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A diathesis-stress conceptualization of expressed emotion and clinical outcome

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Abstract

A sizeable body of research has demonstrated that expressed emotion (EE) predicts clinical relapse in a number of distinct psychiatric disorders. These findings have provided the impetus for the development of interventions that attempt to reduce patients' relapse rates by modifying aspects of the family environment believed to be associated with high levels of EE. Despite the efficacy of these treatments, however, we know little about how EE develops in relatives of psychiatric patients or about the mechanisms through which high EE leads to relapse. Moreover, there is not a coherent theory that attempts to integrate findings concerning the impact of high EE on relapse in different disorders. The purpose of this article is to elucidate a diathesis-stress conceptualization of EE to explain both the development and manifestation of high EE in relatives of disordered patients and the impact of high EE on the course of patients' disorders. In this context, we use a diathesis-stress perspective to examine why EE predicts symptom relapse and poor clinical outcome in schizophrenia, depressive disorders, and borderline personality disorder. We conclude by discussing treatment implications of the diathesis-stress perspective and by outlining what we believe are fruitful directions for future research.

Keywords: Diathesis-stress, Expressed emotion, Relapse

Conventional wisdom regards stress as a bad thing, and the literature that concerns psychopathology is no exception. Indeed, one of the most influential heuristics for understanding the development of psychopathology is the diathesis-stress model (e.g., Monroe & Simons, 1991; Rosenthal, 1970; Zubin & Spring, 1977). According to this model, various forms of psychopathology result from the interaction between stress and some form of vulnerability toward a specific disorder. However, although the diathesis-stress model forms the conceptual underpinning of most current thinking about psychopathology, it does not generally structure the way in which psychopathology research is conducted. This is not to say that we do not study stress or that we do not study vulnerability to psychopathology. We do. Nevertheless, it is broadly the case that the researchers who study vulnerability are not the same investigators as those who study stress.

Given the complexities of scientific research and the high level of expertise needed to study even one construct well, this is hardly surprising. One unfortunate consequence of this conceptual and practical separation is the restriction it can impose on the way we think about the phenomena we study. This problem is particularly acute in the area of expressed emotion (EE) research. EE is a characteristic of the family environment that has been found to predict relapse in a broad range of psychiatric and psychopathological conditions (Butzlaff & Hooley, 1998). Because considerable empirical data link EE and symptom relapse, EE is widely regarded as a form of psychosocial stress. Over the past two decades, the EE construct has stimulated the development of family-based treatments for schizophrenia that seek to reduce patients' relapse rates by modifying construct-relevant aspects of the family environment (Hogarty et al., 1986; McFarlane, Link, Dushay, Marchal, & Crilly, 1995; McFarlane et al., 1995).

The success of these efforts has been gratifying and has resulted in tangible clinical benefit for many patients and their families. However, the move from psychosocial construct to psychosocial treatment has also had negative consequences. It can be argued that intervention efforts have outstripped knowledge of the EE construct and have brought premature closure to theoretical development in this field.

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In this article we focus attention back onto two basic questions that have to be addressed if the EE construct is to make any further contribution to the understanding and treatment of psychopathological disorders. We attempt to move beyond the simple conceptualization of EE as a psychosocial stressor that must be modified if we wish to reduce patients' relapse rates. We contend that both theoretical and clinical developments in this area might be facilitated by some reorganization of the way we think about and use the EE construct. More specifically, we believe that there is much to be gained from reconceptualizing this familiar construct within a diathesis-stress framework. Accordingly, in the first part of the article we use the structure of the diathesis-stress model to organize our thinking about why high EE develops and is manifested. This is not a question that has previously been addressed with an explicit diathesis-stress conceptualization. Nevertheless, we believe that such an approach has much to offer, not least because it moves us away from simple notions that conceptualize EE either as a trait variable or as a state-dependent construct. In the second part of the article we use a diathesis-stress perspective to focus on why EE predicts symptom relapse and poor clinical outcome in schizophrenia, depressive disorders, and borderline personality disorder.

Neither high EE nor psychiatric relapse occurs in a vacuum. The diathesis-stress perspective holds the advantage of requiring us to focus attention away from simple main effects and toward interactions between vulnerability and stress variables. This perspective also encourages us to recognize that both patients and relatives are involved in a system of mutual influence in which each provides the stress that acts on the intrinsic vulnerabilities of the other, even after the disorder has developed. It is our hope that a more systematic focus on these reciprocal influences will lay some much needed theoretical groundwork for future research in this area. Of course, all of our discussions presuppose that EE can be reliably measured and that it plays an independent and causal role in the relapse process. Accordingly, some background about these features of the EE construct is in order.

Expressed Emotion: Description and Measurement

Despite its somewhat inappropriate name, EE is not a measure of emotional expressiveness. Rather, it is a measure of the extent to which an individual family member of a psychiatric patient talks about that patient in a critical or hostile manner or in a way that indicates marked emotional overinvolvement or overconcern. Note that EE is a characteristic of family members and not of the patients themselves. Ratings of criticism and hostility involve both the content of the remark and the changes in voice tone that occur when the relative is speaking about the patient. Critical remarks are those that, explicitly or implicitly, reflect dislike or disapproval of something the patient does (e.g., "I get annoyed when he sits around smoking and doing nothing"). More extreme remarks that criticize the patient for who he or she is rather than his or her specific behaviors (e.g., "This kid is a con-artist") are rated as denoting hostility. In sharp contrast, the rating of emotional overinvolvement (EOI) reflects a dramatic, overprotective, devoted, or self-sacrificing response to the patient's illness that is out of proportion to circumstances (e.g., "I can't leave the house without worrying about him. What if I am at work and he needs me?"). These attitudes are assessed by a trained coder on the basis of a 1 to 2 hour private and audiotaped interview with a patients' key relative or relatives (see Vaughn & Leff, 1976b). Relatives are rated high in EE if they make an above-threshold number[1] of critical comments, show any evidence of hostility, or demonstrate marked evidence of EOI.

Expressed Emotion and Relapse

It is now very clear that high family levels of EE are reliably associated with higher rates of relapse in patients with schizophrenia. In a meta-analysis of 26 studies, Butzlaff and Hooley (1998) demonstrated that living in a high EE home environment more than doubled the baseline relapse rate for schizophrenic patients 9 to 12 months after hospitalization. Overall, the weighted mean effect size of the association between EE and relapse in schizophrenia was $r = .31$. Additional statistical analyses (e.g., file drawer statistic) demonstrated that the magnitude of this effect would not change appreciably even if new replications (or nonreplications) were added to the literature.

If EE were a valid predictor of poor outcome solely in schizophrenia, it would certainly be a construct worthy of empirical attention. However, EE has also been found to be predictive of relapse in other psychopathological conditions. For example, EE predicts relapse in patients with unipolar depression (e.g., Vaughn & Leff, 1976a; Hooley, Orley, & Teasdale, 1986). Depressed patients who return home from the hospital to live with relatives (typically spouses) who are rated as high in EE are at much higher risk of relapse than patients who return to live with spouses who are low in criticism, hostility, and EOI. Similar findings have been reported for manic patients (Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988). Although there are many fewer studies examining the predictive validity of EE in mood-disordered patients (unipolar and bipolar), the effect size of EE in mood disorders ($r = .45$) is still highly significant (see Butzlaff & Hooley, 1998).[2] Moreover, even though one well designed study failed to demonstrate any significant association between EE and relapse of mood disorder (see Hayhurst, Cooper, Paykel, Vearnals, & Ramana, 1997), recalculation of the meta-analytic effect size to include this study still results in a highly significant association between high EE and relapse

[1] For schizophrenia and bipolar disorder, a threshold of six or more critical comments is conventionally used. For unipolar depression, the usual cut-off is two or three critical remarks.

[2] Cut-off for high EE is three or more criticisms.

