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Social Relationships, Adversity and Neurosis: An Analysis of Prospective Observations

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Summary: The effect of deficiencies in social relationships has been studied prospectively in a community sample. In the half who were exposed to the higher level of adversity, these deficiencies explained 30 per cent of the variance in neurotic symptoms four months later. They explained only 4 per cent in those with lower adversity. Contrary to expectation, it was not the lack of relationships, but perception of these as being inadequate under adversity, which had by far the stronger predictive power. This may mean that actual conditions in the immediate social environment are not important for neurosis. Instead, intrapsychic and personality factors may have a much stronger effect.

In previous papers, the association between neurosis and deficiencies in social relationships was reported, firstly in a study of patients, then in a general population sample (Henderson *et al*, 1978a; 1978b; 1980). These investigations showed that persons with neurotic symptoms reported a deficiency in the availability and adequacy of social ties. This deficiency occurred both for close affectional bonds and for more diffuse relationships; it was observed both in persons seeking treatment and in a general population sample. Deficiencies in the social relationships of neurotics was therefore unlikely to be characteristic only of those who sought help, as Mechanic (1963; 1978) has cautioned.

Lin *et al* (1979) reported similar findings in a study of a small sample of Chinese Americans in Washington, D.C. Silberfeld (1978) also found such an association in psychiatric outpatients. The association may hold for medical as well as psychiatric disorders (Cassel, 1976). In an impressive study of mortality in Alameda County, California, Berkman and Syme (1979) used a prospective design and found a relative risk of 2.3 in men and 2.8 in women who had low social support; they showed that this was not related to social class, health status at the initial interview, or to other variables such as bad health practices. There therefore is considerable evidence for a protective effect from social relationships.

Any study of neurosis and social bonds, if made at only one point in time, cannot establish the direction of causality: persons with already established neurotic symptoms may report deficiencies in relationships because of their affective state, because their symptoms may have had an adverse effect on their personal networks, or because underlying variables such as personality attributes, may have rendered them vulnerable to neurosis and therefore less able to establish and maintain mutually satisfying personal relationships (Foulds, 1965; 1976). From the cross-sectional study previously quoted (Henderson *et al*, 1980), a subsample has been studied prospectively to examine the matter of causality. The associations between the variables are complex and the present paper is an attempt to discern the main effects which may be at work.

The hypotheses being tested are: (a) that a lack of social relationships is a causal factor in the onset of neurosis and (b) that a deficiency in either attachment or in social integration is a causal factor in the subsequent onset of neurosis, independent of the load of adversity. That is, a lack of social relationships is postulated to be a causal factor in its own right and not to act only by inducing vulnerability, as has been proposed by Cassel (1976) for social ties in general, and by Brown and his colleagues for close confiding relationships (Brown *et al*, 1975; Brown and Harris, 1978a). The investigation is a step towards assessing the therapeutic or preventive value of social support.

Method

A psychiatric morbidity survey was carried out on a representative sample of residents in Canberra in 1977 (Henderson *et al*, 1978b; 1979; 1980; Duncan-Jones and Henderson, 1978). Of the total sample who were interviewed in the cross-sectional stage of the survey (n = 756, or 85 per cent of those randomly chosen

from the electoral roll), a random subsample were interviewed again on three occasions at intervals of about four months. This is referred to as the Panel Study and the four waves of interviews will be described as Waves 1, 2, 3 and 4. Of an original subsample of 323 taken from Wave 1, 231 completed all four interviews a year later. Attrition of the sample between each of the three follow-up interviews was found to be unrelated to mental health status at the previous examination. Most interviews took place in the respondents' homes. The interviewers were experienced professionals selected by us for this work and some of them had taken part in the pilot study (Henderson *et al*, 1978b). Before embarking on the main study the interviewers were taught and rehearsed the use of the instruments and three further training sessions were held during the course of the study. Respondents were not examined more than once by the same interviewer and interviewers were not informed of the respondents' previous performance. The interviewers were not informed of the hypotheses which were being tested.

At each Wave, three instruments were administered:

(1) The Interview Schedule for Social Interaction (ISSI), an instrument for the systematic assessment of an individual's personal network. An account of its construction, reliability and validity, together with its performance in a general population sample, has been given by Henderson *et al* (1980). The ISSI explores both the respondent's close affectional bonds and his more diffuse ties, together with those provisions which may be derived from social relationships, as proposed by Weiss (1973; 1974). The interview lasts about an hour and yields the following indices of social relationships. These indices have been found to be reliable and strikingly stable over time; their validity has been fairly thoroughly explored, including the use of data from another informant, and has been shown to be satisfactory (Henderson *et al*, 1980).

