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Assessment of Stress-Buffering Effects of Social Networks on Psychological Symptoms in an Inner-City Elderly Population[1]

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Social network researchers have been divided into two camps: those who propose that social networks have a direct effect on subsequent psychological symptoms and those who posit a stress-buffering effect as well. Previous research has been limited by rudimentary measures of social interaction and the absence of longitudinal data as well as by different approaches to the assessment of possible buffering effects. In the present study, using 19 social network variables, the authors followed 133 elderly residents of mid-Manhattan SRO hotels for 1 year. Three different methods of determining buffering effects were examined: (a) Dividing the sample into high- and low-stress groups and contrasting differences in percentage variance accounted for by social networks between the two groups; (b) Examining the group as a whole to assess if any Network Variable × Stress interactional terms are significant; (c) Examining the group as a whole to assess whether there is a reduction in the beta value of stress with respect to psychological symptoms when network variables are added to the analysis. Method 1 indicated a direct network effect, but none of the methods indicated a buffering effect. Of clinical relevance was the nonlinearity of the network effects, that is, depending upon a person's stressor level, different network dimensions must be emphasized and strengthened.

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The interrelationship between stress, social networks, and psychological health has received considerable attention in recent years (Dean & Lin, 1977; Gottlieb, 1981; Greenblatt, Becerra, & Serafetinides, 1982). An attractive feature of this relationship has been its potential clinical applicability. For example, are there particular dimensions of social networks that are especially helpful in promoting and maintaining psychological well-being even in times of stress? Conversely, are there social network features that render an individual more at risk? Identification of such patterns could greatly assist therapists in planning support system intervention strategies for high-risk individuals.

Although it is essential that efforts continue to be made to modify those factors that aggravate psychological symptoms and produce stress, as an immediate practical strategy it may be more expedient to intervene at the network system level. As Cassell (1976) suggested, "It would seem more immediately feasible to attempt to improve and strengthen social supports rather than reduce the exposure to the stressors" (p. 121).

Two hypotheses have been proposed that singly or in combination were thought to explain the effects of social network support on psychological symptoms. One hypothesis proposed that social networks have a direct effect on the level of symptoms. The second hypothesis posited a buffering explanation in which social networks exert their greatest effect on symptoms as the level of stressors increases. Social network researchers have generally belonged to one of two camps: those whose analyses imply that social networks exert only a direct effect on symptoms (Lin, Simeone, Ensel, & Kuo, 1979; Pinneau, 1976; Williams, Ware, & Donald, 1981), and those who assert that social networks exert a buffering effect as well (Cassell, 1976; Cobb, 1976; LaRocco, House, & French, 1980).

Williams et al. (1981) have observed that this lack of consensus regarding the role of social networks with respect to stress and outcome appears to be due in part to the failure of researchers to define clearly what is meant by buffering or modifying effects as contrasted with direct effects, and to the failure to conduct statistical tests for these effects. There have been three principal ways used to elicit buffering effects:

Method 1. Social networks are divided into high and low support, and buffering is said to exist if under high stress persons with high support exhibit significantly fewer symptoms than those with low support. The differences between the groups with high and low support under conditions of low stress should be minimal or absent (e.g., Gore, 1978; Lin et al., 1979; Nuckolls, Cassel, & Kaplan, 1972).

Method 2. Buffering is thought to exist if the interactional term "Network × Stress" in a regression analysis has a statistically significant effect in terms of explaining symptom level. If this term is not significant, but the

social network variables alone significantly explain symptom level variance, then an "additive" (direct) effect exists (e.g., LaRocco et al., 1980; Monroe, 1983; Sandler & Barrera, 1984; Williams et al., 1981).

Method 3. Buffering exists if the effect of stressors on symptoms significantly diminishes when the network variables are added to a regression analysis (e.g., Aneshensel & Frerichs, 1982; Eckenrode & Gore, 1981).

