-lagged panel correlations. Rogosa (1980), however, demonstrated that the null hypothesis of spuriousness either may be rejected under conditions of equal reciprocal or third variable causality, or conversely, may not be rejected when, in fact, causal effects are not equal. These inconsistencies prompted Rogosa to reject the technique, even when used with additionally restrictive conditions, and to call for it to be "set aside as a dead end" (p. 257). Therefore we did not include studies using either of these approaches in the present review.

We applied two final restrictions in selecting research for this review. First, we selected only those studies in which symptoms were evaluated with a measure specifically designed to assess depression, as opposed to nonspecific disorder. Much good longitudinal research has been conducted using measures of general psychological distress (e.g., Henderson, Byrne, & Duncan-Jones, 1981). Although the distinction between this construct and depression in nonclinical samples is not clear (Gotlib, 1984), we attempted to sharpen the focus of this review by discussing only research on depression. As a related point, we also did not include studies using self-report measures of the severity of depressive symptoms which were modified for use in retrospective studies (e.g., Zuroff, 1981). The use of these inventories to diagnose cases of current depression has been questioned (Depue & Monroe, 1978; Hammen, 1980), and their validity as retrospective diagnostic instruments must be considered even more suspect.

Our second restriction was that only studies of depression in adult samples be included in this review. Although there is a growing literature examining diverse aspects of depression in children, there is serious concern that the findings obtained with samples of depressed children may not be meaningfully compared with those obtained with depressed adults. In a recent examination of this issue, Digdon and Gotlib (1985) noted that in an effort to understand child depression, some investigators have simply extrapolated downward from adult depression, without consideration of the implications of specific developmental differences between adults and children. Digdon and Gotlib reviewed biological, psychodynamic, cognitive, and behavioral theories of depression and discussed a number of developmental issues in relation to each theory's formulation of the etiology, maintenance, and treatment of this disorder. They presented evidence clearly indicating that developmental

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of initial symptoms. This procedure involves entering the product term for T1 depression and the T1 predictor variable after entering both of these variables into a regression equation, with T2 depression as the dependent variable. If this interaction term is significant, it indicates that the relationship between the predictor at T1 and depression at T2 is significantly different at different levels of initial symptoms. This method may be used regardless of whether T1 depression is a categorical or a continuous variable. Various methods are available for obtaining more specific information about the nature of the interaction using the regression equation (Wise & Barnes, 1986) or the Johnson-Neyman technique (cf. Pedhazur, 1982).

differences do exist in the manifestation of depression: Depression in children is both qualitatively and quantitatively different from depression in adults. Consequently, to avoid the confounds of this issue, we restricted the present review to research conducted with depressed adults.

In summary, research investigating the relationship of adult depression to at least one of the six psychosocial variables listed earlier was included for review if it used one of three basic methods of controlling for the effects of concurrent symptoms. A comparison of this research with cross-sectional studies involving symptomatic depressives will identify those variables that are not robustly related to depression and that therefore may be viewed more parsimoniously as symptoms of depression rather than stable impairments.

### Research Review

Tables 1 through 6 summarize the relevant results of studies in each of the six content areas. We first review the research on attributional style, followed by the research on dysfunctional attitudes, personality, social support, marital adjustment, and coping style.

#### *Attributional Style*

According to the reformulated learned helplessness model of depression (Abramson et al., 1978; Peterson & Seligman, 1984), vulnerability to depression derives from a habitual style of explaining the causes of life events, known as attributional style. The onset of a depressive episode is precipitated by the occurrence of a negative event that triggers the expectation of the uncontrollability of future negative events. Although this expectation itself is sufficient to cause the appearance of symptoms, a self-deprecating attributional style fosters this expectation. Thus, the invocation of internal, stable, and global factors to explain negative events and, to a lesser extent, the attribution of positive outcomes to external, specific, and unstable causes (Seligman, Abramson, Semmel, & von Baeyer, 1979) comprise a depressogenic or self-deprecating attributional style.

Attributional style is viewed as a trait; that is, individuals are believed to exhibit cross-situational (or cross-event) and temporal consistency in their causal explanations for positive and negative events (Peterson & Seligman, 1984). A stable attributional style is often inferred from respondents' causal explanations for hypothetical events, such as those presented in the Attributional Style Questionnaire (ASQ; Peterson et al., 1982). Some studies using the ASQ have found that self-deprecating attributional biases are associated with current depression in both student (Seligman et al., 1979) and patient samples (Persons & Rao, 1985; Raps, Peterson, Reinhard, Abramson, & Seligman, 1982; Zimmerman, Coryell, Corenthal, & Wilson, 1986). Furthermore, attributions for a single recent upsetting event have been found to differentiate depressed from nondepressed patients (Gong-Guy & Hammen, 1980; Miller, Klee, & Norman, 1982). Together, these results suggest that symptomatic depressives, compared with their nondepressed counterparts, exhibit self-deprecating attributional biases in response to both hypothetical and real-life events.

Studies that have examined the relationship between depres-

sion and attributional style while controlling for the effects of concurrent symptoms are summarized in Table 1. The questions generated by the learned helplessness model that can be addressed with available research are these: First, do premorbid depressives exhibit more negative attributional tendencies than are reported by control subjects? Second, does attributional style predict a change in future depressive symptoms? Third, is a self-deprecating attributional style a stable cognitive trait that distinguishes people known to be at risk for depression (i.e., remitted depressives) from control subjects?

In the only study to examine premorbid attributional biases, Lewinsohn et al. (1981) found no differences between the causal attributions of subjects who became depressed during the course of the study and the attributions of subjects who did not. This result clearly suggests that a self-deprecating attributional style does not precede the onset of depression. These negative results might have been due to methodological weaknesses that include the use of nonstandardized measures and the assessment of only the internality of subjects' attributions. Furthermore, it might be argued that this study did not test the diathesis-stress hypothesis proposed by the theory, which is that the interaction of cognitions and stress causes depressive symptoms to appear. However, as mentioned earlier, given the large number of subjects in this study, this interaction would not mask a main effect for attributional style unless cognitions and life events were strongly negatively correlated (Coyne & Gotlib, 1986).

The results of research examining the effect of attributional style on change in level of depression are not as straightforward and require more detailed discussion. In two studies, attributional style was found to be a significant predictor of subsequent symptoms (Cutrona, 1983; O'Hara et al., 1982). Methodologically, these two studies are similar: The symptoms of postpartum depression were predicted from ASQ scores obtained during pregnancy, with T1 symptoms partialled out. Cutrona found that attributional style for negative events accounted for about 10% of the variance in postpartum depression. This was true, however, only for women who had low scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at T1; attributional style was not a significant predictor of the postpartum symptoms of women with higher initial BDI scores. This pattern of results provides indirect evidence of an interaction between individual differences in initial symptoms and attributional style. It suggests that attributional biases may influence depressed mood within a "normal" or nondepressed range: Nearly all of the women who were nondepressed at T1 remained nondepressed at T2. For women with mild to severe depression, however, the influence of attributional style was not significant. In comparison, O'Hara et al. (1982) found that although the ASQ significantly predicted postpartum depression, subjects' symptoms improved significantly from T1 to T2. This suggests that attributional style may have an effect on recovery from depression, with subjects who exhibit more self-deprecating attributions being expected to show less improvement than subjects with fewer negative biases. As discussed earlier, however, the failure to evaluate the interaction between initial symptoms and attributional style means that our interpretation of these data is tentative. However, this interpretation is consistent with results obtained by Lewinsohn

**Table 1**  
*Attributional Style*

Study	Subjects	Time lag	Measure of cognitions	Measure of depression	Events	Dimensions	Results
<b>Predicting future depression</b>							
<i>Premorbid case-control comparison</i>							
Lewinsohn, Steinmetz, Larson & Franklin, 1981	Community volunteers	1 year and RD	MMCS	CES-D, RDC	Hypothetical (-) events	I only	No premorbid differences between cases and controls on MMCS.
<i>Two-wave panel variations</i>							
Cochran & Hammen, 1985	Students	2 months	Own measure	BDI	5 upsetting events	I, G, S	Attributions not related to subsequent depression.
Cutrona, 1983	Pregnant women	5 months	ASQ	BDI, HRS-D	ASQ (-) events	I, G, S	ASQ (T1) predicted postpartum symptoms only for women who had T1 BDI < 9.
Manley, McMahon, Bradley, & Davidson, 1982	Pregnant women	1 month	ASQ	BDI	ASQ (+) and (-) events	I, G, S	ASQ (T1) nonsignificant predictor of postpartum symptoms.
Metalsky, Halberstadt, & Abramson, 1987	Students	3 weeks	Modified ASQ	MAACL	ASQ (-) events	G, S	ASQ (T1) nonsignificant predictor of subsequent negative mood. Interaction of ASQ (T1) with negative event significant predictor of mood 4 days, but not 2 days, after event.
O'Hara, Neunaber, & Zekoski, 1984	Pregnant women	6 months	ASQ	RDC, BDI	ASQ (+) and (-) events	I, G, S	ASQ (T1) nonsignificant predictor of postpartum symptoms.
O'Hara, Rehm, & Campbell, 1982	Pregnant women	6-20 weeks	ASQ	BDI	ASQ (+) and (-) events	I, G, S	ASQ (T1) predicted postpartum symptoms.
Peterson, Schwartz, & Seligman, 1981	Students	6 weeks	ASQ	BDI	ASQ (-) events	I only	ASQ (T1) nonsignificant predictor of T2 symptoms.
Rush, Weissenburger, & Eaves, 1986	Recovered depressed patients	6 months out	ASQ	BDI, HRS-D	ASQ (-) events	I, G, S	ASQ (T1) nonsignificant predictor of T2 symptoms.
<b>Postmorbid functioning</b>							
<i>Prospective case-control comparison</i>							
Eaves & Rush, 1984	In- and outpatients	Not specified	ASQ	RDC, BDI, HRS-D	ASQ (+) and (-) events	I, G, S	D, RD > ND on all subscales and composite score of ASQ.
Hamilton & Abramson, 1983	Inpatients	Not specified	ASQ	RDC, BDI	ASQ (+) and (-) events	I, G, S	D > ND at T1, RD = ND at T2 on ASQ.
<i>Cross-sectional remitted case-control comparison</i>							
Fennell & Campbell, 1984	In- and outpatients	Cross-sectional	Own measure	RDC, BDI	Hypothetical (+) and (-) events	I, G, S	RD = ND on ASQ.
Lewinsohn, Steinmetz, Larson & Franklin, 1981	Community volunteers	Cross-sectional	MMCS	CES-D, RDC	Hypothetical (-) events	I only	RD = ND on MMCS.

*Note.* ASQ = Attributional Style Questionnaire; MMCS = Multi-dimensional Multi-attributional Causality Scale; BDI = Beck Depression Inventory; CES-D = Centre for Epidemiological Studies (Depression Inventory); HRS-D = Hamilton Rating Scale for Depression; RDC = Research Diagnostic Criteria; MAACL = Multiple Affect Adjective Check List; (-) = negative; (+) = positive; I = Internality; G = Globality; S = Stability; RD = remitted depressives; D = depressives; ND = nondepressives; T1 = first assessment; T2 = second assessment.

et al. (1981), who found that initially depressed subjects who improved had significantly fewer negative cognitions than did those who did not improve.

These were the only two studies meeting our selection criteria in which a main effect for attributional style was found. However, support for the diathesis-stress aspect of the learned helplessness model has recently been obtained in a study of negative mood (Metalsky et al., 1987). In an earlier study that did not meet the selection criteria for this review because there was no control for initial symptoms, Metalsky, Abramson, Seligman, Semmel, and Peterson (1982) examined the interaction between attributional style and a stressful event among students. On the basis of their results, Metalsky et al. (1982) concluded that the relationship between attributional style and future mood disturbance was significant only among subjects who had experienced the negative event. However, in a reanalysis of these data, Williams (1985) demonstrated that the correlation between attributional style and negative mood was not significantly greater among subjects who had experienced a negative event than among subjects who had not, thereby suggesting that the hypothesized interaction did not pertain. In the more recent study, Metalsky et al. (1987) again examined the predictive power of the interaction of students' attributional styles with the receipt of disappointing exam results, but they strengthened the design by statistically controlling for initial dysphoria. The results suggested that attributional style had no effect either on students' initial reactions to the receipt of a poor grade or on the severity of their mood disturbance during the entire course of the study. Four days following receipt of the grade, however, students with more negative attributional styles who had been disappointed in their grade continued to report some mood disturbance, whereas other students did not. It should be noted that Metalsky et al. used an affective adjective checklist, rather than a measure of depression, as their dependent measure, making the comparison of these results to those of other research on depression difficult (cf. Gotlib & Cane, in press). In addition, the authors did not assess subjects' internal attributions because they suggested that the mood disturbance under study did not include a loss of self-esteem. Nevertheless, these results appear to be similar to those reported by Cutrona (1983), suggesting that attributional style may be a useful predictor of certain parameters of normal fluctuations in mood experienced in response to negative life events.

