The relationship between relative's Expressed Emotion and schizophrenic relapse: an Australian replication

K. Vaughan¹, Mary Doyle², N. McConaghy³, A. Blaszczynski², A. Fox², and N. Tarrier⁴

¹ Palmerston Unit, Hornsby Hospital; ² Prince of Wales Hospital; ³ Prince of Wales Hospital, University of New South Wales;

⁴ University of Manchester, UK

Accepted: September 18, 1991

Summary. We report a predictive study, carried out in Sydney Australia, investigating the association between the Expressed Emotion (EE) status of the household to which the patient is discharged and schizophrenic relapse. Expressed Emotion was not related to illness severity either at admission or discharge, but was related to variables reflecting chronicity and employment history. There was a significant association between returning to a high EE household and both re-hospitalisation and relapse. The significant association between EE and relapse held only for: patients not on medication, males, and those patients in high contact with their relatives. A discriminant function analysis found that decline in occupational status and the number of critical comments expressed by the relative were the strongest predictors of relapse. The results presented here are consistent with the majority of published reports on EE and relapse and contradict the negative findings of a recently published but smaller study also carried out in Sydney.

There is now a considerable body of research investigating the association between schizophrenic relapse and the level of Expressed Emotion (EE) of the relatives to whom the patient returns to live with after hospital discharge (Kuipers and Bebbington 1988; Falloon 1988). Typically patients are recruited into the study during an admission for an acute schizophrenic episode. During the admission the relatives living in the same residence are interviewed using the Camberwell Family Interview (Leff and Vaughn 1985) which is audiotaped. From this audiotape three measures: criticism, hostility, and marked emotional overinvolvement (EOI) are assessed and on the basis of these scores the relative is assigned to either a high EE or low EE category. After discharge the patients are followed up over a nine to twelve month period during which relapses, defined in terms of a worsening or recurrence of positive symptoms, are noted. A comparison between the number of relapses in patients returning to live with high and low EE relatives is then made. A number of studies in England (Brown et al. 1972; Vaughn and Leff 1976; MacMillan et al. 1986; Tarrier et al. 1988), North America (Vaughn et al. 1984; Moline et al. 1985; Nuechterlein et al. 1986; Karno et al. 1987), India (Leff et al. 1987), Italy (Cazullo et al. 1988) and Poland (Rostworowska et al. 1987) have used this methodology and found a significantly greater number of relapses in patients returning to live with high EE relatives. Two studies, one carried out in Germany (Kottgen et al. 1984) and the other in Australia (Parker et al. 1988) have failed to find any association between relapse and EE. These two studies show a number of methodological differences and defects especially in relapse identification which could explain their negative results.

The majority of evidence supports the association of living with a high EE relative and increased relapse rates. However the interpretation of these studies is not entirely unambiguous due to methodological differences in defining relapse and EE and in the failure of some studies to assess all the relatives in the household.

Other factors appear to have a limiting effect on EE. Continuous neuroleptic medication and low face to face contact (below 35 hours per week) have been shown to be associated with reduced relapse rates in patients living with high EE families (Leff and Vaughn 1985). There is also some evidence that EE is a stronger predictor of relapse in males than females (Vaughn et al. 1984; Hogarty 1985) although most studies have a greater percentage of males in their sample which may explain part of this effect.

Clearly the question arises whether EE is a causative factor in precipitating relapse or is a consequence of living with a patient who is more severely disturbed and hence more likely to relapse. However, this cause or consequence question is likely to be overly simplistic. In the initial study conducted by Brown and his colleagues (1972) an association was found between the patients' "behavioural disturbance" prior to index hospitalisation and relapse, but this factor failed to add any further predictive validity to EE status. Furthermore, ratings of severity of psychopathology at discharge have been reports as unrelated to EE status (Vaughn et al. 1985; Miklowitz et al. 1983). MacMillan and colleagues (1986) reported that they had found an association between the preadmission

