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Review Article

Recent Developments in Expressed Emotion and Schizophrenia

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Expressed emotion (EE) has substantial scientific support as a predictor of relapse of positive symptoms. The median relapse rate in a high-EE environment is 48%, compared with 21% in a low-EE environment. This effect does not seem to be due to confounding with other variables, but it is subject to limitations in its scope of application. EE probably determines relapse through its effect on emotions and symptom control. A stress–vulnerability model of relapse is advanced that incorporates biological factors as well as cycles of mutual influence between symptomatic behaviour, life events, and EE. Aversive types of behaviour in patients and their relatives are seen as understandable reactions to stress that are moderated by social perceptions and coping skills. Families have made positive achievements, including the provision of non-invasive support.

The last 30 years of research into schizophrenia has seen considerable interest in the role of social and situational factors in relapse (Brown & Birley, 1968; Birley & Brown, 1970; Katschnig, 1986). An influential part of this research has focused on the concept of expressed emotion (EE; Brown *et al*, 1962, 1972). Although the early studies examined the role of both positive and negative emotions, subsequent research has effectively narrowed the concept to negative or intrusive attitudes that relatives express about the patient.

Since those early studies, EE assessments have usually been derived from a single instrument, the Camberwell Family Interview (CFI), which is a standard interview that is administered separately to each relative and is typically administered during an exacerbation of symptoms (Vaughn & Leff, 1976*b*). The interviews now take 1–1½ hours and ratings are made from audiotapes. The key scores for EE are a frequency count of critical comments about the patient, and global ratings of hostility and 'emotional overinvolvement' (EOI). Critical comments are unambiguous statements of disapproval or resentment, rejecting remarks, or statements that are delivered with a critical tone of voice. Hostility is present when there is rejection of the patient or expression of a global criticism. EOI refers to self-sacrifice, overprotection, or overidentification with the patient. An interview is rated high in EE if there is one or more of the key features: a high level of criticism (usually 6 comments or more), a score above 3 or 4 on the 6-point EOI scale, or any degree of hostility. Only one high-EE interview is required for the family assessment to score as high EE.

Most of the research on EE has focused on the prediction of schizophrenic relapse, but it soon became evident that EE could be a more generalised risk factor for relapse in psychiatry and health psychology. There is now some evidence that EE predicts outcomes in depression (Vaughn & Leff, 1976*a*; Hooley *et al*, 1986; Hooley & Teasdale, 1989), bipolar disorder (Miklowitz *et al*, 1986, 1988; Priebe *et al*, 1989), and weight reduction (Fischman-Havstad & Marston, 1984; Flanagan & Wagner, 1991). A number of other studies are currently in progress.

Even within schizophrenia, not all of the studies have supported the EE hypothesis, and the notion that EE might be a determinant of relapse has attracted scientific controversy (Hogarty *et al*, 1986; MacMillan *et al*, 1986, 1987; J. Mintz *et al*, 1987). Families and health researchers have also expressed concern that EE may be used to blame families for the disorder (Hatfield *et al*, 1987; Kanter *et al*, 1987). The present paper reviews these issues and discusses implications for practitioners.

Outcome studies on schizophrenia

Studies on the prediction of the course of schizophrenia from EE are shown in Table 1. Twenty-six are displayed, with a total sample size of 1323. The median relapse rate over 9–12 months is 21% for low EE, or less than half of the 48% rate in the high-EE group. This suggests that a phenomenon as valuable clinically as medication (30% relapse on neuroleptics, 65% on placebo; Davis, 1975). Among the studies that presented data on both high and low EE, 87% had higher absolute values for relapse when EE was high, and the difference in relapse rates was statistically significant in 70%. These results do not seem to be strongly affected by a publication bias in favour of significant results, since Table 1 is thought to provide an almost exhaustive list of the research at the time of preparation.

Given that the small sample sizes in these studies provided limited opportunity to detect binary

Table 1
Relapse data from prospective outcome studies on expressed emotion

	Percentage relapses and sample size			
	0 to 9–12 months		0 to 24 months	
	low EE % (*n*)	high EE % (*n*)	low EE % (*n*)	high EE % (*n*)
Brown *et al* (1962)[1]	28 (47)	76 (50)***	–	–
Brown *et al* (1972)	16 (56)	58 (45)***	–	–
Vaughn & Leff (1976*a*), Leff & Vaughn (1981)	6 (16)	48 (21)**	20 (15)	62 (21)*
Vaughn *et al* (1984)	17 (18)	56 (36)*	–	–
Moline *et al* (1985)[1]	29 (7)	71 (17)[2]	–	–
Dulz & Hand (1986)	65 (17)	48 (29)	–	–
MacMillan *et al* (1986)[1]	41 (34)	68 (38)*[3]	–	–
Nuechterlein *et al* (1986)[1]	0 (7)	40 (20)*	–	–
Karno *et al* (1987)	26 (27)	59 (17)*	–	–
Leff *et al* (1987, 1990*a*)	9 (54)	31 (16)*[4]	33 (46)	50 (14)
McCreadie & Phillips (1988)[1]	20 (35)	17 (24)	–	–
Parker *et al* (1988)	60 (15)	48 (42)	–	–
Cazullo *et al* (1988)	27 (11)	63 (8)*[5]	–	–
Gutiérrez *et al* (1988)[1]	10 (21)	54 (11)*	–	–
Tarrier *et al* (1988*b*, 1989)[6]	21 (19)	48 (29)*	33 (18)	59 (29)
Rostworowska *et al* (1987), Budzyna-Dawidowski *et al* (1989)[1]	9 (11)	60 (25)**	18 (11)	72 (25)**
Mózný *et al* (1989)[1]	29 (38)	60 (30)**	–	–
Montero *et al* (1990)[7]	19 (36)	33 (24)	–	–
Arévalo & Vizcarro (1989)	38 (13)	44 (18)	–	–
Ivanović & Vuletić (1989)	7 (31)	66 (29)**	–	–
Buchkremer *et al* (1991)	28 (40)	37 (59)[8]	–	–
Barrelet *et al* (1990)	0 (12)	33 (24)*	–	–
Vaughan *et al* (1992)	25 (40)	53 (47)**	–	–
Median relapses[9]	21	48	27	61
High-EE control groups in treatment studies				
Leff *et al* (1982, 1985)	–	50 (12)	–	75 (12)
Falloon *et al* (1982, 1985)	–	44 (18)	–	83 (18)
Hogarty *et al* (1986, 1987)	–	28 (29)	–	66 (27)
Median relapses across studies[9]	21	48	27	66
Totalling subjects across studies	23 (605)	50 (718)	29 (90)	66 (146)

*$P \leqslant 0.05$, **$P \leqslant 0.01$, ***$P < 0.001$.

1. These studies measured relapses over 12 months. All other studies had follow-ups lasting 9 months. A 12-month follow-up in Leff *et al* (1987) was measured from admission and approximated 9 months from discharge.
2. High EE was based on critical comments ⩾6 and EOI ⩾4. The result using a cut-off point of ⩾10 critical comments was low EE 31% (13), high EE 91% (11)**.
3. Follow-up varied from 6 months to 2 years. The calculation excluded five subjects who were lost during follow-up; if they did not relapse, the rates would be low EE 39% (36), high EE 63% (41).
4. Rates are based on clinical diagnosis from all known information. The rates based on PSE and CATEGO diagnoses were: low EE 14% (37), high EE 33% (12).
5. These rates were for exacerbations of symptoms, using critical comments ⩾6 and EOI ⩾4. There was no significant predictive effect for EE when the EOI cut-off point was 3. If psychiatric admissions were used as the criterion for relapse, low EE had 9% and high EE 63%*.
6. The Tarrier *et al* (1988, 1989) results are from control groups in a treatment study.
7. With a criterion of critical comments ⩾4 and EOI ⩾3, the results became low EE 10% (30), high EE 40% (30)*.
8. These data are 9-month re-admission rates read from a graph.
9. A study by Straube *et al* (1989) could not be included in this table because relapse proportions were not reported. EE had a correlation of 0.22, $P < 0.10$, with relapses over 2 years.