The remaining six studies in this area do not support the predictions of the reformulated learned helplessness model. When initial symptoms were statistically controlled, attributional style was not found to be a significant predictor of (a) the severity of symptoms of postpartum depression in two community samples (Manley, McMahan, Bradley, & Davidson, 1982; O'Hara, Neunaber, & Zekoski, 1984), (b) the severity of depressive symptoms in either students (Cochran & Hammen, 1985; Metalsky et al., 1987; Peterson, Schwartz, & Seligman, 1981) or remitted depressives (Rush, Weissenburger, & Eaves, 1986), or (c) the diagnostic status of women in samples of either community residents (O'Hara et al., 1984) or formerly depressed patients (Rush et al., 1986).

It is worth noting here that the disappointing results of this body of research stand in contrast to the more positive findings obtained in two longitudinal studies with samples of normal

children (Nolen-Hoeksema, Girgus, & Seligman, 1986; Seligman et al., 1984). In both of these studies, attributional style was found to predict future depressive symptoms. Furthermore, Nolen-Hoeksema et al. found that the interaction of attributional style and life events was also a significant predictor of subsequent symptoms. These discrepant results in research conducted with adults versus children appear to support the notion discussed earlier that the processes involved in childhood depression may be different from those involved in depression among adults.

Generally nonsignificant results have also been found by research investigating depressives' postmorbidity functioning. Two studies compared the attributional style of depressives with that of nondepressed controls both during and following the depressive episode (Eaves & Rush, 1984; Hamilton & Abramson, 1983). Similarly, two studies conducted cross-sectional comparisons of the attributional styles of groups of currently depressed, remitted depressed, and nondepressed subjects (Fennell & Campbell, 1984; Lewinsohn et al., 1981). Although many of the subjects in these studies received pharmacotherapy, no treatments designed explicitly to alter their cognitive style were implemented. Thus, the results of these studies should demonstrate that the enduring cognitive vulnerability to depression represented by a self-deprecating attributional style is evident in former depressed patients. In three of these four studies, and consistent with the cross-sectional literature, currently depressed patients were found to have significantly more self-deprecating attributional styles than were nondepressed controls (Eaves & Rush, 1984; Fennell & Campbell, 1984; Hamilton & Abramson, 1983). These differences, however, do not appear to be stable. Hamilton and Abramson, for example, found that patients' ASQ scores fell to normal levels following symptomatic recovery. Similarly, both Lewinsohn et al. and Fennell and Campbell found no significant difference between the attributional styles of remitted depressives and never-depressed controls. Only Eaves and Rush found recovered patients to have a more self-deprecating attributional style than that of control subjects. It is possible that the inconsistency in these results is due to differences among the criteria used to determine patients' remitted status. Silverman, Silverman, and Eardley (1984b), for example, suggested that some patients assessed by Eaves and Rush following discharge may not have been sufficiently recovered to be classified as remitted. Thus, it appears that formerly depressed people, following symptomatic recovery, do not exhibit abnormally negative biases in their attributional style.

In summary, research with adults that has controlled for the effects of concurrent symptoms has, by and large, failed to support the causal hypotheses of the reformulated learned helplessness model. A self-deprecating attributional style was not found to be a temporal antecedent of depression, nor did it appear to predict an increase in depressive symptoms over time, although there is evidence of a predictive relationship between attributional style and negative affect among children and nondepressed adults. Finally, remitted depressives did not exhibit more negative attributional biases than control subjects. Two cautionary points should be considered in evaluating this evidence. The first was mentioned already; that is, there have been no adequate tests in adults of the full diathesis-stress model pro-

**Table 2**  
*Dysfunctional Cognitions*

Study	Subjects	Time lag	Nature and measure of cognitions	Measure of depression	Results
<b>Predicting future depression</b>					
<i>Premorbid case-control comparison</i>					
Hammen, Marks, deMayo, & Mayol, 1985 (Expt. 1)	Students	4 months	Negative self-schema (SRET with recall task)	RDC, BDI	No premorbid differences between cases and controls on negative self-schemata.
Lewinsohn, Steinmetz, Larson & Franklin, 1981	Community volunteers	1 year	Irrational beliefs (own measure)	CES-D, RDC	No premorbid differences between cases and controls on irrational beliefs.
<i>Two-wave panel variations</i>					
P. A. Barnett & Gotlib, in press	Students	3 months	Dysfunctional attitudes (DAS)	BDI	DAS (T1) did not predict T2 symptoms. Interaction of DAS (T1) with social support (T2) predicted T2 symptoms.
Hammen, Miklowitz, & Dyck, 1986	Students	1 month	Negative self-schema (SRET with recall task)	BDI	Negative schema did not predict symptoms at T2.
Hewitt & Dyck, 1986	Students	2 months	Perfectionistic attitudes (PFS)	BDI	Neither attitudes nor the interaction of attitudes with stress predicted T2 symptoms.
O'Hara, Rehm, & Campbell, 1982	Pregnant women	6–20 weeks	Dysfunctional attitudes (DAS)	BDI	DAS (T1) did not predict postpartum symptoms.
Rholes, Riskind, & Neville, 1985	Students	5 weeks	Hopelessness (HS), loss cognitions (CC)	BDI	Hopelessness, but not loss cognitions, predicted T2 symptoms.
Rush, Weissenberger, & Eaves, 1986	Inpatients	6 months	Dysfunctional attitudes (DAS)	BDI, HRS-D clinical course	DAS (T1) was a significant predictor of only one of three criteria of depression at T2.
<b>Postmorbid functioning</b>					
<i>Prospective case-control comparison</i>					
Dobson & Shaw, 1986	Inpatients	3 months	Dysfunctional attitudes (DAS), distortions (CRT), automatic thoughts (ATQ)	HRS-D, RDC	D > ND on all depressive cognitions. DAS scores were stable from T1 to T2. RD = ND on DAS and CRT. RD > ND on ATQ.
Dobson & Shaw, 1987	Inpatients	2 months	Negative self-schema (SRET)	RDC	D > ND in negative self-schema, but RD = ND.
Eaves & Rush, 1984	In- and outpatients	Not specified	Dysfunctional attitudes (DAS), automatic thoughts (ATQ)	HRS-D, RDC, BDI	D > ND on DAS and ATQ, but both decreased with improvement in depression. RD > ND on DAS at T2.
Hamilton & Abramson, 1983	Inpatients	Not specified	Dysfunctional attitudes (DAS), hopelessness (HS)	RDC, BDI	D > ND on all cognitions at T1. RD = ND at T2.



Table 2 (continued)

Study	Subjects	Time lag	Nature and measure of cognitions	Measure of depression	Results
<b>Postmorbid functioning</b>					
Hammen, Marks, deMayo, & Mayol, 1985 (Expt. 2)	Students	2 months	Negative self-schema (SRET with recall task)	RDC, BDI	T2 D > (RD = ND) in amount of negative information retrieved on recall task.
Miller & Norman, 1986	Inpatients	9 months	Cognitive biases (CBQ)	DSM-III criteria, BDI	RD = ND on CBQ. Subgroup of D (T1) who had many biases at T1 also had more than normal number of biases when remitted at T2.
Reda, Carpiniello, Secchiaroli, & Blanco, 1985	Inpatients	1 year	Dysfunctional attitudes (DAS)	DSM-III criteria, HRS-D	D > ND on DAS at T1. RD = ND at T2 and T3 on DAS. RD > ND on subset of attitudes.
Silverman, Silverman, & Eardley, 1984a	Outpatients	Not specified	Dysfunctional attitudes (DAS)	DSM-III criteria	DAS decreased significantly following remission. RD = ND scores from A. N. Weissman & Beck, 1978.
<i>Cross-sectional remitted case-control comparison</i>					
Blackburn & Smyth, 1985	Community	Cross-sectional	Dysfunctional attitudes (DAS), distortions (CST), automatic thoughts (ATQ)	HRS-D, BDI	RD = ND on all depressive cognitions.
Hollon, Kendall, & Lumry, 1986	Outpatients	Cross-sectional	Dysfunctional attitudes (DAS), automatic thoughts (ATQ)	DSM-III criteria, RDC	RD = ND on DAS and ATQ.
Lewinsohn, Steinmetz, Larson & Franklin, 1981	Community volunteers	Cross-sectional	Irrational beliefs (own measure)	CES-D, RDC	RD = ND on measures of irrational beliefs.
Wilkinson & Blackburn, 1981	Outpatients	Cross-sectional	Hopelessness (HS)	BDI	D > RD = ND = psychiatric controls on HS.
<i>Prospective cases only (stability)</i>					
Blackburn & Bishop, 1983	Outpatients	12–15 weeks	Hopelessness (HS), cognitive triad (semantic differential)	RDC, BDI	Cognitions became significantly more positive following remission.
Simons, Garfield, & Murphy, 1984	Outpatients	12 weeks	Dysfunctional attitudes (DAS), distortions (CRT), automatic thoughts	BDI, HRS-D	All depressive cognitions decreased significantly following remission.

*Note.* ATQ = Automatic Thoughts Questionnaire; CBQ = Cognitive Bias Questionnaire; CC = Cognitive Checklist; CRT = Cognitive Response Test; CST = Cognitive Style Test; DAS = Dysfunctional Attitudes Scale; HS = Hopelessness Scale; PFS = Perfectionism Scale; SRET = self-referent encoding task; BDI = Beck Depression Inventory; CES-D = Centre for Epidemiological Studies (Depression Inventory); DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.); HRS-D = Hamilton Rating Scale for Depression; RDC = Research Diagnostic Criteria; T1 = first assessment; T2 = second assessment; T3 = third assessment; D = currently depressed group; ND = nondepressed control group; RD = remitted depressed group.

posed by Peterson and Seligman (1984). The positive results obtained by Metalsky et al. (1987) in their study of negative mood are promising, and suggest that the interaction of attributional style and negative life events should be investigated in prospective research using proper measures of depression to explore the issue more fully. However, there are a number of reasons to expect negative results in research of this type. For example, Coyne and Gotlib's (1986) argument concerning the necessary presence of an improbable negative correlation between cognitions and negative life events has already been noted. In addition, our review of the literature suggests that an abnormal attributional style is not characteristic of the cognitive functioning of either premorbid or remitted depressives; that is, research has been unable to demonstrate the postulated stable cognitive diathesis in people vulnerable to depression.

The second possible explanation for the negative results of the studies reviewed here concerns the psychometric shortcomings of the ASQ. The reliability coefficients both of the individual scales (e.g., internality) and of the composite score have been reported to be "too low, even for the experimental use of the measure" (Cutrona, Russell, & Jones, 1985, p. 1046). Not surprisingly, test-retest correlations are also relatively low (Cutrona et al., 1985; Peterson et al., 1982). Thus, the low reliability of the measure may account for the general lack of significant results, due to the attenuation of all correlations involving the ASQ. A different and more substantive interpretation, however, is that the low reliability of the ASQ is not purely a psychometric problem; rather, it may accurately reflect the lack of cross-situational consistency in subjects' causal attributions (Cutrona et al., 1985; Miller et al., 1982). This interpretation is consistent with the paucity of empirical evidence of a trait-like cognitive vulnerability to depression and suggests the need to revise the reformulated learned helplessness model of depression.

### *Dysfunctional Attitudes*

Beck (1967, 1976) implicated three cognitive constructs in the etiology and phenomenology of depression: the cognitive triad, schemata, and cognitive distortions. Superordinate schemata, or personality tendencies (Beck, 1983; Beck & Epstein, 1982), are discussed in the following section of this review. Schemata are particularly germane to the general theme of this review because they are thought to be stable and enduring cognitive structures. As Kovacs and Beck (1978) stated, "[Certain] cognitive processes seem chronically atypical among depressed patients and may represent a *stable characteristic of their personality*" (p. 530, italics added). Depressogenic schemata, which develop as a result of certain early life experiences (Shaw, 1982), take the form of excessively rigid and inappropriate beliefs or attitudes about the self and the world (Beck & Epstein, 1982; Beck et al., 1979). These dysfunctional attitudes represent unrealistic, often perfectionistic standards by which the self is judged (A. N. Weissman & Beck, 1978). Activation of the depressogenic schemata by stress leads to the appearance of specific negative cognitions that, in turn, cause the onset of the somatic, affective, and motivational symptoms of depression (Beck et al., 1979).

The most widely used measure of cognitive vulnerability to depression is the Dysfunctional Attitudes Scale (DAS; A. N.