There have been two major categories of difficulty associated with prior studies: (a) failure to develop adequate operational definitions of "social networks" and associated constructs; and (b) reliance upon retrospective experimental designs. For example, Method 1 requires that the construct social network be essentially unidimensional, cast in terms of relative degree of social support. Such an assumption is based upon the value judgment that exchanges between individuals are ipso facto beneficial—that more is generally better (e.g., Weinberg & Marlowe, 1983)—whereas, in fact, social networks must be taken as considerably more broad constructs. While encompassing social supports, the construct social network includes a variety of forms of social interaction that are not necessarily beneficial (e.g., density, configuration). From this it follows that experimental and analytic designs cannot rest upon the attribution of high or low social support as the central measure of social networks. In related fashion, there has been a notable lack of precision in defining network dimensions (DiMatteo & Hays, 1981; Gottlieb, 1981; Mitchell & Trickett, 1980). No consistent position has been taken regarding the type of relationship (e.g., friends, kin), or the qualities of relationship (e.g., information, affective support, number of ties) that are important. Generally, investigators have selected one or two such variables, thus imposing a narrow framework which constricts the investigation of relationships between network and selected outcomes.

Reliance upon retrospective designs, in which both network and outcome variables have been measured simultaneously (Dean & Lin, 1977), has made it difficult, if not impossible, to identify causal relationships among variables. If research is to determine whether or not social networks influence psychological symptoms, then analyses must focus upon the relationship between network and symptoms having controlled for initial symptom levels; that is, longitudinal study is required in order to establish the causal sequence. This is particularly the case when dealing with outcome measures such as health and psychological well-being for which it is known that, all other things being equal, initial levels are the best predictors of levels at time of follow-up. Added to this basic conceptual issue are tactical issues relating to the unreliability of recall measures or the confounding by current psychological problems of social network reporting.

There are additional problems relating to prior studies, at what might be regarded as the tactical level (i.e., in analytic interpretations). For example, if the interactional term in Method 2 is significantly related to symptom level, this does not necessarily reflect buffering: It could indicate only that network effects are conditioned by increases in stress. The same reservation applies to findings obtained using Method 1, among high-stress subjects. Essentially, the "purest" definition of buffering is provided by Method 3. However, this has been attempted only rarely.

Turning to the question of the sample used in the current study, there is ample support in the literature for the contention that older adults living in single-room occupancy (SRO) hotels are excellent subjects for studying the relationships among social factors, disease, stress, and adaptation. Blazer (1982) enumerates three important reasons for using an elderly sample. First, by limiting the age range to that of older subjects, one can simultaneously control for the effect of aging and, to some extent, cohort effects. Second, certain social events which are both stressful and deplete the social environment are prevalent in late life. Third, the aged may be more susceptible to the deleterious effects of the social environment, for example, diminishment of perceptual and other adaptive capacities may render them especially susceptible to social influences.

Research has shown how important the social support system, albeit small, is for the aging SRO resident's survival in the community (Cohen & Sokolovky, 1980; Erickson & Eckert, 1977). More commonly than their more affluent age peers, SRO dwellers must depend on their social networks for material as well as emotional support (Cohen & Rajkowski, 1982). Although there are some loners among the SRO elderly, the notion that these hotels are constituted of isolates has been shown to be a myth (Cohen & Sokolovsky, 1980). In the present sample, the mean network size was 11.4 contacts, with only 1% of the sample reporting no contacts (Cohen, Teresi, & Holmes, 1985). Nine-tenths of all linkages involved two or more types of transactions (e.g., food exchange, advice giving, money lending). Moreover, these individuals were not averse to enlisting network members. For example, 76, 81, 92, and 93% indicated a willingness to contact others if they had problems with cash, food, illness, or required information, respectively.

Although Leavy's (1983) extensive review of the literature has shown that the generalizability of findings regarding social support must be tempered by differential effects of culture, economic status, age, and other demographic factors, the SRO hotels can still provide an excellent laboratory for studying the ability of social networks to cushion against adverse conditions. Moreover, learning how social networks help these individuals can suggest strategies for interventions with other high-risk elderly groups.

