## Paths to Relapse: Possible Transactional Processes Connecting Patient Illness Onset, Expressed Emotion, and Psychotic Relapse

### KEITH H. NUECHTERLEIN, KAREN S. SNYDER and JIM MINTZ

A vulnerability/stress framework for schizophrenia is one means by which the strong evidence for genetic and other biological factors in schizophrenia can be combined in a useful way with persistent evidence that stressful environments may play a role in precipitating psychotic episodes (Gottesman & Shields, 1972; Zubin & Spring, 1977; Nuechterlein & Dawson, 1984; Nuechterlein, 1987; Ciompi, 1989). Within a large longitudinal study of the early course of schizophrenia, we have been attempting to examine several possible ways in which both psychobiological vulnerability factors in the patient (Dawson & Nuechterlein, 1987; Nuechterlein et al, 1991) and external environmental stressors (Ventura et al, 1989) individually and jointly influence the course of schizophrenia. At the Second International Symposium on Schizophrenia in Bern, we focused on two promising mediating factors in schizophrenia - those involving persistent information-processing abnormalities and stresstriggered autonomic arousal (Nuechterlein et al, 1989). We focus here on recent analyses that relate to current controversies in the literature on interpersonal attitudes that are typically called expressed emotion (EE) - socio-environmental attributes that have been statistically associated with psychotic relapse in schizophrenia.

A series of studies has indicated that the presence of high levels of critical comments, hostility, or emotional overinvolvement among the significant others of schizophrenic patients is predictive of a higher likelihood of psychotic relapse in the 9 to 24 months following hospital discharge (Brown et al, 1962, 1972; Vaughn & Leff, 1976a; Leff & Vaughn, 1981; Vaughn et al, 1984; Moline et al, 1985; Nuechterlein et al, 1986b; Karno et al, 1987; Leff et al, 1987). Although we will follow the convention of referring to such attitudes and behaviour as high EE, several caveats are needed to put our approach to this topic into context. Firstly, the association of 'high expressed emotion' with higher relapse rates does not imply that all expressions of emotion by significant others are correlated with heightened relapse risk. In practice, attitudes towards patients are classified into high v. low EE categories based on the level of highly critical comments, hostility, or intrusive and overprotective behaviour, rather than on the expression of all types of emotion. Positive comments and warmth were also rated in initial studies, but have been de-emphasised in more recent research, because detection of their potential protective role is complicated by the observation that emotionally over-involved attitudes are often accompanied by very high levels of positive comments (Leff & Vaughn, 1985). Secondly, an important feature of this EE research is the focus on the course of schizophrenia rather than on aetiology. We do not endorse a psychogenic aetiology of schizophrenia, but rather assume that its causes involve critical genetic and other biological factors that lead to an ongoing vulnerability to psychotic episodes. Thirdly, since only some relatives of schizophrenic patients exhibit highly critical or emotionally overprotective attitudes, these attitudes are not general characteristics of all relatives. Finally, these attitudes are not specific to relatives of schizophrenic patients, but rather appear to be environmental attributes that are non-specific predictors of relapse in several disorders (Vaughn & Leff, 1976a; Hooley et al, 1986; Miklowitz et al, 1988). Such attributes are probably best viewed as possible relevant characteristics of social environments in general, rather than of familial environments in particular.

The basic predictive relationship has been a strong one in most studies, with psychotic relapse rates for schizophrenic patients with relatives who show attitudes of high EE typically being 2 to 4 times higher than those for patients whose relatives do not (Leff & Vaughn, 1985; Bebbington & Kuipers, 1989). The mechanism by which these attitudes are related to relapse remains to be clarified, however. It has usually been assumed that these attitudes have an impact on the patient by contributing to a high level of ongoing stress, which increases the vulnerable individual's liability to a psychotic episode (Brown et al, 1972; Leff & Vaughn, 1985). Autonomic activation of the patient has been postulated as a mediating psychophysiological process, and has reasonable empirical support (Brown et al, 1972; Leff & Vaughn, 1985; Dawson & Nuechterlein, 1987; Tarrier, 1989).

