

# 5. Short-Term Relapse in Young Schizophrenics: Can It Be Predicted and Affected by Family (CFI), Patient, and Treatment Variables? An Experimental Study\*

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## The Hamburg Schizophrenia and CFI Project

The Hamburg Schizophrenia and CFI Project evolved from an attempt by various academic and nonacademic members of the Psychiatric University Clinic to develop more reliable and effective means for predictive assessment and treatment of younger, mainly first- or second-breakdown schizophrenic patients at high risk for a chronic course of illness. We decided to work with younger schizophrenics as there had been much less systematic treatment research performed with them than the more chronic conditions.

The development of our research design (Hand and Köttgen; details in Hand and Gross, 1980) was strongly influenced by the following sources:

1. The results published concerning experience with the Camberwell Family Interview (CFI) (extensive literature reviews by Hooley, Chap. 2, and Vaughn, Chap. 8, this volume) and direct support from C. Vaughn and K. Snyder regarding the training of raters
2. The frank and stimulating presentations and discussions of the schizophrenia research groups participating in the 1979 conference of the Society for Psychotherapy Research in Oxford, England
3. The results of the three major long-term studies in German-speaking countries (Bleuler, 1972; Ciompi and Müller, 1976; Huber et al., 1979) on the course of schizophrenia over decades
4. The continuous flow of information from the ongoing research work of R. Liberman and his colleagues in Camarillo and Los Angeles.

Within the context of our study, we conducted the first and so far only attempt at

replication of the Anglo-American CFI studies in a German-speaking culture. At the same time, with our diagnostic and treatment interventions, we wanted to investigate what the "meaning" of the published results and of our own CFI results could be for a conceptualization of the findings that until then had been purely empirical. Much dispute had occurred and still seems to be going on about whether or not the CFI literature implicitly "blames" the relatives for the patients' relapse and illness. Researchers investigating expressed emotion (EE) felt themselves misunderstood by these critics. Yet, they themselves had not made clear whether high EE-related behaviors of relatives were supposed to "cause" relapse of psychosis or vice versa. Does the assumption of a behavioral feedback loop really answer this question?

We shall first summarize the main aims of this project and some general data on outcome before turning to our detailed analyses of relapse and the variables that possibly contribute to relapse.

### Replication of Previous CFI Results and Direct Evaluation of the CFI

The main intentions of the following investigations were to replicate previous CFI results and to directly evaluate the CFI.

Replication of previous CFI results consisted of:

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- Identification of the proportions of high EE relatives in younger schizophrenic patients in a German-speaking culture.
- Comparison of relapse rates in patients from high EE and low EE families.
- Comparison of relapse rates in patients from high EE families who either did or did not receive regular medication and who did or did not participate in group therapy.

Direct evaluation of the CFI consisted of:

- Assessment of the stability of initially high and initially low EE ratings over time (no such investigation had been published at the start of this project). If high EE ratings (in relatives) at the time of the patient's discharge do predict relapse (in patients) 9 months later, one ought to expect some kind of causal interaction and, hence, stability of high EE ratings over this period of time.
- Comparison of global EE ratings over time with ratings on the EE subscales of emotional overinvolvement (EOI), criticism, and hostility. All published studies attribute a high EE "total" score to a relative as soon as she or he has reached high ratings on at least one of the EE subscales. Yet, there is no published evidence that these subscales are directly related to a basic, unifying concept. If ratings over time on these subscales were not correlated or went into opposite directions, this would not necessarily question the clinical usefulness of the EE total score as a "melting pot" for different relapse-related variables but it would certainly question its "meaning" as a construct.
- Comparison of repeated assessments over time of EE ratings and nuclear symptoms (according to the Present State Examination, PSE). Only such an analysis of correlations over time can produce empirical evidence for a conceptualization of the CFI that may stop the arguments about the "meaning" of CFI results obtained in the "classic" way (EE at discharge, relapse at the 9-months follow-up).
- Comparison of EE ratings and self-ratings of multiple neurotic symptomatology and neurotic traits over time in (a) the relatives (e.g., are obsessive relatives

more critical than nonobsessional?) and in (b) the patients (do specific neurotic symptoms or traits in patients specifically correlate with high scores in EE scales of their relatives?).

### **Indirect Evaluation of the CFI: Group Therapy for Patients and Relatives Separately**

According to the CFI literature in the late 1970s, "immunization by neuroleptic medication" (of the patient) and reduction of face-to-face contact between the patient and high EE family members (less than 35 h/week seemed to be powerful means of protecting vulnerable patients from relapse-inducing, high EE-related behaviors of their relatives. (It ought to be remembered here that this causal attribution was implicit in most of the studies available until the late 1970s.) These interventions seemed easy to administer and were the most frequently applied.

Surprisingly, most of the CFI-related treatment studies in those years employed *long-term neuroleptic medication* and only *short-term psychosocial treatment* (often only on an inpatient basis). Assuming that family and social learning factors have a major impact on the course of schizophrenia, one would rather have expected the reverse proportion of both treatment ingredients – medication for patients as intermittent crisis intervention and psychosocial treatment as the long-term stabilizer.

As far as the psychosocial treatments themselves are concerned, they did not seem to systematically use interventions derived from information gathered with the CFI.

In recent years, there has been a major change in the content of CFI-related treatment programs. A variety of approaches to directly affect negative family interactions are now being investigated: (a) social skills training groups for patients (Lieberman, Wallace, Vaughn, and Snyder, 1980; Wallace, Vaughn, and Snyder, 1982), (b) "psychoeducational" groups for relatives, in combination with family sessions (Anderson, Hogarty, and Reiss, 1980), (c) relatives' groups, with a main focus on CFI-derived themes to achieve reduction of high

EE-related attitudes and behaviors (Berkowitz, Kuipers, Eberlein-Vries, and Leff, 1981), (d) mixed relatives' groups with high and low EE members in an attempt to employ low EE relatives as co-therapists in teaching skills for coping with schizophrenia to family members with high EE ratings (Leff, Kuipers, and Berkowitz, 1979), and (e) treatment of the single family unit in its natural environment emphasizing CFI-related themes (Falloon, Boyd, and McGill, 1984).