duration of the illness and EE in first admission schizophrenics, a finding which they interpreted to signify that EE was not a significant predictor of relapse. But Mintz and colleagues (1989) subsequently produced data to suggest that this relationship was an artifact of differential exposure of the relatives to the early stages of the patients' disorder. Studies by Tomaras et al. (1988) and Milne (1988) indirectly support the supposition that relatives' attitudes are not purely determined by characteristics of the patient. Both these studies assessed relatives' attitudes but used measures other than EE, hence their results can only be of limited support. However both studies found that relatives' attitudes did not change even though there were improvements in the patient's condition and behaviour, thus indicating that relatives' attitudes do not simply reflect the patients' clinical condition.

There is also evidence which suggests that EE can influence relapse. This evidence comes principally from family intervention studies in which changes in the relatives' EE from high to low were associated with decreased relapse rates (Hogarty et al. 1986; Tarrier et al. 1988; Leff et al. 1989). Further evidence that the relative's EE is not purely a consequence of the patient's behaviour or illness comes from a series of psychophysiological studies which demonstrated that the physiological reactions of the patient are influenced by the EE of their relative (Turpin et al. 1988; Tarrier and Turpin 1991).

Despite this evidence it is probable that a large array of factors influence both schizophrenic relapse and EE, and EE is more productively viewed as an index of a developmental and interactive process than as a simple cause or consequence. This position would be strengthened with further positive reports of significant associations of EE and schizophrenic relapse. This paper reports on the results of a predictive study examining the association of EE and relapse. This is especially important since the publication of another subsequent but smaller (n = 57) study, carried out in the same city, has indicated a higher relapse rates in patients returning to live with low EE relatives (Parker et al. 1988).

Method

Subjects

Five hospitals in Sydney, Australia gave permission for a psychiatrist (KV) to examine case records of patients with diagnoses of schizophrenia who had been admitted to their psychiatric wards in the previous week. Those patients in whom the diagnosis was supported by reports of the presence of delusions, hallucinations or bizarre behaviour, not obviously due to an affective or organic illness and who agreed to take part in the study were interviewed with the Present State Examination (PSE). Patients were excluded if: (1) their diagnosis was not confirmed by the PSE/Catego, (2) if they were not expected to return to live with their family after discharge, (3) if they had a history of heroin abuse, or (4) if there were indications of primary organic brain damage. In the week prior to discharge a second PSE was administered to act as a baseline to assess relapse over the follow-up period.

Ninety-one suitable patients agreed to participate in the study. Fifty-seven (63%) were male and 34 female. The mean age of the men was 27.6 years (SD 8.5 years), for the women 40.2 years (SD 13.6 years) and 32.2 (SD 12.3) for the sample as a whole. They had a mean of 3.3 admissions (SD 3.8, range 1 to 20) and 28 (31 %) were experiencing their first admission. Mean age at first admission of the men was 22.9 years (SD 6.6 years) and of the women 31.1 years (SD 12.5 years). There was evidence of long-standing impairment in many patients, as indicated by their classification of occupation on the Congalton 7-point scale of occupation (Congalton 1969) in Australia (where 1 is high status and 7 is low). The mean rating for the best occupational levels achieved was 5.7 (SD 1.1). In the preceding year 47% of the patients had been employed at a level lower than their best occupational level, and 45% had been unemployed for at least twelve months. The mean rating of 13.2 (SD 6.0) on the Phillips Premorbid Personality Scale and 17.3 (SD 6.5) on the UCLA Premorbid Personality Scale suggests that their adjustment in adolescence was poor for many of these patients.