Weissman & Beck, 1978). Research using the DAS with university students has demonstrated that mildly depressed subjects endorse significantly more dysfunctional attitudes than do non-depressed subjects (Dobson & Breiter, 1983; Gotlib, 1984; A. N. Weissman & Beck, 1978). Similar studies have reported that depressed psychiatric patients exhibit higher scores on the DAS than do normal controls, although they do not differ significantly from nondepressed psychiatric patients (Zimmerman et al., 1986). The results of validation studies suggest that the schemata measured by the DAS are more stable than are self-rated symptoms of depression across both 6-week (Oliver & Baumgart, 1985) and 2-month (A. N. Weissman, 1980) time lags. Finally, the interaction of dysfunctional attitudes with negative life events has also been investigated (Olinger, Kuiper, & Shaw, 1987; Wise & Barnes, 1986), and the results of this research suggest that the depressogenic effect of negative events is more potent among cognitively vulnerable (i.e., high-DAS) subjects than among those subjects who endorse a low number of dysfunctional attitudes.

Thus, research has generally supported the hypotheses of Beck's cognitive model of depression concerning the relationship between schemata and current depression. The model also predicts, at least implicitly, that dysfunctional attitudes are predictive of future depression, particularly through their interaction with stressful life events, that they are relatively stable, and that they are elevated in depressive probands who are asymptomatic. Research addressing these issues will now be reviewed. Table 2 contains a summary of those studies involving the DAS that have controlled for the effects of concurrent symptoms.

Prospective studies that have statistically controlled initial symptoms have produced mixed results (P. A. Barnett & Gotlib, in press; O'Hara et al., 1982; Rush et al., 1986). Rush et al. regressed data from three different measures of depression at T2 on remitted depressives' DAS scores at T1. When T1 symptoms were partialled out, the DAS was a significant predictor of only one of the three dependent measures. O'Hara et al. found that dysfunctional attitudes measured during pregnancy did not significantly predict severity of postpartum depression. Finally, P. A. Barnett and Gotlib found no main effect for the DAS and were unable to replicate the significant findings of Olinger et al. (1987) and Wise and Barnes (1986) concerning the interaction of dysfunctional attitudes with stressful life events. However, the interaction of the DAS administered at T1 with a measure of social support given at T2 did account for a significant proportion of the variance in T2 depression after controlling for T1 symptoms. Analysis of this interaction suggested that subjects who endorsed a high number of dysfunctional attitudes were more likely to report a higher number of depressive symptoms when they perceived social support to be low. It should be noted that subjects' symptoms improved significantly during the course of the study, which suggests that dysfunctional attitudes may be useful predictors of recovery from existing depression under certain social conditions.

It appears from this research that a main effect for the DAS in predicting future depression is not a robust finding. Additional research may be required to clarify the role of dysfunctional attitudes in mediating the effects of stress on depression. In the absence of this latter research, however, it is informative to turn to studies that have examined a related assumption of this di-

athesis-stress model, that is, that dysfunctional attitudes represent a stable characteristic of depressed patients' personality (Kovacs & Beck, 1978).

This assumption has been investigated in six studies involving the assessment of depressed patients both during and following an episode of depression (Dobson & Shaw, 1986; Eaves & Rush, 1984; Hamilton & Abramson, 1983; Reda, Carpiniello, Secchiarioli, & Blanco, 1985; Silverman Silverman, & Eadley, 1984a; Simons, Garfield, & Murphy, 1984). It is important to note that none of the patients in these studies received cognitive therapy and, therefore, no change in their habitual cognitive style due to treatment would be expected. In only one of these six studies were dysfunctional attitudes found to be stable (Dobson & Shaw, 1986). Although Reda et al. concluded that total DAS scores are mood-state dependent, they also observed that remitted patients continued to endorse a subgroup of DAS items at discharge and at a 1-year follow-up assessment. As a group, these items appear to reflect the need to please others, combined with an attitude of perfectionistic self-reliance. The possible importance of this apparent conflict concerning dependence and autonomy is interesting and is discussed in our subsequent section on personality.

Several studies have examined the stability of between-group differences on the DAS (Dobson & Shaw, 1986; Eaves & Rush, 1984; Hamilton & Abramson, 1983; Reda et al., 1985; Silverman et al., 1984a). Across studies, symptomatic depressives were found to have higher DAS scores than nondepressed subjects; only Eaves and Rush, however, found that this difference was maintained following the remission of depressive symptoms.<sup>3</sup> Two cross-sectional studies that compared carefully diagnosed remitted depressives with normal controls also found no differences between these two groups on the DAS (Blackburn & Smyth, 1985; Hollon, Kendall, & Lumry, 1986). Thus, there is nearly uniform evidence that a higher-than-normal level of dysfunctional attitudes is limited to the duration of a depressive episode: Dysfunctional attitudes return to normal levels when depression remits. This result was found despite the absence of any interventions that would be expected to alter directly patients' characteristic cognitive functioning.

Overall, then, there is little empirical support for the contentions of the cognitive theorists that dysfunctional attitudes represent a stable vulnerability to depression. Before proceeding to a more detailed consideration of this conclusion, we present a brief review of research examining other types of cognitions.

Although dysfunctional attitudes are identified by Beck et al. (1979) as being the primary link in the causal chain that leads to the onset of a depressive episode, the stability and predictive power of diverse cognitions postulated to be associated with depression have been investigated in a number of studies that have controlled for the effects of concurrent symptoms. These cognitions include thoughts of hopelessness (Blackburn & Bishop, 1983; Hamilton & Abramson, 1983; Rholes, Riskind, & Neville, 1985; Wilkinson & Blackburn, 1981), thoughts of loss (Rholes et al., 1985), negative self-schemata (Dobson & Shaw, 1987; Hammen, Marks, deMayo, & Mayol, 1985; Hammen, Miklowitz, & Dyck, 1986), negative construct accessibility (Gotlib & Cane, 1987), the cognitive triad (i.e., negative view of self, future, and world; Blackburn & Bishop, 1983), perfectionistic attitudes (Hewitt & Dyck, 1986), irrational beliefs (Lewin-

sohn et al., 1981), cognitive distortions (Blackburn & Smyth, 1985; Dobson & Shaw, 1986; Miller & Norman, 1986; Simons et al., 1984), and negative automatic thoughts (Blackburn & Smyth, 1985; Dobson & Shaw, 1986; Eaves & Rush, 1984; Hollon et al., 1986; Simons et al., 1984).

With two exceptions (Dobson & Shaw, 1986; Rholes et al., 1985), the results of this research support the hypothesis that abnormal cognitive activity is a concomitant or symptom of depression. Dobson and Shaw (1986) obtained data indicating that negative automatic thoughts were more frequent among remitted depressives than among normal controls.<sup>4</sup> This finding is somewhat contrary to recent formulations of cognitive theory in which negative automatic thoughts are seen as relatively unstable, symptomatic cognitions (cf. Beck & Epstein, 1982). Furthermore, it should also be noted that this finding was not replicated in the studies cited earlier that also examined negative automatic thoughts. In the second study, Rholes et al. found that hopelessness cognitions significantly predicted subsequent depression among initially nondepressed subjects, although no information was given regarding subjects' level of depression at T2. Future research should be directed to an exploration of the predictive relationship between hopelessness cognitions and more serious depression.

In summary, research that has controlled for the effects of concurrent symptoms has found little support for the concept of a cognitive vulnerability to depression. Four conclusions are possible based on these results. First, dysfunctional attitudes, like many other negative cognitions, may be episode markers of depression that return to normal levels following symptomatic recovery. Second, the pharmacotherapy that most patients in the studies of postmorbid cognitions received may have had a direct effect on patients' attitudes, thereby leading to subsequent nonsignificant group differences. Third, depressogenic schemata may be unconscious or otherwise inaccessible cognitive structures. Riskind and Rholes (1984), for example, argued that negative schemata may need to be primed by negative life events before they become accessible to measurement. Finally, a defense of the null hypothesis must rule out methodological insensitivity. The DAS may be too general a measure of vulnerability to depression, and its interaction with a general measure of negative life events may be too diffuse to reveal significant relationships. These issues are addressed in greater detail in the concluding sections of this article.

### *Personality*

Recent interest in personality traits associated with depression has led to a "rediscovery" of the contributions of psychoanalytic and object relations theories and to the development of

<sup>3</sup> Dobson and Shaw (1986) did not explicitly compare remitted depressives' DAS scores with those of control subjects. However, we conducted independent one-tailed *t* tests using the group means and standard deviations reported by Dobson and Shaw. These analyses revealed that the DAS scores of remitted depressives assessed at T2 did not differ significantly from the scores of either normal or psychiatric controls assessed at T1,  $t(30) = 1.04, p < .05$ , and  $t(29) < 1$ , respectively.

<sup>4</sup> We evaluated this between-group difference using an independent one-tailed *t* test,  $t(30) = 6.05, p < .001$ .

new scales for conducting empirical tests of these theories. We devote space to a description of this theory and to relevant cross-sectional research both because the research has not been reviewed elsewhere and because it is methodologically diverse.

Developmentally acquired traits, such as interpersonal dependency and labile self-esteem, have been identified in clinical case studies as characteristic of the personalities of people prone to depression (see reviews by Chodoff, 1972; Hirschfeld et al., 1976; Masserman, 1970). Vulnerable individuals are hypothesized to depend primarily on the love and attention of others for the maintenance of their fragile self-esteem. When these extreme dependency needs are frustrated, the resulting threat to self-worth is defended against by increasing demands for support or by denying interpersonal dependency and developing obsessive, perfectionistic tendencies. Thus, these two traits of dependency and perfectionism are thought to share a common etiology of excessive dependency needs.

Descriptions of a similar dichotomy of correlated traits associated with depression are found in the works of other theorists (e.g., Arieti & Bemporad, 1980; Beck, 1983; Blatt, 1974). Although the respective personality styles identified by these theorists appear to be similar, different terms have been used to describe them: anaclitic, or dependent, and introjective, or self-critical (Blatt, 1974; Blatt, D'Afflitti, & Quinlan, 1976); dominant other and dominant goal (Arieti & Bemporad, 1980); and sociotropic and autonomous (Beck, 1983). Although dependent and autonomous tendencies are postulated to be dominant modes of personality, they may nevertheless coexist within a single individual. Both personality modes are hypothesized to predispose one to depression: Whereas the dependent individual is particularly at risk when sources of interpersonal support are threatened, the autonomous person is sensitive to setbacks in goal attainment.

A number of self-report inventories have been developed to measure these personality styles, among them the Depressive Experiences Questionnaire (DEQ; Blatt et al., 1976) and the Sociotropy-Autonomy Scale (SAS; Beck, Epstein, Harrison, & Emery, 1983). Despite the apparent similarity of the constructs being measured by these two inventories, correlations between the corresponding subscales across measures (e.g., self-criticism with autonomy) suggest that they may be quite different (Robins, 1985). Furthermore, the DEQ and the SAS produce different patterns of results with respect to the relationships of dependency and autonomy, or self-criticism, with depression. Whereas the Self-Criticism factor of the DEQ has a stronger and more robust relationship with depression than does the Dependency factor (Blatt et al., 1976; Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982; Pilon, Olioff, Bryson, & Doan, 1986), the Sociotropy scale of the SAS has consistently been found to be related to depression whereas the Autonomy scale has not (Robins, 1985, 1986; Robins & Block, 1986).

The relationship to depression of the interaction of personality and life events has also been investigated, using both the SAS (Robins, 1986; Robins & Block, 1986) and a schema-based method for assessing personality type (Hammen, Marks, Mayol, & deMayo, 1985). The results of this research suggest that dependency, or sociotropy, mediates the depressogenic effects of negative life events, but the more specific link between sociotropy and negative *social* events has not been well demon-

strated. Little support has been found for the role of autonomy as a mediator of either social or achievement-related events.

Hirschfeld et al. (1977) also developed a measure of interpersonal dependency, a trait involving thoughts, feelings, and behaviors associated with the need to interact with and rely on others. As Hirschfeld et al. (1976, p. 385) stated,

Individuals possessing higher amounts of this trait desire more support and approval from important others, are more anxious about being alone or abandoned, have more fragile feelings, have low social self-confidence, have difficulty in making decisions on their own . . . , lack confidence in their own judgement, and are never able to get enough care and attention.

This measure, the Interpersonal Dependency Inventory (IDI), has three subscales: Emotional Reliance on Another Person, Lack of Social Self-Confidence, and Assertion of Autonomy. The first two subscales have been found to correlate with the severity of depression in a patient sample, whereas the latter subscale, a measure of the pseudo-autonomy described by Chodoff (1970), has not been found to be related to depression (Hirschfeld et al., 1977).

As is apparent from Table 3, research on personality and depression that has controlled for the effects of concurrent symptoms has focused exclusively on the measurement of personality in remitted depressives. Four such studies have been conducted using the IDI (Hirschfeld, Klerman, Clayton, & Keller, 1983; Hirschfeld, Klerman, Clayton, Keller, & Andreasen, 1984; Pilowsky & Katsikitis, 1983; Reich, Noyes, Hirschfeld, Coryell, & O'Gorman, 1987). The results of these studies are uniform: Compared with never-depressed control subjects, remitted depressives report high emotional dependency and low social self-confidence. In addition, and consistent with the cross-sectional findings, autonomy scores did not differentiate recovered depressives from control subjects.