In our design, we (Hand and Kötting, in Hand and Gross, 1980) decided to try yet another approach, *indirect* family therapy: The families were treated in separate groups for patients and their relatives with the same therapists treating those groups into which the members of each single family had been separated. In half of these families, the patients received "high-intensity" (HI) treatment (weekly group sessions) and their relatives a corresponding "low-intensity" (LI) treatment (monthly group sessions); in the other half, the reverse application of group treatments was made. Treatment was entirely on an outpatient basis and was meant to go on for 2 years. CFI-derived information provided the main themes, both in the patient's and relatives' groups.

It was hoped that a comparison of the outcome in the patient-centered group therapy would allow evaluation of the following questions within one single study:

Can high EE ratings be directly affected by intensive group treatment of relatives, and how does this affect patients?

Can patients and their course of illness be directly changed by intensive treatment, and how does this affect high EE ratings in their relatives?

Are separate group treatments for patients and their relatives a helpful and economical means of treating families with problematic or traumatic interactions?

The main treatment aims originally were to (a) reduce (interactional) vulnerability in the patient, (b) reduce harmful (interactional) events in daily life, in particular from high EE-related attitudes and behaviors in relatives, (c) reduce patients' behaviors that might cause or maintain high EE behaviors in their relatives, (d) increase coping and

other skills in patients and relatives, and (e) offer training in recognizing the occurrence of schizophrenic symptoms such as an expression of the illness, an attempted coping response for more severe (e.g., social) deficits, and an "interactional maneuver."

Additionally, basic information about the illness of schizophrenia was given. In contrast to the "psychoeducational" approach, the delivery of "knowledge" to patients and relatives was tried in a much less directive manner, spaced over much longer periods of time, with smaller amounts of information to be digested in a given session, and with more room for the patients to influence the structure and content of the treatment session.

The treatment plan is described in some detail by Kötting, Sönnichsen, Mollenhauer, and Jurth (1984). This final strategy was developed on an eclectic-psychodynamic basis derived from the cooperation and close mutual supervision of the therapist dyads (for each treatment group) composed of therapists from the project and from the hospital. It was not possible to develop the originally intended, structured behavioral therapy program. It was also not possible to continue the project for the originally planned 6-years period.

### General Design and Overall Outcomes

One-hundred and twenty patients were referred from two hospitals based on admission interviews as part of the project. Criteria for inclusion were: nuclear symptoms (PSE) exhibited during the month before admission; age, 18–30 years; total length of previous inpatient treatments no longer than 1 year; maximum number of previous hospital admissions, 3; key relative willing to participate in assessments and, if offered, treatment. The results of the application of these selection criteria are shown in Figure 1. Only 13% of the originally referred sample eventually received treatment.

Originally, 52 patients and their 79 relatives were included in the project (further reduction of participants, cf. p. 64). An attempt was made to secure complex assessments of patients and relatives: with self-rating scales on psychotic and multiple neurotic symptomatology, personality, social