The aim of this paper is to address those deficiencies outlined above, using as subjects elderly SRO residents. Commonly used support variables have been replaced with four broad categories of social network dimensions, which comprise a total of 19 network variables. Longitudinal data are used so that the antecedent variables can be controlled adequately. Three analytic strategies are used to examine the various buffering potentials of social networks, paralleling the three methods presented earlier:

Analysis I: Test for Method 1 Type Buffering Effect. How do the network effects on psychological symptoms compare for individuals experiencing relatively fewer stressful life events as contrasted with experiencing higher levels of stressful events? Operationally, using regression procedures, are there any differences between the high- and low-stress groups in terms of the percentage variance of Time 2 psychological symptoms accounted for by the network variables?

Analysis II: Test for Method 2 Type Buffering Effect. For the group as a whole, are there certain social network dimensions that serve to buffer against untoward psychological symptoms caused by increased stress? Operationally, using regression analysis are there any Network Variable $\times$ Stress Interactional terms that are able to account for a significant percent of the explained variance of Time 2 psychological symptoms?

Analysis III: Test for Method 3 Type Buffering Effect. For the group as a whole, do the network variables reduce the effects of stress on psychological symptoms? Operationally, with Time 2 psychological symptoms as the dependent variable, is there a reduction in the standardized regression coefficient (β value) of stress when the network variables are added to the analysis?

METHODS

Sample

Utilizing a single-stage clustering technique (Van Dalen, 1973), interviews were conducted in 21 single-room occupancy (SRO) hotels in a midtown sector of Manhattan. Within each selected hotel an effort was made to interview all residents aged 60 and over on a roster furnished by the management. Vigorous efforts were made to recruit respondents by providing a $5 remuneration, initiating a series of letters and telephone calls, using flexible interview hours, and working with neighborhood agencies and hotel personnel. The initial sample (Time 1) consisted of 161 residents, representing 40% of the persons on the roster. The size of the sample we obtained reflected, in part, the reluctance of some hotel managers to provide complete rosters and to cooperate with the study. On 1-year follow-up

(Time 2), 133 individuals were reinterviewed. The final sample consisted of 58 men and 75 women; 93% were white and their mean age was 72.3 years.

In assessing the representativeness of the sample, it is interesting to note that our sample in many ways resembled that of a national census survey of 157,000 elderly SRO residents (Haley, Pearson, & Hull, 1981). There were no differences between the two samples with respect to age (72.3 years NYC, 72.8 National) and percentage having spent 5 years at their current address (53% NYC, 51% National), and there were only minimal differences in percentages having completed eighth grade (52% NYC, 42% National) and in median income ($3,900 NYC, $4,760 National). There were greater differences in sex (56% female NYC, 38% female National) and race (93% white NYC, 66% white National) that reflected the particular section of Manhattan that we studied.

Instruments

Physical and mental health and social functioning were determined by the Comprehensive Assessment and Referral Evaluation (CARE) developed by Gurland et al. (1977). Internal consistency reliability was high for most scales, with alpha coefficients ranging from .51 to .92. The interrater reliability for all scales ranged from 0.56 to 1.0.

Variables derived from the CARE that were used in the analysis were (a) *Psychological symptoms:* Sum of an individual's z scores on four mental health scales derived from the CARE (Depression, Organic Brain Syndrome, Alcoholism, Schizophrenia). (b) *Stress:* A 15-item scale based on categories derived from the PERI Life Events list (Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978). Events had to occur during the 1-year study period. Typical items included "deterioration in the neighborhood," "family illness," "mugging in past year," "personal injury due to crime," and "financial problems." Items that were redundant with social network variables were removed. Since stress is a skewed variable with persons farther from the mean experiencing more substantive levels of stress, and because it was necessary to have a sample size that permits valid conclusions to be drawn from the data, we defined "high stress" as being a mean score of 0.5 standard deviations above the mean.