(weighted z-transformed $r = .38, p < .001$). In other words, although nonreplications can be found in both the schizophrenia and mood disorders literature, these do not call the predictive validity of EE into serious question.

A complete discussion of the range of psychopathological disorders that have been studied with respect to EE is beyond the scope of this article. However, recent meta-analytic evidence has suggested that EE is also a significant predictor of poor clinical outcome for patients with eating disorders (Butzlaff & Hooley, 1998). High levels of EE in spouses have also been linked to more rapid relapse in male patients with alcoholism (O'Farrell, Hooley, Fals-Stewart, & Cutter, 1998) and to more negative treatment outcomes in anxiety disorders (Chambless & Steketee, 1999) and posttraumatic stress disorder (Tarrier, Sommerfield, & Pilgrim, 1999).

Directions of Effect

Of course, the fact that there is a reliable association between EE and relapse tells us nothing about why this is the case. Although it is often assumed that high levels of EE are independent of patient factors and causally related to relapse, the existence of a correlation between EE and relapse does not, in itself, warrant such an assumption. For instance, patients who are at elevated risk for relapse might simply be those who, for any of a number of reasons, are likely to engender criticism in their relatives. However, a review of the literature provides no strong support for this assumption (see Hooley, Rosen, & Richters, 1995). Similarly, even when potentially important patient variables are controlled statistically, EE still makes a significant and independent contribution to relapse (Nuechterlein, Snyder, & Mintz, 1992). This suggests that the link between EE and relapse is not simply a result of their common association with another (unmeasured) third variable.

Perhaps the strongest evidence supporting a causal role for EE in relapse comes from family intervention research. A growing body of literature now suggests that intervention efforts designed to reduce high levels of EE in relatives also result in a decline in patients' relapse rates. The vast majority of these family-based treatment studies concern schizophrenia. It is also the case that, for obvious ethical reasons, these studies do not employ a true experimental design. This would involve randomly assigning participants to comparison conditions in which attempts were made to increase levels of EE with the expectation that this would increase patients' relapse rates! Nonetheless, across the majority of studies conducted to date, it appears that when efforts are made to decrease family levels of criticism, hostility, and EOI through behaviorally oriented family treatments, patients fare much better than they do if they are assigned to a comparison condition of medication and routine clinical care (Lam, 1991; Mari & Streiner, 1994). These findings are consistent with the idea that EE is functioning as a causal risk marker and suggest that EE is somehow involved in the relapse process.

Why Are High EE Attitudes Manifested?

Living with a patient who suffers from some form of mental disorder is not easy. Psychiatric illness taxes the emotional and economic resources of the best of families. Indeed, inspection of published estimates of the prevalence of high EE indicates that somewhere between 45% and 75% of the relatives of patients with schizophrenia are rated as high in EE (Hooley et al., 1995). Far from being unusual, therefore, high EE attitudes are quite normative.

Although high EE attitudes are quite common, it is nonetheless the case that significant numbers of relatives remain low in EE despite the stresses associated with dealing with psychiatric illness in a close family member. This raises the very important question of why, given the same stressor (an ill family member), some family members exhibit high EE and others evidence low EE. A diathesis-stress approach to this question seems particularly suitable and leads to a consideration of factors that might lie within the relative, conferring some kind of intrinsic liability toward criticism, hostility, or EOI. It also demands that we consider the nature of the stresses or stressors that translate such a diathesis in the relative into measurable and manifest behavior (i.e., high EE). We begin with a survey of candidate diatheses and then turn to an examination of these stressors.

Potential Vulnerability Factors

Empirical research suggests that high and low EE relatives differ from each other in important ways. For example, personality measures suggest that high EE relatives of patients with schizophrenia are less flexible and less tolerant than are relatives who are low in EE (Hooley & Hiller, 2000). The demonstration of significantly different levels of flexibility in high EE versus low EE relatives coincides nicely with earlier clinical impressions that flexibility is the most important feature differentiating low from high EE relatives (Leff & Vaughn, 1985). In addition, high EE relatives, particularly those high in criticism, have been shown to have a more internally based locus of control than do low EE relatives (Hooley, 1998) and to be more self-critical (Docherty, Cutting, & Bers, 1998). Taken together, these findings suggest that high levels of EE may reflect underlying characteristics of relatives that are highly stable over time. These may include certain personality traits. However, other, as yet unexplored factors may also play an important role.

Within a diathesis-stress formulation, such characteristics might be viewed as placing relatives along a continuum of vulnerability to develop and manifest high EE attitudes in the face of the stress that comes with exposure to severe psychopathology. In cases where intrinsic vulnerability is high, relatives are likely to become high EE quite readily (i.e., with exposure to comparatively low levels of stress). For such people, test–retest assessments might indicate a more trait-like style of high EE or of the characteristics that may facil-

itate the adoption of high EE attitudes. In cases where intrinsic vulnerability is low, higher levels of stress (or perhaps more chronic exposure to the stress variables) might be necessary to move the relative into the high EE range (cf. Zubin & Spring, 1977). This more reactive form of high EE would be expected to be less stable over time (e.g., more state-like).

Perhaps reflective of differences in vulnerability are differences in attribution styles across high and low EE relatives. More than a decade ago, it was suggested that criticism and hostility in relatives might reflect their underlying belief that patients could do more to control some of the problematic aspects of their lives and their illnesses (Hooley, 1985, 1987). Empirical research in the intervening period has provided a great deal of support for this hypothesis. Analysis of the causal attributions that relatives of schizophrenic patients spontaneously make when talking about patients has demonstrated that high EE relatives are more likely than low EE relatives to make attributions to causes that are controllable and personal (idiosyncratic) to the patient (Barrowclough, Johnston, & Tarrier, 1994; Brewin, MacCarthy, Duda, & Vaughn, 1991; Weisman, Lopez, Karno, & Jenkins, 1993). In other words, when they make reference to the causes of the patients' problems or problem behaviors, high EE relatives are much more inclined to view the problem as unique or personal to the patient (rather than common to all patients with the disorder) and also as at least somewhat controllable by the patient. High EE relatives say such things as "If she would take her medicine, she would be a whole lot better." Low EE relatives, in contrast, are more likely to accept that the patient is doing all that he or she can for the moment or to make remarks that indicate that they view the patient's problems as more universal (as opposed to personal) and uncontrollable by the patient (e.g., "He has schizophrenia. I can't expect him to be just like everyone else").

Importantly, the association between high levels of EE and a tendency to make more attributions of patient responsibility (attributions coded as internal, personal, and controllable by the patient) appears to transcend diagnosis. Licht (2000) has demonstrated that such attributions are highly characteristic not only of relatives of patients with schizophrenia, but also of relatives of unipolar depressed patients and relatives of patients with bipolar disorder. Although correlational data tell us little about whether attributions precede EE or whether EE precedes attributions, it is not unreasonable to suggest that personality characteristics in relatives incline them to have certain expectations of patients and consequently to hold patients more accountable when problems arise. To the extent that this is true, attributions about the patient's illness may be at the heart of relatives' emotional reactions toward patients. This formulation is consistent with Weiner's (1993) notions concerning anger and attributions of responsibility.

Of course, even the most inflexible and intolerant person has to have something to react to. A diathesis without a stressor is merely a diathesis. A person predisposed to respond to an emotionally ill family member in a particular manner will not evidence such a response in the absence of an ill family member or a comparable interpersonal challenge. This raises the question of what it is that psychiatric patients do that triggers the manifestation of high EE attitudes in their susceptible relatives.