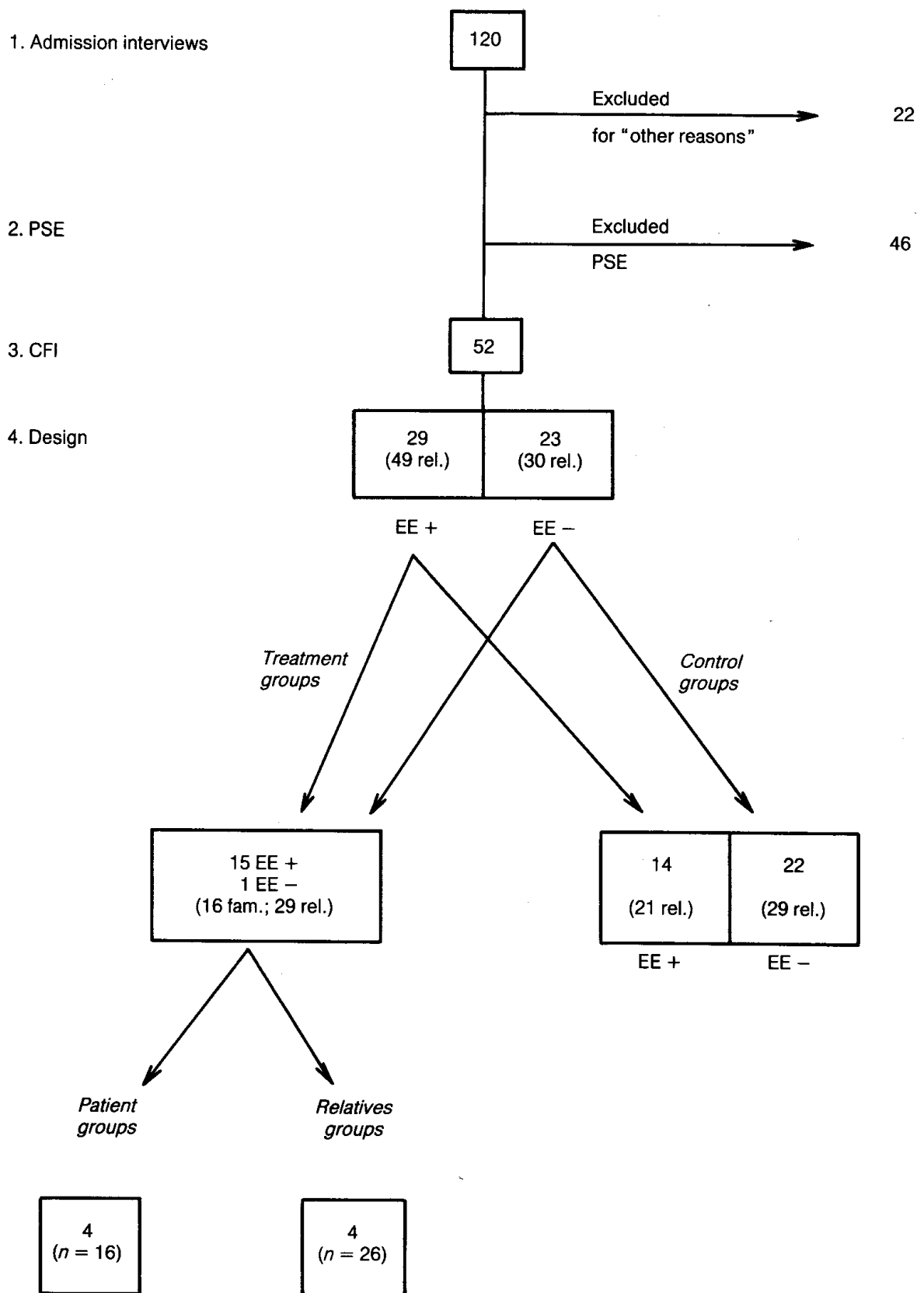


Fig. 1. Selection of patients and relatives.

skills and deficits, and socioeconomic development. Structured interviews were done with the PSE, CFI, and a relapse questionnaire. Except for the PSE and CFI, an attempt was made to perform most assessments with both the patients and relatives. Assessment intervals, except for relapse (first follow-up after 9 months), were planned as usual: during admission and 6, 18, and 30 months after discharge. The CFI was assessed with the abbreviated version by Vaughn and Leff. A family was rated as high EE when at least one relative scored 4 or 5 on EOI and made six or more critical remarks. One project rater (C. Köttgen) received CFI training with C. Vaughn and K. Snyder. She then trained two other raters in the project. For 12 families, interrater reliability between her and one of the additionally trained raters was reported to be high ( $r = 0.95$ ).

Of the 16 patients who received treatment, only 3 were female. Of the 26 relatives, 14 were mothers, 10 fathers, and 2 siblings. All relatives of high EE families were offered participation in the treatment program (if they were low EE themselves, they are called low EE co-relatives in the subsequent analyses). Allocation to high- or low-intensity treatment groups was mainly influenced by the sequence in which patients were referred to the project. Because of the loss of families due to the various selection steps, it often took months from identification of the first "suitable" pa-

tient and relative to the last to form a group.

The patients had a mean age of 23 (18–30), their relatives of 53 years. Two-thirds of the relatives interviewed were mothers, and two-thirds of the parents were still married.

The results for patients and relatives with most of these assessment instruments are reported in detail by Köttgen et al. (1984) as well as a direct comparison with the previous studies in London (Vaughn and Leff, 1976) and Los Angeles (Vaughn et al., 1984). As differences may be important for a discussion of our results, some are summarized here (LO = London; LA = Los Angeles; HH = Hamburg):

Percentage of families where both parents were interviewed:

LO 27; LA 52; HH 38

Percentage of parents in total sample:

LO 46; LA 94; HH 85

Percentage of high EE families:

LO 57; LA 67; HH 56

Percentage of male patients:

LO 41; LA 77; HH 65

Percentage of first admissions:

LO 57; LA 17; HH 69 (first and second admissions together: 92!) Percentage of relapse in EE+: EE– families:

LO 48:6; LA 56:17; HH 41:57!

The EE and relapse results in our own study here differ slightly as we – unlike Köttgen et al., but similar to the L.A. study – did not include the patients with "persisting symptoms" in all the analyses.

## Experimental Results

We shall start our analyses of predictor variables for the course of schizophrenia (relapse) with the family variables from the CFI, to be followed by the patient variable "nuclear symptoms" (NS) and the treatment variables "medication" and "group treatments." Finally, results from a combination of two to five variables for predictions of relapse will be presented.

### Family Variables

Before investigating the CFI variables in detail, we will first present results with the "classic" CFI analysis: assessment of re-

lapses at the 9-month follow-up and their correlation with single EE ratings at the time of the patients' discharge. This will be followed by a series of analyses with repeated assessments of EE variables (at discharge and the 6-month follow-up, CFI-FU), eventually again related to relapse rates (at the 9-month follow-up, REL-FU).

### "Classic" EE Assessment and Relapse

#### *Relapse Rates at the 9-Month Follow-up: Control and Treatment Groups Combined*

The course of illness in our patient sample is

summarized in Table 1. We formed then three categories of "no relapse," "relapse," and "special course." As the operationalization of relapse in the literature on schizophrenia is still a largely unresolved problem, we decided to use criteria similar to those described by Vaughn et al. (1982). The six criteria which constitute the three main courses of illness in Table 1 are:

1a: No psychotic symptomatology since discharge from the hospital; 1b: psychotic symptomatology without deterioration in the quality of daily life, maximum duration of 1 week, with or without hospital admission; 1c: intermittent, short-term psychotic symptomatology without deterioration of the quality of daily life.

2a: Psychotic symptomatology without nuclear symptoms, for longer than 1 week, without hospital admission;

2b: nuclear symptoms in addition to 2a.

3a: Readmission to the hospital for more than 1 week, but no nuclear symptoms; 3b: 3a with nuclear symptoms.

4: Persisting symptoms with deterioration of the everyday quality of life.

5: Hospitalization for more than 50% of the follow-up time.

6: Suicide.

Relapse was defined with regard to its individual as well as social relevance. Hospital readmission of less than 1 week is interpreted as one means of preventing relapse in a short-term psychotic crisis. Likewise, intermittent short-term psychotic symptomatology was not defined as relapse when the private and (or) professional life of the patient was not affected by the symptomatology.