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outcomes, this is substantial support for the predictive utility of EE. If the true difference in outcomes for low v. high EE were 0.20–0.50, a sample size of 43 in each group is required before 70% of the studies are reliably expected to show a significant difference at $P<0.05$ (Fleiss, 1973). Only four studies approach this number in each group, and the median number is only 21 (low EE) to 25 (high EE).

Methodological issues

It remains to be seen whether the methods used in the studies in Table 1 allow a conclusion in favour of EE as a predictor.

Assessment of symptoms and diagnoses

The studies typically derived an initial diagnosis of schizophrenia, schizoaffective disorder or paranoid psychosis from a standard interview schedule such as the Present State Examination (Wing *et al*, 1974; cf. Parker *et al*, 1988). In most of the studies a major exacerbation in florid schizophrenic symptoms was used as the sole criterion for relapse, and the relapse assessments were undertaken by clinicians who were blind to EE status (e.g. Nuechterlein *et al*, 1986; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984). Although some of the confirming studies relied heavily on retrospective reports of symptoms (e.g. Vaughn & Leff, 1976*a*; Možný *et al*, 1989; Vaughan *et al*, 1992), ones that had more frequent follow-up assessments had comparable results (e.g. Vaughn *et al*, 1984).

Assessment of EE

All of the researchers in Table 1 used the CFI for their assessment of EE. While the status of the concept should not rest on a single operationalisation, the similarity in assessment provides comparability across the studies. The inter-rater reliability of CFI scores is well established (Brown & Rutter, 1966; Rutter & Brown, 1966; Vaughn & Leff, 1976*b*), and is maintained by a training course and reliability assessments. Like other self-report instruments, the CFI is subject to self-presentation effects (Parker *et al*, 1989), but this would not increase the predictive efficacy of EE unless the ability to 'fake good' produces a more positive outcome.

When a dichotomy of high v. low EE is formed from a series of continuous variables (criticism, EOI), predictive results could be inflated by shifting the criteria to suit the observed outcomes in each study (Parker *et al*, 1988). Criteria for high and low EE are displayed in Table 2. Since the cut-off points were established in the initial studies by Brown *et al* (1972) and Vaughn & Leff (1976*a*), the only consistent change has been a reduction in the EOI cut-off from 4 to 3 in most of the research published after 1986. Changes in cut-offs were used to 'rescue' initially non-significant results in only three papers (Moline *et al*, 1985; Cazullo *et al*, 1988; Montero *et al*, 1990), and the data in Table 1 are based on the standard criteria wherever possible. The data do not support the contention that EE results are significantly affected by changing criteria. Given the variety of cultural settings in which the CFI has been employed, it is in fact remarkable that there have not been more changes to the criteria across the studies.

The status of the EE concept does not rest on the CFI alone. Similar predictive effects have been obtained with a five-minute speech sample by relatives, using indices of anxiety and hostility towards others (Gottschalk *et al*, 1988). Furthermore, it is sometimes forgotten that the original predictive study by Brown *et al* (1962) was not based on the CFI, but on observation of relatives and patients in a joint interview. Negative or intrusive family interactions have also been associated with schizophrenic relapse in studies on affective style (Doane *et al*, 1981, 1986), a concept that is closely related to EE. A combination of the CFI and a standardised family interaction task seems to provide the most powerful predictive measure of the EE/affective style concept (Goldstein, 1987; Miklowitz *et al*, 1988), and may prove to be the dominant method in future research.

Some data from the Parental Bonding Instrument (PBI; Parker, 1983) has also supported a relationship between family relationships and the course of schizophrenia. In a study by Parker *et al* (1982), patients who reported receiving low levels of care and high levels of protection from their parents had an elevated readmission rate over the next nine months. Consistent with EE, predictive effects were strongest for patients in high contact with their relatives. However, a second study did not replicate the predictive results (Parker & Johnston, 1987) and PBI scales were only weakly associated with EE variables (Parker *et al*, 1989).

Representativeness of the samples

Refusal rates in these studies are usually within acceptable limits (e.g. 3% in Barrelet *et al*, 1990; 5% in Brown *et al*, 1972; 12% in Vaughn & Leff, 1976*a*; 15% in Leff *et al*, 1987; 26% in MacMillan *et al*, 1986). If losses to later assessments are added, these rates sometimes increase (e.g. to 10% in Barrelet *et al*,

Table 2
Proportions of high-EE households, criteria for high EE, and location of studies

	% high EE	Criteria for high EE[1]		Location
		Critical comments (number)	EOI (0–5 scale)	
Brown *et al* (1962)[2]	52	-	-	London, UK
Brown *et al* (1972)[3]	45	⩾7	⩾4	London, UK
Vaughn & Leff (1976*a*)	57	⩾6	⩾4	London, UK
Vaughn *et al* (1984)	67	⩾6	⩾4	Los Angeles, USA
Moline *et al* (1985)	71	⩾6	⩾4	Chicago, USA
Dulz & Hand (1986)	56	⩾6	⩾4	Hamburg, Germany
MacMillan *et al* (1986)	53	⩾6	⩾3	London, UK
Nuechterlein *et al* (1986)	74	⩾6	⩾4	Los Angeles, USA
Karno *et al* (1987)	41	⩾6	⩾4	Los Angeles, USA (Mexican-Americans)
Leff *et al* (1987)	23	⩾6	⩾3	Chandigarh, India
Wig *et al* (1987)	54	⩾6	⩾3	Aarus, Denmark
McCreadie & Phillips (1988)[4]	42	⩾6	⩾3	Nithsdale, UK
Parker *et al* (1988)	74	⩾6	⩾3	Sydney, Australia
Cazullo *et al* (1988)	42	⩾6	⩾4	Milan, Italy
Gutiérrez *et al* (1988)[4]	34	⩾6	⩾3	Galicia, Spain
Rostworowska *et al* (1987)	69	NK	NK	Crakow, Poland
Tarrier *et al* (1988*b*)	77	⩾6	⩾3	Salford, UK
Možný *et al* (1989)	44	⩾7	⩾3	Kromeriz, Czechoslovakia
Montero *et al* (1990)	40	⩾6	⩾3	Valencia, Spain
Arévalo & Vizcarro (1989)	58	⩾6	⩾3	Madrid, Spain
Ivanović & Vuletić (1989)	48	⩾6	⩾3	Belgrade, Yugoslavia
Buchkremer *et al* (1991)	60	NK	NK	Munster, Germany
Barrelet *et al* (1990)	67	⩾6	⩾4	Geneva, Switzerland
Vaughan *et al* (1992)	54	⩾6	⩾3	Sydney, Australia
Median across studies	54			

NK, not known.
1. These are criteria for the outcomes described in Table 1. High EE is scored if either criticism or emotional overinvolvement exceed the criterion level, or if hostility is present.
2. Not rated from the CFI.
3. Brown *et al* (1972) also used measures from a joint interview with the client and a key relative (2 critical comments or marked EOI).
4. The CFI was not administered during a relapse.

1990; and to 40% in MacMillan *et al*, 1986), but in most cases they do not present significant problems for representativeness. If exclusions are also taken into account, such as patients living alone, living with non-English-speaking relatives, or not being discharged, participation rates sometimes fall considerably (e.g. to 30% in MacMillan *et al*, 1986; and to 52% in Parker *et al*, 1988). These exclusion rates do not detract from the methodological rigour of the EE studies, but they indicate that the EE literature is based on a subset of the schizophrenia population.

Limitations to the predictive effect

Contact with relatives

If EE is an index of problematic interactions within the home, patients should be more at risk when they are in greater contact with their relatives. In three of the outcome studies, EE had little effect on relapse when contact with relatives was below 35 hours per week (Brown *et al*, 1972; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984). Furthermore, some of the studies that have not observed a predictive effect for EE have had relatively few subjects who were in high contact with their relatives (e.g. Dulz & Hand, 1986; McCreadie & Robinson, 1987).