An important and very plausible alternative explanation for the predictive role of the family EE level, however, stems from the possibility that these attitudes are elicited by the exposure of the relatives to the patient's illness. Indeed, Brown et al (1962) noted this possible source for some instances of emotional overinvolvement. More recently, Kanter et al (1987) and Lefley (1989) pointed out that the extreme strain of living with a mentally ill relative has not received sufficient attention in the conceptualisations of the interaction between patients and their families. Perhaps this strain is particularly high when the illness is more severe, increasing the likelihood that significant others will make critical comments or display hostility and/or emotional overinvolvement. In this view, the association between such attitudes and relapse is an epiphenomenon, resulting from the fact that the more severely ill patient is both more likely to elicit such attitudes and also to have psychotic relapses, the attitudes themselves having no direct influence on relapse.

A particular version of the latter view of EE has been endorsed in two recent studies. MacMillan et al (1986) found that a high family EE level predicted higher relapse rates among first-episode schizophrenic patients, but this relationship was no longer significant when both the duration of illness before hospital admission and neuroleptic treatment following discharge were controlled statistically. Noting that families of patients with a long duration of illness before admission (one year or longer) were exposed to the patient's illness for a longer period, they suggested that this was a source of highly critical or emotionally overinvolved attitudes. The fact that controlling for this exposure factor reduced the predictive value of EE to a non-significant level, they reasoned, supported the view that the EE attitudes were a correlated, but not causal, factor in relapse. A similar view has been expressed by Parker et al (1988), who suggested that a younger age at first admission and other indicators of a poor illness course may elicit high-EE attitudes in relatives, and thereby make the primary contribution to the link between EE and relapse.

This recent controversy about the possible interpersonal processes connecting the characteristics of patients, relatives' EE attitudes, and psychotic relapse led us to examine these possible alternatives in our own longitudinal project.

#### Method

The sample of 43 schizophrenic patients and their immediate families is participating in a longitudinal study of the initial phase of schizophrenic disorder, 'Developmental Processes in Schizophrenic Disorders' (Nuechterlein et al, 1986a, 1989, 1991). The patients were recruited from consecutive admissions to four public hospitals in the Los Angeles metropolitan area (UCLA Neuropsychiatric Institute & Hospital, Harbor/UCLA Medical Center, Olive View Medical Center, and Camarillo State Hospital) and admissions to the out-patient service of the UCLA Neuropsychiatric Institute and Hospital. Criteria for inclusion in the study are: (a) a diagnosis of schizophrenia or schizoaffective disorder, mainly schizophrenic, by Research Diagnostic Criteria (Spitzer et al, 1978), with psychotic symptoms lasting at least two weeks, based on an expanded version of the Present State Examination (PSE) (Wing et al, 1974) administered to the patient and on any additional information from relatives; (b) the first onset of major psychosis occurred not longer than two years before first contact with the project; (c) age between 18 and 45 years; and (d) Anglo-American, Native American, or acculturated Asian or Hispanic background (including fluency in English). Patients are excluded from the study if they have: (a) a known organic central nervous system disorder (e.g. epilepsy, traumatic brain injury); (b) significant and habitual alcohol or drug abuse in the six months before the current episode, or evidence that substance abuse triggered the psychotic episode, makes the diagnosis ambiguous, or will be a prominent factor in the course of illness; or (c) mental retardation (premorbid IQ<70).

The present report involves 43 of the first 49 schizophrenic patients who completed a one-year, standardised-medication out-patient trial. Of the six excluded patients, three did not have EE data, two were missing Strauss-Carpenter prognostic scale data, and one had such severe and continuous psychotic symptoms throughout the one-year outcome period that a psychotic relapse was, by definition, impossible. The current sample includes the 26 patients from our preliminary EE report from this study (Nuechterlein *et al*, 1986*b*) plus additional patients who have since completed this standardised-medication phase.

The 43 schizophrenic patients had a mean age of 22.7 years (s.d. 3.3, range 18-32 years) and a mean of 12.4 years of education (s.d. 2.0, range 8-16 years) at entry to the project. The sample was predominantly male (35 males, 8 females) and Anglo-American (40 Anglo-American, 2 Hispanic, and 1 of mixed race). The patients' families were from all social classes, with a mean of 3.0 (s.d. 1.2) on the 5-point Hollingshead-Redlich Index of Social Position (Watt, 1974).