Table 1 does not include the data of four patients (which are included in the publication by Köttgen et al., 1984), as these patients had moved away and information had come only from their relatives. The remaining 48 patients include those 5 from categories 5 and 6 ("special course") who will be excluded from all those subsequent analyses that rely on follow-up data (resulting in a  $n$  of 43). Category 5 was reached by three an  $n$  of 43). Category 5 was reached by three female patients who had not been discharged from the hospital during the REL-FU. The two patients in category 6 had committed suicide while on the waiting list for their assigned project group.

The overall outcome of approximately 50% relapse in young schizophrenics during the first 9 months after discharge is in accordance with previous reports in the literature.

*EE at Discharge and Relapse at the 9-Month Follow-up*

Of all our patients from the relapse categories 1–5 ( $n = 46$ ; cf. Table 1), 25 (54%) had relapses. The relapses occurred in 14 of the 29 (48%) patients with high EE relatives, but – contrary to all previous publications – even in 11 of the 17 (65%) from low EE families.

**Table 1.** Relapse rates at the 9-month follow-up: control and treatment groups combined.

Relapse category					
	$n$	%	$n$	%	
1 a	16	33	21	43	No relapse
1 b, c	5	10			
2 a, b	3	6	22	46	Relapse
3 a, b	13	27			
4	6	13			
5	3	6	5	10	Special course
6	2	4			
	48		48		

*Course of EE Ratings over Time*

In all subsequent analyses and figures with EE results, we shall first report the "total" EE score (EE), followed by the results on the subscales of emotional overinvolvement (EOI) and criticism (CRIT). The subscale of hostility is not included as relatives never reached the cutoff point for a high score. Simultaneously high ratings on EOI and CRIT at the first assessment were only observed in 4 of 63 relatives; one remained high at FU only in CRIT, two scored low on both scales, and the fourth did not participate in the FU.

*Course of EE Ratings over Time  
Treatment and Control Groups Combined*

The results on all scales together are shown in Table 2: 63 relatives of 46 patients participated in admission interviews as well as in CFI-FU. Of these 63, 46 relatives came from high EE families. These 46 included 13 low EE co-relatives from families with at least one high EE relative. Therefore, the EE+ ratings in Table 2 are from a total of 33 relatives (46 minus 13), whereas the EE+ ratings are from 30 relatives (17 from the low EE control group and 13 from low EE co-relatives of high EE families). This differentiation cannot be made in both subscales be-

cause any of the EE+ patients could score initially low on one of the two subscales.

On the EE score, 16 of 33 initially high EE relatives scored low at FU, whereas only 5 of 30 initially low EE relatives changed to high ratings at FU. Only 8 of all these relatives were spouses, 7 of whom scored low on both occasions.

Results on the total EE score resembled a labile, state-like quality of high EE and a rather stable, trait-like quality of initially low EE ratings. The EOI and CRIT scales showed almost identical developments of ratings over time, with a particularly high stability of low EOI ratings.

*Course of EE Ratings over Time:  
Control Groups*

Whereas in the treatment groups all relatives participated in both CFI ratings, the control groups, in particular the one with low EE relatives, showed a much lower retest compliance: 12 of 29 (41%) of the low EE relatives and 3 of 21 (14%) of the high EE relatives refused to participate in the second assessment. Understandably, retest compliance is higher in treated than in untreated persons, but it does not appear to be as obvious as to why the dropout rate was so much higher in the low EE relatives.

**Table 2.** Course of EE ratings over time: treatment and control groups combined.

		2nd CFI			
		EE scales			
		+	-		
1st CFI	EE+	17	16	33	EE total score
	EE-	5	25	30	
				63	
	EOI+	7	8	15	EE emotional overinvolvement
	EOI-	2	46	48	
				63	
CRIT+	10	12	22	EE criticism	
CRIT-	8	33	41		
			63		

**Table 3.** Natural course of EE ratings over time: control group (high and low EE groups separately).

		2nd CFI					
		EE scales					
		+	-				
1st CFI	EE+	6	7	13	High EE control grp.	EE total score	
	[EE-]	2	3	5			
	EE-	2	15	17	Low EE control grp.		
	EOI+	2	2	4	High EE control grp.		EE emotional overinvolvement
	EOI-	1	13	14			
	EOI-	0	17	17	Low EE control grp.		
	CRIT+	3	6	9	High EE control grp.	EE criticism	
	CRIT-	2	7	9			
	CRIT-	2	15	17	Low EE control grp.		

Table 3 shows the results for the high EE and the low EE control group separately. The low EE co-relatives of high EE families are now separated (the separation being indicated by brackets around EE- ratings) from the low EE relatives in the low EE control group. We introduced this additional subgroup, as we wanted to investigate whether low EE co-relatives resemble more their high EE counterparts or the low EE control group in the course of ratings (cf. p. 74).

At CFI-FU, 7 of 13 initially high EE relatives scored low, whereas only 2 of the initially 17 low EE relatives had changed in the reverse direction. Results in the untreated control groups thus resembled those from the total sample. The same holds true for the EOI ratings. The scale for CRIT even showed a decrease of high scores in two-thirds of the subjects (without treatment). Of the five low EE co-relatives, two changed to high ratings.

#### *Course of High EE Ratings over Time: Treatment and Control Groups*

Direct comparison of treated ( $n = 20$ ) and untreated ( $n = 13$ ) initially high EE rela-

tives on all three scales surprisingly did not show a specific treatment effect (in both conditions, some 50% of initially high scores were low at FU, whereas low scores showed little change). Paradoxically, the CRIT ratings even seemed to indicate a beneficial control group effect. In the low EE co-relatives, two of eight became high in the treatment groups compared with two of five in the control groups (Table 4).

Comparison of high-intensity groups for relatives (HI rel.), with 23–26 treatment sessions offered to participants, and low-intensity groups for relatives (LI rel.), with 8–24 sessions offered, showed no differences between groups, and both resembled the results reported before on the total EE scores.