Contact has had little bearing on the results of some of the outcome studies (e.g. Karno *et al*, 1987; Parker *et al*, 1988). Assessment of the level of contact before a relapse would be expected to moderate EE only if it had substantial stability over time. There are some indications that contact may now be subject to substantial fluctuation (Parker *et al*, 1988) because of the range of community programmes and short-term accommodation that is currently available. In addition, most of the work on EE and contact has relied on a retrospective estimation that is made at the same time as the CFI and has not usually been checked against monitoring data. As a result of these factors, the role of contact between family members is difficult to evaluate.

If the effects of EE are substantially reduced when contact time is low, this has important implications for clinical practice. When there are few people with schizophrenia in the population who are in regular contact with their families, EE may seem to have less practical relevance. For example, McCreadie & Phillips' (1988) epidemiological sample had only 13% of their subjects in high contact with their families. However, some other studies have observed much higher rates of contact. In Barrelet *et al* (1990), which examined first episodes of illness, 74% were living at home and 70% had more than 35 hours of contact a week with their relatives.

Clearly, the rate of high contact with relatives will vary across populations. However, I argue that EE is potentially relevant even where contact is low. One reason is that contact time is only an indirect measure of the probability that patients will be exposed to negative interactions, and there are some circumstances where low levels of contact may still be associated with significant levels of distress. EE may also be relevant to non-familial households, although there are few data on that issue.

When contact does moderate EE effects, it is not clear how this should be interpreted. Contact is probably confounded with higher levels of skills and attainments: clients may be in low contact because they are working or attending day centres (Leff *et al*, 1982), and active relatives may be functioning at a higher level as well. Reduced contact is also confounded with beneficial effects from other activities such as their impact on negative moods (Lewinsohn & Graf, 1973). This point is important for practitioners, since it suggests that simply recommending a reduction of contact may not be as effective as helping patients and relatives to gradually increase independent activities that they find enjoyable (cf. Leff *et al*, 1982).

Neuroleptic medication

Vaughn & Leff (1976), Vaughn *et al* (1984), and Vaughan *et al* (1992) observed that a regular intake of neuroleptics appeared to reduce the predictive effect of EE. However, a number of other studies have not confirmed that view (e.g. Leff & Vaughn, 1981; Moline *et al*, 1985; Karno *et al*, 1987).

In most of the research, effects of medication have been confounded with compliance, and thus with other variables that may promote a positive outcome. Nuechterlein *et al* (1986) eliminated the effects of compliance by giving subjects regular injections of fluphenazine decanoate. Despite receiving medication, 40% of the high-EE group relapsed over nine months, compared with none from low-EE environments. This suggests that clinicians cannot rely on medication alone to protect the patient from the impact of a stressful interpersonal environment, although it remains an important factor in reducing relapse (Davis, 1975). The optimum treatment strategy appears to be a combination of medication with a social intervention (Goldstein & Kopeikin, 1981); this may often allow medication doses to be reduced (Falloon *et al*, 1985).

Variations in the EE assessment

The current assessment of EE requires only one high-EE interview for a social environment to be rated high in EE. This is based on the assumption that criticism or overprotection from one member of the environment is sufficient to increase risk of relapse, even when there are others of low EE. There is some evidence that appears to support this proposition. When the patient lived in a two-parent family, most studies that supported EE as a predictor seem to have interviewed both parents in at least 75% of cases. On the other hand, in the studies by Dulz & Hand (1986) and MacMillan *et al* (1986), most CFI assessments included only one relative. Omission of some relatives with high-EE attitudes may have contributed to the relatively high relapse rates for low EE in those studies (65% and 41% respectively). This issue is important for the concept, since it suggests that the presence of low EE from one person in the environment may often be insufficient to moderate the impact of high EE from another person.

The predictive efficacy of EE also appears to be more established when the CFI is administered in the usual manner. For example, the CFI is most sensitive to high EE during a relapse (Brown *et al*, 1972; Tarrier *et al*, 1988*a*); if applied at other times it may have less ability to predict relapse (McCreadie & Phillips, 1988; cf. Gutiérrez *et al*, 1988). The normal CFI format was departed from in at least one disconfirming study (Parker *et al*, 1988), although its results were substantially the same when ambiguous EE assessments were excluded.

Prediction of subjects at extremes of risk

As the overall relapse rate departs from 0.50, it becomes progressively more difficult to detect true differences of the same relative magnitude unless the sample size increases. This may account for some of the predictive failures of EE (e.g. McCreadie & Phillips, 1988). Additional problems are posed by the inclusion of some subjects who are at very high risk of relapse. Studies that confirmed the predictive power of EE usually excluded subjects who have a high degree of persisting symptoms from their previous episode, since they present difficulties in identifying an exacerbation (Vaughn *et al*, 1984). The inclusion of such subjects in Dulz & Hand (1986) and Parker *et al* (1988) could have reduced the power of social factors in those studies.

There is another way to read the data on extremes of risk. Perhaps the course of the disorder for subjects with persisting symptoms is so strongly influenced by biological determinants that there is little remaining variance to be explained by social factors (Vaughn, 1986). Similarly, subjects at very low biological risk may be less sensitive to the effects

of EE. However, the fact that some studies with relatively low relapse rates do obtain significant effects for EE (e.g. Leff *et al*, 1987; Barrelet *et al*, 1990) suggests that low overall rates do not impart immunity from an EE effect.

Chronicity

The prediction from EE does not appear to be restricted to subjects with long-established disorder (cf. Barrelet *et al*, 1990). Time since the initial hospital admission does not usually moderate EE effects (Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984; Karno *et al*, 1987), and most studies including early-onset subjects have supported EE as a predictor (Leff *et al*, 1987; MacMillan *et al*, 1987; Nuechterlein *et al*, 1986; cf. Dulz & Hand, 1986).

Gender

The evidence in favour of EE is more strongly established for male than for female patients (Vaughn *et al*, 1984; Hogarty, 1985; Vaughan *et al*, 1992). However, significant interactions with gender are not usually obtained. Most studies have fewer women than men in their sample, and this makes it more difficult to obtain a statistically significant effect for gender. Gender did not moderate EE effects in recent studies with Mexican-Americans (Karno *et al*, 1987) and Indian families (Leff *et al*, 1987), so it is premature to conclude that EE effects are limited to men across all cultural groups. Future research on the issue should have sufficient numbers of men and women to allow a more powerful test.

Even if EE predicts relapse in both sexes, tolerance of specific types of client behaviour may be affected by views about the gender appropriateness of the behaviour (Goldstein & Kreisman, 1988). These views may have important implications for the development of criticism, retention of clients in the home, and the decision to seek admission.

Period of high risk

The primary risk period for EE-related relapse appears to be in the first nine months after an admission. Subjects from high-EE environments who have *not* relapsed by that time are not at a significantly higher risk of a major relapse than those from less stressful environments (Hogarty *et al*, 1988; Leff & Vaughn, 1981). One reason for this is that a substantial proportion of the sample at high risk (the ones who have already relapsed) are omitted from such an analysis. Another reason for the result may be the limited stability of the CFI: some of the households that originally displayed high EE will have become low EE.

While there appears to be a fall in the relative risk of relapse if subjects have not relapsed for a significant period, there are some data suggesting that EE may continue to have some subtle effects. Hogarty *et al* (1988) found that subjects in a high-EE environment who had not relapsed in the first six months had more minor episodes at around 18–30 months if they were being maintained on a low dose of neuroleptic medication (20% of their stabilisation dose). In the absence of family intervention, these people may require a higher maintenance dosage.

Prediction of functioning, negative symptoms

The predictive effects in Table 1 refer to relapses of positive symptoms. Up to now the EE literature has paid little attention to prediction of negative symptoms or indices of social and occupational functioning (El-Islam, 1989). However, a plausible argument could be made that criticism and over-protection could undermine patients' confidence in their capabilities and that this in turn would reduce their motivation to engage in challenging activities and to persist in the face of setbacks (Bandura, 1982). Similarly, overprotection should limit patients' opportunities to develop specific skills, because they will often be prevented from engaging in the relevant activity, or will have it performed for them. Together, these factors should produce a difference in the motivation, activity level, and functional capability of people from high-EE and low-EE environments.