Possible Stressors

The finding that levels of high EE are common in the relatives of patients with a wide range of psychiatric problems makes it unlikely that the specific symptoms of any one disorder will be sufficient and necessary to evoke criticism, hostility, or EOI. If high EE were caused by a negative reaction to patients' hallucinations, for example, why should spouses of nonhallucinating unipolar depressed patients also express critical attitudes? This is not to say that relatives will be unreactive to formal psychiatric symptoms. However, at a general level, it seems more reasonable to assume that the stressors that trigger the development and manifestation of high EE attitudes belong to a class of "psychological irritants" that must be more universal than specific. In all probability, the behavioral or functional triggers for criticism cut across formal diagnostic boundaries. In some cases they might also be quite idiosyncratic to the individual relative involved.

The attribution model of EE described earlier would predict that triggers for criticism would be behaviors that relatives believe patients can do something to change. Criticism, by definition, is a tangible expression of a wish for a person to behave differently. Behavioral candidates for the evocation of criticism are therefore likely to be behaviors that relatives do not like and that they believe patients can change (see also Hooley, 1987). Within such a formulation, tangible and unambiguous signs of "legitimate illness" such as hallucinations, a fever, or a broken leg are less likely to attract criticism than are symptoms such as psychomotor retardation, loss of interest, or poor hygiene. This model would also predict that relatives would be more critical in situations in which patients are seen as violating family norms of appropriate social behavior (e.g., refusing to help, being rude) or engaging in behaviors that appear to relatives to make a bad situation worse (e.g., not taking medication, rejecting their efforts to help, etc.).

Consistent with this view is the demonstration that relatives of schizophrenic patients were much more inclined to criticize negative symptoms (emotional withdrawal, motor retardation, blunted affect, and disorientation) than they were to criticize positive symptoms (hallucinations, conceptual disorganization, unusual thoughts, and grandiosity) (see Weisman, Nuechterlein, Goldstein, & Snyder, 1998). Moreover, high EE relatives were more likely than low EE relatives to criticize negative symptoms, even though the frequency of negative symptoms did not differ across the two patient groups. Weisman et al. (1998) reported that high EE relatives criticized long-standing personality characteristics of the patients (e.g., stubbornness) more often than did low EE relatives and complained more about nonsymptomatic

behaviors such as the patient's career choice. Together, these findings suggest that high EE relatives are more overtly responsive to perceived behavioral deficits in patients than are their low EE counterparts. It is important to note that, consistent with the diathesis-stress formulation of EE, actual behavioral deficits in patients do not occur more frequently in patients with high EE relatives than they do in patients with low EE relatives. Rather, it may be the case that the same levels of symptomatology in patients trigger different reactions in relatives and that these different responses can be explained by underlying differences in the relatives themselves.

If some of the variance in EE can be explained by how reactive relatives are to aspects of psychopathology, we might also expect that clinical improvements in patients might be associated with decreases in relatives' levels of EE. Interestingly, evidence suggests that this might be the case. Brown, Birley, and Wing (1972) were the first to observe that around one-third of high EE relatives showed a spontaneous change to low EE over a 9-month period—in large measure because of declines in criticism. Since then, other investigators have reported similar decreases in criticism in the months following the patients' discharge from the hospital (e.g., Tarrier, Barrowclough, Porceddu, & Watts, 1988). It is interesting to note that, although levels of criticism do decline significantly during periods of relatively better patient functioning, the most critical relatives at the time of the patients' admission to the hospital are still the most critical relatives several months later (Hooley et al., 1995). In other words, regardless of how well or poorly the patient is doing, relatives' preserve their rank order with respect to how critical they are, at least as indicated by the high test–retest correlation for criticism. In the context of our diathesis-stress formulation, this preservation of rank order suggests that the relatives' potential for criticism is a somewhat stable characteristic. And as would be predicted by the diathesis-stress formulation, EE can look both traitlike (e.g., Schreiber, Breier, & Pickar, 1995) and statelike (e.g., Tarrier, Barrowclough, Porceddu, & Watts, 1988).

Finally, there is some reason to believe that yet another potential stressor for relatives is how long he or she has been coping with the patient's illness. Although chronicity of patient illness does not explain the relationship between EE and relapse (EE typically remains a significant predictor of relapse even when symptom severity and illness chronicity are considered), preliminary evidence suggests that patients who have been sick longer are more likely to have high rather than low EE relatives. In a cross-sectional analysis, Hooley and Richters (1995) found that the typical relative of a schizophrenic patient in his or her first year of illness was low in EE (71%). In contrast, after five or more years of illness, the great majority of relatives received a high EE rating (83%). Of course, these differences could simply reflect clinical differences in the patients such as the type and severity of clinical symptoms. However, analyses of the clinical data suggested that this was not the case. Rather, the important difference concerned how long the patients had been ill and, by implication, how long their relatives had been coping with psychopathology and behavioral impairment. In other words, although the findings demonstrated that low levels of EE can be found at all stages of the illness, they also suggested that there may be a tendency for relatives to move from low to high EE attitudes as exposure to severe psychopathology increases. From a diathesis-stress perspective, time itself may be an important stressor that takes its toll on relatives's attitudes.

Summary

A diathesis-stress approach to EE allows us to move beyond simple formulations of EE as a trait or a state and recognize that EE is truly a relational variable. Rather than being a measure of something about the relative or a reaction of the relative to something about the patient, EE is almost certainly a product of the interaction of both patient and relative characteristics. It is our hope that, by conceptualizing EE within the familiar diathesis-stress framework, it will become easier to think of EE in interactional terms. Researchers and clinicians can then begin to explore the factors that might place relatives at the lower or higher end of the vulnerability continuum. This kind of approach highlights the importance of understanding high EE relatives. A related line of research might focus on the characteristics of low EE relatives that could protect them from developing critical, hostile, or emotionally overinvolved attitudes under stressful circumstances. Articulation of the precise stressors might also be important. However, our sense is that, beyond such general stressors as psychiatric symptoms and impaired functioning, the stress variables will be strongly linked to the nature of the relatives' vulnerabilities. Stress is very much in the eye of the beholder. For this reason we believe that researchers in this area would do well to begin by exploring the nature of vulnerability.

With this in mind, the attributional model of EE provides a theoretical framework within which much of what we currently know about this construct can be understood and organized. Relatives' emotional responses to patients, especially relatives' criticism and hostility, appear to be linked to how they understand and interpret events involving the patient. Regardless of the patients' formal psychiatric diagnoses, one hallmark of high EE relatives is their tendency to view patients as responsible for their problems and life difficulties. This is not to say that they blame patients for developing a psychiatric illness, although in our experience this sometimes does happen. More typically, however, high EE relatives make remarks that indicate they believe some of the patient's current difficulties are caused by (and therefore potentially resolved by) factors that are under the patients' control. It is our impression that the overwhelming motivation of high EE relatives is to improve patients' current level of functioning. High EE relatives genuinely want patients to get better. Unlike their low EE counterparts, however, they believe

that volitional factors on the part of the patient can play a pivotal role in this process.

These differences in attribution styles may reflect underlying differences between high and low EE relatives concerning controllability of problems more generally. In all probability, high EE relatives are expecting patients to do what they themselves think they would do if faced with a similar problem or set of difficult circumstances. Because flexibility is not a trait associated with high levels of EE, it may be more difficult for such relatives to see the situation from the perspective of the patient. Low EE relatives, in contrast, may be more buffered from the stress of coping with severe psychopathology because of their more tolerant personalities. Greater acceptance of the current situation, combined with less of a need for control, may help protect relatives from developing critical or hostile attitudes toward patients. Interestingly, increasing relatives' acceptance of their spouses or family members is at the heart of recent approaches to marital and family therapy (e.g., Hayes, Strosahl, & Wilson, 1999; Jacobson & Christensen, 1996).