However, as Table 5 indicates, HI rel. groups seemed to specifically affect ratings in both subscales; while initially high EOI ratings decreased in this group, initially low CRIT ratings increased (the LI rel. group did not show this effect).

#### *Course of EE Ratings and Relapse*

Since EE ratings at discharge did not prove



**Table 4.** Course of high EE ratings over time: treatment and control groups.

		2nd CFI						
		Treatment groups			Control groups			
		EE scales			EE scales			
		+	-		+	-		
1st CFI	EE+	11	9	20	6	7	13	EE total score
	[EE-]	2	6	8	2	3	5	
				28			18	
	EOI+	5	6	11	2	2	4	EE emotional overinvolvement
	EOI-	1	16	17	1	13	14	
				28			18	
	CRIT+	7	6	13	3	6	9	EE criticism
	CRIT-	4	11	15	2	7	11	
				28			18	

to be useful predictors of relapse in our study, we shall now investigate whether repeated EE ratings can increase the predictive power of this instrument. We shall first investigate EE ratings and relapse in high EE families ( $n = 46$  relatives), both in treatment and control groups (again including the low EE co-relatives separately under

**Table 5.** Course of high EE ratings over time: effects of high-intensity vs low-intensity relatives groups.

		2nd CFI						
		Treatment groups (LI pat. groups)			Control groups (HI pat. groups)			
		EE scales			EE scales			
		+	-		+	-		
1st CFI	EE+	5	5	10	6	4	10	EE total score
	[EE-]	1	2	3	1	4	5	
				13			15	
	EOI+	2	5	7	3	1	4	EE emotional overinvolvement
	EOI-	1	5	6	0	11	11	
				13			15	
	CRIT+	4	3	7	3	3	6	EE criticism
	CRIT-	3	3	6	1	8	9	
				13			15	

the total EE score), then in low EE families ( $n = 17$  relatives).

*Results from High EE Families (Treatment and Control Groups Combined):*

As already mentioned, some 50% of the high EE relatives changed to low EE total scores at FU, independently of relapses in patients. Initially low EE co-relatives remained the same only when patients did not relapse; three of four initially low EE co-relatives of the patients who relapsed changed to high EE. It is difficult to interpret this result, as the initially low EE relatives in the control group in the vast majority remained low, even when patients relapsed (Table 6).

In contrast to high total scores, high EOI scores showed a positive correlation between relapse and their development over time. Four of six initially high EOI relatives remained the same at FU when patients relapsed, as compared with four of ten, when patients had suffered no relapse. Low EOI ratings remained the same, independently of relapse.

Results became even more inconclusive with the CRIT ratings: high ratings changed to low in about 50% when patients showed

no relapse, but two-thirds changed to low even when patients relapsed!

Do such diverse developments of the three EE scales over time question the validity of the (most commonly used) total score?

*Results from Low EE Families (Control Group):*

Of the 17 relatives in the low EE control group (10 with a relapsed patient), 15 remained the same at FU on all three scales. The other two relatives, both with a relapsed patient, remained low in EOI, but became high in CRIT.

**Patient Variables**

*Nuclear Symptoms and Relapse*

All seven patients with nuclear symptoms (NS) at discharge (two) or at discharge and FU (five) "relapsed" during FU, five of them with persisting symptoms (relapse category 5).

Of the 36 patients without NS at discharge, 15 (42%) relapsed. Of the 21 patients without NS at discharge and no relapse, only 1 developed NS at FU.

**Table 6.** Course of EE ratings over time: high EE families and relapse.

		2nd CFI						
		High EE relatives of patients						
		Without relapse EE scales			With relapse EE scales			
		+	-	=	+	-	=	
1st CFI	EE+	10	9	19	6	7	13	EE total score
	[EE-]	1	9	10	3	1	4	
				29			17	46
	EOI+	4	6	10	4	2	6	EE emotional overinvolvement
	EOI-	1	18	19	2	9	11	
				29			17	46
	CRIT+	6	6	12	3	6	9	EE criticism
	CRIT-	3	14	17	2	6	8	
				29			17	46

Of the 22 patients with relapse, about one-third ( $n = 8$ ) showed no NS at discharge and FU, the second third ( $n = 7$ ) NS only at FU, and only the last third ( $n = 7$ ) NS at discharge or on both assessments.

taken into consideration. In fact, 9 of 12 patients who relapsed in spite of CNM complained about side effects, whereas only 6 of 11 who received CNM and did not relapse expressed the same complaint.

**Treatment Variables**

*Continuous Neuroleptic Medication and Relapse*

Continuous neuroleptic medication (CNM) was defined as a neuroleptic dosage comparable to 100 mg Haldol Decanoat in monthly intervals for at least over half of the follow-up time (the majority of patients received depot neuroleptics, e.g., haloperidol decanoate, independently of the project).

Although CNM compliance seemed to be much higher in patients from high EE families (17 of 26 = 65%) – among those particularly high in the treatment groups – than from low EE families (6 of 17 = 35%) medication did not seem to affect relapse rates specifically. Fifty percent of the patients with and without medication relapsed (Table 8).

Medication may show specific effects when their side effects are additionally

*Group Therapy and Relapse*

Of the patients from high EE families, 7 of 13 (54%) relapsed in the control and 5 of 14 (36%) in the treatment groups. We shall now look at both treatment conditions separately.

High-intensity group treatment (HI pat.) was given to eight patients in two groups. One of them only had the chance to attend 9 sessions, the others were offered between 28 and 43 sessions. Five of these patients remained without relapse. They as well as two of the three who relapsed attended more than 75% of the treatment sessions offered.