AVAT — the availability of affectionally close relationships (attachments);

ADAT % — the perceived adequacy of what comprises these close relationships, expressed as a percentage of what is available;

NONAT — in those who lack close relationships, there might nevertheless be acceptance of this. The NONAT index is a measure of such satisfaction despite the absence of attachment.

ATTROWN — the number of attachment persons with whom the respondent has been having rows in the last month.

AVSI — the availability of more diffuse relationships, as with friends, work associates and acquaintances, here called social integration.

ADSI — the perceived adequacy of these more diffuse relationships.

(2) The List of Recent Experiences (LRE) which is an interview to assess the respondent's exposure to adverse events or difficulties in the previous twelve months, and has been described elsewhere (Steele *et al*, 1980). A score is obtained for exposure to adverse experiences in the previous twelve months, or in the case of the panel interviews, in the four months since the previous one. It is a 71-item inventory, based on a list constructed for an Australian population by Tennant and Andrews (1976) and similar to the instruments of Holmes and Rahe (1967) and Paykel *et al* (1971). It differs from other instruments in that longstanding difficulties as well as temporally discrete events are identified. The distress caused by each nominated experience is scored according to criteria which are independent of the individual's report of his own affective response. These scores are summed to obtain a cumulative score for exposure to adverse experiences. The reliability of the instrument has been found to be satisfactory for cumulative scores but not for individual experiences (Steele *et al*, 1980).

(3) The 30-item General Health Questionnaire (GHQ) (Goldberg, 1972; Goldberg *et al*, 1976) is an instrument for the detection of non-psychotic psychiatric disorder. We had the choice of using the GHQ scores themselves or the estimated case rates on the Present State Examination (Wing *et al*, 1974) derived from them by a logit regression equation (Duncan-Jones and Henderson, 1978). We have used the former as a simpler and continuous measure of the dependent variable but we recognized that the morbidity expressed by this may be different, either in its severity or duration from that encountered in psychiatric practice. One could not expect to have many new formal cases of neurosis appear in the present sample over the time period studied.

Results

The first hypothesis. The correlations were examined between the GHQ scores in Waves 1, 2, 3 and 4 and the two independent variables, social relationships and adversity, as reported in Wave 1. These are shown in Table Ia, which provides some information on the relationship between these variables and the GHQ scores at the same point in time, then four, eight and twelve months later. Overall, there is a decline in the strength of the association between Wave 1 and 4.

But the reporting of social relationships and of adversity in Wave 1 would have been open to contamination by the respondents' affective state at that time; and the GHQ scores at increasing intervals could have been influenced by subsequent changes in social relationships and subsequent exposure to adversity; the longer the time, the greater the chance of this. We shall therefore focus our attention on associations between measures made in Waves 1 and 2. Further analyses will be required to allow all three sets of variables to be examined over all the Waves. Methods for this are being explored.

A correlation matrix (Table Ib) for those who were psychiatrically well at the time of the Wave 1 examination, was then examined. This has been done by excluding those respondents who had a GHQ score of more than 5 in Wave 1, leaving a cohort of 177, as shown in Table I(b). This cutting score is one point higher than recommended by Goldberg (1972) for recognition of a case. In the four months up to Wave 2, 12 of these 177 respondents developed symptoms leading to a GHQ score over 5.

The indices most strongly associated with the subsequent GHQ score were the two measures of perceived adequacy, social integration being slightly greater than attachment. When all of the Wave 1 ISSI indices were combined in a multiple regression equation, they accounted for 12.7 per cent of the variance in GHQ score four months later. By contrast, adversity in Wave 1 explained only 4 per cent. If adversity reported for the four months between Waves 1 and 2 was allowed into the calculation, it accounted on its own for 3.4 per cent of the variance in fresh neurotic symptoms at Wave 2; and when it was combined with the adversity measure reported for the previous year, when all the sample were still well at Wave 1, 5.2 per cent of the variance in Wave 2 GHQ was explained. Because persons with symptoms may report an excess of recent adversity for reasons other than a direct causal effect from the latter (Brown *et al*, 1973a; 1973b), such an observation must remain of limited value. It is therefore preferable to consider the effect of predictor variables measured only in persons who are as yet well. This means forfeiting information about adversity or social relationships which may have been obtained closer in time to the onset of symptoms, such as for the present Wave 2 indices, but it is methodologically safer to accept this.