The fact that we have suggested several personality characteristics that might function as vulnerability factors for the development of high EE attitudes should in no way be taken to mean that we now have a full understanding of the EE construct. In all probability, other factors are at play here too. Our discussion also fails to answer the more fundamental question of why relatives should differ in such characteristics as tolerance, flexibility, and locus of control. We know little of how and why people become who they are. Differences in basic temperament between high and low EE relatives obviously warrant exploration. In addition, we should anticipate the ways in which expectations of ourselves and of others are shaped by cultural influences, media, and so on (Jenkins & Karno, 1992). In this regard, it is interesting to note that, although levels of high EE are quite normative in industrialized societies such as the United States and Europe, the majority of relatives of patients with schizophrenia in India are rated as being low in EE (Leff et al., 1987). Cultural differences in such variables as locus of control might provide a possible explanation for this decrease in the base rate of high EE. Without question, cultural influences in the United States reflect the considerable premium that is placed on notions of personal control and potential for change ("Be all that you can be"). Although we might predict that such exhortations are less firmly embedded in Indian culture, empirical data on this issue would no doubt be most informative.

Finally, we would note that our attributional model is consistent with evidence that high EE relatives of schizophrenic patients are less well informed about the illness than are their low EE counterparts (Cozolino, Goldstein, Nuechterlein, West, & Snyder, 1998). In a related vein, it is important to note that family-based intervention efforts that are designed to reduce levels of EE almost always attempt to educate relatives about the illness. Schizophrenia is a complex disorder, and many of its symptoms can easily be mistaken for signs of obstinacy or laziness. The more relatives know about how schizophrenia affects patients, the more they may be able to modify their expectations of patients and temper their attributions of control. To the extent that the attributional styles of high EE relatives reflect more underlying and traitlike personality differences, however, we might expect that attributions would be rather resistant to rapid change. Although this issue has not received a great deal of attention to date, preliminary evidence suggests that this may well be the case, especially for attributions of control (Brewin, 1994).

Why Is Relapse Associated With High EE?

To the extent that we view high EE as a form of psychosocial stress, the question of why patients are at greater risk of relapse when they live in high EE home environments suggests the need to examine patients' diatheses that may be interacting with this stress to produce relapse. Yet to explore this issue we first need to know more precisely what high EE relatives do. We then need to evaluate this in light of what we currently understand about the nature of psychiatric vulnerability. In the sections that follow we consider these issues for three distinct disorders: schizophrenia, unipolar depression, and borderline personality disorder. Of course, EE has been studied within a diagnostic range that is much broader than this, but we have neither the space nor the expertise to speculate about the link between EE and relapse for all possible conditions. Instead, we focus on disorders with which we have the most clinical and empirical experience in an effort to provide a way in which future thinking about EE and relapse might profitably be structured. It is our hope that others will adopt the diathesis-stress approach we use and outline possible vulnerability and stress factors that interact to precipitate relapse in disorders that we are unable to consider.

High EE As a Stressor

There is no shortage of evidence for the concurrent validity of EE. Studies that have examined the behavior of high and low EE relatives during face-to-face interactions with patients have consistently shown that high EE relatives are more behaviorally negative than are their low EE counterparts. For example, high EE relatives talk more and listen less (Kuipers, Sturgeon, Berkowitz, & Leff, 1983). They also make more critical remarks directly to schizophrenic patients than do low EE relatives (Miklowitz, Goldstein, Falloon, & Doane, 1984; Valone, Norton, Goldstein, & Doane, 1983). Behavioral observations of the spouses of depressed patients paint a similar picture. High EE spouses are more critical than are low EE spouses. They also disagree with patients more frequently than do low EE spouses and show less accepting behavior toward their depressed partner (Hooley, 1986).

Moreover, it is not just the frequency of negative behaviors that differentiates high from low EE relatives. Sequential analyses have indicated that there is more reciprocal neg-

ativity and less reciprocal positivity in interactions involving a high EE family member (Hahlweg et al., 1989; Hooley, 1990; Simoneau, Miklowitz, & Saleem, 1998). When a high EE relative is involved, negative verbal or nonverbal behavior tends to be reciprocated, regardless of who started the negative interaction sequence to begin with. Low EE interactions, in contrast, are less affectively charged. Positive interaction sequences have a high probability of continuing, and negative interaction sequences are quickly terminated. Importantly, these patterns of reciprocal exchange tend to characterize high EE and low EE interactions regardless of the specifics of the patient's diagnoses. Although EE is operationalized as an attitude about a patient expressed in private to researcher, it also appears to be indexing something very meaningful about how the relatives interact with patients on a day-to-day basis.

Given all that we have discussed, it should come as little surprise that another behavioral characteristic of high EE relatives is their tendency to be controlling. In a logical extension of research on attributions and EE, Hooley and Campbell (2000) analyzed interviews with the relatives of both schizophrenic and depressed patients. After identifying all statements that made reference to the patient's capacity (or lack thereof) to control his or her disorder or problem behavior, trained raters made a global rating of each relative on a scale of 1 to 5 (1 = no perceived control; 5 = a great deal of perceived control). In a similar manner, raters also coded the degree of behavioral control exercised by the relative over the patient. Ratings were based on the severity and frequency of remarks relatives made about efforts to control the patient. Mildly controlling statements were ones that described a suggestion or polite request for the patient to do something specific (e.g., "I asked her to please hang up her coat"). Statements such as "I told her to stop smoking so many cigarettes" were regarded as evidence of more moderate levels of control, whereas remarks such as "I pulled the plate out of her hand to make her stop eating" led to relatives being rated as highly controlling.

Consistent with the attribution model, the high EE relatives of the schizophrenic patients were rated higher on attributions of control than were the low EE relatives. The same was true for the high EE relatives of the depressed patients. Importantly, the results revealed that, regardless of diagnosis, high EE relatives were rated as being more controlling in their behavior than were low EE relatives. In other words, high EE relatives not only believe that patients can do more to deal with their own problems and problematic behaviors, but also try to influence patients directly through their own actions. The idea that high EE reflects a form of social control has also been suggested by Greenley (1986).

In summary, available evidence suggests that high and low EE relatives differ in how they think about the illness as well as in how they behave when they are with patients. High EE relatives behave more negatively toward patients and show less positive verbal and nonverbal behavior toward them. Family discussions that involve high EE relatives are likely to be characterized by sequences of reciprocated negative verbal and nonverbal escalation. Finally, there is some evidence that the attributional differences between high and low EE relatives are paralleled by differences in how much behavioral control they try to exert over patients. It seems clear, therefore, that high EE relatives are engaging in a range of behaviors that might reasonably be expected to be stressful for the people with whom they interact.

This is not to say that the behaviors of high and low EE relatives might not be reactions to what the patients themselves are doing in the same interactions. Indeed, evidence suggests that there are behavioral differences between patients in high and low EE interactions. For example, depressed patients tend to be less self-disclosing when they are interacting with high EE spouses (Hooley, 1986), whereas bipolar patients are more nonverbally negative in such circumstances (Simoneau et al., 1998). Patients with schizophrenia make fewer statements that reflect autonomy when they are with their high EE versus low EE relatives and tend to be more critical in discussions involving high EE relatives (Strachan, Feingold, Goldstein, Miklowitz, & Nuechterlein, 1989). In other words, these more negative behaviors in their high EE relatives.

In all probability, patients are reacting to relatives and relatives are reacting to patients. Consistent with this notion, Wuerker (1994) observed that mutual competition for control is highly characteristic of both patients and relatives in high EE dyads. However, regardless of who is reacting to whom at any particular point in time, one thing should be clear. Interactions that involve high EE relatives are likely to be more stressful for all participants than are interactions that involve low EE relatives (cf. Hubschmid & Zemp, 1989).

Of course, stressful interactions are an unfortunate and unavoidable fact of life. What is so appealing about the diathesis-stress approach is its assumption that, in the absence of intrinsic vulnerability, stressful family interactions will not constitute a risk factor for either the onset of psychopathology or for symptom relapse. For someone with established vulnerability, however, the consequences of stress exposure may be quite different. In the following sections we examine this issue with respect to schizophrenia, depression, and borderline personality disorder.