Sixty-two patients (53 men) were discharged to live with parents; 36 with both parents, 25 with mothers only and one with his father only. Twenty-six (23 women) returned to live with spouses. One returned to live with a sister, one with his son, and one with a homosexual partner. Thirty-six patients who were discharged to live with their high EE parents also took part in an intervention study (Vaughan et al. 1991), however since the 18 patients who received the intervention did not show significantly decreased relapse rates compared to those in the control group we feel justified in including them in the prospective study. If this small group did effect the results it would be in the direction of reducing high EE relapses. Assessment of sex differences demonstrated that fewer women reported using marijuana than men ($\chi^2 = 5.64$, P < 0.02); more males came from parental homes, whereas more females lived with their spouses ($\chi^2 = 42.7$, P < 0.001); females were older than males (t = 4.85, P < 0.001), and older at first admission (t = 3.55, P < 0.001), and the number of years since first admission was greater for females (t = 2.23, P < 0.05); and the males had significantly higher scores on the Phillips Premorbid Personality Scale (t = 2.89, P < 0.005) but not the UCLA scale. This was probably due to the latter not being strongly related to marital status. There were no other significant differences.

Procedure

During each patient's admission, all key relatives were interviewed by one of the authors (MD) using the Camberwell Family Interview (Leff and Vaughan 1985). All interviews were audiotaped and ratings of high EE were made using the following criteria: if the relative made 6 or more critical comments, or showed the presence of hostility, or scored 3 or more on a rating of marked emotional over-involvement (EOI).

The rater had been trained in the CFI/EE at the Institute of Psychiatry, London, UK and achieved an accept-

able level of inter-rater reliability (Pearson correlation, r = 0.81) with the instrument trainers. The households were classified as high EE if there were one or more high EE relatives present. Thirty (49%) of the mothers, 15 (41%) of the fathers and 12 (46%) of the 26 spouses were classified as high EE. Forty-two patients (46%) returned to low EE households and 49 (54%) to high EE households. For the mothers: the mean number of critical comments was 5.78, 12 (20%) made no critical comments and 17 (28%) made ten or more; 12 (20%) were scored as 3 or higher on EOI; and 30 (49%) exhibited hostility. For the fathers the mean number of critical comments was 5.37. 7 (19%) made no critical comments and 12 (32%) made ten or more. 3 (8%) were scored as 3 or higher on EOI, and 15 (41%) exhibited hostility. For the husbands the mean number of critical comments was 6.43. 5 (22%) made no critical comments and 7 (30%) made ten or more. 2 (9%) were scored as 3 or higher on EOI, and 11 (48%) exhibited hostility. The number of wives or other relations was too small to be presented separately. These EE scales did not significantly differ either between mother and father, or between parent and spouse.

Follow-up

Nine months following discharge, or at a subsequent admission if this occurred beforehand, patients were again interviewed using the PSE by one of the authors (AB) who was blind to the EE status of the household. The PSE does not provide an index of severity of schizophrenic symptoms. Delusions, hallucinations, and thought interference were measured independently on a 0-4 scale. Incoherence of speech was measured on a 0-3 scale, and catatonic symptoms was measured on a 0-2 scale. Each point was operationally defined in terms of the PSE item score. An increase from the discharge interview to the follow-up interview of one point on two more scales, or two or more points on any one scale was considered evidence of schizophrenic relapse. At the time of the third interview a relative or partner was interviewed to determine the time the patient had spent in face to face contact with the relative or partner with whom he or she was living. In four of the households this information was unavailable. Five patients committed suicide during or soon after the study, four of these before the follow-up assessment. Information obtained from their treating psychiatrist indicated that these patients had experienced an intensification in their schizophrenic symptoms in the month prior to suicide. These patients were classified as relapses. Two other patients were not formally re-assessed, one had returned to live abroad and the other's sister had committed suicide just prior to the assessment follow-up. Since all the available evidence from telephone contact and their treating doctors suggested that they had remained well, they were so classified.

Medication

Medication was assessed solely on self-report. Thirty-eight patients (42%) reported taking continuous medication and 49 (54%) reported taking no medication at fol-

low-up. The remainder appeared inconsistent in their medication use.