Low-intensity treatment (LI pat.) was given to another eight patients in two groups. Of these, one refused treatment and another one could not participate for other reasons (both relapsed). Of the remaining six, who were offered between 8 and 14 sessions, two relapsed. These two attended less than 50% of the sessions offered, but similarly so did three of the four who did

**Table 7.** Treatment intensity and relapse.

		Group treatments					
		HI pat. group (LI rel. group)			LI pat. group (HI rel. group)		
		Relapse			No relapse		
+	-	+	-				
Patients	>75%	2	5		0	1	
	50%–75%	0	0		0	0	
	<50%	1	0		2	3	
		3	5	8	2	4	6
Relatives	>75%	1	3		0	2	
	50%–75%	2	2		2	0	
	<50%	0	0		0	2	
		3	5	8	2	4	6

Compliance with group treatments

not relapse (cf. Table 7, upper section). These short-term relapse data do not show better results for high-intensity treatments (with higher appointment compliance of patients) than for low-intensity treatments (with also lower appointment compliance of patients).

As the relatives of LI pat. groups themselves were offered HI rel. groups., with regard to family problems, this may have been a compensation: Every single family unit in both patient groups received the same total amount of treatment.

If HI rel. groups were to have such a compensatory effect, this could only be expected from high compliance of the relatives. As data in Table 7 (lower section) show, this was not the case.

Whereas most patients in HI pat. groups also showed optimum compliance and those in LI pat. groups showed low compliance (i.e., they intensified by their compliance the separation of treatment conditions by design), the relatives did not show the same response pattern. We found that in the HI rel. groups the four relatives of patients who did not relapse were equally distributed to both extreme pools of compliance, whereas the two relatives of relapsed patients showed medium compliance. In the LI rel. groups, no relative attended less than 50% of the sessions offered! Four of the eight relatives reached medium, the other four optimum compliance.

With regard to the single family units, it became clear that HI pat./LI rel. treatment produced by far the best compliance of both family members in the treatment of-

fered. It appears that patients' compliance with treatment motivated their relatives to participate regularly rather than the other way around.

### Multivariate Prediction of Relapse

As we have seen so far, the single variables investigated in most analyses separated the total patient sample into two or three almost equally sized subsamples – with the exception of the presence of NS “at discharge.” Can the combination of these variables enhance their predictive power?

#### *Relapse: Family and Treatment Variables*

Neither CNM nor group therapy alone specifically affected relapse rates. If we regard both together, a third variable, high EE, implicitly (by design) affects the outcome.

A look at the data of patients from high EE families only (treatment and control groups combined) reveals that 15 of them had suffered no relapse, 9 of whom had been in treatment groups. Of the nine with and the six without group therapy, one-third in each condition had received no medication. Of the patients in group therapy, there were only five who (together with one relative) attended more than 50% of the sessions offered, whereas four did not (cf. Table 8).

In the treatment groups, a combination of EE+, CNM, and group therapy (Table 8) only prevented relapse in 6 of 11 patients. This result seems to be even worse than for patients from high EE families with CNM,

**Table 8.** Relapse: family and treatment variables.

		EE ratings								Total
		Patients with CNM				Patients without CNM				
		Treatment groups +	Control groups +   -		<i>n</i>	Treatment groups +	Control groups +   -		<i>n</i>	
Relapse	+	5	2	5	12	2	2	6	10	22
	-	6	4	1	11	3	2	5	10	21
		11	6	6	23	5	4	11	20	43

but without additional group therapy; in this group, four of six patients did not relapse. In the small subsamples of patients from high EE families without CNM, group treatment was not revealed to have exerted a specific effect compared with no treatment at all.

In the patients from low EE families, CNM did not show any "protective" effect regarding relapse rates; five of these six patients relapsed; on the other hand, only 6 of 11 patients without medication relapsed.

alone or in combination enhance predictability of relapse, what then can additional application of a patient variable ("no NS at discharge", no NSd) bring about?

No NSd and no CNM led to relapse in 9 of 19 and no NSd with CNM in 6 of 17 patients. Of the five patients with no NSd and CNM without side effects (SE) (cf. p. 69), only one relapsed. In all following analyses, we shall only use CNM without SE as the medication variable, as this outcome as well as the analysis of side effects tentatively support the clinical impression of their relevance.