### Psychiatric and social history data

Information for a Psychiatric and Social History Schedule was collected from the patients and their family members and from any prior psychiatric treatment records. The items of the prognostic scale from the WHO Pilot Study of Schizophrenia examined by Strauss & Carpenter (1974,

1977) were rated as part of this schedule. The age of onset of the illness was also determined, based on all sources of information. For patients who were functioning marginally before the onset of clear psychosis, the age at which an accelerated downhill prodrome occurred was used as the age of onset. Mean age of onset was 21.5 years (s.d. 3.2, range 17-31 years). Considering that the mean age at entry to the project was 22.7 years, this mean age of onset of illness documents the fact that these patients had become ill very recently. By a very strict definition of 'first episode', 25 of these patients were in the midst of their first psychotic episode during the index admission. For this tally, first episode was defined as being within the first six months of the initial onset of any psychotic symptoms at entry to the project and never having had a period of a month or more without psychotic symptoms since that onset. The remainder were either in the midst of a first psychotic period that had lasted more than six months or had experienced their first psychotic episode within the last two years.

#### Assessment of expressed emotion attitudes

Family members who were in significant contact with the patient during the three months before the index admission were administered the abbreviated version of the Camberwell Family Interview (CFI) individually (Vaughn & Leff, 1976b) within one month of admission. Significant contact was defined as living with the patient, or being in at least weekly contact for at least one of the three months before the admission. The abbreviated version of the CFI is a 1.5-hour, semi-structured, standardised interview that focuses both on the onset and development of the illness episode and the impact of the illness on family life in the three months before admission. The relative's behaviour, self-reports of emotion, and spontaneous expressions of feelings are noted and the interview is tape-recorded.

Ratings of critical comments, hostility, and emotional overinvolvement were made by the interviewers shortly after the CFI, following procedures used in the original British studies. Interiewers had been trained by Christine Vaughn, PhD, or Karen Snyder, MA, and had achieved inter-rater reliability on CFI scales of at least r=0.80 (Pearson correlation), by comparison with criterion ratings. Co-rating of randomly selected interviews by Ms Snyder was used to maintain high inter-rater reliability.

Following the usual practice (Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984), an immediate social environment was categorised as high in EE if any family member made six or more critical comments, was rated as showing hostility, and/or was rated a '4' or '5' on the 5-point emotional overinvolvement scale.

#### **Out-patient treatment**

Patients were followed up at the UCLA Aftercare Clinic and placed on a standardised dosage of 12.5 mg fluphenazine decanoate every two weeks as soon as their clinical state could be stabilised, which typically took 2 to 3 months after hospital discharge. (One patient was on oral fluphenazine because she refused injections. Intolerable side effects led to reductions to 6.25 or 10 mg in 5 patients.) Individual case management and supportive, behaviourally-orientated therapy were also provided. The treating staff was unaware of the EE level of the family. The one-year period for outcome evaluation started when a battery of informationprocessing, psychophysiological, personality, and psychiatric symptom measures was administered, one month after the standardised medication was established.

#### Assessment of psychotic relapse

One-year outcome was placed into one of nine categories (Nuechterlein et al, 1987), based on ratings on the expanded Brief Psychiatric Rating Scale (Overall & Gorham 1962; Lukoff et al, 1986) completed every two weeks by the individual case manager. The unusual thought content, hallucinations, and conceptual disorganisation items were used to assess the course of psychotic symptoms. The following three subtypes of return or significant worsening of psychotic symptoms were combined into an overall psychotic relapse category. "Remission followed by relapse" involves an increase on one of the three scales to a rating of severe (6) or extremely severe (7), after an earlier period of ratings of mild (3, defined as a non-psychotic level) or less for at least a month on all three psychotic scales. "Remission followed by significant exacerbation" involves an increase to moderately severe (5), either for more than a month or with an accompanying increase of at least two points on another psychotic scale, after an earlier period of ratings of 3 or below for at least a month on the three psychotic scales. Finally, "persisting psychotic symptoms followed by significant exacerbation" is defined as having psychotic symptoms rated as moderate (4) or greater throughout the follow-up period, but with a 2-point increase at some point to severe (6) or extremely severe (7) or a 1-point increase to this level and an accompanying 2-point increase on another of the three psychotic scales.

#### Results

Of the 31 patients from high-EE families, 12 (39%) had a psychotic relapse during the one-year, standardisedmedication period, whereas none of the 12 patients from low-EE families had such a relapse. This basic predictive relationship is significant ( $\chi^2 = 6.44$ , d.f. = 1, P < 0.011, r = 0.39).