There is some evidence supporting an association of EE and social functioning. Somewhat different results have been obtained when functioning is examined during different time periods. In the seminal study by Brown *et al* (1972), patients from high-EE environments had poorer social functioning and more behavioural disturbance in the previous two years. Miklowitz *et al* (1983) found that poor premorbid social functioning was not related to EE status, but was associated with EOI. In contrast, current scores on recreation, independence and overall functioning were poorer when subjects came from a high-EE environment in the study by Barrowclough & Tarrier (1990): these results were due to an association with hostility rather than EOI.

None of these results involved predicting later levels of functioning from EE. One study does examine this question. Hogarty *et al* (1988), who were examining the effects of maintenance neuroleptic treatment, observed the outcomes of subjects who had not relapsed during an initial stabilisation period of around six months. In this population, patients from low-EE settings had better social

functioning during the next two years. Unfortunately, this study does not establish whether this represents an effect of EE on functioning or the reverse. It does, however, suggest that we should pay more attention to social functioning as an outcome variable.

Prediction of hospital admission

The predictive value of EE is more consistent in studies that have used exacerbation of symptoms as the sole criterion of relapse than in others that included admission to psychiatric hospital among their relapse criteria (Dulz & Hand, 1986; McCreadie & Phillips, 1988; Parker *et al*, 1988). The only studies to find significant predictive effects of EE on admission rates are those by Cazullo *et al* (1988) and Vaughan *et al* (1992). (My reanalysis of the results by Gutiérrez *et al* (1990) shows an effect of EE on admission of $P<0.10$; Buchkremer *et al* (1991) report a significant effect over 18 months using a log-rank test, but the χ^2 results do not reach significance.)

While the use of relapse rather than admission restricts the ambit of EE, it does not necessarily present theoretical problems for the concept. Admissions can be affected not only by the patient's symptoms but also by the patient's consent, the treatment policies of clinicians or hospitals, and other people's tolerance of bizarre behaviour (Goldstein & Kreisman, 1988). In some settings, these factors can degrade the integrity of admission as a criterion for relapse of symptoms to the point where admissions and symptoms bear little relation to each other (Vaughn *et al*, 1984).

The nature of EE

The predictive status of EE appears to be on relatively firm ground, even if its application is more limited than was first thought. But what is the nature of EE, and what could be the mechanism for the predictive results?

Components of EE

As we might expect, critical comments and ratings of hostility are significantly correlated, and criticism is usually more common (Vaughn *et al*, 1984). However, cultural factors can result in hostility being evidenced even when levels of criticism are low (e.g. Leff *et al*, 1987).

While criticism and hostility are usually related, the data offer dubious support for a global EE concept that encompasses both criticism and EOI (Vaughn *et al*, 1984; Szmukler *et al*, 1987). In the study by Vaughn *et al* (1984) the correlation between the two components was 0.30 for fathers, but only −0.03 for mothers. Psychophysiological measures have been unable to distinguish between subjects from households of high EOI and those from households of high criticism (Tarrier *et al*, 1988*a*), but this does not mean that the variables are equivalent in other respects.

The limited correlation between EOI and criticism has probably had little impact on the predictive studies. Criticism usually exerts more power over EE classification than does EOI, because of its greater frequency in most samples (e.g. Brown *et al*, 1972; MacMillan *et al*, 1986; cf. Vaughn *et al*, 1984). There is also a substantial overlap between households that are above the cut-off points for EOI and criticism (Brown *et al*, 1972). A low correlation between the components as continuous variables is of little detriment to a dichotomous EE assessment.

Among the EE variables, criticism seems to make the greatest contribution to relapse in most of the studies that have examined the issue (e.g. Moline *et al*, 1985; MacMillan *et al*, 1986; Hogarty *et al*, 1986; Barrelet *et al*, 1990). However, some studies have found hostility (Parker *et al*, 1988; Leff *et al*, 1990*a*) or EOI (Gutiérrez *et al*, 1988) to be more sensitive predictors. The relative effects of components may differ across samples according to their relative frequency and the sensitivity of the assessment to cultural differences in their expression.

Stability over time

Expressed emotion is most evident on the CFI during an admission. If subjects are retested on the CFI 6–12 months after discharge, 50% or more of the people who initially showed high EE obtain low-EE ratings (Dulz & Hand, 1986; Tarrier *et al*, 1988*b*; Leff *et al*, 1990*a*). This is not a regression effect, since only one in six people with low EE show high EE at retest (Dulz & Hand, 1986). There is some evidence that criticism is more unstable than EOI (Brown *et al*, 1972), but this difference is not found in all studies (Dulz & Hand, 1986; Hogarty *et al*, 1986; Tarrier *et al*, 1988*b*). Changes in CFI scores do not present problems for outcome studies if they routinely take the EE index when the patient is experiencing a symptom crisis, as most of the studies in Table 1 did (cf. Gutiérrez *et al*, 1988; McCreadie & Phillips, 1988).

Fluctuations in CFI ratings do have important theoretical implications. If EE were only evident during a relapse, it would be unlikely to affect the later course of the disorder. Is it just that the CFI is insufficiently sensitive to detect EE between relapses, or do the interactions change in quality?

Miklowitz *et al* (1989) show that the changes are not restricted to the CFI. In their study there were no significant relationships between EE and family interactions in the laboratory some eight weeks later. While these data do not show that interactions in the home are unaffected, they are consistent with the interactions changing with alterations in symptom status. On the other hand, Tarrier *et al* (1979) obtained some consistency in schizophrenia sufferers' affective reactions to their relatives. Sufferers' electrodermal responses were measured after a relative entered the testing room. High EE was associated with less adaptation than low EE, even when the EE interview had taken place as long as two years previously. Sufferers are reacting as though the relative's behaviour is stable over time: it is unclear whether this means that past events are still affecting them, or whether undetected changes are continuing at home.

The development of EE

High EE is not related to a poorer pre-morbid adjustment, but the EOI component is (Miklowitz *et al*, 1983). EOI seems to develop as the disorder becomes more chronic and is relatively rare among families of first-episode patients (MacMillan *et al*, 1986). To some extent this may be an artefact of the EOI scale, which may take insufficient account of the patient's disability when overprotection is being assessed. Alternatively, relatives may overestimate patients' deficits after observing several florid episodes.

Since critical comments on the CFI are higher when patients' symptoms are worse and their behaviour is more disturbed (e.g. MacMillan *et al*, 1987), it is plausible to assume that this is because the behaviour is more aversive and difficult to manage (Greenley, 1986). Aggressive behaviour is especially provocative (Creer & Wing, 1974; Hatfield, 1979), and mothers of sufferers of paranoid schizophrenia are more critical than those who have children with disorganised features (Ivanović & Vuletić, 1989). This 'coping strategy' view of EE is consistent with observations that one-parent families are more likely to show high EE (Parker *et al*, 1988).

Both EOI and criticism are probably affected by knowledge about the disorder and attributions of the behaviour. People rated high in EE appear less informed about schizophrenia and more often ascribe difficult behaviour to the person rather than the disorder (Cozolino *et al*, 1988; Brewin *et al*, 1991). 'Negative symptoms' are especially prone to misattribution (Leff & Vaughn, 1985), since they are less obviously related to the disorder than are hallucinations or delusions. This is not to say that simply giving the family information about schizophrenia will automatically change these reactions. Brief informational interventions have been relatively ineffective at changing family attitudes and reactions (Smith & Birchwood, 1987; Tarrier *et al*, 1988*b*). Experience with these interventions suggests that education needs to address entrenched attitudes and be sensitive to factors such as unresolved grief about the disorder. If it is to produce sustained changes in attitudes, the educational programme should encourage families to test ideas between sessions (Tarrier *et al*, 1988*b*), since enactive information often provides the most potent challenges to pre-conceptions (Bandura, 1982).