Research has not yet examined the relationship between interpersonal dependency and self-esteem. Although some data suggest that remitted depressives may have chronic self-esteem deficits (Altman & Wittenborn, 1980; Cofer & Wittenborn, 1980; Wittenborn & Maurer, 1977), other studies have found no evidence of this relationship (Billings & Moos, 1985; Lewinsohn et al., 1981). Hirschfeld et al. (1976) pointed out that individuals who are high in interpersonal dependency are able to maintain their self-esteem by satisfying their dependency needs. Thus, this theory would not necessarily predict that chronic low self-esteem represents a vulnerability to depression; rather, it is the reactivity of self-esteem to positive and negative interpersonal experiences that puts one at risk. Research testing this model would be expected to find that individuals who are prone to depression have labile self-esteem that is reactive to interpersonal stress.

To summarize, theoretical formulations based primarily on clinical observations identify two personality styles that may predispose to depression: excessive dependency and autonomy. Cross-sectional research has generally supported the relationship of dependency, but not autonomy, with depression. Research with remitted depressives has shown that formerly depressed people report higher-than-normal levels of interpersonal dependency, a finding that suggests remitted patients may be unusually dependent on the positive emotional support of

**Table 3**  
*Personality*

Study	Subjects	Time lag	Trait and measure	Measure of depression	Results
<b>Postmorbid functioning</b>					
<i>Prospective case-control comparison</i>					
Benjaminsen, 1981	Inpatients	Not specified	N, E (EPI)	Clinical diagnosis	RD < published norms on E. Remitted nonendogenous and neurotic, but not endogenous D, higher than norms on N. RD > ND controls on dependency and N. RD < ND controls on E.
Hirschfeld, Klerman, Clayton, & Keller, 1983	Inpatients	1 year	Interpersonal dependency (IDI), N, E (MPI)	RDC	RD > ND controls on dependency and N. RD < ND controls on E.
Kendell & DiScipio, 1968	Inpatients	3 months	N, E (EPI)	Clinical diagnosis	Neither N nor E changed when depression remitted. RD > norms on N; RD > norms on E. RD = ND on N, RD < ND on E.
Liebowitz, Stallone, Dunner, & Fieve, 1979	Outpatients	6 months	N, E (MPI)	Feighner criteria	
Reich, Noyes, Hirschfeld, Coryell, & O'Gorman, 1987	In- and outpatients	1 year	Interpersonal dependency (IDI)	RDC	D > ND in interpersonal dependency both during and following the depressive episode.
Wittenborn & Maurer, 1977	Inpatients	1 year	11 variables, each with multiple items (Own measure)	Clinical diagnosis	Displacement of hostility, dissident role low self-esteem, dysphoric mood did not change with remission.
<i>Cross-sectional remitted case-control comparison</i>					
Altman & Wittenborn, 1980	Recovered inpatients	Cross-sectional	Self-esteem, failure preoccupation, unhappy outlook, narcissistic vulnerability, self-confidence (Own measure)	Clinical diagnosis	Items comprising these factors differentiated RD from ND.
Hirschfeld, Klerman, Clayton, Keller, & Andreason, 1984	Recovered inpatients	Cross-sectional	Interpersonal dependency (IDI)	RDC	RD > ND controls on dependency.
Pilowsky & Katsikitis, 1983	Inpatients	Cross-sectional	Interpersonal dependency (IDI)	LPD	RD > ND on interpersonal dependency. ND data were published norms.
<i>Prospective cases only (stability)</i>					
Bailey & Metcalfe, 1969	Inpatients	6 weeks	N, E (MPI and EPI)	Clinical diagnosis	N decreased following remission. E increased for men, not women with one measure, and vice versa with another.
Coppen & Metcalfe, 1965	Inpatients	Not specified	N, E (MPI)	Clinical diagnosis	N decreased, E increased following remission.
Garside, Kay, Roy, & Beamish, 1970	Inpatients	5-7 years	N, E (MPI)	Clinical diagnosis, HRS-D	N, but not E, correlated with severity of symptoms. N, but not E, decreased following remission.
Hirschfeld & Klerman, 1979	Inpatients	2 years	N, E (MPI)	RDC	N decreased following remission. E did not increase.
Hirschfeld, Klerman, Clayton, Keller, McDonald-Scott, & Larkin, 1983	Inpatients	1 year	N, E (MPI)	RDC	N decreased following remission. E did not increase.
Kerr, Schapira, Roth, & Garside, 1970	In- and outpatients	4 years	N, E (MPI)	Clinical diagnosis	N decreased as depression remitted. E did not increase.
Perris, 1971	Inpatients	3 years	N, E (MPI)	Clinical diagnosis	N decreased but E did not change following remission.
Wretmark, Astrom, & Eriksson, 1970	Inpatients	6 months	N, E (MPI)	Clinical diagnosis	N decreased, but E did not change following remission.

*Note.* N = neuroticism; E = extraversion; EPI = Eysenck Personality Inventory; IDI = Interpersonal Dependency Inventory; MPI = Maudsley Personality Inventory; HRS-D = Hamilton Rating Scale for Depression; LPD = Levine Pilowsky Depression Questionnaire; RDC = Research Diagnostic Criteria; ND = nondepressives; D = depressives; RD = remitted depressives.

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others for the maintenance of their self-esteem. Although these results are congruent with the hypothesis that interpersonal dependency represents a stable, enduring predisposition to depression, there is at present no prospective evidence demonstrating the temporal antecedence of dependency to the onset of depression. Therefore, conclusions regarding interpersonal dependency's role as a vulnerability factor for depression must remain tentative. For example, it may be that this trait is increased only as a result of a person's having been depressed. Additional research is also required to examine the interactive effect of high interpersonal dependency and negative interpersonal events on self-esteem and depression.

The relationship with depression of a second pair of personality dimensions, introversion and extraversion, has been the focus of another large body of literature. Neuroticism and extraversion-introversion are postulated by H. J. Eysenck and M. W. Eysenck (1985) to be the most universal and fundamental dimensions of human personality. The highly neurotic individual is postulated to have a low threshold for autonomic nervous system activation, to be prone to anxiety and fear responses, and to be emotionally labile. Although neuroticism is known to correlate significantly with anxiety, the empirical basis for other descriptors of neuroticism is not well established (H. J. Eysenck & M. W. Eysenck, 1985). More is known about the correlates of extraversion-introversion. Extraverts have been found to engage in more social interaction, to initiate conversation and verbalize more often, and to be less avoidant of stressful situations than are introverts (A. Campbell & Rushton, 1978; Carment, Miles, & Cervin, 1965; Furnham, 1981).

In general, whereas neuroticism correlates positively with depressive symptoms, extraversion is inversely related to depression (Garside, Kay, Roy, & Beamish, 1970; Kerr, Schapira, Roth, & Garside, 1970). Symptomatic depressives have been found to be more neurotic and less extraverted than nondepressed people (Hirschfeld & Klerman, 1979; Kendell & DiScipio, 1968). Finally, neuroticism, but not extraversion, significantly predicted treatment outcome in one study (M. M. Weissman, Prusoff, & Klerman, 1978); this finding, however, was not replicated in a subsequent investigation (Zuckerman, Prusoff, Weissman, & Padian, 1980). Research on the stability and relative levels of neuroticism and extraversion in remitted depressives is summarized in Table 3. We will review the research on neuroticism first and then discuss the results on extraversion.

Although relatively stable in the general population, neuroticism is clearly affected by depression. Out of 10 studies that assessed patients both during and following a depressive episode, neuroticism was found to decrease significantly in 9 (Bailey & Metcalfe, 1969; Coppen & Metcalfe, 1965; Garside et al., 1970; Hirschfeld & Klerman, 1979; Hirschfeld, Klerman, Clayton, Keller, & Andreasen, 1984; Kerr et al., 1970; Liebowitz, Stallone, Dunner, & Fieve, 1979; Perris, 1971; Wretmark, Astrom, & Eriksson, 1970). In contrast to these 9 studies, Kendell and DiScipio (1968) found that depressed patients' neuroticism scores were stable and remained higher than published norms following recovery. Four other studies that compared the neuroticism scores of remitted depressives with normative data did not produce consistent results. Hirschfeld, Klerman, Clayton, and Keller (1983) found no difference between former patients'

scores and published norms. Hirschfeld and Klerman (1979) found that recovered patients who were tested at discharge were abnormally neurotic; however, a small subsample tested 2 years later had scores in the normal range. Finally, Benjaminsen (1981) found that remitted nonendogenous, but not endogenous, patients had higher-than-normal neuroticism scores, whereas Kerr et al. (1970) found the opposite pattern of results.

The lack of consistent results from these studies may be due to the use of published norms as a substitute for a control group. For example, Hirschfeld and Klerman (1979) compared data collected from university students with scores obtained from recovered depressed patients whose mean age was 34. Because neuroticism is significantly correlated with age, sex, and socioeconomic status, and has been found to vary across nationalities and occupations (H. J. Eysenck & S. B. Eysenck, 1968, 1975), it is likely that inappropriate comparisons were made in some of these studies, resulting in conflicting findings.

Finally, two studies compared remitted depressives' level of neuroticism with that of controls, but again, the results are inconsistent. Whereas Hirschfeld, Klerman, Clayton, and Keller (1983) found that recovered patients were significantly more neurotic than were never-depressed controls, Liebowitz et al. (1979) obtained a between-group difference that narrowly missed significance.

In conclusion, neuroticism has been shown to be mood-dependent, decreasing as depressive symptoms abate. Given the high correlation between neuroticism and anxiety, the decrease in neuroticism may simply reflect the amelioration of anxiety symptoms associated with depressive illness. Although there is some evidence that remitted depressives remain abnormally neurotic following recovery from depression, this finding is not consistent across studies, and additional research with properly matched control groups is clearly required.

The evidence linking the extraversion-introversion personality dimension with previous depression is more consistent. In general, it has been found that extraversion scores are not significantly affected by recovery from depression (Garside et al., 1970; Hirschfeld & Klerman, 1979; Kendell & DiScipio, 1968; Kerr et al., 1970; Liebowitz et al., 1979; Perris, 1971; Wretmark et al., 1970). Three additional studies, however, found evidence of instability in extraversion scores, in one case for both males and females (Coppen & Metcalfe, 1965) and, in another, for females but not for males (Hirschfeld, Klerman, Clayton, Keller, McDonald-Scott, & Larkin, 1983). In the third study, there was an interaction of gender and measurement instrument (Bailey & Metcalfe, 1969). Four of the five studies that included comparisons with published norms found that depressed patients had higher-than-normal introversion scores following recovery (Benjaminsen, 1981; Hirschfeld & Klerman, 1979; Hirschfeld, Klerman, Clayton, & Keller, 1983; Kendell & DiScipio, 1968). Kerr et al.'s (1970) results differed slightly from this trend: Whereas recovered endogenous depressives were abnormally introverted, remitted reactive depressives' scores did not differ from the norms. Although the use of normative data in these five studies is potentially problematic, extraversion appears to be relatively stable with respect to various sociodemographics (H. J. Eysenck & S. B. Eysenck, 1968, 1975). Furthermore, these results are consistent with those of studies employing never-depressed controls, in which remitted depressives were

found to be more introverted than the control subjects (Hirschfeld, Klerman, Clayton, & Keller, 1983; Liebowitz et al., 1979).

Overall, these results suggest that although extraversion-introversion covaries with depression, recovered depressives remain more introverted than never-depressed people. This conclusion is supported by related data reported by Hirschfeld, Klerman, Clayton, and Keller (1983). Hirschfeld, Klerman, Clayton, and Keller (1983) found that, in addition to being more introverted, formerly depressed patients, compared to never-depressed controls, were significantly less sociable, less dominant, less active, and lower in social self-confidence. Remitted patients' scores on these variables, as well as on a measure of social restraint, also differed significantly from published norms.

H. J. Eysenck hypothesized that introverts are chronically more aroused than are extraverts and that the increased arousal associated with social interaction becomes aversive to them. Attempts to demonstrate chronic cortical arousal in introverts, however, have produced mixed results (H. J. Eysenck & M. W. Eysenck, 1985). Therefore, in keeping with the empirical evidence of differences in social behavior between introverts and extraverts, it seems reasonable to conclude only that remitted depressives tend to engage in less social activity, to be more restrained in their social interactions, and to avoid stressful situations to a greater extent than do never-depressed people.

In conclusion, a paradoxical combination of personality traits appears to distinguish remitted depressives from never-depressed controls. On the one hand, formerly depressed patients have greater interpersonal dependency needs; that is, they desire approval, attention, and help from others to a greater extent than do nondepressed controls. On the other hand, there is also some evidence to indicate that they socialize less and participate in social situations less fully than do never-depressed individuals. That recovered depressives exhibit these opposing tendencies suggests that they may experience some difficulties or conflict in their interpersonal relationships. Research on the relationships with depression of social support, marital adjustment, and coping style may elucidate these difficulties, and it is to the literature on these variables that we now turn.