These findings strongly suggest that deficiencies in social relationships, and particularly their perceived adequacy, had an effect on the early development of neurotic symptoms and that collectively this effect was stronger than the effect of adversity but they do not demonstrate that the lack of social relationships had a direct causal effect. The evidence is consistent with the first hypothesis but does not prove it, as will be discussed below.

The second hypothesis. To test the second hypothesis, that the effect of deficient social bonds in the onset of neurosis is independent of the load of adversity, the correlations have been calculated for the Wave 1 ISSI indices and the Wave 2 GHQ scores, but with the 177 respondents who were well at Wave 1 divided into two groups: those with low and those with high exposure to adversity in the twelve months before the Wave 1 interview. The adversity score was dichotomized at its mean of 89 (s.d. = 62.4, range 0 to 320). The results were striking (Table II). Deficient social bonds at Wave 1 were much more likely to be followed by the onset of neurotic symptoms in those persons who were also experiencing adversity. This effect was again strongest for the two adequacy indices. For those with low adversity before Wave 1, deficiencies in social relationships had little effect, only the ADSI index reaching the 5 per cent level of significance. By contrast, in those with the higher level of adversity, the reported adequacy of attachment accounted for 20.3 per cent ($.45^2$) and the reported adequacy of social integration for 19.4 per cent ($.44^2$) of the variance in GHQ score four months later. The evidence is therefore against the second hypothesis: a deficiency in social relationships is more strongly associated with subsequent symptoms if there is also high adversity. This is now examined further.

Explained variance. Table III shows the results of a multiple regression equation in which all the ISSI indices in Table II have been entered in the sequence shown. This order has been imposed, and was based on the assumption that it followed a logical hierarchy, similar to that used in analysis of the cross-sectional data (Henderson *et al*, 1980). The availability and then the adequacy of attachment is taken first, followed by the index of satisfaction when it is absent. The availability and adequacy of social integration follows, then finally the index of rows with close others. Together, the ISSI indices explain only 4.1 per cent of the variance in Wave 2 GHQ score for those exposed to low adversity at Wave 1, but 30.0 per cent in those with high adversity. These findings suggest that there may be an appreciable interaction effect between deficiencies in social relationships and exposure to adversity.

Discussion

Prospective data of this type have not, to the writer's knowledge, been previously available for a general population sample. The closest to the present data are those of Hagnell (1966), although his variables were somewhat different and the period between observations much longer. The main strengths of the

TABLE I(a)

Correlations between ISSI indices at Time 1 and GHQ scores in the four waves (Total panel sample n = 231)

	Wave 1	Wave 2	Wave 3	Wave 4
AVAT (Availability of close relationships)	−.18‡	−.04	−.09	−.02
ADAT% (Perceived adequacy of close relationships)	−.38†	−.27†	−.20†	−.17‡
AVSI (Availability of diffuse relationships)	−.16‡	−.17‡	−.17‡	−.15‡
ADSI (Perceived adequacy of diffuse relationships)	−.33†	−.29†	−.17‡	−.21†
NONAT (Acceptance of not having close relationships)	.00	.02	.10	−.05
ATTROWN (Rows with close others)	.29†	.23†	.10	.04
ADVERSITY	.30†	.24†	.16‡	.23†

‡ P < .01; † P < .001.

TABLE I(b)

Cohort well at Time 1 (N = 177): Correlations of the ISSI and adversity indices at Time 1 with subsequent GHQ scores

	Wave 1	Wave 2	Wave 3	Wave 4
AVAT (Availability of close relationships)	−.09	.01	−.10	.06
ADAT% (Perceived adequacy of close relationships)	−.23‡	−.22‡	−.12	−.15*
AVSI (Availability of diffuse relationships)	.00	−.10	−.09	−.09
ADSI (Perceived adequacy of diffuse relationships)	−.12*	−.31†	−.06	−.21‡
NONAT (Acceptance of not having close relationships)	−.02	.04	.10	−.09
ATTROWN (Rows with close others)	.15*	.15	.06	.02
ADVERSITY	.15*	.20‡	.13*	.05

* P < .05; ‡ P < .01; † P < .001.