EE and Vulnerability to Relapse

Schizophrenia. Conventional wisdom regards patients with schizophrenia as being highly sensitive to environmental stress. Indeed, one assumption of the diathesis-stress model of the etiology of schizophrenia is that some form of stress is needed in order for an intrinsic liability toward the illness to be clinically expressed (Kendler & Eaves, 1986; Zubin & Spring, 1977). Stress is also implicated in the relapse process. We know that independent stressful life events occur more frequently in the period prior to a psychotic relapse than they do during comparison periods (Ventura, Nuechterlein, Hard-

esty, & Gitlin, 1992; Ventura, Nuechterlein, Lukoff, & Hardesty, 1989). Independent life events, by definition, are not caused by the patients' behavior. Nonetheless, they appear to presage the worsening of symptoms. And as we have already noted, family stress in the form of high EE is an important predictor of early relapse in schizophrenia. At a general level, environmental stress is thought to interact with preexisting biological vulnerabilities to increase the probability of relapse in schizophrenic patients (Nuechterlein et al., 1992).

But what is the mechanism through which high EE and schizophrenic relapse might be related? Brown was the first to suggest that the problem might be one of environmental overstimulation (Brown et al., 1972). In a highly influential article that has obvious connections to this basic vulnerability idea, Nuechterlein and Dawson (1984) suggested that autonomic hyperarousal in patients might act as a common pathway mediating the effects of psychosocial stress on schizophrenic vulnerability and bringing about relapse. What we know about the behavioral correlates of high EE attitudes makes it reasonable to hypothesize that interactions with high EE family members are more stressful for patients than are interactions involving low EE relatives. Data concerning electrodermal arousal suggest that this is indeed the case.

In an early study, Tarrier and his colleagues collected psychophysiological data on schizophrenic patients in remission (Tarrier, Vaughn, Lader, & Leff, 1979). The patients were tested in their own homes, and electrodermal and blood pressure data were collected both before and after their high and low EE relatives entered the room. In the 15 minutes prior to the entry of the relative and while only in the presence of the experimenter, no differences were apparent between the patients in the two groups. However, subsequent to the entry of the relatives, patients with high EE relatives showed an elevation in diastolic blood pressure. In contrast, patients with low EE relatives showed a decrease in the frequency of nonspecific skin conductance responses (NS-SCRs). Subsequent testing in a laboratory setting revealed no significant differences in heart rate, electroencephalogram, or electrodermal activity between the patients with high and low EE relatives. The absence of psychophysiological differences between the patients indicated that the differences between them during the at-home testing were triggered by the presence of the relatives. These findings suggest that the presence of high EE relatives is more arousing to patients, whereas the presence of low EE relatives is calming.

Subsequent studies have broadly supported this view. In a similar investigation conducted when patients were still in an acute episode of illness, patients with high EE relatives were found to have significantly more NS-SCRs than patients with low EE relatives throughout the entire period of testing. This was the case regardless of whether the relative was present or not (Sturgeon, Kuipers, Berkowitz, Turpin, & Leff, 1981; Sturgeon, Turpin, Kuipers, Berkowitz, & Leff, 1984). A second series of studies by Tarrier, also conducted with patients in an acute episode, again indicated that the frequency of NS-SCRs decreased in patients with low EE relatives after those relatives entered the room (Tarrier, Barrowclough, Porceddu, & Watts, 1988). The general finding, then, is that greater electrodermal arousal is associated with high EE relatives (see Tarrier & Turpin, 1992, for a detailed review).

In a sophisticated extension of this line of research, Altorfer, Käsermann, and Hirsbrunner (1998) collected psychophysiological data from patients with schizophrenia and bipolar disorders while they were involved in problem-solving discussions with their families. What was most important about this study was that patient cardiovascular data could be time-locked to specific points in the interactional sequences. Although no EE data were collected on the relatives, Altorfer et al. (1998) used the EE literature to identify conversational sequences that were thought to be either stressful or neutral in their content. Examples of stressful content included criticism, guilt induction, or intrusiveness. Using this approach, Altorfer et al. (1998) reported that stressful statements by relatives were linked to increased cardiovascular activity in patients. To our knowledge, this is the first empirical demonstration that high EE verbal behavior in relatives is directly associated with psychophysiological indices of stress in patients.

Taken together, available evidence supports the view that patients with schizophrenia are physiologically challenged when they interact with high EE relatives. There is also reason to believe that patients may become more soothed when they interact with low EE family members. However, there is a great deal of distance between stress responses and symptom relapse. By what mechanism might physiological stress develop into symptom exacerbation?

A provocative model outlined by Walker and Diforio (1997) provides some guidance on this issue. In a thoughtful review, Walker and Diforio noted that one of the primary manifestations of the stress response in animals and humans is activation of the hypothalamic–pituitary–adrenal (HPA) axis. Exposure to stress is associated with increases in the release of glucocorticoids (specifically, cortisol in humans) from the adrenal cortex (e.g., Kirschbaum et al., 1995). Of relevance to our discussion of schizophrenia are empirical data suggesting that baseline levels of plasma cortisol are elevated in schizophrenic patients relative to normal controls (Altamura, Guercetti, & Percudani, 1989; Breier & Buchanan, 1992). Elevations in baseline cortisol are also found in people with schizotypal personality disorder relative to other Axis II and normal controls (Walker et al., 1996). This suggests that heightened cortisol release might be linked to vulnerability to schizophrenia rather than simply being a correlate of manifest schizophrenic illness.

Also of interest is the association between cortisol and dopamine. Dopamine has long been considered an important neurotransmitter with respect to schizophrenia (Carlsson, 1995). It is therefore intriguing to discover that animal studies have shown that cortisol release triggers subcortical dopamine activity (Rothschild et al., 1985). Human studies

have also shown a positive correlation between cortisol release and dopamine activity (McMurray, Newbould, Bouloux, Besser, & Grossman, 1991). The neurotransmitter glutamate has been implicated in schizophrenia (Olney & Farber, 1995). The fact that glucocorticoid secretion has effects on glutamate release is particularly exciting (Horger & Roth, 1995; Walker & Diforio, 1997).

Might Walker and Diforio's (1997) neural diathesis-stress model of schizophrenia provide a possible explanation for the link between high levels of EE and schizophrenic relapse? At the present time, we regard this as an intriguing possibility. Although necessarily speculative, it is nonetheless plausible to suggest that patients with schizophrenia have a heightened vulnerability to psychosocial stress (see Fowles, 1992), and that one commonly occurring form of psychosocial stress involves interactions with high EE family members. If high EE interactions are characterized by criticism, negative affectivity, and direct efforts to control the patient, these behaviors are likely to be perceived by patients as unwelcome and stressful. To the extent that high EE behaviors are perceived as stressful, they are likely to trigger release of cortisol. This in turn could function to stimulate the dopaminergic and glutamatergic neurotransmitter systems—perhaps ultimately leading to a recrudescence of symptoms.

With this model in mind, a study by Rosenfarb, Goldstein, Mintz, and Nuechterlein (1995) is noteworthy. This investigation focused on the behavior of schizophrenic patients interacting with their high and low EE relatives. They observed that when patients made an unusual remark (e.g., "If that kid bites you, you'll get rabies."), high EE relatives were more likely than were their low EE counterparts to respond with a criticism directed toward the patient. Of particular interest, however, was that when this happened, it tended to be followed by another unusual remark from the patient. In other words, there was an increase in patients' unusual thinking immediately after being criticized by a family member. These data are consistent with the idea that negative (stress-inducing) behaviors by relatives trigger increases in unusual thinking in schizophrenic patients.