As in our earlier analysis, which was restricted to strictly defined first-episode cases (Mintz *et al*, 1989), we found that the best estimate of the total duration of illness before the index admission was not related to high-EE attitudes among relatives or to psychotic relapse. In addition, analyses of two summary variables from the Strauss-Carpenter prognostic scale did not yield predictors of psychotic relapse that might help account for the relationship between EE and relapse. However, we also extended our earlier analysis, which found a relationship between the patient's place of residence before the hospital admission that preceded entry to the project and high-EE attitudes among relatives (Mintz *et al*, 1989). In the current sample, which was followed up for the initial one-year evaluation period, patients who were living with their

relatives before the admission that preceded entry to the project were again more likely to have relatives with high-EE attitudes than those who were living away from relatives (r=0.37, P<0.02). This correlation suggests the possibility that the degree of exposure to the symptomatic patient is a factor in eliciting high-EE attitudes.

Finally, we noted that the patients who were living with their parents before the index admission and recruitment to the study were significantly younger when they had first become ill than those who were living away from their parents (r = -0.43, P < 0.005). Because this admission and recruitment occurred relatively shortly after the initial onset of symptoms, many of the patients who were living with their parents had not yet reached an age at which one would typically establish a separate residence. This relationship raises the possibility that another confounding variable – age at onset of illness – might influence the association between family attitudes and clinical course.

# Path analyses among the observed variables to model the effects on relapse

These significant relationships were examined through two path models. Path analyses allow various possible relationships between variables to be examined, with the effects of other variables statistically controlled (Loehlin, 1987). Because path analysis does not involve experimental manipulation of a variable, this statistical modelling procedure needs to be supplemented by such experimental methods, to establish causal relationships. However, path analysis can clarify the directional influences that would fit a set of correlational data. In the initial two path models in which all of the variables were directly observed, the path coefficients are equivalent to standardised partial regression coefficients in multiple regression analyses.

First, as depicted in Fig. 1, we examined whether the predictive relationship between high EE among relatives and heightened relapse rate could be accounted for as an artefact of direct effects on relapse of being younger at onset of illness or of living with relatives before the key admission. An early age of onset might be expected to indicate a more severe form of schizophrenia, characterised by a higher relapse rate; if this led directly to a higher relapse rate, perhaps the predictive relationship between EE and relapse could be accounted for as an epiphenomenon of an early age of onset. Thus, perhaps such patients would still be living at home with their parents before the index admission, leading their parents to experience the patients' symptoms more directly. This experience might elicit high levels of critical comments, hostility, or emotional overinvolvement, but these high-EE attitudes might have no direct effect on the likelihood of relapse.

As seen in Fig. 1, the possibility that the relationship between the level of EE and relapse might be an epiphenomenon is not supported, because the direct paths from age at onset of illness to relapse, and from living with relatives before admission to relapse are negligible. Therefore, the predictive relationship between high-EE attitudes and relapse is not accounted for by direct predictive effects of these patient variables. A very interesting chain of effects through EE attitudes is suggested, however. Age at onset of illness does have a significant effect on whether the patient is living with relatives before the index admission, as would be expected in the normal process of attaining adulthood and moving out of the parental home. Furthermore, living with relatives before admission continues to be related to a high EE level



Fig. 1 Path analysis of chain of effects from age of onset of patient's illness through patient's residence before key hospital admission and expressed emotion level to likelihood of psychotic relapse.



Fig. 2 Path analysis results for alternative model in which expressed emotion level is hypothesised to affect illness onset age, patient's residence with relatives before key hospital admission, and psychotic relapse rate.

among the relatives, even with age at onset of illness controlled, which suggests that exposure to the patient's developing psychosis does play a part in eliciting critical comments, hostility, and emotional overinvolvement. This model indicates that presence of high-EE attitudes, in turn, then predicts an increased likelihood of psychotic relapse. A test of the goodness of fit of this model with the non-significant paths removed (calculated by Peter Bentler's EQS program) indicates that this model provides an excellent fit to the data ( $\chi^2 = 0.097$ , d.f. = 3, P = 0.992, Bentler-Bonett normed fit index = 0.996).

