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Expressed-emotion development and course of schizophrenic illness: considerations based on results of a CFI replication

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Abstract This study examines the correlation between development of expressed emotion (EE) in relatives and course of illness of 99 DSM-III schizophrenic patients. Patients whose relatives were high EE at baseline and at the 2nd CFI approximately 20 months later had a poor prognosis at the very outset of the study and an unfavourable course of illness. They had a higher rehospitalisation rate, more symptoms, lower psychosocial assessment, and a poorer 2-year and even 8-year outcome. Patients from families with a fluctuating EE or a consistently low EE had better courses. Expressed emotion is therefore a valid predictor not only of symptomatic relapses, but also of other important aspects of schizophrenia. The connection between EE index and course of illness seems not to be simply reactive or causal, but complex and non-uniform.

Key words EE development · CFI replication · Course of illness · Causality hypothesis

Introduction

Several studies have confirmed that a high EE index among relatives is associated with higher symptomatic relapse rates and, to a lesser extent, with higher rehospitalisation rates (Bebbington and Kuipers 1994; Schulze Mönking and Buchkremer 1995). Several long-term follow-up studies (McCreadie et al. 1993; Tarrier et al. 1994) have shown EE to be not only a short-term but also a valid long-term predictor of relapse. Huguelet et al. (1995) have shown EE to be correlated not only with relapse, but also with the development of psychosocial adaptation.

These results are all the more impressive as EE describes a partially changing condition. The percentage of relatives changing spontaneously from high to low EE has a wide range: from 25% (Leff et al. 1982; Hogarty et al. 1986) to 79% (Leff et al. 1990). Most studies on the stability of EE cover a period of 9 months to 1 year (cf. McCreadie et al. 1993). Most studies describe the fluctuation of primarily high-EE relatives (Brown et al. 1972; Leff et al. 1982; Hogarty et al. 1986; Tarrier et al. 1988; Leff et al. 1990), but some from low to high EE (Tarrier et al. 1988).

The longest period was covered by McCreadie et al. (1993). Within 5 years three Camberwell Family Interviews (CFIs) were assessed among relatives of 30 patients. From the first study (McCreadie 1982) only those patients were included who had continued to live with the same relatives. Expressed emotion was stable in 63% of the relatives. It did not necessarily change only at the time of relapse, but relapse rates were significantly lower for patients living in a consistently low-EE home.

Most studies examine the relationship between EE of relatives and symptomatic relapse, some between EE and rehospitalisation (cf. Schulze Mönking and Buchkremer 1995). Apart from the above-mentioned study by Huguelet (1995), there are no studies on the relationship of development of EE and aspects of the illness other than relapses.

In the present study we examine the following questions: Are there correlations between different types of development of EE and course of illness (rehospitalisation, development of symptoms and psychosocial functioning)? Do our results allow conclusions to be drawn on the type of relationship between EE and course of illness?

Methods

This study was carried out within the framework of the Münster Families Study (Buchkremer 1984). This prospective evaluative intervention study funded by the German Ministry of Research and

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Table 1 Description of patients. GAS Global Assessment scale; EE expressed emotion

Age (years/SD)	27	(6.6)
Male (%)	73	
Mean duration of illness (years/SD)	5.5	(4.3)
Mean age at first onset (years/SD)	21.5	(4.9)
Mean hospitalisation rate (SD)	2.6	(1.3)
Mean GAS score	53.0	(15.1)
Mean symptom score	23.9	(15.3)
Key relatives high EE (%)	60	

Technology was aimed at examining the impact on relapse prevention of therapeutic work with relatives of chronic schizophrenic patients in a randomised experimental group design. The interventions were confined to relatives, with the patients continuing to receive their outpatient care in its existing, uninfluenced form. The primary aim of the study was to alleviate symptoms through therapeutic work with relatives. As no therapeutic effects were recorded with respect to rehospitalisation rates, symptoms or social functioning of the patients and no significant changes in EE of relatives – as reported elsewhere (Buchkremer et al. 1995) – the course of illness up to the time of the control examination can be described with some justification as spontaneous. The study covered 99 patients who are described in Table 1. The symptoms were measured with the AMDP system (AMDP 1981), a German symptom rating scale for schizophrenic and affective disorders.