The CFI and family interactions

Electrodermal data suggest a mechanism for the effects of EE on the patient, but they do not show what it is that is triggering the affective responses. Data on interviews and interactions in the laboratory suggest that the mediating factors involve the types of communications between family members. Relatives who are more highly critical on the CFI make more critical remarks to the patient during interaction tasks, and those who have high EOI are somewhat more intrusive (Valone *et al*, 1983; Miklowitz *et al*, 1984; Strachan *et al*, 1986*a*; Szmukler *et al*, 1987; Hahlweg *et al*, 1989). People who show high EE also talk more in a family interview and listen less effectively (Kuipers *et al*, 1983). The laboratory tasks also highlight the reciprocal influences within the household. For example, patients in high-EE environments are more critical of their relatives and less autonomous in their coping style (Goldstein *et al*, 1989; Strachan *et al*, 1989).

There are no published observations of EE-related interactions in the home, but reported incidents show more 'helping and protecting' with low EE, and fewer 'belittling and blaming' responses (Hubschmid & Zemp, 1989). When EE is low, patients and relatives report that they tailor their responses to the behaviour of other members: high EE is associated with less flexibility, more coercion, and more vigilance (Hubschmid & Zemp, 1989). As a result, the home environment appears to be more prone to conflict.

Another reported feature of the high-EE environment is that interpersonal responses are more inconsistent over time (MacCarthy *et al*, 1986). Since the disorder induces deficits in information processing, ambiguity and inconsistency may be problematic for a sufferer of schizophrenia. People with more severe deficits are likely to find unpredictability in the home

environment especially confusing. This raises the intriguing possibility that such patients may be especially vulnerable to the effects of EE on the development of delusions. Inconsistency in social interchanges would also produce difficulties for less ill patients, since it would deprive them of information they could use to prevent aversive interchanges.

Cultural variations in EE

As Table 2 shows, high EE is much more frequent in the West than in India. Among rural Indian families the incidence of high EE was only 8% (Wig *et al*, 1987). Kuipers & Bebbington (1988) present an analysis that uses the low incidence of EE to explain the reduced risk of relapse that is seen in developing countries (World Health Organization, 1979; Waxler, 1979). Part of the reason for reductions in EE and relapse rates may be the greater involvement of extended families in these cultures (Wig *et al*, 1987). El-Islam (1982) found that extended families in an Arab culture were more tolerant of eccentric behaviour and temporary withdrawal than were nuclear families. They also encouraged more social activity without overly taxing the patient's social resources. Such behaviour is similar to low-EE responses to the patient's problem. Reactions like these by the extended family not only would produce a less stressful environment for the patient, but would model low-EE behaviour of members of a nuclear family. If these results transfer to other cultures, they would emphasise the importance of maintaining traditional links with extended families.

EE and affective responses

Brown *et al* (1972) suggested that EE produces relapse by raising the patients' arousal beyond an optimal level. Data on emotional arousal and EE are particularly interesting, because they attempt to test this proposition. Reviews are presented by Turpin *et al* (1988) and Tarrier (1989). When patients are first tested, subjects from high-EE settings either have a higher frequency of skin conductance responses (Sturgeon *et al*, 1984), or they show less adaptation after the relative enters the room (Tarrier *et al*, 1979, 1988*a*). As already mentioned, the differences are present even when the CFI assessment had been undertaken a considerable time before. However, the differences do not recur when subjects are retested three to nine months later (Tarrier *et al*, 1979, 1988*b*) unless the subjects are in high contact with their relatives. The authors argue that the stress of an initial testing allows more sensitive measurement of affective reactions to high EE.

Support for the view that these negative emotions are implicated in relapse is provided by the observation that features such as depression and hostility frequently precede a relapse (Herz & Melville, 1980; Subotnik & Nuechterlein, 1988; Birchwood *et al*, 1989). However, there is little information on the process by which relapse might be triggered by these emotions. One possibility is that the emotional states could have direct effects on the biochemical course of the disorder. An alternative view looks at psychological effects of the emotions. Increases in anxiety, depression or anger that are sustained or severe evoke behavioural sequelae such as insomnia, lack of interest in activities, social withdrawal, and aggression (Herz & Melville, 1980). When such behaviour triggers aversive social interactions, it may provide apparent substantiation for paranoid ideas. When the behaviour cuts the person off from social contact, it reduces the patient's sources of assistance and corrective information. Negative emotions also colour interpretations of events and are likely to undermine the client's ability to concentrate, test the validity of ideas, and control emerging symptoms (Falloon & Talbot, 1981; Breier & Strauss, 1983; Glynn *et al*, 1990). If these effects of the emotions occurred when the person was physiologically vulnerable to positive symptoms, they would increase the probability of relapse.

One apparent problem with this hypothesis is that people with schizophrenia are, in laboratory tests, notoriously prone to error in verbally identifying emotional reactions of other people (e.g. Cramer *et al*, 1989), but there are several reasons for caution in interpreting these data. One is that much of the research has been undertaken with in-patients or with ratings being made of people who are unknown to the client. Naturalistic interactions also differ from the laboratory tasks in the presence of a substantial degree of redundancy and of emotion labelling by others in the social environment. Further potential problems involve the verbal facility of subjects and their willingness to confide their true reactions to a tester. All of these factors could lead to exaggerations of deficits if the studies are used to predict everyday perceptions of emotion and emotional reactions by people with schizophrenia.

The psychophysiological evidence demonstrates that people with schizophrenia do experience powerful emotional reactions in response to negative interactions. The restriction of predictive effects in the EE literature to interactions that have a critical, hostile or intrusive character provides further support for the proposition that patients are perceiving the

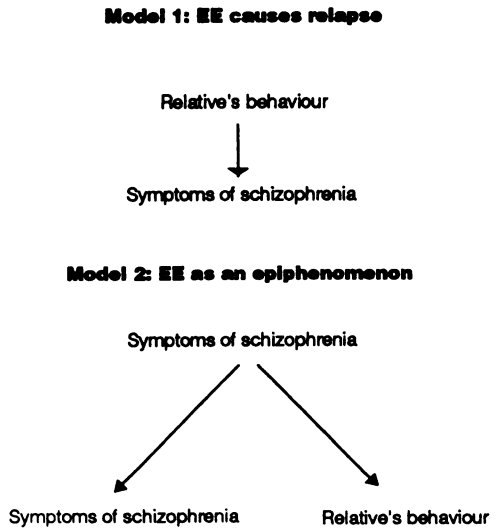

Fig. 1 Unidirectional models of EE and schizophrenia.

interactions accurately on sufficient occasions for this to have a substantial effect.

Status of EE as a determinant of relapse

The proponents of EE were not only advancing it as a predictor of relapse, but argued that EE was involved in producing it (Brown *et al*, 1972; Leff & Vaughn, 1985). The issues in the preceding section obviously bear on this issue. However, it remains to evaluate it against alternative hypotheses. Two causal models are displayed in Fig. 1. Model 1 represents a simplified version of EE as a determinant of relapse, where the family's behaviour triggers an exacerbation of symptoms in the patient. This is a popular conception of the EE hypothesis that is fed by some reviews and by the CFI's focus on assessment of relatives. Understandably, many families resent the implication that they are responsible for relapse, and feel that the model is inadequate. It is important to remember that the proponents of EE (Brown *et al*, 1962, 1972; Vaughn, 1989) have not been arguing for such a simplistic view. Implicit in their work is some form of interactive model that recognises the role of biological factors and acknowledges the impact that a patient's behaviour can have on the responses of relatives.

If symptoms do alter family behaviour, an alternative view of EE is that the family attitudes are epiphenomenal to relapse (model 2 in Fig. 1). Symptoms are produced by other variables such as an underlying disease process, and high EE is simply correlated with more severe forms of the disorder. However, most studies find that the relationship between EE and relapse is not explained by patient characteristics such as severity of symptoms or premorbid adjustment (Brown *et al*, 1962, 1972; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984; Karno *et al*, 1987; Gutiérrez *et al*, 1988; cf. Leff *et al*, 1987). While these studies have tested individual variables, two studies have demonstrated the independent contribution of the EE variables in multivariate analyses, albeit with small sample sizes (Barrelet *et al*, 1990; Straube *et al*, 1989; Vaughan *et al*, 1992).