### *Social Support*

The construct of social support and the many operational definitions of it that appear in the literature on depression are so broad that a comprehensive discussion of them is well beyond the scope of this article (see reviews by Barrera, 1986; Cohen & Wills, 1985; Leavy, 1983; Thoits, 1986). However, because different operationalizations may produce different results (cf. Cohen & Wills, 1985), it may be instructive to offer briefly some form of classification. Three levels of conceptual analysis are evident in different uses of the term *social support*. First, at the broadest level, social support has been conceptualized variously as an objective quantity of social resources (Monroe, Imhoff, Wise, & Harris, 1983); a process by which one develops, uses, and maintains resources (Leavy, 1983); a cognitive appraisal (Turner, Frankel, & Levin, 1983); and a transaction between person and environment (Coyne & Holyroyd, 1982). Second, social support has been dichotomized as either structural or functional (Cohen & Wills, 1985). Structural support

refers to the number and degree of integration of relationships, whereas functional support comprises various content dimensions, such as esteem, informational support, social companionship, and tangible support. Finally, the most fine-grained analysis reveals from whom support is received. Perhaps the most important distinction to be made at this level is that between marital and extramarital support. Coyne and DeLongis (1986) argued that the failure of previous research to recognize the qualitative difference between marital and extramarital support may have obfuscated the nature of the relationship between social support and emotional well-being. For this reason, research examining marital support is presented separately in the next section.

There is consistent evidence of a negative relationship between many facets of social support and concurrent depression (e.g., Bell, LeRoy, & Stephenson, 1982; Billings, Cronkite, & Moos, 1983; Billings & Moos, 1984; Blazer, 1983; Dean & Ensel, 1982; Gore, 1978; Mitchell & Moos, 1984; Schaefer, Coyne, & Lazarus, 1981). Smaller social networks, fewer close relationships, and less perceived adequacy of relationships are all related to depressive symptoms. The precise nature of the relationship of social support with depression, however, may depend to some extent on the nature of the support measure used. On the basis of a review of research that examined diverse psychological and physical symptoms, Cohen and Wills (1985) concluded that the perceived availability of functional support buffers the effects of stress by enhancing broadly applicable coping abilities. In comparison, the degree of integration in a social network, or structural support, was found to have a direct positive effect on well-being, reducing negative outcomes in both high- and low-stress circumstances. We therefore note the differential relationships of various aspects of support with depression in the following review.

We found only six studies that met the design criteria for inclusion in this review, and they are summarized in Table 4. The studies are divided according to the operationalization of social support, yielding four groups of studies: those examining quantity of social resources, those assessing the perceived adequacy of support, those investigating different dimensions of functional support, and those using a composite variable that includes both size and perceived availability of information.

The results of the two studies that examined social network size provide some evidence of a negative relationship between social support and depression. Monroe et al. (1983) assessed number of best friends and social group memberships, as well as whether respondents lived with their parents. Living away from home significantly predicted an increase in depression, after the effects of concurrent symptoms were partialled out, whereas main effects for the other indicators of support were not significant. In addition, the interaction between total number of best friends and an index of perceived impact of stress was also significant, with fewer friends and more undesirable versus desirable life events predicting a higher level of depressive symptoms. However, with little control for Type I error in this study, these results may represent chance findings and need to be replicated. Billings and Moos (1985) found that remitted depressives tested 12 months after admission to treatment had fewer friends and fewer close relationships, but not fewer social network contacts, than did normal controls. These results suggest that although

Table 4  
Social Support

Study	Subjects	Time lag	Type and measure of support	Measure of depression	Results
<b>Predicting future depression</b>					
<i>Premorbid case-control comparison</i>					
O'Hara, 1986	Pregnant women	6 months	Functional support from a single confidant (SSI)	RDC	No premorbid differences between cases and controls in support received from a single extramarital confidant.
Phifer & Murrell, 1986	Community sample	6 months	Social participation index, amount of help available in a crisis (LSSS)	CES-D	Low social support (T1) and its interactions with loss events and poor health discriminated cases from controls at T2.
<i>Two-wave panel variations</i>					
Cutrona, 1984	Pregnant women	4 months	6 dimensions of support, e.g., attachment, social integration (SPS)	BDI, HRS-D	Total support score and social integration score (T1) each predicted postpartum symptoms.
Lin & Ensel, 1984	Community sample	1 year	Enough close friends, presence of close companion	CES-D	Social support (T1) predicted symptoms at T2.
Monroe et al., 1983	Students	3 months	Living with parents, number of best friends, social group membership, comfort confiding in friends.	BDI	Living with family (T1) significant predictor of T2 symptoms. Interaction of number of best friends with life event index was only significant interaction.
<b>Postmorbid functioning</b>					
<i>Prospective case-control comparison</i>					
Billings & Moos, 1985	In- and outpatients	1 year	Number of friends, social network size, number and quality of close relationships, family and work support (FES, HDL, WES)	DSSI	RD < ND in number of friends, number of close relationships, supportive family interactions. RD = ND quality of close relationships, work support, and size of social network.

*Note.* FES = Family Environment Scale; HDL = Health and Daily Living Form; LSSS = Louisville Social Support Scale; SPS = Social Provisions Scale; SSI = Social Support Interview; WES = Work Environment Scale; BDI = Beck Depression Inventory; CES-D = Centre for Epidemiological Studies (Depression Inventory); DSSI = Depression Symptom Severity Index; HRS-D = Hamilton Rating Scale for Depression; RDC = Research Diagnostic Criteria; RD = remitted depressives; ND = nondepressives; T1 = first assessment; T2 = second assessment.

remitted depressives maintain normal levels of superficial relationships, they may have fewer meaningful relationships than do never-depressed people.

Decreased perceived adequacy or availability of social support has also been found in some studies to predict the future level of depressive symptoms and to differentiate remitted patients from normal controls. Lin and Ensel (1984) asked respondents in a community sample to indicate on a 4-point scale whether they had a close companion and enough close friends. With initial symptoms included in the model, a path analysis revealed a direct negative effect of T1 social support on change in depressive symptoms at T2. Billings and Moos (1985) observed that remitted depressives perceived the quality of their familial interactions to be less supportive than that reported by normal controls; however, recovered patients did not differ from normal controls in the quality of a significant relationship or work support.

A number of dimensions of support were measured in two studies investigating postpartum depression. Cutrona (1984) obtained information on six dimensions of social support from women during their pregnancies. Many of these aspects of support would most probably have been provided by the women's husbands, but at least one concerned extramarital support: social integration. With stringent control for Type I error, none of the support items significantly predicted depression at 2 weeks postpartum, after T1 symptoms were partialled out. However, depressive symptoms at 8 weeks postpartum were negatively related to both the social integration and total support scores obtained at T1. Social integration accounted for 9% of the variance in postpartum depression independent of initial symptoms and, moreover, none of the interactions between support and stress was significant when alpha was controlled experimentwise. In contrast, O'Hara (1986) was unable to differentiate women who became depressed postpartum from those who



did not on the basis of respondents' perceptions of emotional and instrumental support received from a single extramarital confidant during pregnancy. O'Hara's use of rigorous diagnostic criteria to divide subjects into depressed or nondepressed groups, which entails a loss of information about the covariance of independent and dependent variables, may account for the discrepancy in these results. It is more probable that by narrowing the source of support to a single person, O'Hara failed to detect differences that may have been revealed if Cutrona's more general approach had been taken.

Finally, additional evidence of the independent negative effect of social support on depression was found in a study that combined an index of social participation with a measure of the perceived availability of help in a crisis (Phifer & Murrell, 1986). All subjects in this study were nondepressed at T1. Social support measured at T1 and the interactions of support with health at T1 and with loss events at T2 were significant contributors to a discriminant function that differentiated people who were depressed 6 months later from those who were not, after controlling for initial symptoms. However, it should be noted that only 12% of the variance in depressive onset was accounted for by a discriminant function that comprised initial symptoms, six other significant factors, and 18 nonsignificant predictors. Therefore, these results should be interpreted with caution.

In conclusion, there is some evidence that the relationships with depression of different aspects of extramarital social support remain significant when the effects of concurrent depressive symptoms are controlled. Substantively, the results of this research suggest that low social integration—measured with a multi-item questionnaire (Cutrona, 1984), by assessing subjects' social participation (Phifer & Murrell, 1986), or by examining the number of important relationships (Billings & Moos, 1985; Lin & Ensel, 1984)—may be characteristic of people prone to depression. This interpretation is based on evidence that level of social integration predicts the onset of depression (Phifer & Murrell, 1986) and the course of existing symptoms (Cutrona, 1984; Lin & Ensel, 1984) and distinguishes remitted depressives from control subjects (Billings & Moos, 1985). Conversely, receiving adequate support from a single extramarital confidant does not appear to be a prophylactic against the onset of a depressive episode (O'Hara, 1986). Finally, whereas global assessments of functional support significantly predicted the subsequent severity of depression, their interactions with stress did not (Cutrona, 1984), which suggests that these dimensions of support may also have a direct effect on the course of depression.

### *Marital Adjustment*

The relationship between disturbance in intimate interpersonal functioning and depression has received increased attention over the past decade. Three converging lines of research have provided evidence of this relationship. First, descriptive studies have suggested that marital conflict correlates highly with concomitant depression (Crowther, 1985; M. M. Weissman & Paykel, 1974) and with the course of depressive illness (Rounsaville, Weissman, Prusoff, & Herceg-Baron, 1979). In addition, marital therapy has been found to be effective in re-

ducing the symptoms of depression, both alone (Beach & O'Leary, 1986) and in combination with pharmacotherapy (Friedman, 1975). Second, researchers studying the interpersonal behavior of depressed people have identified dysfunctional patterns of communication in couples with a depressed spouse (Biglan et al., 1985; Hautzinger, Linden, & Hoffman, 1982; Kahn, Coyne, & Margolin, 1985; Kowalik & Gotlib, 1987; Merikangas, Ranelli, & Kupfer, 1979; Ruscher & Gotlib, in press). Compared with their nondepressed counterparts, depressed couples have been found to exhibit asymmetrical influence in settling disagreements, increased expressions of dysphoria, and lower levels of constructive problem solving, mutual self-disclosure, and reciprocal support.

A third line of research has suggested that the lack of a confiding, intimate relationship leaves individuals vulnerable to depression (Brown & Harris, 1978; Brown & Prudo, 1981; Costello, 1982; Roy, 1978). Although in some cases respondents in these studies had no partner or attachment figure, in others their primary relationship was assessed to be of low intimacy. This research points to the potentially pathogenic consequences of distressed intimate relationships; however, in many studies supporting the vulnerability hypothesis, retrospective assessment of subjects' interpersonal circumstances was conducted when respondents were symptomatic. Studies investigating the relationship between marital adjustment and depression that are less confounded by concomitant depression are listed in Table 5.

The majority of these studies compared the marital adjustment of remitted depressives with that of normal controls (Beach et al., 1983; Bothwell & Weissman, 1977; Dobson, 1985; Gotlib, 1986; Hinchcliffe, Hooper, & Roberts, 1978; Merikangas, 1984; Paykel & Weissman, 1973). With one exception (Dobson, 1985), the results of these studies support the hypothesis that marital dysfunction is an enduring aspect of former depressives' interpersonal functioning; however, a number of methodological issues are relevant to an interpretation of these results.

Most important, diverse dependent measures have been used in this research. In one study, for example, the foci of investigation were the patterns of communication between spouses (Hinchcliffe, Hooper, & Roberts, 1978).<sup>5</sup> Unfortunately, the extremely high number of statistical contrasts performed on these data renders the meaningfulness of significant results somewhat dubious. At a descriptive level, couples in this study with a currently depressed spouse exhibited an interactional style characterized by emotional outbursts, negative tension release, and mutual interruptions. Observed again following recovery from depression, these couples continued to demonstrate a high frequency of negative expression and to disrupt the flow of their conversations with more tension release behaviors. Although these results must be interpreted with caution because of the lack of control of Type I error rate, they do suggest that couples with a formerly depressed spouse may engage in more emotional discharge behaviors than do normal couples. This inter-

<sup>5</sup> The results of studies combined in Hinchcliffe, Hooper, and Roberts's (1978) book *The Melancholy Marriage* were also published separately. Four of the five individual references are listed in Table 5; the other is Hooper, Roberts, Hinchcliffe, and Vaughn (1977).