Results

EE and demographic variables

There was a significant relationship between living in a high EE household and 1) a decline in occupational status ($\chi^2 = 5.34$, P < 0.05); 2) a higher mean number of previous admissions (t = 2.18, P < 0.05); 3) a lesser number of months since the last admission (t = 2.03, P < 0.05); 4) a longer period of unemployment (t = 2.51, P < 0.02); 5) a shorter period of employment during follow-up (t = 2.35, t = 2.05); 6) and higher scores on the Phillips Premorbid Personality Scale (t = 2.28, t = 2.05).

EE and PSE syndromes

Comparisons between patients living with high and low EE households indicated that there were no significant differences between these groups on the presence or absence of PSE syndromes at admission (using Bonferroni correction) and discharge. The 38 PSE syndromes were divided into four major categories: schizophrenic, depressive, manic, and non-specific neurotic symptoms. The total score for each of these four major categories was calculated as the sum of the syndrome scores. Comparisons of patients from high and low EE households at admission, discharge and follow-up indicated that there were no significant differences on the mean total symptom score.

Males had significantly higher scores on the schizophrenic (t = 2.09, P < 0.05) and depression (t = 2.16, P < 0.05) scales than females at admission.

EE and relapse and re-hospitalisation

Outcome at follow-up was divided into the following classifications: a) remission to relapse (23 patients); b) persisting symptoms to exacerbation (12 patients); c) remission to remission (36 patients); d) persisting symptoms to improved (6 patients); e) persisting symptoms to remission (10 patients); f) persistent severe symptoms to persistent severe symptoms (4 patients). By the end of the nine month follow-up period 44 patients had been re-admitted. There was a significant association between living in a high EE household and re-hospitalisation. The percentages from each type of household are presented in Table 1. Since it was considered impossible to determine whether the four patients with severely persistent symptoms had relapsed or not they were not included further in the analysis. Of the remaining 40 who had been re-admitted, 27 were assessed as having relapsed. Eight of the 47 not re-admitted were also considered to have relapsed. There was a significant relationship between rehospitalisation and relapse ($\chi^2 = 22.9$, P < 0.001). The association between relapse and EE household was significant and is shown in Table 1. Of the 13 patients who were re-admitted but not assessed as relapsed, six had

Table 1. Re-admission and relapse rates

	Number of admissions	Percentage of admis- sions	2
Complete sample HEE LEE	29/49 15/42	59 % 36 %	$\chi^2 = 5.0 P < 0.03$
	Number of relapses	Percentage of relapses	
Complete sample HEE LEE	25/47 10/41	53 % 24 %	$\chi^2 = 7.1 P < 0.01$
Not on medication HEE LEE	17/28 4/21	61 % 19 %	$\chi^2 = 8.5 P < 0.005$
On medication HEE LEE	8/19 6/19	42 % 32 %	$\chi^2 = 0.45 \text{ ns}$
High contact HEE LEE	15/22 2/17	68 % 12 %	$\chi^2 = 12.41 P < 0.0005$
Low contact HEE LEE	8/23 6/21	35 % 29 %	$\chi^2 = 0.2 \text{ ns}$
Males only HEE LEE	15/28 6/25	54 % 27 %	$\chi^2 = 4.83 \ P < 0.03$
Females only HEE LEE	10/19 4/15	53 % 27 %	$\chi^2 = 2.33 \text{ ns}$

Table 2. Summary table from stepwise discriminant function analysis using demographic variables and EE dimensions

Variable	Wilks Lambda	P value	Residual variance	Stan- dard coeffi- cient	Correlation with function
Decline in occu- pational status	0.87	0.0013	0.8611	0.74	0.59
Number of critical comments	0.78	0.0002	0.7754	0.47	0.58
Parental vs. non- parental home	0.75	0.0001	0.7390	-0.31	-0.15
Number of pre- vious admissions	0.72	0.0002	0.7179	0.35	0.50
Duration of un- employment	0.70	0.0002	0.6972	-0.45	0.12

been discharged to a high EE household and seven to a low EE household. This proportion of admitted but not relapsed patients is consistent with that reported from North America (Vaughn et al. 1984).