A second path model was examined using the EQS program, to evaluate the alternative possibility that high EE among relatives contributed to an earlier initial onset of illness, rather than that early age of onset served as an indirect influence on development of high-EE attitudes. As shown in Fig. 2, this model proposes that high EE is a source of both early onset of illness and of living with relatives before admission. Because we do not have a direct measure of the level of EE before the onset of illness, we use the EE level at the admission before entry to the project as an index of earlier EE attitudes. It must be recognised, of course, that the EE attitudes migh have of this model is limited by this possibility.

As Fig. 2 shows, the results do not support the view that high-EE attitudes influence the age of initial onset of the patient's illness. In this model, the only significant path from EE to relapse is a direct one. Living with relatives before admission, on the other hand, is found to be significantly affected by direct paths from both early onset of illness and from high-EE attitudes of relatives. The critical point, however, is that high-EE attitudes do not account for an earlier onset of the patient's illness, making the initial model (Fig. 1) a more plausible explanation of the observed inter-relationships.

# Analyses incorporating a latent variable: severity of the patient's illness

Additional analyses were completed to examine the possibility that a latent variable - the severity of the patient's illness – was determining the observed relationships. In this data analytic approach, the latent variable is not observed directly, but rather is measured indirectly from multiple observed variables (Loehlin, 1987). First, we evaluated the possibility that the measured variables were actually all indicators of this single latent variable, a shown in Fig. 3. We felt that this model, which is often proposed as an alternative to a causal influence of EE attitudes on relapse, would be parsimonious if it fitted well. This model was evaluated through structural equation analysis with a maximum likelihood method, using Bentler's EQS program. The coefficients are represented in a standardised form for ease of comparison to the coefficients in the previous two figures. As is evident in Fig. 3, this model yields significant relationships between the latent variable severity of the patient's illness - and earlier onset of illness, living with relatives before the hospital admission, and high-EE attitudes. The relationship between severity of the patient's illness and relapse is not quite significant (P=0.09). This model yields only a moderate goodnessof-fit index (Bentler-Bonett normed fit index = 0.782) and approaches  $\chi^2$  criteria for rejection ( $\chi^2 = 4.68$ , d.f. = 2, *P*<0.10).

An alternative latent variable model was compared with this initial simple model. In this model, as shown in Fig. 4, the latent variable – severity of the patient's illness – is



Fig. 3 Path analysis in which the severity of patient's illness is hypothesised to be the common source of all observed relationships.

hypothesised to influence age at onset of the illness, living with relatives before admission, and development of high-EE attitudes, but such attitudes are then, in turn, postulated to increase the likelihood of relapse. The maximum likelihood solution indicates that this model yields coefficients that are significant for all hypothesised relationships. Furthermore, this model fits the data much better than the previous one. The  $\chi^2$  value indicates very little deviation from the data ( $\chi^2 = 0.026$ , d.f. = 2, P = 0.99) and the Bentler-Bonett normed fit index is 0.999, suggesting an excellent fit. Thus, these structural equation results suggest that highly critical or emotionally overinvolved attitudes in the social environment play a mediating role in the likelihood of psychotic relapse, even if the other measured variables can be accounted for by the severity of the patient's illness.

### Discussion

Path and structural analyses of the inter-relationships between the patient's age at onset of illness, the



Fig. 4 Path analysis in which the severity of patient's illness is hypothesised to be the source of age at onset of illness, living with relatives, and expressed emotion level, with latter then related to psychotic relapse rate.

patient's residence before the index admission, the level of critical or emotionally overinvolved attitudes of significant others, and later psychotic relapse support the likelihood of a sequence of processes linking these variables. These path models suggest that high expressed emotion attitudes may develop, in part, through living with the patient during the period preceding admission, which is more likely to occur if the patient has an earlier age of onset of illness.

An alternative view of these inter-relationships that is also supported by these analyses is that a latent variable - greater severity of the patient's illness may be an underlying factor in early onset of illness, living at home with relatives, and high EE among relatives. Thus, these analyses support the view that a more severe illness may contribute to development of a high level of critical and emotionally overinvolved attitudes among relatives, as several investigators have suggested. It should be noted that the approach used here, determining whether a latent variable could account for inter-relationships of early age of onset of illness, living at home with relatives, and high EE among relatives, differs from that of prior studies which examined whether high-EE attitudes are associated with the cross-sectional presence or severity of psychiatric symptoms (e.g. Miklowitz et al, 1983; Vaughn et al, 1984; Miklowitz et al, 1989; Glynn et al, 1990) or level of behavioural disturbance (Brown et al, 1972; Vaughn & Leff, 1976a).