The degree of EE in the relatives was measured with the CFI (Vaughn and Leff 1976). The key relative was the relative with the most face-to-face contact with the patient. The cutpoints of the high EE were more than six critical remarks and a level of more than three on the EOI scale. The rater had been trained in Hamburg (Köttgen) and London (Vaughn) and had an interrater correlation of 0.90. Admission to the study and thus CFI interviewing did not take place immediately after hospitalisation, but only when patients had been in outpatient therapy for at least 8 weeks (up to several years) and had been prescribed neuroleptic long-term medication. At the outset of the study they all were living with their relatives.

The patients were re-examined 1, 2, 3 and 8 years after the beginning of the study, with the symptom scores, psychosocial functioning (GAS; Spitzer et al. 1976) and number and duration of hospitalisations being recorded each time. A second CFI was carried out with 76 key relatives after approximately 20 months. In 1992–1993 an 8-year follow-up was made with 69 of the original 99 patients.

The prognosis was estimated with the Strauss-Carpenter scale (Strauss and Carpenter 1974) and the Münster Prognosis score. This short dichotomous prognostic instrument proved to have the best short-term prognostic validity of our study in an 18-month follow-up and good prognostic validity even in an 8-year follow-up (Schulze Mönking 1994). The patients are rated on four items: number of previous episodes, stability of course of illness in the past year, premorbid development and stability of social environment.

Results

To examine whether there are any correlations between course of illness and development of expressed emotion, Table 2 compares patients whose relatives displayed varying EE developments. This yields four groups: relatives who were either high EE or low EE at both interviews and relatives who changed from high EE to low EE, and vice versa. The parameters used for the course of illness are rehospitalisation for at least 24 h within 2 years, and symptoms and psychosocial functioning (GAS) at baseline and after 2 years (i.e. ca. 3–6 months after the second CFI). Statistical differences were recorded between the patients of the consistently high-EE group and all others together.

Of the relatives, 32% underwent a change in EE, and 68% were stable. Patients whose relatives were high EE at both interviews were significantly more often hospitalised and had a significantly poorer outcome than all others.

To examine whether the correlation between different types of EE development and course of illness is just a temporal one for the time between the CFI measurements or whether there is a longer-term correlation, the results of the 8-year follow-up are shown in Table 3. To establish whether the course of illness is correlated to other types of prognostic assessment instruments, the results of the Münster Prognosis scale (MPS) and the Strauss-Carpenter scale (Strauss and Carpenter 1974) are added.

As can be seen, the patients from consistently high EE families had a significantly poorer prognosis at the very outset of the study and a significantly poorer outcome even in an 8-year follow-up with respect to duration of illness and development of symptoms and psychosocial functioning.

Table 2 Differences in baseline and outcome criteria in the course of 2-year follow-up of patients whose relatives displayed a divergent development of the EE index within 2 years. CFI Camberwell Family Interview

	1	2	3	4	5	6
	1st and 2nd EE index high (n = 32)	1st EE index high, 2nd EE index low (n = 14)	1st EE index low, 2nd EE index high (n = 10)	1st and 2nd EE index low (n = 20)	Low EE at least at one of the two CFIs (n = 44)	P between column 1 and column 5
AMDP baseline (mean)	27.3	20.5	16.8	21.5	20.1	0.047 (t-test)
AMDP after 2 years (mean)	24.6	7.6	13.2	12.8	11.3	0.006 (t-test)
GAS baseline (mean)	48.2	57.5	60.1	56.6	57.7	0.009 (t-test)
GAS after 2 years (mean)	49.9	68.6	57.9	59.8	62.6	0.001 (t-test)
Rehospitalisation 2 years (%)	71	38	40	55	47	0.036 (chi^2)

Table 3 Differences in baseline and outcome criteria in the course of an 8-year follow-up of patients whose relatives displayed a divergent development of the EE index within 2 years. MPS Münster Prognosis scale

	1	2	3	4	5	6
	1st and 2nd EE index high (n = 22)	1st EE index high, 2nd EE index low (n = 10)	1st EE index low, 2nd EE index high (n = 7)	1st and 2nd EE index low (n = 16)	Low EE at least at one of the two CFIs (n = 33)	P between column 1 and column 5
AMDP after 8 years (mean)	20.9	7.4	11.4	12.2	10.5	0.014 (t-test)
GAS after 8 years (mean)	50.0	67.5	59.9	59.8	62.2	0.016 (t-test)
No. of hospitalisations 84–92 (mean)	3.8	1.4	0.8	3.1	2.1	0 023 (t-test)
Duration of hospitalisation 1984–1992 (months, mean)	12.0	4.0	1.8	4.8	4.0	0.000 (t-test)
Prognosis good (MPS)	25%	64%	60%	55%	59%	0.003 (chi^2)
Strauss-Carpenter Scale (mean)	41.6	46.4	51.5	44.5	46.5	0.018 (t-test)