The significant relationship between discharge to a high EE household and relapse was present in patients who were not receiving medication but not in those on medication, in patients who were in high face to face contact with their relatives but not in those with low contact, and in men but not women. However, the relative odds of relapse in high EE households was identified in males and females (see Table 1). The relationship between high EE house-

hold and returning to live with parents was significant ($\chi^2 = 5.0$, P < 0.05) but was not significant in patients returning to live with spouses ($\chi^2 = 2.16$, ns). However, there is a confounding between sex and type of home the patient returns to, with the majority of male patients returning to live with parents, and the majority of women returning to live with their spouses. Thirty-four (58%) of the 59 parental homes were high EE as compared to 13 (46%) of the non-parental homes. This difference was not statistically significant. Of the 34 subjects who returned to high EE parental homes, 18 (53%) relapsed compared to 6 (24%) of the 25 who returned to low EE parental homes. Similar percentages (54% and 27%) relapsed of the 28 subjects returning to high and low EE non-parental homes.

Relapse and demographic variables

Relapse was also significantly associated with a decline in occupational status ($\chi^2 = 6.2$, P < 0.05) and the number of previous admissions (t = 2.11, P < 0.05).

Prediction of relapse using demographic variables and EE

As there were a number of variables which were related to EE status of the family and to relapse, it is possible that the relationship between EE and relapse reflects these variables. To examine this possibility a stepwise discriminant function analysis was carried out to determine which variables were significant in predicting relapse.

Independent variables assessed were both demographic (number of previous admissions, number of months since the last admission, decline in occupational status over the last year, employment impairment in the three months prior to admission, length of unemployment, number of months unemployed at follow-up, Phillips Premorbid Personality score, gender, family type [parental or marital], hours in contact with relative and medication), and CFI variables (number of critical comments, hostility, EOI, warmth and number of positive remarks). When the subject returned to live with both parents, the parent with the higher EE was used to provide these data. The variables which emerged as significantly predicting relapse are presented in Table 2. The remainder did not significantly contribute to further reduction in variance between relapsers and non-relapsers. Thus the EE dimensions of EOI and hostility did not predict relapse once demographic factors and critical comments were 'controlled' for in the discriminant analysis. However, the number of critical comments was very important in predicting relapse, as can be seen by the magnitude of the standard discrimination coefficient and its correlation with the discriminant function. Next to decline in occupational status, the frequency of critical comments contributed most to the reduction in variance between the relapse and non-relapse groups.

Discussion

In accordance with most previous studies, schizophrenic patients in the Sydney sample who were discharged to high EE households had a significantly higher relapse rate

than those discharged to low EE households. This relationship holds both for symptomatic relapse and for rehospitalisation. This result does not, however, establish that the relationship between discharge to a high EE household and subsequent schizophrenic relapse is causal. But in the light of previous results of the bulk of prospective studies, and the results of family intervention studies which demonstrate a relationship between reduced relapse rates and changes in EE from high to low, it is suggestive of such a relationship. High face to face contact and a lack of continuous medication appear to act as risk factors for patients returning to live with high households, as was reported by the earlier London study (Vaughn and Leff 1976). The high percentage of patients who were not on continuous medication (56%) is perhaps surprising, and may reflect differences in the local psychiatric services in Sydney. It would appear that reducing contact with a high EE relative and prophylactic use of medication act as protective factors and reduce the difference in relapses between the high and low EE groups to a non-significant one. The results reported could be taken to support past findings that EE is a stronger predictor of relapse in males than in females (Falloon 1988), but the non-significant findings in females probably reflects a type II error.

The complex nature of EE is further indicated by the significant relationship shown by both EE and relapse with variables reflecting chronicity and employment history, but not symptom severity or type. This may support a developmental or coping model of EE.