Although these analyses provide support for the possibility that critical and emotionally overinvolved attitudes of family members are partly a response to characteristics of the patient and living circumstances, the supported models differ in a critical way from those suggested by MacMillan *et al* (1986) and Parker *et al* (1988). Rather than an epiphenomenon, these analyses suggest that high-EE attitudes among relatives, once they have developed, operate as an important mediating variable that may influence the likelihood of psychotic relapse.

Considered more broadly, these analyses are consistent with a diathesis/stress or vulnerability/ stress model of schizophrenic relapse (Gottesman & Shields, 1972; Nuechterlein & Dawson, 1984; Liberman, 1986; Nuechterlein, 1987) in which genes and other early biological factors establish an ongoing level of vulnerability to psychosis that interacts with a variety of possible biological and psychosocial sources of stress to influence the likelihood of relapse. Additional work with experimental designs that alter key variables, such as programmes to increase the ability of significant others to cope with the severe strains of mental illness in a family member, is necessary to establish more clearly the directional relationships suggested by these path analyses. Evidence that psychoeducational, problem-solving approaches with patients and their significant others can lower patients' risk of relapse is thus far also consistent with a mediating role for socioenvironmental factors in relapse (Goldstein *et al*, 1978; Falloon *et al*, 1982; Leff *et al*, 1982; Hogarty *et al*, 1986; Tarrier *et al*, 1988).

Further elaborations of the path models described here may also benefit from incorporation of a possible relationship between the presence of psychiatric disorder in some biological relatives of schizophrenic patients and likelihood of highexpressed emotion attitudes that endure into the patient's post-discharge period, as suggested in the article by Goldstein *et al* in this volume (pp. 97– 102).

#### Acknowledgements

This research was supported by NIMH Research Grants MH-37705 to K. H. Nuechterlein, PhD, and MH-30911 (Clinical Research Center for the Study of Schizophrenia) to R. P. Liberman, MD. The authors gratefully acknowledge the research contributions of the patients and their family members, the efforts of Pamela Dirham, BA, Portia Loughman, BA, Marianna Lopez, MA, and Sandra Rappe, MSW, in collection of Camberwell Family Interview data, and the dedication of Michael Gitlin, MD, David Fogelson, MD, Joseph Ventura, MA, David Lukoff, PhD, Deborah Gioia-Hasick, MSW, and Sally Friedlob, MSW, in the clinical care and assessment of the patients. Data analysis was supported by the Methodology & Statistical Support Unit of the Clinical Research Center.

#### References

- BEBBINGTON, P. & KUIPERS, L. (1989) Social influences on schizophrenia. In Schizophrenia: The Major Issues (eds P. Bebbington & P. McGuffin), pp. 201-225. Oxford: Heinemann.
- BROWN, G. W., MONCK, E. M., CARSTAIRS, G. M., et al (1962) Influence of family life on the course of schizophrenic illness. British Journal of Preventative and Social Medicine, 16, 55-68.
- ——, BIRLEY, J. L. T. & WING, J. K. (1972) Influence of family life on the course of schizophrenic disorders: a replication. *British Journal of Psychiatry*, 121, 241–258.
- CIOMPI, L. (1989) The dynamics of complex biological-psychosocial systems: four fundamental psycho-biological mediators in the long-term evolution of schizophrenia. *British Journal of Psychiatry*, **155** (suppl. 5), 15-21.
- DAWSON, M. E. & NUECHTERLEIN, K. H. (1987) The role of autonomic dysfunctions within a vulnerability/stress model of schizophrenic disorders. In *Psychopathology: An Interactional Perspective* (eds D. Magnusson & A. Öhman), pp. 41-51. Orlando, Florida: Academic Press.
- FALLOON, I. R. H., BOYD, J. L., MCGILL, C. W., et al (1982) Family management in the prevention of exacerbations of

schizophreia: a controlled study. New England Journal of Medicine, 306, 1437-1440.