Total variance in Wave 2 GHQ scores explained by ISSI indices: At Time 1 = 12.7 per cent; in GHQ 1 = 7.9 per cent

TABLE II

Testing hypothesis 2: The effect of adversity Cohort well at time 1. Correlation of ISSI indices at time 1 with Wave 2 GHQ, by adversity at Time 1

ISSI Index	Low adversity (n = 115)	High adversity (n = 62)
AVAT	.07	−.16
ADAT%	.04	−.45†
NONAT	−.07	−.26*
AVSI	.06	−.29*
ADSI	−.14	−.44†
ATTROWN	−.04	−.16

* P < .05.
† P < .001.

TABLE III

Cohort well at Wave 1. Cumulative variance (%) in Wave 2 GHQ explained by all ISSI indices at Wave 1

	Low adversity (n = 115)	High adversity (n = 62)
AVAT	0	2.7
ADAT%	0.5	13.0
NONAT	0.5	19.4
AVSI	0.7	23.0
ADSI	4.0	28.7
ATTROWN	4.1	30.0

present study are that it was conducted on a representative sample of a general population and it was conducted prospectively, so that the measures of social bonds and of adversity are less likely to be contaminated by already established neurotic symptoms. These are two features advocated by Paykel (1978) in his examination of the relative risk of neurosis caused by life events. The measure of adverse experiences was of demonstrated reliability and was appropriate for an enquiry in which the principal independent variable was social relationships. While it is more comprehensive than other life event inventories, it is still methodologically less sound than the alternative method developed by Brown and his colleagues, where contextual factors are taken into account in determining the severity of each adverse experience (Brown and Harris, 1978a). In the present study such contextual factors, which might be connected with social relationships, would have introduced a further source of contamination between the independent variables unless steps were taken specifically to avoid this in rating sessions. Thirdly, this study has employed a newly developed method for systematically examining an individual's range of social relationships and their consequences.

The main deficiency of the study is the measure of the outcome variable, neurosis. A standardized clinical interview, such as the Present State Examination, given to all respondents, would have provided a much more secure and diagnostically differentiated estimate of this. We shall present evidence in a later paper that the symptomatic states which developed over the whole 12 months were fairly short-lived episodes. It cannot be claimed in the present study that the dependent variable was neurosis in the clinical sense of the term; it was neurotic symptoms as detected by the GHQ. It would not have been practicable for the Unit to undertake 231 PSE's in both Wave 1 and 2. In the GHQ, three of the thirty items refer to social performance. To exclude such contamination by the independent variable, the analyses were re-run with these three masked out. The results were unaffected.

A second deficiency is the sample size, which naturally yielded only a very modest inception rate of morbidity in four months, although this was sufficient to give results of high statistical significance. Methods are now being developed to examine the three sets of variable over the whole twelve months.

Interpretation: The findings are in keeping with the first hypothesis, that a lack of social relationships is a causal factor in the onset of neurosis; they are against the second hypothesis, that this effect is independent of the load of adversity. Other interpretations have to be considered. First, it is conceivable that the real reason for the increased morbidity in those with high adversity at Wave 1 might have been that they had more subsequent adversity during the four months between Waves 1 and 2. The measure of adversity at Wave 2 is, of course, open to contamination by the symptoms which had developed in some by then. Nevertheless, the possibility has been checked by examining the mean adversity scores in Wave 2. Indeed, those who had high adversity at Wave 1 also had it at Wave 2; but this applied as much to those with adequate as those with inadequate social relationships. This interpretation can therefore be discounted.

A second interpretation is that persons who were still well at Wave 1 contained a subgroup who had already damaged their personal relationships as part of a prodromal process, prior to developing a neurotic disorder by Wave 2. This would account for the present observations without the need to invoke a direct causal effect from the lack of social bonds. Such an interpretation is plausible but has not been investigated further on the present data. It would require a method for identifying latent neurotic

illness, which does not currently seem practicable in epidemiological studies.

A third interpretation is that social relationships only postpone the effects of adversity, lengthening the incubation period before the onset of symptoms. This was tested by examining the mean GHQ scores in Waves 2, 3 and 4 for those with high adversity at Wave 1, but doing so separately for those with high and those with low levels of social relationships. The peak means were in Wave 2 for both these groups. The lag interpretation can therefore be discounted.