Discussion

Our results show that the stability of EE in our patients was similar to that recorded in other studies, especially in those by Leff et al. (1982) and Hogarty et al. (1986); this suggests that our population is comparable with those of other studies. For a large proportion of the families there is a close correlation between EE development, course of illness and prognosis: With high EE at both the first and second CFI, the prognosis was unfavourable at the very outset of the study, and the number of rehospitalisations and the extent of symptoms remained high not only in a 2-year, but even in an 8-year, follow-up. With consistently low or fluctuating EE the short-and long-term prognosis was better and the illness had a favourable course.

With respect to EE and outcome, the results are comparable with those of McCreadie et al. (1993) except in one point: McCreadie et al. recorded a better outcome only for patients from consistently low-EE families, whereas we found a better course also for patients from families with fluctuating EE. In fact, the designs of the two studies are not completely comparable because McCreadie et al. performed three CFIs in 5 years, whereas we carried out only two CFIs in approximately 20 months. Moreover, our target criteria were different, with McCreadie et al. recording psychotic relapses.

One striking aspect of our study is that there were differences not only in rehospitalisation rates and duration of hospitalisation over a long period, but also in psychosocial functioning and development of symptoms. Therefore, our results suggest that EE is a predictor not only a psychotic relapse, but also of other important aspects of the course of illness.

Nevertheless, the results do not show the type of correlation clearly. One possible explanation for the results is that EE causes relapse and other parameters of a poor outcome. In this case the patients of the group with consistently high-EE relatives must have been influenced by their relatives at the very beginning of the illness. This led to a poor course and to two CFIs with high EE. Assuming that EE remained high, it also could have caused a continued poor outcome.

If we accept this hypothesis, we have to assume that there are two types of high-EE relatives: One group is consistently high EE and has a bad influence on the disease, and others are consistently low EE or just temporarily high EE. This difference may represent a continuum with consistently high EE on the one hand, and consistently low EE on the other, and fluctuating relatives in between. Consistently high EE may just be the consequence of a higher threshold. On the other hand, our results cannot prove this with a different number of critical comments or EOI assessment for the groups with consistently high and fluctuating EE (the question is whether this might be a valid indicator). The most important argument against a single causal hypothesis is that more than 50% of the patients left their homes in the first 3 years of the study, but the correlation between EE and course of illness remained close over 8 years (Schulze Mönking 1996).

Another possible explanation for the results is that relatives react to aspects of the illness. For the consistently high-EE group this would mean that the relatives reacted to the severe symptoms and other aspects of the poor prognosis. The course of the illness was unfavourable and the relatives remained high EE. This line of reasoning is valid in a similar form for the consistently low-EE group. For the relatives who changed from high to low EE the reaction hypothesis seems most convincing, for the symptoms were high at the outset of the study and lower at follow-up. One argument against a simple reaction hypothesis for all patients is that in one group the relatives changed from low to high EE, but the patients remained well.

Knowledge of the temporal relationship between EE and relapse might indicate which of the two factors is the cause of the other. McCreadie et al. (1993) rated relatives' EE within 2 months of a relapse in 10 patients. They found no sign of synchronisation of EE and time of relapse, which speaks against a simple causality or a simple reaction hypothesis. Therefore, the possibility of a common cause must also be considered: Do relatives of patients with a primarily unfavourable course tend espe-

cially towards high EE? Might high-EE relatives and patients with an unfavourable course have a common cause, e.g. a common vulnerability of any kind whatsoever which is manifest in the one as an emotional reaction and in the other as an illness? Might the described correlation be the outcome of more than one cause? However, our results give no indication of a higher genetic susceptibility among the relatives.

The combination of all arguments suggests that the relationship between EE and course of illness is complex and non-uniform with reactive, causal and correlative factors which may not only be different between families, but may also change in the course of time within one family. Our results speak clearly against a simple causal hypothesis in which the relatives have a major responsibility for an unfavourable course of illness.

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