- GLYNN, S. M., RANDOLPH, E. T., ETH, S., et al (1990) Patient psychopathology and expressed emotion in schizophrenia. British Journal of Psychiatry, 157, 877-880.
- GOLDSTEIN, M. J., RODNICK, E. H., EVANS, J. R., et al (1978) Drug and family therapy in the aftercare treatment of acute schizophrenia. Archives of General Psychiatry, 35, 169-177.
- GOTTESMAN, I. I. & SHIELDS, J. (1972) Schizophrenia and Genetics: A Twin Study Vantage Point. New York: Academic Press.
- HOGARTY, G. I., ANDERSON, C. M., REISS, D. J., et al, and the Environmental/Personal Indicators in the Course of Schizophrenia Research Group (1986) Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia: I. One-year effects of a controlled study on relapse and expressed emotion. Archives of General Psychiatry, 43, 633-642.
- HOOLEY, J. M., ORLEY, J. & TEASDALE, J. D. (1986) Levels of expressed emotion and relapse in depressed patients. *British Journal of Psychiatry*, 148, 642-647.
- KANTER, J., LAMB, H. R. & LOEPER, C. (1987) Expressed emotion in families: a critical review. Hospital and Community Psychiatry, 38, 374-380.
- KARNO, M., JENKINS, J. H., DE LA SELVA, A., et al (1987) Expressed emotion and schizophrenic outcome among Mexican-American families. Journal of Nervous and Mental Disease, 175, 143–151.
- LEFF, J. P., KUIPERS, L., BERKOWITZ, R., et al (1982) A controlled trial of social intervention in the families of schizophrenic patients. British Journal of Psychiatry, 141, 121-134.
- & VAUGHN, C. (1981) The role of maintenance therapy and relatives' expressed emotion in relapse of schizophrenia: a two-year follow up. British Journal of Psychiatry, 139, 102-104.
- & (1985) Expressed Emotion in Families. New York: Guilford Press.
- —, WIG, N. N., GHOSH, A., et al. (1987) Expressed emotion and schizophrenia in North India. III. Influence of relatives' expressed emotion on the course of schizophrenia in Chandigarh. British Journal of Psychiatry, 151, 166–173.
- LEFLEY, H. P. (1989) Family burden and family stigma in major mental illness. American Psychologist, 44, 556-560.
- LIBERMAN, R. P. (1986) Coping and competence as protective factors in the vulnerability-stress model of schizophrenia. In *Treatment of Schizophrenia: Family Assessment and Intervention* (eds M. J. Goldstein, I. Hand & K. Hahlweg). Berlin: Springer.
- LOEHLIN, J. C. (1987) Latent Variable Models. Hillsdale, New Jersey: Erlbaum.
- LUKOFF, D., NUECHTERLEIN, K. H. & VENTURA, J. (1986) Manual for Expanded Brief Psychiatric Rating Scale (BPRS). Schizophrenia Bulletin, 12, 594-602.
- MACMILLAN, J. F., GOLD, A., CROWN, T. J., et al (1986). The Northwick Park Study of First Episodes of Schizophrenia. IV. Expressed emotion and relapse. British Journal of Psychiatry, 148, 133-143.
- MIKLOWITZ, D. J., GOLDSTEIN, M. J., DOANE, J. A., et al (1989) Is expressed emotion an index of a transactional process? I. Relative's affective style. Family Process, 28, 153-167.
  - —, —, & FALLOON, I. R. H. (1983) Premorbid and symptomatic characteristics of schizophrenics from families with high and low levels of expressed emotion. *Journal of Abnormal Psychology*, **92**, 359–367.
  - \_\_\_\_, \_\_\_\_, NUECHTERLEIN, K. H., et al (1988) Family factors and the course of bipolar affective disorder. Archives of General Psychiatry, 45, 225-231.
- MINTZ, L. I., NUECHTERLEIN, K. H., GOLDSTEIN, M. J., et al (1989) The initial onset of schizophrenia and family expressed emotion: some methodological considerations. British Journal of Psychiatry, 154, 212-217.