What is particularly appealing about the glucocorticoid-based model of EE and relapse is that it holds the potential to explain another interesting and clinically important observation. In their meta-analysis of the EE and schizophrenia literature, Butzlaff and Hooley (1998) found that, although EE predicts relapse regardless of the chronicity of the patient sample being studied, EE was a significantly stronger predictor of relapse for patients with more long-standing illnesses. In their discussion, these authors suggested that patients might become more sensitive to EE over the course of their illnesses. With this in mind, it is interesting to note that animal studies have suggested that exposure to stressors can result in a sensitization effect (Plotsky & Meaney, 1993). In other words, under certain conditions, repeated stress can result in persistent elevations of cortisol (Levine, 1993). Although we have no direct data linking EE levels in relatives and cortisol levels in patients, it is intriguing to speculate that, in addition to the general stress sensitivity that characterizes vulnerability to schizophrenia, high EE environments might serve to make it even more difficult for HPA activity to stabilize. In contrast, low EE environments might directly protect patients by working to calm them (see Tarrier & Turpin, 1992) and thus to suppress HPA overactivity.

It is worth noting that antipsychotic medications reduce cortisol release in schizophrenia patients (see Walker & Diforio, 1997), and that antipsychotic medications are thought to be especially important for patients in high rather than low EE homes (Brown et al., 1972). Although neuroleptic medications are highly advisable for all patients with schizophrenia, they may play a less important role for patients who live in low EE homes. Very possibly, the protective effects of low EE families and the protective effects of antipsychotic medications operate via a more common biological pathway than would seem obvious at first glance.

Let us consider the potential influence of moderator variables. As we noted earlier, not all patients who live in high EE families relapse and not all patients who live in low EE homes stay well, even though the majority do. Although the glucocorticoid model of EE and relapse is intuitively appealing and has some indirect support from the empirical literature, the question of differential vulnerability to relapse still remains. With this in mind, we offer a few final suggestions about the variables that might interact with EE to confer more or less vulnerability to relapse.

At the biological level it seems obvious that there are individual differences in stress responsivity to high EE-like behaviors. For example, of the thirteen patients studied by Altorfer et al. (1998), three showed no physiological responses to identified stressful comments from their relatives. This suggests that all patients are not physiologically equal when it comes to EE stimuli. Two potential sources of such variability in vulnerability to EE stressors are underlying baseline HPA levels and responsivity of neurotransmitter systems to phasic cortisol release. These, of course, could be a consequence of earlier stress sensitization occurring at any stage of development. Moreover, to the extent that stressful home environments can sensitize the HPA axis, we would predict that duration of exposure to sustained high EE behavior would make patients more biologically vulnerable to the subsequent effects of high EE.

Another indicator of the integrity of HPA regulation might come from using the Dexamethasone Suppression Test (DST). Dexamethasone is a potent synthetic glucocorticoid that can be used in a challenge paradigm to test for preexisting dysregulation on the HPA axis (Mossman & Somoza, 1989). Initially it was hoped that the DST would provide a biological marker for the diagnosis of depression. However, research soon revealed that not all depressed patients showed DST nonsuppression (Hubain, Van Veeren, Staner, Mendlewicz, & Linkowski, 1996). DST nonsuppression has also been found in patients with other disorders and in nonpa-

tients—a considerable problem for a potential diagnostic marker (Baldessarini & Arana, 1985; Ismail, Murray, Wheeler, & O'Keane, 1998). However, the fact that DST nonsuppression is found in patients with schizophrenia and mood disorders and that patients with these disorders are more susceptible to relapse in high EE homes is potentially interesting with respect to our glucocorticoid model. Future studies should explore the extent to which DST data might be used to identify patients at greater risk of relapse in the face of high EE environments. We would predict that patients who are DST nonsuppressors (and who thus show evidence of more abnormal HPA regulation) would be overrepresented in the group of patients who relapse in the face of high EE—regardless of diagnosis. On the other hand, we would expect that patients who have a less dysregulated HPA axis might be more likely to do well, even in the context of a high EE home environment. Although no studies of this nature have yet been conducted, information about the possible interactions between markers of biological vulnerability to stress and stress itself would be very valuable.

Finally, we note that vulnerability to the influences of high EE need not necessarily be biologically based. Psychological variables are no doubt of importance here. For example, schizophrenia patients' ratings of their current feelings toward their relatives made using a 5-point Likert-type scale (mostly strong negative thoughts; mostly strong positive thoughts) have been found to predict psychotic exacerbation over a one-year follow-up (Lebell et al., 1993). This suggests that how patients appraise the behavior of their relatives might be important. In cases where high EE behaviors are not perceived as either negative or stressful, we might expect that patients would do well regardless of how the family environment is objectively rated.

With this in mind, we note that the effect size for the association between EE and relapse is significantly lower for schizophrenia than it is for either mood disorders or eating disorders (Butzlaff & Hooley, 1998). The possibility that the information-processing deficits that are so characteristic of schizophrenia might actually serve to protect these patients from high EE stress is an intriguing possibility. Patients with schizophrenia are known to have difficulties in understanding the nuances of social exchanges and in interpreting emotional cues (e.g., Corcoran, Mercer, & Frith, 1995; see Hooley & Candela, 1999, for a review). To the extent that some patients fail to detect affectively negative behavior expressed toward them by high EE family members, we might predict that they would be less susceptible to any stressful physiological sequelae of such behaviors. We would also hypothesize that patients who are relatively disengaged affectively from their families, patients with better social skills, patients who dismiss or ignore relatives' criticisms, or patients who have other supportive alternative social networks couldbe relatively more buffered from the stress-related consequences of high EE interactions. Nearly three decades ago Brown noted that patients who spent less time with their high EE relatives did better than patients who spent more time in face-to-face contact with their families. Viewed now in the context of our current discussion, this seems all the more prescient.

Depression. As we noted earlier, high levels of EE have been found to predict relapse in patients with unipolar depression (e.g., Hooley et al., 1986; Vaughn & Leff, 1976a). However, the mechanisms or processes by which high levels of EE might be related to an increased probability of relapse in depression have not been elucidated. Although some of the same general mechanisms that we described previously to explain the association between EE and relapse in schizophrenia may help us to understand how high EE leads to relapse of depression, the presence of important differences between these two disorders impels us to make specific predictions about depressive relapse in the context of a high EE environment.

With respect to common mechanisms, we have described research indicating that schizophrenic individuals are characterized by tonic autonomic and neuroendocrine hyperarousal, which may interact with a high EE environment to produce schizophrenic relapse. There is evidence to suggest that depressed individuals, although not tonically electrodermally hyperaroused (see Sponheim, Allen, & Iacono, 1995), do show greater physiologic reactivity to aversive stimuli than do nondepressed individuals. In two studies, Lewinsohn, Lobitz, and Wilson (1973) assessed autonomic functioning of depressed, psychiatric control, and normal subjects before, during, and after they were given a mild electric shock or exposed to aversive noise bursts. They found that, in contrast to the psychiatric control and normal participants, the depressed subjects exhibited significantly greater autonomic reactivity during (but not before or after) the presentation of the aversive stimuli. Similar results have been reported more recently by Rottenberg, Najmi, Wilhelm, Gross, and Gotlib (2000), who found that depressed patients responded to affectively negative film clips with greater autonomic arousal than did nondepressed patients and nondepressed nonpatient controls subjects.

These findings are important from a diathesis-stress perspective in elucidating the nature of the association between high EE and relapse in depression. As we noted earlier, diathesis-stress conceptualizations can clarify why some relatives of psychiatric patients are high in EE. The diathesis-stress model we outlined here is intended to provide a broad context within which to understand the development of high EE attitudes. At a more specific level, it is likely that for depression a number of other important factors come into play. For example, in contrast to schizophrenia, most relatives in studies of EE in depression are spouses (often husbands) of the depressed proband and not parents (cf. Gotlib & Hooley, 1988). Studies of this type consistently demonstrate that spouses of depressed persons are themselves characterized by high levels of psychopathology, a pattern consistent with the notion of "assortative mating" (e.g., Hammen, 1991; Merikangas, Weissman, Prusoff, & John, 1988). Thus, a high

proportion of spouses of depressed persons would be expected to be characterized by elevated levels of negative affect and, consequently, by high rates of criticism. To the extent that this is true, high negative affect might be expected to incline a relative to develop high EE attitudes under stressful circumstances (viz. coping with a depressed partner).

