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Guest editorial

Interpreting the evidence on social support

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Social support may contribute to mental health. There is currently much attention being given to the several hypotheses embraced by this statement. Indeed, one predicts that the subject will now move quickly to a position of prominence in the literature comparable to that accorded to life events during the last two decades. With such a growing intensity of research on social support, it was appropriate that the WPA Section of Epidemiology and Community Psychiatry made this the subject of a Symposium at the 1983 World Congress in Vienna. This issue of Social Psychiatry presents a selection of papers loosely based on some of the presentations at that meeting.

The hypothesis has considerable intuitive appeal for many, in that it proposes an aetiological factor of some importance outside the individual, in his social environment. Such a paradigm has long been a mainstay of social psychiatry. But the main attraction is that confirmation of the hypothesis could provide a rationale for treatment, or possibly for prevention; and that a better specification of its effective ingredients could be deployed towards groups deficient in social support and demonstrated to be at increased risk. The genesis of the hypothesis has been described in some detail by Henderson et al. (1981), Greenblatt et al. (1982) and Broadhead et al. (1983). From these reviews, it can be seen that the idea took shape in the mid-seventies, arising from a confluence of theories about social networks (Barnes 1954; Litwak and Szelenyi 1969), social disorganisation (Leighton et al. 1963) and the basic human need for social transactions (Maslow 1968; Hamburg 1968a, b). In his important paper "The contribution of the social environment to host resistance", Cassel (1976) put forward evidence that social support gives protection against stress-related physical morbidity through cushioning individuals against stressful experiences. The protective effect of a confiding relationship was proposed by Brown et al. (1975) for depressive illness. Miller and Ingham (1976) suggested from their general practice study that not only a confidant but also more diffuse relationships protected against neurotic symptoms. Both of these frequently cited papers have undoubtedly stimulated many subsequent investigations. As Cassel predicted, the target disorders have not been confined to psychiatry, but have included the complications of pregnancy (Nuckolls et al. 1972), increased mortality from physical disorders (Berkman and Syme 1979; Blazer 1980) and coronary heart disease (Reed et al. 1983). Thus no specificity of effect has been apparent.

At this point, it is important to recognise that the generic hypothesis must be broken into several parts, because this new independent variable is a rubric for many conceptually distinct components. Its measurement is therefore more demanding than many investigators have recognised. It may be examined for a buffering effect against adversity, as in the original proposition by Cassel (1976), or it may have a direct pathogenic effect when it is deficient. A separate class of hypothesis is that social support has a therapeutic effect after the onset of a particular disorder, or that its presence promotes positive well-being. The former would be largely in keeping with what is known about effective psychotherapy (Frank 1973, 1974). It may have any of the above effects but only for some demographic groups, such as younger women; or certain personality types, such as those with strong dependency or affiliative needs. Lastly, social support could conceivably be toxic for some: in studies of elderly populations, both Lowenthal (1968) and Kay et al. (1964) concluded that some individuals show a long-standing preference to have only limited social interaction.

The papers given at the Vienna Symposium illustrate some of the challenges faced in the investiga-

Table 1. Requirements for the Measurement of Social Support

Requirement	Comments
Specification of what is meant by support	Instrumental or affective Caring and unburdening, or endorsement and feedback Close or diffuse relationships Reported availability of relationships and what they provide Reported adequacy of these
Indices	Indirect, through coarse sociodemographic variables Purpose-built instruments Reliability Validation
Time-reference	Social networks may undergo changes following life events or episodes of illness
Not affected by symptoms	Symptoms may alter a personal network; or the validity of reports about it
Not confounded by recent life events	Social support and life events overlap; or they influence each other; their measures must be disaggregated
Not confounded by personality traits	Social support is the product of competence in personal relationships Individuals vary in their requirements for supportive relationships Dissatisfaction and neurosis

tion of these hypotheses and point to those areas in methodology which now call for attention. It is the measurement of social support which presents by far the greatest challenge. Some of the most important issues are set out in Table 1.

It is firstly necessary to specify which of the elements within the general rubric are to be measured. Where this has not been done, the measure of support can regrettably become a mélange of the instrumental and affective components which Weiss (1974) took pains to distinguish; or it may combine those facets of social relationships which may prevent demoralisation through encouragement and the unburdening of painful affect, as opposed to the feedback function which both Cassel (1976) and Cobb (1976) considered likely to be important. It should also be made clear whether the measure is of the availability of supportive social relationships or the perceived adequacy of these for an individual in a given context.

The measurement of social support has been approached in four ways. Firstly, coarse sociodemographic variables have been used as indices of isolation and of not having an intimate relationship. These carry the limitation of reflecting only indirectly the actual social environment of the individual. A second method has been to derive indices of social support from a few questionnaire items concerning marital status, living arrangements, social functioning and involvement with group activities. This was essentially the approach used by Eaton (1978), Berkman and Syme (1979), Lin et al. (1979), Williams et al. (1981), Aneshensel and Stone (1982) and Blazer (1980, 1983). A third method has been to construct ratings for specific elements within the personal network. This was done by Brown et al. (1975) in determining the presence of a confiding relationship. It has the strength of being more observer-based than the responses of the patient or interviewee; but it does not wholly escape the possibility of confounding by personality factors. Individuals who do not have an affectionally close relationship and some social interaction may have personality attributes which also render them vulnerable to psychiatric disorders, particularly depression. Lastly are purpose-built instruments, such as the Psychosocial Assets Questionnaire (Nuckolls et al. 1972), the instrument developed for sociological research by McCallister and Fisher (1978), the Social Support Questionnaire (Sarason et al. 1983) and the Interview Schedule for Social Interaction (ISSI) (Duncan-Jones 1981a, b; Henderson et al. 1980, 1981). The latter has indices which allow a distinction to be maintained between instrumental and affectional facets of support, between intimate and more diffuse relationships and between availability and reported adequacy. This latter distinction is also made in the questionnaire by Sarason and his colleagues. The reliability of such instruments has usually been found to be satisfactory, but there are considerable technical problems in validation, as discussed by Henderson et al. (1981). Validation of availability measures can be achieved by obtaining information from a collateral source; but this approach fails for the adequacy indices which are open to confounding by contextual factors and personality traits.