Table 5  
*Marital Distress*

Study	Subjects	Time lag	Type and measure of adjustment	Measure of depression	Results
<b>Predicting future depression</b>					
<i>Premorbid case-control comparison</i> O'Hara, 1986	Pregnant women	6 months	Marital support, adjustment (DyAS, SSI)	RDC	Cases had lower premorbid marital adjustment than did controls, but there were no premorbid differences in social support from spouse.
<i>Two-wave panel variations</i> Menaghan & Lieberman, 1986	Community sample	4 years	Feelings about daily life with spouse (Own measure)	Depression items from HSCL	Feelings about marriage (T1) predicted T2 symptoms.
Monroe, Bromet, Connell, & Steiner, 1986	Community women	1 year	Marital support, conflict (Own measure)	Depression items from HSCL	Marital support (T1) predicted T2 symptoms when initial symptoms, but not marital conflict, were partialled out.
<b>Postmorbid functioning</b>					
<i>Prospective case-control comparison</i> Beach, Winters, Weintraub, & Neale, 1983	Unipolar & bipolar patients	3-4 years	Marital adjustment (MAT)	RDC, DSM-III criteria	Recovered schizophrenics and RD > ND in distress at discharge. After 3-4 years, only unipolar RD had higher frequency of poor marital course than did ND.
Bothwell & Weissman, 1977	Formerly D patients	4 years	Role adjustment (SAS)	RDS	RD > ND in interpersonal friction and impairment in marital role.
Gotlib, 1986	Inpatients	1 month	Marital distress (MAT)	BDI, DSM-III criteria	Couples in which wife was remitted patient reported more distress than ND couples. Couples with RD husbands = ND couples in marital adjustment.
Hinchcliffe, Hooper, Roberts, & Vaughn, 1977	Inpatients and their spouses	3-12 months	Marital communication (behavioral observation)	Clinical diagnosis	RD couples > ND couples in negative expressiveness.
Hinchcliffe, Hooper, Roberts, & Vaughn, 1978	See Hinchcliffe et al., 1977				RD couples > ND couples in tension release behaviors.
Hinchcliffe, Vaughn, Hooper, & Roberts, 1978	See Hinchcliffe et al., 1977				D = ND before and after depressive episode in responsiveness during interactions.
Hooper, Vaughn, Hinchcliffe, & Roberts, 1978	See Hinchcliffe et al., 1977				Some differences between RD and ND couples in mutual interruptions and length of utterances.
Merikangas, 1984	Inpatients	12-36 months	Divorce	RDC	Divorce rate among RD eight times higher than that for general population.
Paykel & Weissman, 1973	Female patients—see Bothwell & Weissman, 1977	8 months	Role adjustment (SAS)	RDS	RD > ND in interpersonal friction and inhibited communication.
<i>Cross-sectional remitted case-control comparison</i> Dobson, 1985	Formerly D patients	Cross-sectional	Marital adjustment (SAS)	SADS, HRS-D	RD = ND in marital distress.

Note. DyAS = Dyadic Adjustment Scale; MAT = Marital Adjustment Scale; SAS = Social Adjustment Scale; SSI = Social Support Interview; DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.); HRS-D = Hamilton Rating Scale for Depression; HSCL = Hopkins Symptom Checklist-90 (Depression subscale); RDC = Research Diagnostic Criteria; RDS = Raskin Depression Scale; SADS = Schedule for Affective Disorders and Schizophrenia; RD = remitted depressives; D = depressives; ND = nondepressives; T1 = first assessment; T2 = second assessment.

actional style, in turn, may have deleterious effects on marital satisfaction and emotional well-being.

In two other studies divorce was used as the criterion. Merikangas (1984) found that the divorce rate for couples with a formerly depressed spouse, at a 1- to 3-year follow-up, was eight times greater than the rate for the general population. Similarly, Beach et al. (1983) included divorce, as well as self-reports of marital distress, as evidence of a poor marital course when former depressed patients were assessed at discharge from hospital and at a 3- to 4-year follow-up. Recovered unipolar and bipolar depressives, and schizophrenics, all reported a higher level of marital conflict at discharge than did normal controls; moreover, the three patient groups did not differ from each other with respect to reported level of marital distress. At follow-up, only the former unipolar depressed patient group differed significantly from the normal controls in having a higher frequency of poor marital course. One potential problem in interpreting the results of these studies is that the depressed/nondepressed status of probands at follow-up was not specified, and it is possible that some proportion of the "remitted" depressed patients were symptomatic. Although Beach et al. found differences between recovered patients and normal controls at discharge, patients who had relapsed between discharge and follow-up may account for the long-term differences between groups.

Judging from the results of the remaining studies with former patients whose recovery status was made explicit, this does not appear to be a viable explanation. Using an interview-based inventory, Paykel and Weissman (1973) found evidence of disturbed intimate interpersonal functioning in asymptomatic formerly depressed women 8 months after the remission of their symptoms. Compared to never-depressed controls, remitted female depressives exhibited inhibited communication and interpersonal friction in their intimate relationships. Four years after the remission of their symptoms, these recovered female depressives continued to experience more interpersonal friction and to be more impaired in their marital roles than control subjects (Bothwell & Weissman, 1977). Dobson (1985), however, was unable to replicate these results. Although remitted female depressives were found to score higher in their reports of marital adjustment than currently depressed women, and lower than normal controls, these differences did not attain statistical significance.

In a longitudinal study, Gotlib (1986) found evidence of gender effects in the relationship between marital distress and depression. Using a self-report measure of marital satisfaction, Gotlib assessed male and female depressed patients and their spouses both shortly after admission to hospital and again following discharge, at which time the patients were no longer depressed. Preliminary analyses indicated that when they were symptomatic and admitted to hospital, the patients' marital satisfaction scores were significantly lower than were those of normal controls. Following recovery, couples in which the wife had been depressed reported no significant change in their marital satisfaction, whereas couples in which the husband had been depressed reported significant improvement in their marital satisfaction. In fact, couples with a formerly depressed husband were not significantly different from the control couples in their reported levels of marital adjustment on discharge. It is interesting that the husbands of formerly depressed women reported

being the least satisfied with their marriages. One interpretation of this pattern of results is that women may be more tolerant of the interpersonal behaviors associated with depression than men are. Alternatively, depressive interpersonal tendencies may differ between males and females such that formerly depressed men and women behave differently with their spouses. Additional research is required to examine these issues.

On the basis of this research involving remitted depressives, the conclusion that marital distress, particularly among women, is an enduring consequence of depression appears warranted. The results of three prospective studies suggest that marital dysfunction may also be involved in the etiology of depression. Menaghan and Lieberman (1986) found that feelings elicited in respondents at T1 by having them reflect on their daily lives with their spouses significantly predicted depressive symptoms 4 years later, after controlling for T1 depression, age, sex, parental responsibilities, economic problems, and employment status. It is difficult to know how to interpret these results: That a certain level of marital distress would be directly related to a change in depression 4 years later is questionable. As Cohen and Wills (1985) pointed out, relatively short time lags might be most appropriate for studying depression when using a design of this type. Nevertheless, Menaghan and Lieberman's results do suggest that marital distress may have long-term effects on emotional health.

The two remaining prospective studies attempted to examine the independent relationships with depression of marital support and marital conflict or adjustment. Monroe et al. (1986) measured marital support with six items assessing positive aspects of the time the couple spent together. The operationalization of conflict focused on thoughts, feelings, and actions concerning marital problems. The correlation between the two measures was negative and significant, but not extremely high ( $r = -.48$ ). When both initial symptoms and marital conflict were statistically controlled, marital support was not found to be a significant predictor of subsequent depression; with only initial symptoms regressed out, however, a significant main effect for marital support was found. A third pattern of results, obtained when only data for initially nondepressed women with low marital conflict were used, suggests that there was an interaction between the control and predictor variables. After statistically controlling for both initial symptoms and conflict (a procedure that produced nonsignificant results using the full sample), support was found to be a significant predictor of subsequent depression. These findings indicate that the independent assessments of marital conflict and support, constructs usually combined as marital adjustment, may be feasible and useful. Furthermore, the pattern of results highlights the need for future investigations that employ a two-wave panel design to examine the interaction between initial symptoms and predictor variables.

Whereas Monroe et al. (1986) broadly equated support with positive aspects of the marital relationship and conflict with negative aspects, O'Hara (1986) used more conventional measures of social support and marital adjustment. Nondepressed women who became depressed postpartum did not differ from women who remained nondepressed in their reports of emotional or instrumental support received from their husbands during pregnancy. They did, however, report significantly lower

marital adjustment, as measured by the Dyadic Adjustment Scale (Spanier, 1976). This inventory has four subscales that measure Marital Conflicts, Mutual Activities, Marital Satisfaction, and Affectional Expression. O'Hara did not present data for these subscales, which makes the interpretation of the different findings for marital support and adjustment difficult because the two constructs appear to overlap considerably. It should be noted that the depressed group did report receiving less marital support postpartum than did the nondepressed group; a process whereby marital conflict leads to both a loss of marital support and depression, therefore, is consistent with these results.

In summary, research that has attempted to control for the effects of concurrent depression provides considerable support for the hypothesis that disturbances in intimate interpersonal functioning are both antecedents and sequelae of depression. The positive relationship between marital distress and depression is robust with respect to research design (Bothwell & Weissman, 1977; Monroe et al., 1986; O'Hara, 1986), the operationalization of marital adjustment (Merikangas, 1984; Monroe et al., 1986), and time lag between assessments (Bothwell & Weissman, 1977; Gotlib, 1986). Preliminary results further suggest that residual impairment in marital functioning may be gender specific (Gotlib, 1986). Finally, recent evidence indicates that differentiating between the constructs of marital support and marital conflict may yield more specific knowledge concerning the role of intimate relationships in the process of becoming depressed (Monroe et al., 1986; O'Hara, 1986).

### *Coping Style*

Coping style refers to habitual cognitions and behaviors that an individual uses to minimize the impact of stressful circumstances (Billings et al., 1983; Pearlin & Schooler, 1978). Two broad categories of both the cognitions, or appraisals, and behaviors comprising coping have been suggested (Lazarus & Folkman, 1984). Primary appraisal involves the determination of the overall character of the event (e.g., threatening, pleasurable, or important). Secondary appraisal is the comparison of available resources with those deemed necessary to cope with a given situation. The particular coping behavior used in a given situation will depend on the outcome of these appraisal processes, which in turn are affected by the success of past coping efforts. Coping behaviors also have been conceptualized as a dichotomy. Whereas problem-focused behavior is directed toward removing or reducing the source of stress, emotion-focused behavior is aimed at affect regulation or reduction.

The idea that coping style may be involved in the etiology or maintenance of depression has only recently been the focus of empirical research. Nevertheless, the available evidence suggests that symptomatic depressives do exhibit coping styles that are different from those of nondepressed persons. Specifically, compared with nondepressed individuals, depressed persons have been found to perceive themselves as having more "at stake" when appraising stressful situations (Folkman & Lazarus, 1986) and as needing more information before being ready to act (Coyne, Aldwin, & Lazarus, 1981). With respect to behavior, the results of a number of studies suggest that depressives engage in more emotion-focused coping than do nonde-

pressed individuals. This class of behaviors includes hostile confrontation (Folkman & Lazarus, 1986), emotional discharge (Billings et al., 1983; Billings & Moos, 1984; Mitchell, Cronkite, & Moos, 1983), and seeking emotional support (Coyne et al., 1981; Folkman & Lazarus, 1986). Although depressed individuals in one sample of subjects were found to use fewer problem-solving behaviors than did nondepressed persons (Billings et al., 1983; Billings & Moos, 1984; Mitchell et al., 1983), other studies have not been able to replicate this finding (Coyne et al., 1981; Folkman & Lazarus, 1986). Finally, Mitchell and Hodson (1983) obtained a relationship between depression and a high level of avoidance coping combined with a low level of active cognitive and behavioral coping strategies.

Considered collectively, the results of these cross-sectional studies suggest that the coping style of depressed people differs from that of nondepressed people. By definition, coping behaviors mitigate the pathogenic effects of major life events as well as those of chronic role strains and "microstressors" (Coyne et al., 1981). If a coping style is effective, the onset of depression is less likely. By corollary, therefore, the coping style of people who are prone to depression should differ from that of people who are not prone to becoming depressed.

We found only two studies of coping that met the design criteria established for this review; both assessed probands' coping style during the depressive episode and again following recovery (see Table 6). Billings and Moos (1985) asked subjects to select a recent stressful event and to rate the frequency of their use of a number of coping behaviors falling into the two classes discussed above. Problem-focused coping included information seeking and problem solving; emotion-focused coping was composed of affect regulation and emotional discharge. At intake, depressed patients reported significantly more information seeking and emotional discharge, and fewer problem-solving behaviors, than did nondepressed controls. After recovery, the remitted patients differed from the nondepressed controls only in reporting more emotional discharge behaviors. This post-morbid difference was found despite a significant decrease in the patients' emotional discharge coping between intake and follow-up.