- MOLINE, R. A., SINGH, S., MORRIS, A., et al (1985) Family expressed emotion and relapse in schizophrenia in 24 urban American patients. American Journal of Psychiatry, 142, 1078-1081.
- NUECHTERLEIN, K. H. (1987) Vulnerability models for schizophrenia: state of the art. In Search for the Causes of Schizophrenia (eds H. Häfner, W. F. Gattaz & W. Janzarik), pp. 297-316. Heidelberg: Springer.
- & Dawson, M. E. (1984) A heuristic vulnerability/stress model of schizophrenic episodes. Schizophrenia Bulletin, 10, 300-312.
- —, EDELL, W. S., NORRIS, M., *et al* (1986*a*) Attentional vulnerability indicators, thought disorder, and negative symptoms. *Schizophrenia Bulletin*, **12**, 408–426.
- —, SNYDER, K. S., DAWSON, M. E., et al (1986b) Expressed emotion, fixed-dose fluphenazine decanoate maintenance, and relapse in recent-onset schizophrenia. *Psychopharmacology Bulletin*, 22, 633-639.
- —, GOLDSTEIN, M. J., VENTURA, J., et al (1989) Patientenvironment relationships in schizophrenia: information processing, communication deviance, autonomic arousal, and stressful life events. British Journal of Psychiatry, 155 (suppl. 5), 84-89.
- , DAWSON, M. E., VENTURA, J., et al (1991) Testing vulnerability models: stability of potential vulnerability indicators across clinical state. In Search for the Causes of Schizophrenia, Vol. II (eds H. Häfner & W. F. Gattaz), pp. 177-191. Heidelberg: Springer.
- OVERALL, J. E. & GORHAM, D. R. (1962) The brief psychiatric rating scale. *Psychological Reports*, 10, 799-812.
- PARKER, G., JOHNSTON, P. & HAYWARD, L. (1988) Parental "expressed emotion" as a predictor of schizophrenic relapse. Archives of General Psychiatry, **45**, 806–813.
- SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1978) Research Diagnostic Criteria: rationale and reliability. Archives of General Psychiatry, 35, 773-782.
- STRAUSS, J. S. & CARPENTER, W. T., JR. (1974) The prediction of outcome in schizophrenia: II. Relationships between predictor and outcome variables. Archives of General Psychiatry, 31, 37-42.
- & —— (1977) Prediction of outcome in schizophrenia:
  III. Five year outcome and its predictors. Archives of General Psychiatry, 34, 159-163.
- TARRIER, N. (1989) Electrodermal activity, expressed emotion and outcome in schizophrenia. *British Journal of Psychiatry*, 155 (suppl. 5), 51-56.
- ——, BARROWCLOUGH, C., VAUGHN, C., *et al* (1988) The community management of schizophrenia: a controlled trial of a behavioural intervention with families to reduce relapse. *British Journal of Psychiatry*, 153, 532–542.
- VAUGHN, C. E. & LEFF, J. P. (1976a) The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed neurotic patients. *British Journal* of Psychiatry, 129, 125-137.
  - 4 (1976b) The measurement of expressed emotion in the families of psychiatric patients. British Journal of Social and Clinical Psychology, 15, 157–165.
- , SNYDER, K. S., JONES, S., et al (1984) Family factors in schizophrenic relapse: a California replication of the British research on expressed emotion. Archives of General Psychiatry, 41, 1169-1177.
- VENTURA, J., NUECHTERLEIN, K. H., LUKOFF, D., et al (1989) A prospective study of stressful life events and schizophrenic relapse. Journal of Abnormal Psychology, 98, 407-411.
- WATT, N. (1974) Amherst Modification of the Hollingshead Two-Factor Index of Social Position. Unpublished. Available from N. Watt, University of Denver, Denver, Colorado.
- WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) The Measurement and Classification of Psychiatric Symptoms: An

Instruction Manual for the PSE and CATEGO programs. London: Cambridge University Press. ZUBIN, J. & SPRING, B. (1977) Vulnerability - a new view of schizophrenia. Journal of Abnormal Psychology, 86, 103-126.

\*Keith H. Nuechterlein, PhD, Professor of Medical Psychology, Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, 300 UCLA Medical Plaza, Room 2251, Los Angeles, California 90024-6968, USA; Karen S. Snyder, MA, Research Associate, Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles; Jim Mintz, PhD, Professor of Medical Psychology, Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, West Los Angeles Veterans Administration Medical Center, Brentwood Division, Clinical Research Center for Schizophrenia (B117), Wilshire and Sawtelle Blvds., Los Angeles, California 90073, USA

\*Correspondence