The diathesis of the spouses' tendency to experience and display negative affect will no doubt be exacerbated by the stressful behaviors of their depressed partner. Gotlib and his colleagues, among others, demonstrated that depressed people behave in ways that engender negative affect in those with whom they interact (e.g., Gotlib & Meltzer, 1987; Gotlib & Robinson, 1982). This effect is even more pronounced in intimate relationships such as marriage (Gotlib & Beach, 1995). Thus, the tendency of many spouses of depressed partners to experience negative affect and to be critical of their spouses (Hammen, 1991) can be expected to interact with their depressed partners' behaviors and affect to produce a high EE environment.

In addition to its ability to capture processes contributing to the development and manifestation of EE, the diathesis-stress perspective may help us understand why a significant proportion of formerly depressed patients who live with high EE relatives relapse. From this perspective, the diathesis involves the depression-vulnerable person's tendency to attend to negative aspects of his or her environment as well as the meaning he or she attaches to the critical comments of a spouse or other relatives. There is now a large empirical literature documenting the attentional and memory biases of depressed individuals. Numerous studies have demonstrated that depressed people find it particularly difficult to "disattend" from negative aspects of their environment (e.g., Gotlib & McCann, 1984; Gotlib & Neubauer, 2000), that they attend more readily to negative than to positive stimuli (Mathews, Ridgeway, & Williamson, 1996; Westra & Kuiper, 1997), and that they have better memory for negative than for positive events (Denny & Hunt, 1992; Matt, Vazquez, & Campbell, 1992). Given this information-processing style, it is very likely that depressed (and perhaps formerly depressed) people will attend to and remember the critical behaviors and comments of their relatives (often emitted, unfortunately, in response to the depressed person's own aversive behaviors).

Criticisms and negative behaviors by the relatives of depression-vulnerable people are especially salient, given the typically elevated levels of interpersonal dependency characteristic of people prone to depression (cf. Barnett & Gotlib, 1988; Hirschfeld et al., 1989; Lewinsohn, Rhode, Seeley, Klein, & Gotlib, in press). Given the strong dependency needs of many people who experience episodes of depression, it is easy to understand that criticisms from their relatives may represent especially potent threats of being rejected, thereby increasing the importance and aversive impact of the criticisms. Consistent with this formulation, Hooley and Teasdale (1989) found that patients' ratings of "perceived criticism" from their spouses were more powerful predictors of relapse than were the actual levels of criticism. The increased dependency of depressed people, coupled with their greater attention to negative stimuli, might explain why a lower threshold of two or three critical comments has been found to have the most predictive validity for depression whereas a cut-off of six is typically used as the basis for a high EE classification in relatives of schizophrenic patients.

Although psychophysiological studies have not yet been conducted with depressed patients interacting with high and low EE relatives, we would hypothesize that depression-prone people would respond to stressful family environments with elevated levels of physiologic arousal. This heightened arousal, in turn, would be expected to exacerbate the rumination and self-focus that is characteristic of depressed people (e.g., Ingram, 1994; Nolen-Hoeksema, 1987) as well as to increase their depressive symptoms. These symptom displays would, of course, increase the level of criticism from their relatives (Gotlib & Hammen, 1992), and thus the vicious cycle would continue into a full-blown relapse of depression.

Finally, we would like to raise two possibilities that might protect patients under such circumstances. First, there may be a subtype of depression that does not involve negative attentional bias (Traill & Gotlib, 1999); this would limit the impact of critical comments or behaviors of relatives on depressed patients. Second, emotional disengagement from the critical partner may shield the depressed patient from the impact of high EE. In this context, the presence of other supportive (noncritical) relationships could be protective. Hooley et al. (1986) noted that some of the depressed patients in their study who did not relapse despite having high EE spouses were those patients who were involved in extramarital affairs. Although purely anecdotal, this observation suggests that the impact of criticism may be attenuated in cases where emotional dependency on the high EE family member is reduced.

Borderline personality disorder. Research in the area of EE and Axis II disorders is only just beginning. Only one study to date has examined the link between EE and clinical outcome in borderline personality disorder (BPD). Ordinarily, this would incline us to wait for additional contributions to the empirical literature before speculating on the factors that might lie between EE and clinical outcome in such patients. However, there is reason to believe that the nature of the link between EE and outcome in BPD is very different from the link between EE and outcome in schizophrenia and depression. Accordingly, some comments are in order.

In the only published study to date, Hooley and Hoffman (1999) studied 35 psychiatric inpatients diagnosed with BPD. During an index hospitalization, patients' relatives were interviewed in the conventional manner, and levels of EE were assessed. Family EE was then used to predict patients' clinical outcomes one year after hospital discharge. Information from audiotaped interviews with patients and with

their family members was used to rate patients' outcomes reliably on a 1 to 5 global outcome scale, where 1 indicated complete remission of symptoms and 5 indicated rehospitalization.

During the one-year follow-up period, 54% of patients were rehospitalized. Contrary to prediction, the EE variables of criticism and hostility were not significantly predictive of overall clinical outcome or rehospitalization. However, the relatives' level of EOI was significantly predictive of both outcome (rated on the 1 to 5 scale) and hospital readmission. These results held when clinical variables such as illness severity and chronicity were statistically controlled. Interestingly, the direction of the results was opposite to that typically found for schizophrenia, for which high family EOI is associated with poorer clinical outcome. In patients with BPD, high levels of family EOI were predictive of patients doing better overall and not being rehospitalized. Thus, rather than being risk factors for relapse, some aspects of EE (e.g., EOI) appear to be helpful for patients with BPD.

What is both striking and exciting about this study is that the pattern of results is totally different from what is typically found in EE research involving patients with Axis I disorders. This suggests that whether EE is truly a psychosocial stressor or not may depend very much on the nature of the patient population under study. In other words, some patient populations may be more or less vulnerable to high EE family environments. What may be stressful for many patients with schizophrenia or mood disorders may not be stressful for patients with BPD.

Why should criticism be so unimportant in the prediction of clinical outcome for BPD patients? From a clinical perspec-tive we would expect BPD patients to be extremely responsive to affective stimuli. However, recent psychophysiological data have found that reactive autonomic underarousal rather than autonomic hyperarousal is more characteristic of BPD patients. Herpertz and her colleagues (Herpertz, Kunert, Schwenger, & Sass, 1999) studied skin conductance in female BPD patients in response to pleasant, unpleasant, and neutral slides. Compared to a comparison sample of psychiatrically healthy female controls, the BPD patients showed diminished skin conductance responses to all of the emotional stimuli, even though initial baseline assessments of skin conductance did not differentiate the two groups. In other words, the BPD patients demonstrated more physiological hypoarousal to the affective stimuli than did the controls.

Herpertz et al.'s (1999) findings are particularly intriguing in light of data on the predictive validity of EE in BPD patients. Clearly we need to learn much more about how BPD patients process and interpret emotional stimuli. If BPD patients have a down-regulated autonomic response to potentially stressful affective stimuli, this may go some way toward explaining why they do not relapse in the face of high levels of criticism, hostility, or EOI. In addition to being able to create high levels of negative affectivity around them, BPD patients may also be able to tolerate higher levels of affective stimulation in their family environments. Somatic underarousal might be a risk factor for the development of BPD. Heritable decreased autonomic reactivity might be enhanced by the problematic family environments that are thought to be implicated in the etiology of BPD (see Hooley & Dominiak, in press, for a review; Links, 1990). In short, contrary to what we would expect to happen in schizophrenia, BPD patients may increase already down-regulated affective responsivity as a result of exposure to chronic environmental stress. To the extent that either of these possibilities is correct, we would expect BPD patients to be relatively less sensitive to the kinds of psychosocial stressors that are reflected in the EE construct.