A study by MacMillan *et al* (1986, 1987) appeared to support the idea that a relationship between EE and relapse could be due to other factors. Once the investigators controlled for the joint effects of medication and time since the onset of the illness, an apparent association between EE and relapse was explained. However, there are good reasons to be cautious about this evidence. In the case of medication, the study confounded EE with the allocation to active v. placebo treatments, so that subjects treated with neuroleptics were more likely to fall in the low-EE group. By controlling for treatment group, MacMillan *et al* assumed that all of the intergroup differences were due to medication: it could equally be argued that some effects ascribed to the medication could actually have been produced by EE. A similar problem was produced when the authors controlled for the untreated duration of illness, as reported by relatives. Mintz *et al* (1989) showed that parents of low EE report a shorter illness than is established from other sources, whereas relatives with high EE are more accurate. Control by the relatives' duration report could have washed out part of a true EE effect.

A more definitive test of the EE hypothesis would be obtained if a means could be found to examine the impact of producing a change in EE while keeping other factors constant. At first glance, the family intervention literature provides such a test. Family interventions have had remarkable success in controlled comparisons with routine or individual treatments (Table 3), and the impact on relapse does seem to be greater for households that show a reduction in EE (Hogarty *et al*, 1986). However, the data are open to alternative interpretations, since the family interventions also increase skills that could have independently affected relapse. For example, family members may assist patients in controlling their symptoms and in increasing their social functioning. As a result we cannot be sure whether the direction of effects is from clinical improvement to EE or the reverse (Hogarty *et al*, 1986).

The idea that EE is a determinant of relapse is supported by data on the affective responses of patients. In some of the studies, these responses are specific to the presence of people showing high v. low EE (Tarrier, 1989). This specificity is inconsistent with the responses arising from alternative mechanisms such as a generalised affective vulnerability, and suggests a pathway through which EE may affect symptoms.

An interactive model of EE and relapse

Rather than supporting simple, unidirectional models of symptoms and EE, the evidence appears to be pointing to a model that incorporates reciprocal influences between symptoms and the reactions of other people. In an interactive model, patients' symptoms and other problem behaviour elicit frustration, distress and concern from other people, and prompt attempts to cope. Unfortunately, these attempts at coping sometimes involve critical or intrusive interactions. The resultant negative emotions exacerbate patients' symptoms and make it more difficult for them to function effectively.

This interactive approach sees the behaviour of both patients and other people as reactions to stress and as attempts to cope with it. In so doing, it implicitly recognises the role of two moderator variables: the interpretations that each actor makes of the other's behaviour, and the coping skills that each can bring to bear. Interpretations of behaviour that attribute a benign intent (e.g. an attempt to help me) reduce the chance of a negative interaction developing. Similarly, better coping skills (or a less confrontive style of coping) will increase the chance of a more positive outcome to the interaction. I would argue that a model of the interaction should explicitly include these moderator variables, and should embed the interaction within the context of external stressors that are simultaneously operating on the actors.

This description links the EE concept both with a cognitive–behavioural approach to aggression (e.g. Patterson, 1982) and with the literature on stress and social support. To adapt these approaches explicitly to schizophrenia, a further variable will have to be added: a biological vulnerability to schizophrenia which is shown by at least one of the parties. It is this factor that induces psychotic symptoms rather than depression, anxiety, or psychosomatic symptoms, and high levels of this vulnerability factor are expected to produce symptoms even when the level of ambient stress is low.

Figure 2 summarises a model that meets these criteria. It is a variant on a stress–vulnerability view

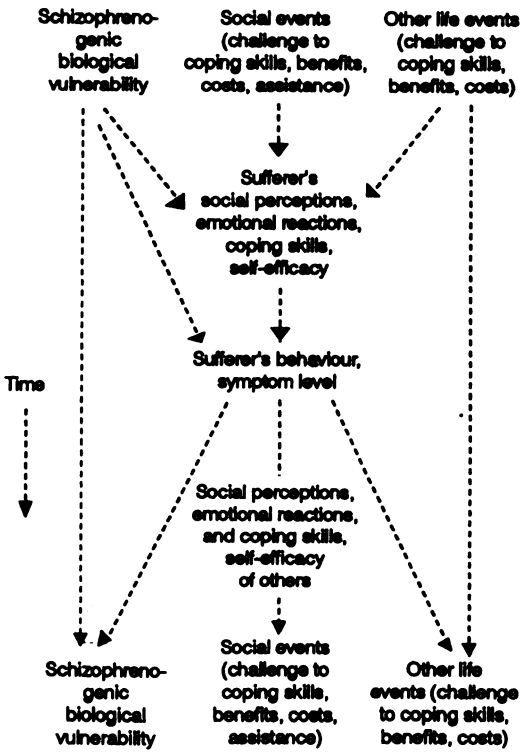

Fig. 2 An interactional model of the course of schizophrenia, describing the reciprocal determinism of risk factors through time.

of schizophrenia (Zubin & Spring, 1977; Nuechterlein & Dawson, 1984) which takes account of patients' biological vulnerability, cognitive processes and skills, and describes the interaction between these factors and their social context. Progression through time is described by a movement from the top to the bottom of Fig. 2.

Within the model, symptoms and behavioural problems that are shown by patients (centre of Fig. 2) are consequences of joint influences from life events (including EE) and problems in brain chemistry or structure that predispose to psychotic symptoms. Events elicit problem behaviour and psychotic symptoms when they are expected to have unpleasant consequences for patients (e.g. criticism or financial loss) or present them with a difficult challenge (e.g. a new relationship or more responsibility at work). This is especially likely if the events occur when the person is biologically very vulnerable. Effects of events are moderated by the interpretations patients place on them, and by their skills and self-efficacy in being able to deal with the challenges that they present (Bandura, 1982). Interpretations and

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coping are affected by the emotional distress that patients experience in response to the situational stressors, including the concerns they have about the disorder and its impact on their lives. Coping is undermined by the direct effects of the biological disorder such as hallucinations and defective information processing that leads to a delusional interpretation of events.

Just as the behaviour of patients is viewed as a stress reaction, so are the responses of other people. When patients engage in behaviour that is potentially self-destructive or has negative effects for others, they trigger attempts by other people to control the behaviour or to minimise its negative effects. When considerable effort is required to monitor the patient's behaviour and reduce its negative impact, the attempt can disrupt other activities and be seen as extremely burdensome (Fadden *et al*, 1987). Individual instances of problem behaviour can also elicit expressions of anger and frustration. Within the model, all of these reactions are moderated by interpretations of the behaviour and by the coping resources the person has available (Potasznik & Nelson, 1984). Sometimes attempts to deal with the situation are based on misunderstandings about the reasons for the patient's behaviour (e.g. believing that negative symptoms are due to laziness). Consistent with this account, people showing low EE are better informed about schizophrenia (Cozolino *et al*, 1988), and greater emotional distress by families is associated with EE *only* when the family does not see the patient as ill (Greenley, 1986). On some occasions the patient's behaviour is correctly interpreted, but people are unable to moderate their own stress reactions – for example, expressing their disappointment over a lack of progress. Reactions are also affected by perceptions of social role that may restrict the range of coping strategies that are available: an example is the notion that a caring mother has to protect a vulnerable son or daughter from every adversity throughout their lifetime.

When the attempts to cope with patients' behaviour involve interactions that are perceived as critical or intrusive, a potentially destructive feedback loop is established. The interactive cycle does not only occur through an increase in psychotic symptoms: as with any interchange, a short-term feedback loop can be created by negative responses from each actor. For example, a relative may try to encourage a patient to get out of bed by shouting to him/her. If the patient views this as an unwarranted intrusion he/she may respond angrily. Frequently both parties then become upset and the patient stays in bed even longer. There is evidence that hostile interchanges do occur in high-EE environments (Strachan *et al*, 1989). When negative interchanges occur frequently, expectations colour social perceptions and coping strategies may be automatised. So, the patient in the example may see a more gentle reminder to get up as the same sort of behaviour as the shouting on previous days. As a result, one person who makes an attempt to relate more positively is often met with unchanged responses from other members of the household (Hubschmid & Zemp, 1989). This does not mean that the model predicts an endless progression of negative interactions will necessarily occur. On a particular occasion a negative interaction cycle is often terminated by one or more people withdrawing from the scene or by new factors affecting behaviour. For example, a series of negative cycles may stop when the patient is admitted to hospital or receives increased medication. However, the emotional impact of interchanges can continue as participants mentally rehearse the events. The negative cycles can also become episodic, triggered by disputes, exacerbation of symptoms, or minor irritations that remind the participants of the previous cycle or happen to coincide with external stress. Recollections of past events colour the participants' perceptions and choice of coping responses, so that cycles of interpersonal distress and symptom development are unwittingly repeated.