Discriminant function analysis to evaluate the contribution of demographic features of the illness as well as the relationship variables on which assessment of EE is based, revealed that occupational decline was the strongest predictor. This variable is hard to interpret due to the difficulty in rating married women who were not working, a good prognosis group, as showing a decline in occupational level. Patients showing a decline were much more likely to be single men living with their parents who both in this study and others tend to be more susceptible to relapse. Occupational decline in this group may relate to severity or chronicity of illness, social drift, or the level of social functioning of the patient. It has previously been reported that patients living in high EE households functioned at a significantly lower level socially than those living in low EE households (Barrowclough and Tarrier 1990). It is possible that a decline in an ability to function is related to the development of maladaptive attitudes and behaviour on the part of the relative which further increase the probability of relapse.

The number of critical comments was the next strongest predictor. Other variables entered accounted for less than 4% of the variance. Interestingly the other EE variables, such as EOI and hostility did not significantly predict relapse.

The demonstration of the significant relationship between returning to live in a high EE household and the increased likelihood of schizophrenic relapse in this study makes it unlikely that the Parker et al. (1988) failure to demonstrate such a relationship in the other Australian study was due to cultural factors. Differences in outcome

in the two Australian studies are difficult to interpret. The high percentage of high EE households in the Parker et al. study compared to the present study (74% compared to 54%) may suggest a sampling difference. This is supported by the fact that this present study was carried out prior to that of Parker et al. and drew patients from the same institutions. Subjects were not included in the study of Parker et al. if they had been included in the study reported here. This may have introduced some bias into the Parker et al. sample, which only included patients returning to the parental home. Nevertheless, the percentage of high EE households in the Parker et al. study is not different from the 77% reported in the Salford, UK study (Tarrier et al. 1988).

There were also a number of other methodological difficulties with the study of Parker et al. which singles it out from the majority of other studies. Firstly diagnosis was on the basis of "clinician's judgement" rather than a standardised diagnostic instrument. Secondly relapse was based on "case status" which is unclear, rather than a specific recurrence or worsening in psychotic symptoms, and perhaps most importantly "interviewing abnormalities" in the administration of the CFI. Similar difficulties with relapse criterion arise with the German study (Kottgen et al. 1984) which also reported negative results. These difficulties tend to reduce the impact of the negative results reported by these two studies. The results of the present Sydney study are more consistent with the published studies from other parts of the world.

Acknowledgement. This project was carried out while the first author was in receipt of a Fellowship from the NSW institute of Psychiatry.

References

Barrowclough C, Tarrier N (1990) Social functioning in schizophrenic patients. I. The effect of expressed emotion and family intervention. Soc Psychiatry Psychiatr Epidemiol 25: 125–129

Brown G, Birley JLT, Wing JK (1972) Influences of family life on the course of schizophrenic disorders: replication. Br J Psychiatry 121: 241-258

Cazullo CL, Bertrando P, Bressi C et al. (1988) Emotivita expressa e schizofrenia: studio prospettico di replicazione. Notizie ARS [Suppl] 88: 16–21

Congalton AA (1969) Status and prestige in Australia. Cheshire Publications, Melbourne

Falloon IRH (1988) Expressed emotion: current status. Psychol Med 18: 269-274

Hogarty GE (1985) Expressed emotion and schizophrenic relapse: implications from the Pittsburgh study. In: Alpert M (ed) Controversies in schizophrenia. Guilford, New York

Hogarty GE, Anderson CM, Reiss DJ et al. (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. Arch Gen Psychiatry 43: 633–642

Kuipers L, Bebbington P (1988) Expressed emotion research in schizophrenia; theoretical and clinical implications. Psychol Med 18: 893–909

Karno M, Jenkins JH, De la Silva A et al. (1987) Expressed emotion and schizophrenic outcome among Mexican-American families. J Nerv Ment Dis 175: 143–151