In the second study, Parker and Brown (1982) derived six factors from a list of behavior changes that normal subjects indicated they would make to cope with two hypothetical threatening interpersonal events. Depressed patients were asked to indicate their preference for, and their expected efficacy of, these coping behaviors in response to the same hypothetical events. Parker and Brown found that differences between depressed and nondepressed subjects, especially evident in the depressed subjects' lower endorsements of socialization and distraction, disappeared almost entirely when these subjects were assessed again following recovery.

Conclusions are difficult to draw on the basis of these two methodologically dissimilar studies. Nevertheless, we should point out that the lack of significant results obtained by Parker and Brown (1982) may not be inconsistent with the single significant finding of Billings and Moos (1985), primarily because there does not appear to be a dimension in Parker and Brown's coping measure that is the equivalent of emotional discharge coping. The second methodological difference between these two studies concerns the nature of the stressors involved.

Table 6  
Coping Style

Study	Subjects	Time lag	Type and measure of coping	Measure of depression	Results
<b>Postmorbidity functioning</b>					
<i>Prospective case-control comparison</i>					
Billings & Moos, 1985	In- and outpatients	1 year	Problem-focused and emotion-focused behaviors used to cope with actual event (HDL)	DSSI	RD > ND on emotional discharge coping only.
Parker & Brown, 1982	Outpatients	14 weeks	Ratings of preference for and effectiveness of 6 dimensions of antidepressive behavior in response to hypothetical events (Own measure)	DSM-III criteria	RD = ND on all dimensions.

Note. HDL = Health and Daily Living Form; DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.); DSSI = Depression Symptom Severity Index; RD = remitted depressives; ND = nondepressives.

Whereas Billings and Moos assessed coping behaviors elicited by one real event chosen by each respondent, Parker and Brown examined subjects' responses to two hypothetical events. Coping behaviors are responses to stress; therefore, the elucidation of the relationship between coping style and depression may require the further specification or classification of the stressful events that elicit similar responses. It does appear, however, that formerly depressed people may engage in more emotional discharge behavior in response to the demands of negative events than do nondepressed people. Clearly, additional research is required both to assess the influence of coping style on future depression and to replicate results suggesting that increased emotional discharge coping is an enduring consequence of depression.

### Summary and Conclusions

In the remainder of this article, we present our conclusions on the nature of the psychosocial deficits associated with depression. We first discuss cognitive variables and then consider the impact of personality and social factors. Finally, we attempt to integrate what is known about the relationships among these latter variables, suggest ways in which they may influence the development of depression, and outline specific issues that must be addressed in future research.

### Cognitions

Our review of the literature suggests that there is little empirical evidence of a stable cognitive vulnerability to depression. A self-deprecating attributional style and a high number of dysfunctional attitudes appear to be two among many cognitive abnormalities that wax and wane with the onset and remission of depression. Little support was found for the causal hypotheses of either the reformulated learned helplessness model

(Abramson et al., 1978) or the cognitive vulnerability model (Beck et al., 1979). Substantial evidence was found of increased numbers of dysfunctional cognitions in symptomatic depressed patients, but these cognitions do not appear to precede the onset of depression, to predict an increase in the severity of subsequent symptoms, or to be evident following remission. Although a negative attributional style was found to be associated with the future severity (Cutrona, 1983) and longevity (Metalsky et al., 1987) of dysphoria among normal subjects, and to be involved in the process of recovery from depression (Lewinsohn et al., 1981; O'Hara et al., 1982), it appears that existing cognitive models of depression do not accurately fit the majority of the accumulated data.

Before turning to a brief discussion of the possible role of cognitive abnormalities in the development of depression, it may be useful to consider more fully alternative explanations for the failure of research to detect a cognitive vulnerability to depression. It has been argued that nonsignificant main effects for cognitions are not inconsistent with the predictions of cognitive theories (Metalsky et al., 1982; Riskind & Rholes, 1984) but that proper tests of the models would examine the relationship with depression of the interaction of cognitions with stressful life events. Several counterarguments to this position have been presented throughout this review. First, if a cognitive vulnerability to depression does exist, it should be evident in remitted patients. That it has not been observed requires explanation and leads directly to the next alternative conclusion—that is, that pharmacotherapy, with which most of the remitted depressives in these studies were treated, has an enduring and specific effect on patients' habitual cognitive style. Although this seems unlikely, it is possible. However, as has been argued elsewhere, if remitted patients exhibit normal, or nondepressed, self-schemata and attributional styles, then their increased vulnerability for future depression is not adequately explained by the cogni-

tive theories of depression (cf. Hollon et al., 1986; Simons et al., 1984). This inconsistency may be accounted for by postulating that dysfunctional cognitions become inaccessible to measurement following symptomatic recovery and need to be primed by stressful experiences (cf. Coyne & Gotlib, 1983; Riskind & Rholes, 1984). Again, this was not found to be the case by Reda et al. (1985), who observed that although recovered depressives no longer endorsed the majority of the dysfunctional attitudes that they had held during their illness, a small number of these negative cognitions concerning dependence and autonomy did remain elevated and accessible postmorbidly. Furthermore, preliminary research suggests that dysfunctional cognitions are not primed by all negative events rated by subjects as being stressful (P. A. Barnett & Gotlib, in press); that is, the interaction between the DAS and a measure of the subjective stressfulness of the various life events experienced by subjects over a 3-month period was not found to be a significant predictor of subsequent depression.

Thus, it is becoming evident that to remain tenable, cognitive theories must invoke increasingly specific environmental stressors and smaller subsets of cognitions. This introduces the final and most convincing alternative explanation for the lack of evidence in support of the cognitive theories of depression: The sensitivity or specificity of the measures of both dysfunctional cognitions and life events may be too low to detect real effects. For example, a recent factor analytic study has suggested that the DAS measures at least two orthogonal dimensions (Cane, Olinger, Gotlib, & Kuiper, 1986). Recent elaborations of the cognitive vulnerability theory of depression have outlined different superordinate schemata, or personality types, that appear to correspond to these substantive dimensions (Beck, 1983). Implicit in these theoretical developments is the recognition of the complexity of both vulnerability factors and personally relevant stressors. It may be that widely used measures of cognitive vulnerability such as the ASQ and the DAS must be modified to increase their sensitivity and specificity. Some modifications to these measures have already been proposed (Cutrona et al., 1985; Metalsky et al., 1987; Olinger et al., 1987), and the development of the Sociotropy-Autonomy Scale by Beck and his colleagues might also be seen as a step in this direction.

Future research on a cognitive vulnerability to depression will likely also benefit from the development of a more precise life-event typology. Research could then focus on the nature and stability of negative biases associated with probands' appraisals of different categories of stressors (e.g., chronic versus acute; interpersonal versus nonsocial). Preliminary research has suggested that cognitions may differentially mediate the depressogenic effects of these different types of stress (P. A. Barnett & Gotlib, in press; Hammen, Marks, Mayol, & deMayo, 1985). Hammen, Marks, Mayol, and deMayo (1985), for example, found that correlations between stressful interpersonal events and depression were higher among subjects whose schemata were interpersonally, or dependency, oriented than among those whose schemata were judged to be achievement oriented. Similarly, within the dependency-schema group, interpersonal events were more highly correlated with depression than were achievement-related events.

Notwithstanding these suggestions for future research, the

broader implications of our conclusions call into question the primacy of cognition in the etiology of depression. The lack of evidence of the stability, cross-situational consistency, and increased frequency among asymptomatic depressives of dysfunctional cognitions suggests that future research should shift away from the conceptualization of depressogenic cognitions as trait-like causal entities that are not affected by current life experiences. In fact, two recent integrative theories of depression have suggested that, rather than being a stable aspect of the individual, depressive cognitions arise as part of a normal dysphoric response to negative life events, particularly those that threaten self-worth (Lewinsohn, Hoberman, Teri, & Hautzinger, 1985; Pyszczynski & Greenberg, 1987). For most individuals, these cognitions and dysphoria are time-limited. However, some individuals do go on to develop a major depressive episode, and these individuals are hypothesized to differ from those who do not, not in their habitual cognitive style, but in their premorbid self-esteem and interpersonal behavior (Pyszczynski & Greenberg, 1987). Individuals with few sources of self-worth are postulated to have a tendency to prolong their use of these negative cognitions, with the result of exacerbating their initial dysphoria and perhaps activating other depressive symptoms. Parenthetically, it is worth mentioning that certain results of the present review, to be discussed next, provide considerable empirical support for the self-worth and social diatheses integrated by Pyszczynski and Greenberg in their self-regulatory perseveration model of depression.

This formulation is quite different from that of the cognitive models examined in this review. Nevertheless, the results obtained by Metalsky et al. (1987), which suggest that making global and stable attributions prolongs an apparently normal dysphoric response to a disappointing event, fit well with the more recent theories. Although a self-deprecatory attributional style may not cause depression per se, it is possible that making an internal, stable, and global attribution for the cause of an event that threatens one's self-esteem and social equilibrium may intensify a negative affective response and may cause other depressive symptoms. Important questions for future research in this area include whether those who make such attributions differ from those who do not in significant ways, and whether such attributions precede the onset of a major episode or emerge as part of the depressive syndrome.

In conclusion, the classification of depressive cognitive abnormalities as concomitants, or symptoms, of depression may be premature. Although there is little evidence of a stable cognitive vulnerability to depression, new theories implicating cognitive functioning in the development of syndromal depression have yet to be fully tested. The results of this review do suggest, however, that theoretical and methodological refinements are required with respect to the reformulated learned helplessness and cognitive vulnerability models of depression. A focus on idiographic cognitive responses to major stressors, rather than on global negative tendencies or biases, might prove to be more fruitful than has the search for a cognitive predisposition to depression.

### *Personality and Social Factors*

Some of the abnormalities in social functioning and personality observed among depressed persons do not appear to have

direct causal relationships with this disorder. Specifically, increased levels of neuroticism and an imbalance in emotion-focused versus problem-focused coping seem to be time-limited concomitants of depression. These phenomena may be responses to the dysphoria associated with depression. As depressive emotions dissipate, these responses also disappear, although some evidence of increased neuroticism (Hirschfeld, Klerman, Clayton, & Keller, 1983; Kendell & DiScipio, 1968) and higher-than-normal frequencies of emotional-discharge coping behavior (Billings & Moos, 1985) has been found among remitted depressives. This latter result is congruent with the results of the series of studies by Hinchcliffe and her colleagues (Hinchcliffe, Hooper, & Roberts, 1978), who found that the interactions of couples with a formerly depressed spouse were characterized by negative emotional expressiveness and tension release behaviors. We must note again, however, that because of the large number of comparisons made in these studies, these may have been chance findings. We recommend, therefore, that future research examine the emotional-discharge behaviors of people who are prone to depression, particularly in the context of their marital interactions. Some more detailed suggestions for this research are outlined in the final section of this article.

In contrast to neuroticism and coping, four psychosocial variables were found to have relationships with past or future depression that remained significant when the effects of concurrent symptoms were controlled. These variables are marital distress, social integration, extraversion-introversion, and interpersonal dependency. There is consistent evidence that some disturbance in each of these four domains characterizes depressives' postmorbid or intermorbid functioning and, further, that marital distress and low social integration may influence the onset of depressive symptoms. Because of a lack of appropriate research, no conclusions regarding the effects of dependency and introversion on the development of depression may be drawn. These two variables are imputed to be stable traits, which suggests that the consistent postmorbid differences between depressed patients and control subjects would also be found premorbidly. This extrapolation must await empirical confirmation, but in the following discussion, we offer some suggestions as to how these traits might influence the development of depression.

For the most part, previous research, both cross-sectional and longitudinal, has investigated the independent relationships of each of these variables with depression. Thus, there is little information to integrate concerning their potential interactive effects. The results of this review might best be integrated by considering their support for a number of recent theories that appear to converge on a single general hypothesis: Depression is caused by the disruption or loss of a central source of self-worth among individuals who do not have satisfying alternative sources of self-esteem (Arieti & Bemporad, 1980; Hirschfeld et al., 1976; Linville, 1985; Oatley & Bolton, 1985; Pyszczynski & Greenberg, 1987). The losses identified in these theories most often involve important interpersonal relationships or social roles, but disappointments in the attainment of achievement-related goals have also been recognized as precipitating events. That this review has identified social factors as potential antecedents or sequelae of depression may be due to a bias in choosing research on such variables for review and does not imply

that nonsocial functioning and events are unimportant. Nevertheless, this possible bias does not diminish the considerable promise that the interpersonal domain appears to hold for future research on the etiology of depression, and in keeping with the specific results of this review, therefore, in the following discussion we focus on the interpersonal factors associated with depression.

As we noted earlier, vulnerability to depression is hypothesized to derive from two related tendencies or conditions. The first is the overinvestment of self-esteem in a single or restricted number of relationships or roles. The second is the failure to develop and maintain secondary roles (Oatley & Bolton, 1985), greater self-complexity (Linville, 1985), or diverse sources of self-esteem (Arieti & Bemporad, 1980; Hirschfeld et al., 1976). Some theories have provided detailed descriptions of the affective, cognitive, and behavioral responses of vulnerable individuals to depressogenic events (Lewinsohn et al., 1985; Pyszczynski & Greenberg, 1987). The social vulnerability factors, however, have received little attention beyond their simple identification.