As important as full-blown disputes may be, two studies draw attention to particular problems that may emerge from an association of hostility or coercion by relatives and withdrawal by patients. Birchwood & Cochrane (1990) showed videotaped excerpts of common types of patient behaviour to families of schizophrenia sufferers and asked them to report their usual coping reactions. The study provides an estimate of family behaviour between major episodes relatively early in the disorder, since testing was undertaken an average of 2.6 years after the first schizophrenic episode. When relatives reported more use of coercive strategies, the patient was more likely to have had a relapse in the previous two years. Coercive strategies in turn were associated with relatives' reports of withdrawal or inactivity by the patient. Converging evidence on the issue is offered by Tarrier & Barrowclough (1990), who found an association between withdrawal and high levels of skin conductance in patients from high-EE environments. The patients who showed higher sustained affective responses to EE were apparently the ones who were withdrawing from the scene. While the direction of influence is not yet clear, these studies suggest that sequences of high-EE behaviour and patient withdrawal may be especially important for both relapse and social functioning. The studies also provide support for the model outlined above,

as they are highly consistent with the model's emphasis on coping strategies.

Up to this point, I have used the interactive model in Fig. 2 to describe negative social influences. However, I am also struck by the possibility of using the model to describe positive effects from each of the factors. For example, social assistance may moderate the challenge that events impose on the coping resources of participants, and pleasurable events may help to reduce negative emotional reactions. Within the model, skills in problem solving, negotiation and anger control can prevent aversive interchanges or resolve the conflict before it has a significant impact on symptoms. These considerations have the potential to moderate the negative conceptual focus of EE (see below).

Interactive models are not new. They have been advanced by a number of previous commentators (Brown *et al*, 1972; Seywert, 1984; L. I. Mintz *et al*, 1987), and Birchwood & Smith's (1987) transactional model in particular bears a close resemblance to aspects of the present one. An interactional view is also implicit in family interventions that have been developed in response to the EE data (Leff & Vaughn, 1981; Falloon *et al*, 1984). The present model attempts to describe the contributing factors in more detail. While it acknowledges the complexity of relapse causation, it retains EE as a determinant and is open to disconfirmation.

Is EE a way to blame families for schizophrenia?

A social interaction model of schizophrenia may help to alleviate concerns that EE represents an attempt to blame families for schizophrenic relapse (Hatfield *et al*, 1987; Parker *et al*, 1988). In place of a unidirectional influence from relatives to patients, the model explicitly recognises the inter-relationship of patients' behaviour and the reactions of relatives, and sees EE as an understandable response to the difficulties that schizophrenia poses for the family. This attitude to EE is consistent with the views of its supporters (Leff & Vaughn, 1987; L. I. Mintz *et al*, 1987), who express concern that high EE should not become a pejorative label (Vaughn, 1986, 1989). Rather than blaming families, L. I. Mintz *et al* (1987) argue that EE puts a moral imperative on treatment agents to assist families better than they did in the past, and they point to the increased support that has been offered as a result.

When listening to the reports of relatives, I am not at all surprised that some become critical, hostile or overprotective when faced with the severely disturbed behaviour that is often displayed by schizophrenic patients. What I find more surprising is that some people manage to cope with the schizophrenia over long periods *without* developing such behaviour and attitudes. A number of other researchers seem to share this view. Christine Vaughn (1986) described an incident where a father was woken by his son who announced that he had a mission from God to kill him. In her account: "The father managed to disarm his son of a knife through gentle persuasion. 'Have a cup of tea, son, and let's talk about this.' He did not contact the hospital until the following morning" (p. 101).

Families frequently impress me with their skills, their courage, and persistence in providing assistance and encouragement for their relatives with schizophrenia, and I have felt that their attainments often surpass the levels that many health professionals could reach. Far be it for us to criticise them when they do not reach these heights of achievement!

However, 'high-EE relative' and 'high-EE family' are correctly perceived as pejorative labels, if not for family members, at least for their coping strategies. This perception has sometimes created problems in developing a therapeutic alliance with families and their representative organisations (Hatfield *et al*, 1987; El-Islam, 1989). The current review has attempted to avoid using EE as an adjective for people or families, since the concept refers to features of responses that change over time – not to features of people that persist, such as personality characteristics. If we want to use a shorthand, we should be referring to the environment or setting as high in EE, not the family. The temptation to label people could be reduced if researchers moved away from a dichotomous EE classification towards continuous predictors (Barrelet *et al*, 1990), and from binary outcomes such as relapsed/not relapsed to continuous outcome variables such as symptom severity and level of social functioning. This would also have the advantage of forcing us to re-examine the relationship between the EE variables and their relative contributions to prediction (Vaughn *et al*, 1984; Parker *et al*, 1989). But the use of different language may be seen as cosmetic if the central concept is thought to have a censorious tone. A further step towards recognising the achievements of families would be made if we examined beneficial family interactions in more detail. Promising areas for further research include the relation between EE and social support.

Continuing questions

Non-invasive social support

The focus on low EE and low contact as positive variables has glossed over potential problems

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with disengagement. If low EE is reached by developing an emotional indifference to patients' problems, the patients may become vulnerable to life events outside the family (Birley & Brown, 1970; Leff & Vaughn, 1980; Leff *et al*, 1987). Elsewhere in the life-event literature the notion of social support has proven useful (Katschnig, 1986). It seems odd that EE research, which grew up in the context of work on life events, has not sufficiently examined the role of appropriate social support and contrasted it with EOI.

The closest that EE has come to incorporating social support in the index has been in its consideration of warmth. Brown *et al* (1972) found that warmth was not a predictor of relapse, but noted that high warmth was confounded with high EOI. When this confound was removed, warmth was related to positive outcomes. When EE was low, the relapse rate dropped from 22% to 9% when warmth was high. Warmth is only an indirect measure of social support, and it might prove useful to obtain a more direct index of 'non-invasive support'. This concept describes support that is provided in response to the patient's request or is offered without pressure to accept. It includes both practical assistance and help in generating solutions to problems, where the aim is to assist patients to reach their own goals, develop their skills, and promote a sense of self-direction.

Non-invasive support is expected to be negatively correlated with the overprotective aspect of EOI and with hostile rejection. When it occurs in the absence of high EE it is expected to reduce the impact of aversive life events, perhaps including high EE from another person. Given that the existing effects of EE are obtained when any one person in the family shows high EE, it is unlikely that effects of high EE will be completely eliminated by the receipt of non-invasive support from another person. But a high level of contact with someone who is giving support may emerge as a predictor of patients' functional capabilities and symptom control at given levels of ambient EE.

What about social support for relatives? Brown *et al* (1972) presented evidence that parents who showed high EE were more often socially isolated, but the size and quality of social networks had no relation to EE in the study by Anderson *et al* (1984) – if anything, high EOI was associated with more network support. This issue clearly requires further study. Support of relatives might prove to be a clearer predictor of EE if it is more closely tied to specific assistance in dealing with the difficulties that schizophrenia poses. Experience with the effects of support groups for relatives (Leff *et al*, 1982, 1989) suggests that a forum to discuss problems and develop solutions does help to decrease EE and relapse.

Part of the difficulty faced by relatives is that people with schizophrenia often have very narrow social networks outside of the family. Crotty & Kulys (1986) observed that perceived burden in the family was inversely related to the number of people in the patient's social network. Increasing access to other sources of social support may significantly reduce the burden on family members.