Measures of social interaction were obtained from the Network Profile Analysis (NAP), which was developed in our previous work with inner-city populations (Sokolovsky & Cohen, 1981). The interrater reliability ranged from 0.83 to 0.92 on various subsections of the profile and was assessed by having each rater score taped interviews of another rater. The NAP comprised six fields on interaction: self–hotel contact, self–outside

nonkin, self-kin, self-hotel staff, self-agency staff, and self-social institution. Although the totality of the profile encompasses the respondent's overall activity field, the present analysis excluded the self-social institution field, which represented linkages to places (e.g., churches, stores) rather than individuals. Any meaningful personal encounters in these places were recorded within the appropriate field of interaction. Only those linkages with a minimal frequency of once every 3 months for hotel contacts and once a year for nonhotel contacts were included.

Numerous criteria have been employed to examine the multifaceted aspects of social networks (Knoke & Kuklinski, 1982). For use here, we have identified four dimensions of social interaction that comprise 19 variables. The four dimensions encompassed aspects of material and emotional exchange (interactional data set), quantitative and morphological features of the overall network (structural data set), characteristics of the network member vis-à-vis the respondent (member attribute data set), and those features of the environment that have been found to influence network formation and composition (environmental attribute data set).

Synchronic Network Variables

A. Interactional Data Set
   1. Numplex: mean number of transactions (e.g., food exchange, advice giving) per person in respondent's network
   2. Sustenance: mean number of persons providing basic support items to respondent (e.g., money, food, medical aid)
   3. Very important: total number of persons rated "very important" or "most important" by respondent
   4. Frequency: mean frequency of contact with persons in the network
   5. Directionality: direction in which aid in a dyadic relationship flows. Mean directionality was calculated for the respondent's hotel, nonkin, kin sectors
   6. Multiplex: total number of persons that have more than one type of transaction with respondent

B. Structural Data Set
   1. Size: total number of persons in respondent's network
   2. Density: ratio of actual linkages each person has with others in respondent's network, excluding linkages with respondent
   3. Degree: average number of linkages each person has with others in the respondent's network, excluding linkages with respondent

4. Number of clusters: number of subunits of network with 100% density
5. Large clusters: number of clusters with five or more members
6. Configuration: graphic measure of network interconnectivity (e.g., cluster, diffuse, mixed)

C. Member Attribute Data Set
1. Length of linkage: mean number of years persons have known the respondent
2. Geographic density: proportion of respondent's network members (informal or formal) in the hotel sector
3. Gender homogeneity: proportion of network members being of the same sex as the respondent
4. Age homogeneity: proportion of network members being 60 years or older

D. Environmental Attribute Data Set
1. Age density: dichotomous variable based on whether percentage of persons in hotel aged 60 and over is 20% or greater
2. Welfare hotel: dichotomous variable based on whether hotel is primarily a public assistance hotel or not
3. Hotel size: number of rooms in hotel

Factor analysis of the 19 network variables revealed that, in general, the network characteristics were statistically independent. Only seven of the variables loaded together (numplex, very important, directionality, number of clusters, size, multiplex, large clusters) with an alpha of .76. Although we developed a scale comprising these seven variables to reduce the number of independent variables (see below), we did not use the scale in the main analyses since it has not been cross-validated. Rather, we maintained the variables within the separate data sets in accord with theoretical conceptualizations described in the literature and discussed earlier.