The results of this review not only provide substantial evidence of the existence of these vulnerabilities but also suggest ways in which they may develop and be maintained. Each of the variables identified in this article as being an antecedent or a consequence of depression describes some aspect of interpersonal or social behavior. Two of these, dependency and introversion, describe remitted patients' interpersonal orientation; that is, these traits reflect global tendencies with respect to social behavior. Dependency is defined as the tendency to rely, almost exclusively, on the positive regard of important others for the maintenance of one's self-esteem (Hirschfeld et al., 1976). High dependency is hypothesized to develop among people who experienced difficulties in establishing adequate secure relationships early in life (Blatt, 1974; Hirschfeld et al., 1976). As a result of this developmental disruption, these individuals are thought to become overly preoccupied with interpersonal security and to experience problems in maintaining positive feelings about themselves without external support.

In contrast to dependency, introversion is a personality style that implies reticence in social interaction and a generalized tendency to avoid social situations (H. J. Eysenck & M. W. Eysenck, 1985). Therefore, introverts are more likely to be socially isolated than are extraverts. We may speculate that introverts prefer to interact with one or two people who are known well, rather than with a larger group of friends, associates, or strangers. Thus, more introverted people would be more likely to have a smaller number of social involvements than would more extraverted people, an implication supported by the results of previous research (Henderson et al., 1981). Furthermore, the tendency to restrict the range of one's social participation might be expected to decrease the number of potential sources of emotional support so important to interpersonally dependent people (cf. Lewinsohn, 1974). Finally, it may be that interpersonal dependency and introversion are moderately correlated: The sense that one's self-esteem is contingent on social approval may exacerbate fears of rejection or disappointment in relationships, thereby increasing the tendency to avoid many kinds of social opportunity.

The consequences of high interpersonal dependency and in-

troversion, therefore, may be precisely the conditions described by some theorists as predisposing to depression; that is, narrowly defined or derived self-worth may co-occur with social isolation among dependent introverts. Direct evidence of the overinvestment of self-esteem in a primary role or relationship among depression-prone individuals is currently lacking and must be inferred from the results of research on interpersonal dependency. Nevertheless, the results of this review do suggest that social integration or, conversely, social isolation, is involved in the etiology of depression. The loss of any meaningful role or relationship by a socially isolated person might elicit a dysphoric response by eliminating one of a restricted number of sources of self-definition and worth. Thoits (1983) obtained some support for this hypothesis in research on general psychological distress. Not only did a lower number of roles at T1 predict a higher level of symptoms at T2 after controlling for concurrent symptoms, but an index of change in the total number of roles for each person from T1 to T2 also significantly predicted distress at T2.

More specifically, however, the results of this review suggest that the disruption of a primary relationship, such as the marital relationship, may lead to depression. Indeed, there is evidence that the other factors identified in this review as antecedents or sequelae may lead indirectly to depression through their effect on the quality of primary relationships. For example, previous research has found that introversion and social isolation are inversely related to marital satisfaction (Barry, 1970; Renne, 1970), whereas self-esteem is positively correlated with marital adjustment (L. R. Barnett & Nietzel, 1979). One explanation for this pattern of relationships is that isolated individuals with vulnerable self-esteem may attempt to decrease their feelings of insecurity, isolation, and impoverishment by making exaggerated demands for support from their spouses. Coyne (1976; Coyne et al., 1987) described a process whereby depressed people alienate those closest to them through escalating demands for support and other depressive behaviors. The results of the prospective research reviewed in the present article suggest that this alienation within the individual's intimate interpersonal system may precede, and possibly precipitate, the onset of depressive symptoms (O'Hara, 1986; see also Gotlib & Hooley, 1988). The results of other research suggest further that certain personality tendencies may serve to maintain this depressogenic system. For example, Kelley and Conley (1987) followed couples for over 40 years in order to assess the relationship between personality and marital adjustment. They found that men who were in lasting but unhappy marriages were more introverted when they got married than were men whose marriages ended in separation. This finding suggests that whereas marital dysfunction may motivate an extraverted man to relieve his distress by pursuing alternative relationships, the more introverted man may tend to remain in an unhappy marriage despite the distress. Thus, the tendency to restrict one's social opportunities, combined with an excessive need for emotional support, may influence the development of depression through its effect on the supportive quality of the interpersonal environment.

Two mechanisms through which marital distress may lead to depression have been suggested by the results of previous research. The first is consistent with the theoretical framework

discussed here; that is, marital conflict may be the "final straw" that precipitates depression. In support of this hypothesis, Paykel et al. (1969) found that an increase in marital disputes in the 6 months prior to seeking treatment was the most frequently reported life event among a group of depressed female patients. Second, marital distress may chronically erode self-esteem and coping resources, leading to the onset of a depressive episode by decreasing the individual's capacity to cope effectively with other stressful environmental demands (Pearlin, Lieberman, Menaghan, & Mullan, 1981; Pearlin & Schooler, 1978). According to this hypothesis, marital distress would be classified as an interpersonal diathesis, rather than a triggering event. It may be that social exits, brought about by divorce or death, have more immediate depressogenic consequences than the mere disruption of the marital relationship.

Marital distress may also exacerbate difficulties experienced in extramarital relationships (Coyne & DeLongis, 1986), thereby increasing introverted behavior and social isolation. In a similar manner, the absence of a marital partner may hasten the onset of depression in otherwise vulnerable people (Brown & Harris, 1978). It may be that a close relationship with family or friends becomes the forum for the increased dependent or demanding behavior of unmarried social isolates. The alienation of these sources of support would serve to increase dysphoria and would leave the isolated individual even more vulnerable to the impact of negative life events.

#### Directions for Future Research

In this section, we present some suggestions for future research by making more explicit some of the research questions formulated or reviewed in the previous section. Although in some cases the relationships between individual variables must be explored, we anticipate that future research will benefit from examining the interactive effects of the antecedents and sequelae of depression identified in this review.

Beginning with the personality variables, the effects that interpersonal dependency and introversion have on the development of depression should be investigated. This may not be advisable, however, until more is known about the nature and measurement of dependency in particular. Although at least three new measures of this trait have been developed recently, each appears to have a number of serious conceptual or psychometric shortcomings, ranging from high correlations with social desirability to questionable weighting systems used in scoring the responses (Beck et al., 1983; Blatt et al., 1976; Hirschfeld et al., 1977). Research is required to demonstrate that measures of interpersonal dependency are not simply inventories of psychological distress, social desirability, low self-esteem, or introversion. Most important, the extent to which high dependency reflects the actual overinvestment of self-esteem in primary relationships is not known. Therefore, we suggest that future research begin with a careful examination of the construct validity of measures of interpersonal dependency.

The task of validating a measure of the tendency to rely principally on positive emotional regard for the maintenance of self-esteem is a formidable one and might best be accomplished using a multitrait-multimethod approach (D. T. Campbell & Fiske, 1959). This research could compare the various mea-



asures of dependency that have been developed with self-report measures of both related and unrelated factors, such as need for affiliation and need for achievement, and with different operationalizations of similar constructs, such as dependency schemata (Hammen, Marks, Mayol, & deMayo, 1985). External criteria such as therapist or spouse ratings might also be included. A different strategy would be required to examine the effect of interpersonal dependency on the lability of self-esteem. This might involve grouping subjects on the basis of the presence or absence of interpersonal losses or other traumas to their relationships. Dependency would be expected to moderate the effects of these traumas on subjects' self-esteem and subsequent level of depression. Finally, the influence of this trait on emotional-discharge coping and behavior during marital interaction may reveal behavioral differences between individuals high and low in dependency. Such preliminary research is central to the future investigation of interpersonal dependency as a risk factor for the development of depression.

With respect to broad patterns of social behavior, we have suggested that low social integration may be the result of certain personality tendencies, the most important of which is introversion. The relationship between extraversion-introversion and the availability and adequacy of social support has been noted elsewhere (Monroe & Steiner, 1986) and has also been reported in research on general psychological distress (Henderson et al., 1981). Although it is possible that this relationship is simply a function of the overlap of measurement instruments, it may also be that social isolation is less a function of the unavailability of resources in the social environment than it is the result of a personal preference for solitude, a discomfort in and avoidance of social interaction, or an alienating interpersonal style. Preliminary investigation has suggested that introverts differ from extraverts in a variety of social behaviors and activity patterns (A. Campbell & Rushton, 1978; Furnham, 1981). Future research might examine the specific deficits in different aspects of social support that correlate with introversion, as well as the emotional responses of introverted people to social interaction.

Another approach to the study of social integration that might benefit from a consideration of personality dimensions concerns the link between a paucity of self-definitional roles and dysphoria (Oatley & Bolton, 1985; Dance & Kuiper, 1987; Linville, 1985; Radloff, 1975; Repetti & Crosby, 1984; Thoits, 1983). Future research might investigate the extent to which interpersonal dependency and introversion mediate the relationship between few identities and depression. Whereas differences in introversion may help to explain individual differences in identity accumulation, interpersonal dependency would be expected to moderate the value placed on a given identity. Social roles providing positive emotional support or approval should be most valued by dependent people, whereas nonsocial roles involving feelings of mastery, for example, would not be as important. Those social roles in which positive emotional regard could not be elicited might be experienced as stressful by people who are high in interpersonal dependency. Finally, the combination of being both dependent and introverted is hypothesized to result in narrow self-definition, with an emphasis on primary relationships and few secondary roles. This central hypothesis could be tested using a number of different methodologies, but Linville's (1985) self-complexity

paradigm, which includes the use of role sorts, might be particularly appropriate. An examination of these personality variables could increase the explanatory power of role theories.

The reciprocal effects of the four variables and their interactive effects on the development of depression may become most evident in the study of intimate interpersonal functioning. Specifically, we have suggested that future research investigate the influence of interpersonal dependency, introversion, and social isolation on the development of marital distress and subsequent depression. We noted earlier that although the relationship between marital satisfaction and interpersonal dependency has not been explored, there is some evidence that introversion and social isolation are negatively related to marital adjustment (Barry, 1970; Renne, 1970). However, these relationships may be different among men and women (cf. Bentler & Newcomb, 1978; Kelley & Conley, 1987). Thus, future research could investigate the effects of introversion and social isolation on the differential development of marital distress in men and women. Furthermore, gender differences in the effect of marital distress on the development of depression could be explored (cf. Gotlib, 1986). This research might be particularly important for establishing the external validity of existing research on the relationship between marital distress and depression. Although it is couples and not individuals who are classified as maritally distressed, the identified patients in most previous research have been female. There are considerably fewer data on whether disturbed intimate interpersonal functioning leads to depression in men.

Finally, one strategy for the future investigation of the relationship between coping behavior and depression would involve the assessment of differences in coping with marital stress. We have suggested that threats to the self-esteem of interpersonally dependent people will increase their demands for support within their primary relationships. However, these demands may be counterproductive. Pearlin and Schooler (1978), for example, found that self-reliance and a reflective, problem-solving approach were more effective in reducing marital stress than was help-seeking or "the eruptive discharge of feelings" (p. 11). Interesting preliminary evidence indicates that a high frequency of emotional-discharge coping may be characteristic of people prone to depression (Billings & Moos, 1985). The relationships among this style of coping, interpersonal dependency, and marital distress could be examined with reference to their ability to predict future depression.

In concluding, we should note that although an empirically based interpersonal approach to the study of depression is a recent development (cf. Coyne et al., 1987), Becker (1964) made the following observations over two decades ago:

The depressed person . . . suffers from a too uncritical participation in a limited range of monopolizing interpersonal experiences . . . [He] has firm patterns of interpersonal behavior, but a narrow repertoire of explicit vocabularies of choice. (pp. 131-132)

The primary purpose of this article was to differentiate concomitant or symptomatic changes in depressed people's thoughts, personality, and behavior from abnormalities that may be antecedents or sequelae of depression. The results of this review suggest, as Becker postulated, that proneness to depression may derive in large part from restricting oneself to "a

limited range of monopolizing interpersonal experiences." Among remitted depressives, these experiences center on the derivation of support and self-esteem from a restricted number of relationships, the most important of which, the marital relationship, is likely to be plagued with conflict. This situation is maintained by interpersonal tendencies or traits that make it distressing and difficult for recovered patients to pursue new social opportunities. We have suggested that these variables may function as vulnerability factors to depression. It is possible, of course, that future research may reveal these interpersonal tendencies instead to be enduring consequences of a depressive episode, although it is unlikely that this distinction can be made without additional premorbid research. In either case, it is our hope that the differentiation in this review of concomitants from antecedents or consequences of depression, and the identification of four particularly promising variables, will contribute to the initiation of this research.

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