Onset of schizophrenia

There is no reason why EE should not be as much related to the timing of the first episode as to later ones. If we accept a stress–vulnerability model of onset, a low-EE environment might even help some individuals to avoid the disorder altogether. Supportive evidence is offered by Goldstein (1987), who examined the 15-year prevalence of schizophrenic spectrum disorders among subjects who had been behaviourally disordered (but not psychotic) as adolescents. The risk of these disorders was increased if their relatives had previously shown high EE or a negative affective style in their family interactions. There are no published tests of EE as a factor in the onset of schizophrenia for individuals not suffering from behavioural disorders. Cross-cultural data appear to disconfirm a causal role for EE in disease onset, since incidence rates are seen as relatively invariant across cultures (Sartorius *et al*, 1986; cf. Torrey, 1989), while EE levels differ dramatically. Studies on genetic factors in schizophrenia (Fulker, 1973) also appear to discount a role for family factors in the lifetime risk of schizophrenia.

Although schizophrenia appears to be primarily determined by biological factors, we would expect a role for EE in the *timing* of the initial episode. Evidence on first admissions and onset of disorder does not confirm this hypothesis (Vaughn *et al*, 1984; Mintz *et al*, 1989). However, data on onset rely on retrospective reports and are subject to problems in dating a disorder which often has an insidious development. Retrospective inferences also assume that current EE ratings reflect family attitudes and behaviour before the disorder commenced; as already discussed, this is a risky assumption. A large longitudinal study with well subjects is required to avoid these problems. If such a study finds no relation between EE and timing of the initial episode, this presents some difficulties for the view that EE is a determining factor in later episodes. At the very least, it would then be incumbent on proponents of the concept to develop hypotheses that account for a change in role across the course of the disorder.

EE in contexts other than the family

Almost all the work on EE has focused on relationships within families, yet around one-third of patients are discharged to live alone or with friends (MacMillan *et al* 1986; McCreadie & Robinson, 1987). Could EE-related behaviour be important in other contexts? Preliminary data suggest that it probably is. Intensive supervision by staff, which may be analogous to familial EOI, does increase risk of relapse (Wing, 1978; Linn *et al*, 1980; Drake & Sederer, 1986). Co-residents and staff sometimes also show high levels of criticism and hostility (Higson & Kavanagh, 1988; Herzog, 1992), and informal observation suggests that interpersonal crises often seem to worsen symptoms. There are still no answers to the call by L. I. Mintz *et al* (1987) for prospective research on EE in hostels and group homes.

Consideration of relationships outside the family may also help to increase the predictive power of EE-related variables within the family literature. When patients spend a significant amount of time outside the home, their relationships with other patients, employers, and health professionals may contribute to their later symptom status. These relationships may help to explain occasions when EE is not a significant predictor.

Development of alternative measures of EE

Although the CFI has established reliability and validity, its application to clinical practice is limited by the time taken to administer it (around 1.5 hours) and the time it requires to score (an additional 2 hours or more). Its accessibility to therapists is also limited by the need for extensive training before it can be applied. Over recent years there have been attempts to develop measures of family atmosphere that are shorter and more accessible to therapists (Kreisman *et al*, 1979; Magãna *et al*, 1986; Schnur *et al*, 1986; Cole & Kazarian, 1988; O'Halloran *et al*, 1989). Some of these measures are showing promise as alternative measures of EE-related variables (e.g. Gottschalk *et al*, 1988), although there are some problems in obtaining sufficient sensitivity to high EE (Magãna *et al*, 1986; McCreadie & Robinson, 1987; Miklowitz *et al*, 1989). The next five years are likely to see advances in this area.

Implications for management

From the social interaction model we would expect that relapses associated with high EE would be reduced if patients and relatives could develop more effective coping strategies to deal with the problems that are posed by schizophrenia. Consistent with this view, skills-orientated family interventions have significantly improved the immediate course of schizophrenia when they are compared with routine or individual treatments. Detailed reviews of the literature are provided by Barrowclough & Tarrier (1984), Strachan (1986), Smith & Birchwood (1990), and Kavanagh (1992), and results of the key studies are summarised in Table 3. Control subjects from high-EE settings had a median relapse rate of 48% over nine months, but only 9% of the family-intervention subjects relapsed over the same period. If these results are compared with Table 1, they show that a family intervention drops the high-EE risk to a level expected for low EE. Over 24 months the relapse figures substantially increase (71% for controls and 33% for family intervention), but they continue to parallel the outcomes of high and low EE in the naturalistic studies. Family interventions can reduce EE or lower contact time for most families (Leff *et al*, 1982; Tarrier *et al*, 1988*b*). Consistent with the EE hypothesis, reductions in relapse are particularly marked when EE becomes low (Hogarty *et al*, 1986; Leff *et al*, 1989). The main impact of the intervention seems to be on prevention of major episodes: minor exacerbations of symptoms may still occur (Falloon *et al*, 1984).

Table 3
Percentage of relapses in treatment studies that pre-selected for high EE

	0–9 months		0–24 months	
	Family intervention	Routine or individual treatment	Family intervention	Routine or individual treatment
Leff *et al* (1982, 1985)	8%	50%	50%[1]	75%[1]
Falloon *et al* (1982, 1985)	6%	44%	17%	83%
Köttgen *et al* (1984)	33%	50%	-	-
Hogarty *et al* (1986, 1987)[2]	19%	28%	32%	66%
Tarrier *et al* (1988*b*, 1989)	12%	48%[3]	33%	59%[3]
Leff *et al* (1989, 1990*b*)	8%	-	33%	-
Median	10%	48%	33%	71%

1. The percentages include subjects who stopped medication and counts suicides as relapses. If those subjects are omitted, the outcomes are: 20% (family treatment), 78% (routine care).
2. Social skills training to individual clients produced a 9-month relapse rate of 20% and a 2-year rate of 42%. A combined intervention of family and social skills training gave 0% relapse in 9 months and 25% in 2 years.
3. Combined results for the brief education and routine treatment conditions.

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Although there were distinctive features to each of the treatments used in the intervention trials, they had a number of elements in common. Almost all of them included education of the family about schizophrenia and assistance in controlling stress. Most of them trained the families in systematic techniques to set goals and solve problems more effectively, and some also included communication training and behaviour management strategies when required. Detailed treatment manuals are provided by Anderson *et al* (1986), Falloon *et al* (1984) and Piatkowska *et al* (1992). As we would expect from an interactional model of relapse, similar improvements can be achieved either by an educational family intervention or by sessions with individual patients that increases their skills in reducing conflict with the family (Hogarty *et al*, 1986). An intervention that combines the individual and family strategies may be better than either of them alone (Hogarty *et al*, 1986), although further research is needed to check that the result is not due to increased contact with therapists in the joint intervention.

There is little evidence that effects from a brief family intervention are maintained once sessions are stopped altogether (Goldstein & Kopeikin, 1981). All of the interventions with sustained effects have continued low-level contact with families and clients (Falloon *et al*, 1985; Tarrier *et al*, 1989). Continued sessions are probably required to remind patients and relatives about the strategies they learned, to prompt the application of the strategies to new situations, and to encourage families to keep trying in the face of setbacks.

At present we do not know which aspects of the family interventions are responsible for its effect. Current studies fail to exclude effects of participation in a special treatment, increased therapist contact with the family, or the provision of non-specific support. Nor do we know which ingredients in the interventions are most effective, although brief didactic programmes do not seem to have a marked effect by themselves (Tarrier *et al*, 1988*b*). We do know that the effects are unlikely to occur through increased medication. Although medication compliance may be better after family intervention (Falloon *et al*, 1982), ingested amounts are lower because of reductions in the prescribed dosage (Falloon *et al*, 1985).

The evidence on family interventions is currently built on a relatively small group of studies and leaves many questions unanswered. However, the current work suggests that these interventions may prove to be the most significant treatment breakthrough in schizophrenia since the discovery of neuroleptic medication. If the concept of EE contributed nothing else, it may be credited with inspiring this potentially important advance.

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