Personal networks are not static structures but can be expected to change in their properties with major life events such as bereavement or relocation; and with the onset of illness, including psychiatric disorders. Blazer (1983) found in a prospective study of older depressed persons that those with depressive states at the beginning of the study had *more* social supports at follow-up 30 months later. A contrasting observation by Henderson and Moran (1983) is that neither the onset nor the remission of substantial neurotic symptoms in a longitudinal community survey had any effect on social relationships other than a change in the frequency of rows with others in the

expected direction. Despite this finding, it would still be wiser to measure social relationships when people are symptom-free, if the investigation is directed at possible causal effects on the incidence of mental disorders.

As in life-event research, there are already many cross-sectional studies reporting a positive association between low levels of support and psychiatric morbidity, but these cannot be taken as evidence to support a causal hypothesis. Prospective longitudinal studies are much rarer. The problem with these is that low levels of support in symptom-free individuals may be due to the prodromata of illness or to personality attributes associated with increased vulnerability. In our own prospective study, quite contrary to our expectations and hypotheses, not one of the social support indices had any significant predictive effect for the subsequent onset of symptoms in persons exposed to low levels of adversity. Similarly, at Vienna, Surtees (1984) reported finding no increase in prevalence, using RDC criteria, for women who did not have an intimate relationship. In the Canberra study, for those having high adversity, the adequacy indices alone had substantial predictive power. We have put forward evidence that this association is likely to be more a function of personality than of actual conditions in the social environment. We found that neuroticism alone explained 69% of the variance in symptom measures, averaged over four waves of interviewing. It thereby dominated any aetiological equation (Henderson et al. 1981).

A personality attribute with particular attraction is the tendency to be dissatisfied. This has a long-established history of being strongly associated with neurotic and affective symptoms (Lord Taylor and Chave 1964; Kay et al. 1964; Krupinski 1979). At the Symposium, Weissman (1984) reported that in her latest New Haven survey, using the Diagnostic Interview Schedule, the case rate was 16.1% in those who were satisfied with their friendships, but 30.4% in those who were unsatisfied. Such an observation clearly has direct relevance to the measurement of social support, where subjective items are so commonly included. The two adequacy indices in the ISSI correlate 0.72, suggesting that both tap some common factor. At the informal workshop held after the Symposium, reference was made to the cognitive set which depressives carry into and out of episodes of illness. Such a set could appreciably influence responses to questions about personal networks and support. Brugha et al. (1982, 1984), in the course of trying to replicate the first Canberra study, reported that depressed out-patients had deficiencies in their personal networks which appeared to be long-standing, considerably antedating their symptoms.

The overlap between life events and the loss of social support is obvious, yet this has often been ignored in studies of the buffering effect of social support on adversity. Of particular concern is the strong representation of losses or exits from the personal network in life event inventories. Persons with high life-event exposure will tend also to have recent losses of social support. The two sets of variables could possibly be disaggregated by considering only life events which are wholly unrelated to the individual's field of social relationships.

To the present writer, the greatest challenge in the whole field is to have measures of the several components of social support, but to have these free of personality attributes. Such a requirement may be logically impossible, because social support is obtainable only through social relationships, which are themselves achieved only by having competence in establishing and maintaining them. Such competence is an attribute of the individual, not of his social environment. The individual is largely the author of his social environment and not just its passive recipient. Individuals who lack social support lack social relationships and this must often be due to personality defects. These in turn may also confer vulnerability to psychiatric disorder. Sarason and Sarason (1982) have provided a lucid analysis of this interface between the individual and support coming from the environment. When this interface is considered in depth, it seems likely that future studies of the aetiological hypothesis will fail because social support and personality are inextricably linked. The challenge for future studies must therefore be to refute the null hypothesis that the lack of social support, uncontaminated by the effect of illness and unconfounded by personality attributes, has neither a direct nor a buffering effect on the onset of mental disorders.

At the informal workshop, Professor Brian Cooper advocated going straight to intervention trials. This recommendation is to be strongly endorsed. Cooper also asked what usefully could be done with findings about personality, if these were indeed found to be more important than current social factors. That question remains unanswered. There may be regret at having to conclude that the aetiological hypothesis is not readily amenable to testing. But the intervention approach, focused on samples of high-risk individuals, would lead more directly to useful information for prevention. The evidence so far suggests that social support cannot easily be shown to contribute to the onset of psychiatric morbidity; but it may have an important effect on the course of some disorders. That is a separate hypothesis which is much more amenable to investigation.

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