A fourth interpretation is that both neurotic symptoms and the perception of social relationships as inadequate arise from a personality attribute and that the effect of this intervening variable is much stronger in the presence of adversity. That is, those who became symptomatic by Wave 2 may have been more vulnerable to adversity by virtue of their personality structure, which at the same time may have made them less competent in personal relationships. It is not all individuals who develop symptoms after exposure to adversity (Rabkin and Struening, 1976; Andrews and Tennant, 1978) and not much is known about those, possibly the majority, who do not succumb. Here, it is necessary to postulate an association between vulnerability attributes, such as neuroticism, and perceiving social relationships as inadequate. Both could be expressions of morbid dependency or anxious attachment, as described by Bowlby (1980). That is, unmet requirements for personal relationships may have been the primary pathogenic factor, activated by the presence of adversity, just as attachment theory predicts.

Fifth, there is another attribute of the individual which might be invoked to explain the present findings. This is the tendency to complain and to be dissatisfied, described as the 'plaintive set' by Gruenberg. It is the opposite of 'pollyannaism', as described by Scott and Peterson (1975). Whether it is seen as a trait or a state, such an attribute could lead to the reporting both of misfortune and a lack of satisfying personal relationships. Conceivably, it might be also associated with increased vulnerability to neurotic symptoms, or at least to positive responses to a questionnaire such as the GHQ. Plaintive set therefore remains a legitimate interpretation, the adequacy indices on the ISSI being a possible expression of it. Further investigation of this would require an instrument designed specifically to tap this construct.

For the second hypothesis, the findings in Tables II and III appear at first to be in conflict with the results in Table I(b), when ADAT% and ADSI were found to correlate significantly with the Wave 2 GHQ. From that Table, it might be proposed that the ISSI indices have an effect in their own right on the later onset of symptoms, and not to require the simultaneous presence of adversity. This apparent conflict is likely to be due to the presence of respondents who had had high adversity in the sample on which these correlations are based. The total non-symptomatic sample of 177 included 62 who had an adversity score above the mean. It is they who would have contributed to these statistically significant correlations. The present findings, therefore, are evidence that a lack of social relationships is a causal factor in the onset of neurotic symptoms, but only when adversity is also present. The second hypothesis has to be rejected.

The conclusion is that subjectively adequate social relationships, both affectional and more diffuse, are probably protective in the face of adversity. The significance of this observation is in advancing our understanding of some of the causes of neurosis. In studies of disasters and extreme environments (Killian, 1952; Beach and Lucas, 1960; Henderson and Bostock, 1977), the presence of others has been found to have a protective effect in the acute situation. This conclusion is in accord with the hypothesis advanced by Cassel (1976) on the contribution of the social environment to host resistance, though it is important to note that this referred principally to medical disorders.

Brown and Harris (1978a; 1978b) and Tennant and Bebbington (1978) have been examining the interaction of adversity and, *inter alia*, close affectional relationships, in the onset of depression. They have used categorical data in contingency tables, for which log linear analysis is appropriate (Everitt, 1977; Everitt and Smith, 1979). In the present study, the prospective data are continuous and to have imposed further dichotomies on the variables would have led to an unnecessary loss of information. More advanced statistical methods for studying interaction effects in continuous variables would have caused major problems in interpretation. Accordingly, the present analysis has not been taken further.

The evidence is that, as a causal factor in neurosis, the crucial property of social relationships is not their availability, but how adequate they are perceived to be when the individual is under adversity. This level of adequacy clearly has two components which cannot be teased apart in the present form of the ISSI: an externally derived judgement of adequacy, taking the context into account; and the intrapsychic needs of the respondent, in terms of dependency or anxious attachment, which would presumably be increased in the presence of adversity. If the former component were judged to be adequate, the present findings would be evidence against a social and in favour of a largely intrapsychic pathogenesis, in which the low adequacy scores are a manifestation of a strong need for supportive relationships. This would mean we had

produced epidemiological data to support the concepts formulated by Rado (1956) and Fairbairn (1952a; 1952b), as considered in some depth by Chodoff (1972) and by Guntrip (1974): persons who view their relationships as inadequate have an increased risk of developing neurotic symptoms under adversity.

Whether the deficiencies in social relationships are real or perceived, this study supports the proposition (Henderson, 1974) that neurotic symptoms emerge when individuals consider themselves deficient in care, concern and interest from others; and that the symptoms themselves can appropriately be seen as care-eliciting behaviour.

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