The hypoarousal model described is helpful in explaining why BPD patients do not fare particularly poorly in critical, hostile, or emotionally overinvolved families. But the data suggest that patients with BPD do better in family environments that are high in EOI. Why should this be? One possibility is that high levels of EOI are viewed by BPD patients as evidence of caring and support rather than as evidence of family intrusiveness or enmeshment. In a disorder where interpersonal concerns and fears of abandonment are common, a validating environment (see Linehan, 1993) may be one that contains a level of emotional engagement that is more than other psychiatric patients (e.g., patients with schizophrenia) are able to tolerate. This might be especially true if we conceptualize EOI as a highly approach-focused behavior. Even if it is well intended, such intrusiveness may be more than the depressed patient or the patient with schizophrenia can handle. For the BPD patient, however, high levels of EOI may well be viewed as evidence of emotional concern and support.

It seems clear that future work concerning EE and BPD must pay attention to several important issues. First, we need to know how high EE relatives interact with BPD patients. Our discussion, of course, is predicated on the assumption that the high EE relatives of BPD patients behave in a similar manner to the high EE relatives of patients with other disorders. Data on this issue are urgently needed. Second, investigations that focus on how BPD patients process and interpret emotionally salient interpersonal stimuli warrant a prominent place on the research agenda. Although EE research with BPD patients is still in its infancy, it has already taught us a valuable lesson. EE is not a one-size-fits-all psychosocial stressor. Some patients may be more or less vulnerable to the consequences of high EE environments by virtue of the nature of their disorders. By recognizing this, we may be able to gain valuable ground in understanding what it is about EE that might be helpful or harmful and for which types of patients.

Treatment Implications

Family-based approaches to the treatment of schizophrenia were developed after it became clear that the EE construct had predictive validity (Leff, Kuipers, Berkowitz, Eberlein-

Fries, & Sturgeon, 1982). Although these interventions were not based on explicit theoretical models of how high EE developed or how EE was linked to relapse, they have nonetheless been successful. Several controlled studies have demonstrated the clinical benefits of psychosocial interventions (e.g., Tarrier, Barrowclough, Vaughn, Bamrah, Porceddu, Watts, & Freeman, 1988; McFarlane, Lukens et al., 1995). Schizophrenic patients whose relatives received family-based interventions had relapse rates of approximately 10% over a 9- to 12- month period. For patients whose families were assigned to the comparison condition, relapse rates hovered around 50%.

With regard to schizophrenia, the common elements of efficacious treatment appear to involve educating relatives about the illness and improving communication and problem-solving skills within the family. Given our discussion, it is easy to see why such approaches might be helpful. Interventions that work to decrease interpersonal tensions in the families of schizophrenic patients would be expected to reduce autonomic arousal in vulnerable patients. Simply decreasing the time that the patient and high EE family member spend together would also facilitate this and hence be a legitimate therapeutic goal (Leff et al., 1982). What would not be predicted to be particularly helpful is simply providing general peer support for patients and giving relatives an opportunity to air their pent-up frustrations. Interestingly, this also appears empirically to be the case (Köttgen, Sönnischen, Mollenhauer, & Jurth, 1984).

It is possible that interventions designed to help patients cope with stressful situations might afford them some protection even in the absence of family-based interventions. Hogarty et al. (1986) noted that one-year relapse rates were reduced in patients from high EE homes who received social skills training compared with comparison patients who received only medication and routine treatment (20% vs. 41%). However, the best outcome was found when patients received social skills training and family treatment. In this group, no relapses occurred during the one-year follow-up. Depending on the type of patient-oriented treatments offered, it may be possible to protect patients enough so that the additional benefits of family treatment become nonsignificant (Linszen et al., 1996).

In short, there may be many ways to optimize treatment outcomes for patients with schizophrenia. Chances of success will be maximized when attention is paid both to reducing the psychosocial stress that patients experience (e.g., decreasing high EE) and to addressing issues of intrinsic vulnerability. For example, it is worth considering whether schizophrenic patients might be better able to handle the stresses inherent in family-based treatments (at least in the early stages) if such interventions were timed to occur when diurnal fluctuations in cortisol are not at their peak. It is also essential that patients receive appropriate neuroleptic medication. Medication clearly affords patients considerable protection, and there is every reason to believe that some of the newer antipsychotic medications will be even more helpful in this regard. Consistent with our diathesis-stress formulation, psychosocial treatment approaches for individual patients and family-based interventions appear to provide incremental benefits. Clinicians need to regard schizophrenic patients as highly "stress sensitive" and to structure treatment accordingly. This message needs to be conveyed effectively to families. When families are provided with the skills to cope with the inevitable stresses of dealing with a psychotic family member, the long-term clinical outcomes can be greatly improved (McFarlane, Link et al., 1995).

It should also be clear from our discussion that different disorders require different types of family-based interventions. EE-based treatments for mood disorders are only just beginning (Vearnals & Asen, 1998). However, we believe that interventions for unipolar depression would be enhanced by a more specific focus on criticism and the meaning of criticism to the depressed patient than would be typical in family-based treatments for schizophrenia or even bipolar disorder (Miklowitz, Wendel, & Simoneau, 1988). Although schizophrenic patients may show elevated psychophysiological arousal to a broad range of stimuli, depressed patients may selectively attend to critical comments or other negative stimuli that have particular relevance for their self-concept or schematic functioning. To the extent that this is true, criticism may be an extremely potent stressor. Moreover, given the dependency issues associated with depression, spending less time with high EE relatives should not be regarded as a desirable treatment goal for these patients. Therapeutic approaches derived from EE research should seek to enhance the quality of the depressed patients' marital and family relationships, not least because marital functioning correlates with EE and predicts patient relapse (Hooley & Teasdale, 1989). In addition, intervention efforts need to focus on decreasing spouses' criticism and helping patients cope with criticism—both behaviorally and cognitively—when it does occur.

Finally, for patients with BPD, we would expect that the most promising interventions would treat all family members together and would emphasize the development of improved coping skills, emotion management, and validation. Based on our reactive hypoarousal model, we would anticipate that high levels of negative affectivity might initially be very characteristic of BPD patients in family treatment, but that this might not be predictive of poor treatment outcome in the longer term. Although family-based treatments for BPD are still in the early stages of development and empirical study, we are seeing some promising beginnings (Gunderson, Berkowitz, & Ruiz-Sancho, 1997; Hoffman, Fruzetti, & Swenson, 1999; Hoffman & Hooley, 1998).

Concluding Remarks

In this article we have offered some speculations about the nature and origins of EE. We have highlighted some vulner-

ability or protective factors that might be important in the link between EE and relapse for three distinct disorders. Space limitations necessitate that many issues remain unaddressed. For example, we have not considered the possible role of EE in the onset of psychopathology. Although some of our models do not preclude this, we believe that any etiological role played by EE will be weak compared to its role in relapse. This position is consistent with kindling models of psychopathology, which suggest that stress may have a larger impact on recurrences of depressive episodes than is the case for first onsets (e.g., Post & Weiss, 1998; Segal, Williams, Teasdale, & Gemar, 1996). We also acknowledge that our attribution model of EE provides a far better explanation for criticism and hostility than it does for EOI. A detailed understanding of this important element of EE must await future empirical findings. Finally, we have not provided anything close to a definitive explanation for why patients relapse or remain well in high EE environments. Rather, our aim has been to provide researchers and clinicians with some ideas that might shape and inform the empirical and intervention efforts of the next decade. To date, there has been very little theory in EE research. We hope that this article will stimulate efforts in this direction.

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