*Relapse: Family, Treatment, and Patient Variables*

If neither family nor treatment variables

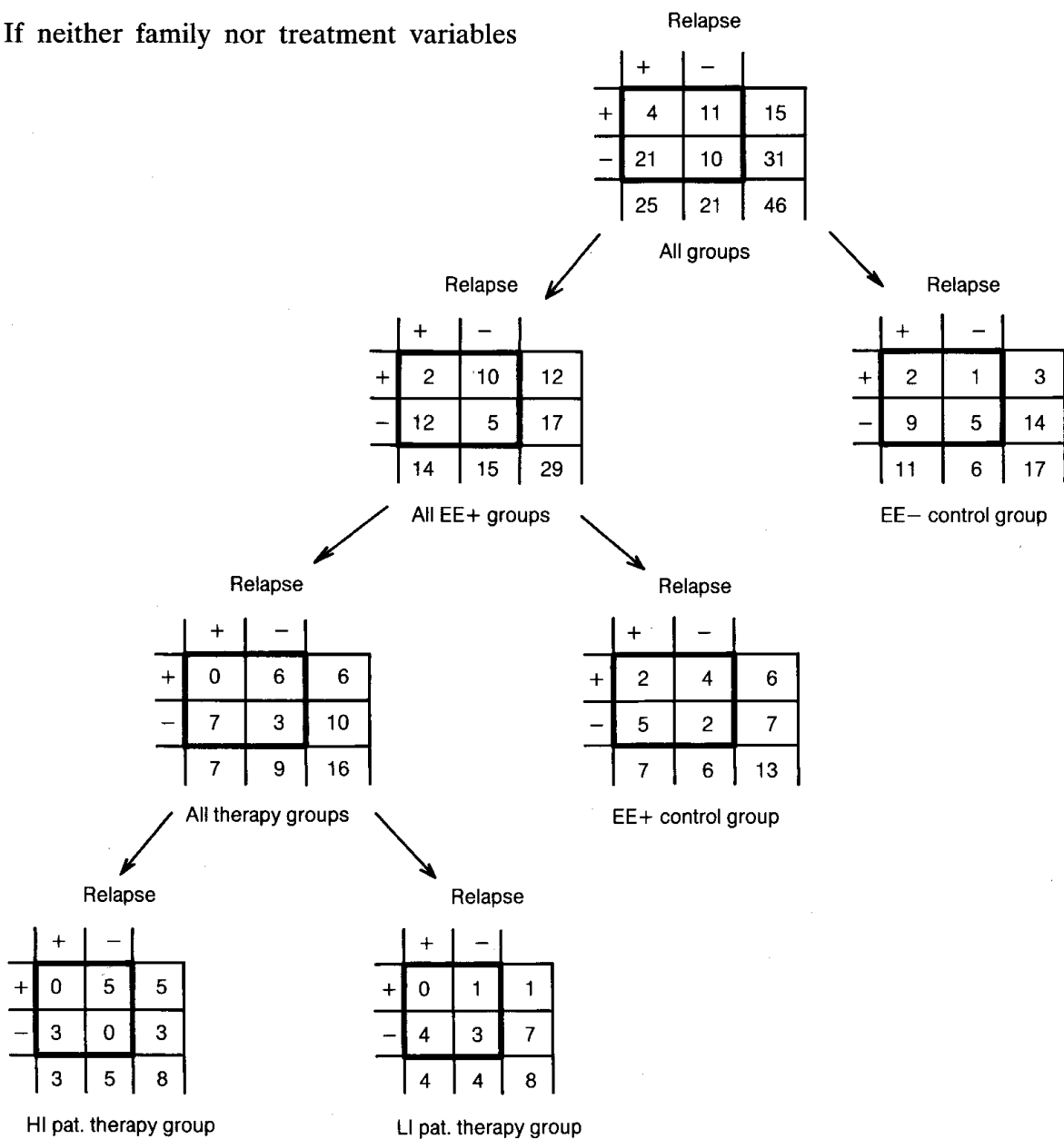


Fig. 2. Prediction of relapse by Family Compliance Index (FCI) for all groups.

Eleven patients had no NSd and group therapy, and only three relapsed. Six patients had no NSd, CNM without SE, and group therapy, and none of them relapsed. However, a fourth and possibly even a fifth variable may have influenced this outcome. These six patients were from a total sample of seven, of whom two relatives participated regularly in group treatment; also, five of these six patients attended the HI pat. groups. Obviously, introduction of the patient variable increased plurivariate predictability of relapse.

Finally, we would like to suggest a multi-variante "*Family Compliance Index*" (FCI) for prediction of the course of illness from the compliance behavior of patients and relatives with regard to the treatment offered. This FCI consists of the following six variables, four of which have to be present in families with group therapy offered and three in those without such an offer to achieve a "high" rating:

1. Regular outpatient psychiatric treatment after discharge.

2. Regular, high-potency neuroleptic medication at least during half of the observation time.
3. No side effects of neuroleptic medication.
4. Regular participation in patients' groups.
5. Regular participation in relatives' groups.
6. If group treatment is not offered, use of alternative treatment modalities (therapeutic hostels, etc.).

A high FCI was shown by 6 of the 16 high EE families in the treatment groups, and none of the respective patients relapsed. Of the 13 high EE families from the control group 6 showed a high FCI; two of the respective patients relapsed.

Of the 19 low EE families, only 3 showed a high FCI – again indicating the specificity of low EE families. Two of these three patients were among the 11 who suffered relapse.

The results of the systematic application of FCI on our total patient sample and its subgroups is shown in Figure 2.

## Summary and Discussion

### Overall Results: Prediction of Relapse

Of 46 mainly (92%) first- and second-breakdown schizophrenic patients, 46% (56%, including those with persisting symptoms) had relapsed at the 9-month follow-up. Relapses occurred in 58% of the high (total  $n = 29$ ) and 65% of the low (total  $n = 17$ ) EE families.

Prediction of relapse with any *single* of the following family or treatment *variables* appeared impossible: EE in single or repeated assessments, continuous neuroleptic medication (CNM), and group treatment for patients and for relatives. With or without any of these variables, relapse rates were about 50%. The apparent, but statistically nonsignificant tendency for a treatment effect (36% relapse in treated as compared with 54% in untreated high EE families) disappeared when HI and LI pat. groups were compared: the HI pat./LI rel. families showed much higher treatment compliance (with group sessions and med-

ication) than LI pat./HI rel. families; yet, relapse rates were identical. Within HI and LI pat. groups, the low and high compliance patients again showed almost identical relapse rates. It should be remembered here that these results refer to the effects of "being under treatment," not to treatment outcome! The patient variable of nuclear symptoms (NS) had equally low predictive power when there was a negative rating at discharge. However, persisting NS during inpatient treatment invariably were correlated with relapse.

Prediction by a *combination of variables* proved hardly more satisfactory: a combination of *relatives'* and *treatment* variables – EE, CNM, and group therapy – did not result in lower relapse rates. In contrast, the results appeared odd, as group therapy did not seem to have an additional effect to CNM in patients from high EE families, and in those from low EE families (who, by design, did not receive group treatment)

CNM even seemed to make outcome worse than no treatment at all.

Only the *additional* application of the *patient variable* of NS (no NS at discharge, no NSd) and the modification of CNM to CNM without side effects (SE) seems to increase predictability of relapse: of the six patients who showed this combination of variables (EE+, CNM without SE, group treatment, and no NSd), none relapsed. However, five of these patients were also in the HI pat. groups; and of all six patients, both parents participated in relatives' groups (which only happened in 7 of all 16 families with group treatment).