Leff JP, Vaughn C (1985) Expressed Emotion in families. Guilford, New York

- Leff JP, Wig N, Ghosh A et al. (1987) Influence of relative's expressed emotion on the course of schizophrenia in Chandigarh. Br J Psychiatry 151: 166–173
- Leff JP, Berkowitz R, Shavit N et al. (1989) A trial of family therapy vs a relatives' group for schizophrenia. Br J Psychiatry 154: 58–66
- MacMillian JF, Gold A, Crow TJ et al. (1986) The Northwick Park study of first episodes of schizophrenia. IV. Expressed emotion and relapse. Br J Psychiatry 148: 133–143
- Miklowitz D, Goldstein MJ, Falloon IRH (1983) Premorbid and symptomatic characteristics of schizophrenics from families with high and low levels of expressed emotion. J Abnorm Psychol 92: 359–367
- Milne D (1988) Organisational behaviour management in a psychiatric day hospital. Behav Psychother 16: 177–188
- Mintz LI, Nuechterlein KH, Goldstein MJ et al. (1989) The initial onset of schizophrenia and family expressed emotion: some methodological considerations. Br J Psychiatry 154: 212–217
- Moline RA, Singh S, Morris A et al. (1985) Family expressed emotion and relapse in schizophrenia in 24 urbane American patients. Am J Psychiatry 142: 1078–1081
- Nuechterlein KH, Snyder KS, Dawson ME et al. (1986) Expressed emotion, fixed dose fluphenazine decanoate maintenance and relapse in recent onset schizophrenia. Psychopharmacolog Bull 22: 633–639
- Parker G, Johnston P, Hayward L (1988) Parental expressed emotion as a predictor of schizophrenic relapse. Arch Gen Psychiatry 45: 806–813
- Rostworowska M, Barbaro B, Cechnicki A (1987) The influence of expressed emotion on the course of schizophrenia: a polish replication. Poster presented at the 17th Congress of the European Association for Behaviour Therapy, Amsterdam
- Kottgen C, Sonnichsen I, Mollenhauser K (1984) Results of the Hamburg Camberwell family interview study, I–III. Int J Fam Psychiatry 5: 61–94

- Tomaras V, Vlachonikolis JG, Stefanis CN et al. (1988) The effect of individual psychosocial treatment on the family atmosphere of schizophrenic patients. Soc Psychiatry Psychiatr Epidemiol 23: 256–261
- Tarrier N, Barrowclough C, Vaughn C et al. (1988) The community management of schizophrenia: a controlled trial of a behavioural intervention with families to reduce relapse. Br J Psychiatry 153: 532–542
- Tarrier N, Turpin G (1991) Electrodermal activity in schizophrenia: a review. Br J Psychiatry (submitted for publication)
- Turpin G, Tarrier N, Sturgeon D (1988) Social psychophysiology and the study of biopsychosocial models of schizophrenia. In: Wagner H (ed) Social psychophysiology: perspectives on theory and clinical application. Wiley, Chichester
- Vaughn C, Leff JP (1976) The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenia and depressed neurotic patients. Br J Psychiatry 129: 125–137
- Vaughn C, Snyder KS, Freeman W et al. (1984) Family factors in schizophrenic relapse: a replication in California of the British research on expressed emotion. Arch Gen Psychiatry 41: 1169–1177
- Vaughan K, Doyle M, McConaghy N et al. (1991) The Sydney intervention trial: a controlled trial of relative's counselling to reduce schizophrenic relapse. Soc Psychiatry Psychiatr Epidemiol 27: 16–21
- Vaughn C (1986) Comments on Dulz and Hand. In: Goldstein MJ, Hand I, Halweg K (eds). Treatment of schizophrenia: family assessment and intervention. Springer, Berlin Heidelberg New York

Dr. K. Vaughan Palmerston Unit Hornsby Hospital NSW 2077 Australia