From these results, we construed a Family Compliance Index (FCI) with six variables. A high FCI indicated a low relapse rate in high EE families, both with and without treatment. It did not do so in low EE families!

This kind of plurivariate prediction of relapse is only of limited practical value. So far, the CFI has been used as a predictor *at the beginning of outpatient aftercare* (hospital discharge), whereas the FCI relies much more on (compliance) variables *during outpatient aftercare*. Any prediction of an event that includes variables more closely related (in time and content) to this event is, of course, bound to achieve better results – with lower generalizability. Further, it cannot be concluded from such an assessment (FCI) whether a high score with a low relapse rate is a consequence of a treatment variable (e.g., HI pat. groups as a “nuclear” treatment, around which the other interventions can be established) or whether an as yet undetected “family health factor” (e.g., benign atmosphere, independent of EE) enabled all family members to respond to any help offered – this health factor possibly being in itself sufficient enough to prevent relapse. Our results with the FCI in treated and untreated high EE families seem to support the latter hypotheses.

### **Specific Results: Direct and Indirect Evaluation of the CFI**

This is the only study so far that has tried to systematically assess the course of ratings on three EE variables (EE, EOI, CRIT) simultaneously and in relation to CNM and CFI-derived group treatment.

The results do not support most of the implicit assumptions in CFI research: High ratings decreased to low on all scales in most analyses in about 50% of the subjects – independently of relapse rates or treatments! They seemed to measure a state-like quality, the state-related variables remaining unidentified. Low ratings in the vast majority remained stable (trait-like) over time on all three scales, with only about 10% changing to high scores.

In comparison with high EE families, the low EE families showed very distinct results: a lower compliance with FU appointments (59% as compared with 86% in high EE control groups); a much lower FCI (only 3 of 17 scored high, and this without relapse prevention, 6 of 16 high EE families with and 6 of 13 without group therapy scored high, with low to zero relapses); finally, patients from low EE families showed a higher relapse rate and lower compliance with medication (the latter hardly being the cause of the first). Yet, as Kötting et al. (1984) described, low and high EE families did not differ in socioeconomic variables.

To complicate matters even further, some of the more puzzling results with EE in subsamples are summarized. The two subscales EOI and CRIT showed a very similar state-like instability over time (like the total EE scores) when ratings were initially high; initially low ratings remained low, particularly in EOI. Thus, there is no support for the assumption that drops in high EE total scores are due to drops in CRIT rather than EOI (cf. Chap. 8, this volume).

On the CRIT scale, more high ratings changed to low in the control than in the treatment groups, and HI pat. groups even seemed to increase initially low ratings (while reducing high CRIT, and even more high EOI). When patients developed no relapse, some 50% of high ratings changed to low – but when patients relapsed, 66% became low.

On the EOI scale, there was a more direct correlation of high ratings with relapse, the ratings remaining high in families with and turning low in families without relapse. Initially low ratings remained the same regardless of relapse. It appears difficult to

subsume these divergent results under one CFI concept.

Ratings of the low EE co-relatives (of high EE families) differed from the low EE control group, as they – like the high ratings of their high EE family members – seemed to correlate with relapse: three of the four whose patients relapsed turned high in their EE.

These small numbers certainly do not question the experimentally supported assumption by Kötting et al. (1984) that the vast majority of low EE families of young schizophrenics may actually be high EE (if instead of one at least two family members are interviewed). However, taking into account all the specific outcomes in low EE families in this study, the overall impression is that low EE families are very special and not just “hidden” high EE families.

Our relapse rates in low EE families seem to support notions that this group of families is inhomogeneous and includes a “burnt-out” type (cf. Chap. 8, this volume), with learned apathy and indifference toward stressful life events. Too little positive stimulation may occur for the patients of these families (cf. Chap. 6, this volume). Additionally, one may have to think of a subgroup of “hypocritical” noncritical relatives, who verbally cope and understand and nonverbally send opposite messages.

### Conclusions

The prediction of relapse and derivation of optimal treatments for young schizophrenic patients remains an unresolved problem.

EE results in this study raise rather than answer questions. Almost none of the current assumptions in the CFI literature could be supported by our data.

Reasons for our contradicting results may be manifold: Kötting et al. (1984) have described in detail differences between our patients' and relatives' samples and those in previous studies with regard to duration of illness, age, sex, and type and number of

relatives interviewed. A particularly important variable from previous studies seemed to be the amount of face-to-face contact between patient and relatives (>35 h/week increasing relapse rates). Again, this could not be reproduced in our study: in the high EE families, some 50% had more than 35 h contact; yet, around 40% relapsed each in the high- and low-contact environments. In the low EE families, 77% had low contact, but nevertheless 69% of them relapsed.

Could there be a relevant amount of erroneous CFI ratings, as there was only an interrater reliability test between the “properly” trained CFI rater in the project and one of the two other raters she had trained herself? This appears unlikely because of the very close cooperation between all three raters throughout the whole project.

Could it then have mattered that 65% of all and 81% of the patients treated were male, whereas two-thirds of the relatives interviewed were mothers and all CFI interviewers and main therapists (in the therapist dyads) were female? Finally, if differences in duration of illness were the decisive factor for differences in EE and relapse results (i.e., treatment effects), then it would no longer appear clinically meaningful to give first- and second-breakdown schizophrenics this diagnosis.

It follows that, at least for young “schizophrenics”, more studies are needed with repeated assessments over time of all variables regarded relevant, and more attention should be paid to low EE families.

The rather short duration of treatment and FU, the loss of the majority of families by the selection criteria, and compliance problems with assessment appointments and treatments severely restrict the conclusions that can be drawn for many of the questions investigated. Unfortunately, previous studies with other outcomes and conclusions were hardly less affected by these problems.

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# Treatment of Schizophrenia

Family Assessment and Intervention

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