Network Change Variables

A. Interactional Set: A representative number of interactional variables were created based on subtracting scores at Time 1 from those at Time 2. The following variables were used:
1. Numplex
2. Sustenance
3. Very important
4. Frequency
5. Directionality

B. Structural Change Set
   1. Number of contacts lost in past year
   2. Number of new contacts in past year
   3. Compensatory contacts: whether there was at least one new contact to replace lost contacts over past year

RESULTS

Consistent with previous work (Rabkin & Struening, 1976), there was a low but positive correlation between life events and subsequent psychological symptoms ($r = .10$). Further examination of the relationship between stress and symptomatology revealed that the mean z scores for intensity of psychological symptoms at Time 1 and Time 2 among those persons having experienced relatively higher levels of stressful events (HS) were greater than for those who had experienced relatively few stressful events (LS group). The higher the z score, the greater the number of symptoms (Table I). The differences in z scores between the two groups at Time 1 attained statistical significance. For both groups, symptoms at Time 1 were powerful predictors at Time 2, particularly for the HS group for which the percentage variance explained by Time 1 symptoms was twice that of the LS group (see Table II). Thus, individuals in the HS group manifested somewhat more severe and sustained evidence of psychological symptomatology.

For Analysis I, the synchronic network data sets (Time 1) and network change data sets were entered in the following respective orders: (a) Psychological Symptoms Time 1, Interactional Data Set, Structural Data Set, Member Attribute Data Set, Environmental Attribute Data Set; (b) Psychological Symptoms Time 1, Interactional Change Set, Structural Change Set.

Analysis I

For the LS group, after controlling for psychological symptoms at Time 1, both synchronic and change network variables were able to account

Table I. Z Scores of Psychological Symptoms by Group

Time assessed	High stress ($n = 33$)	Low stress ($n = 100$)
Time 1	.818	$-.309^b$
Time 2	.225	−.075

[a]Higher the z scores, the greater the symptoms.
[b]t test: $t = 2.46$, $df = 131$, $p < .02$.

Table II. Synchronic Variables: Increment in Explained Variance (R^2) of Psychological Symptoms at Time 2[a]

Variables	High stress (HS) (n = 33)	Low stress (LS) (n = 100)
Time 1		
Psychological Symptoms	.32	.15[c]
Interactional Data set	.10	.10
Structural Data set	.09	.11[b]
Member Attribute Data set	.11	.02
Environmental Attribute Data set	.01	.03
Total variance explained by network sets	.31	.26[b]

[a]Hierarchical Regression Analysis. F test contrasting total variance of psychological symptoms explained by synchronic network variables for HS and LS groups, p = .38.
[b]$p < .05$.
[c]$p < .01$.

for a significant percentage of the variance in psychological symptoms at Time 2 (see Tables II and III). Thus the variance of 26 and 20% explained by the synchronic and change variables, respectively, pointed to a substantial direct effect of social networks on subsequent psychological symptomatology.

Looking at the broader trend of the data sets as a whole proved to be more interesting than the specific differences among individual network variables. In examining the partial correlations, only one of the synchronic network variables, total network size, was significantly associated with symptomatology: Impoverished relationships are strongly related to psychological problems at Time 2. Two change variables, numplex and the number of very important linkages, attained statistical significance. A third

Table III. Change Variables: Increment in Explained Variance (R^2) of Psychological Symptoms at Time 2[a]

Variables	High stress (HS) (n = 33)	Low stress (LS) (n = 100)
Time 1		
Psychological Symptoms	.32	.15[b]
Time 2–Time 1		
Interactional Data set	.07	.17[b]
Structural Data Set	.17	.03
Total variance explained by network change sets	.24	.20[b]

[a]Hierarchical Regression Analysis. F test contrasting total variance of psychological symptoms explained by network change variables for HS and LS groups, p = .40.
[b]$p < .01$.

change variable, frequency of interaction, fell slightly short of significance. This meant that those individuals who showed a decrease in the frequency and in the mean number of transactions per linkage during the 1-year study period were apt to have more psychological difficulties. Moreover, those who had fewer subjectively important contacts over the year were likewise found to have more symptoms.

In order to test for the Method 1 type of buffering effect for social networks, we compared HS and LS groups at Time 2 in terms of the amount of explained variance in psychological symptoms that could be accounted for by the social network variables. Greater percentages in the HS group would suggest that a buffering effect exists.

After controlling for psychiatric symptoms at Time 1, the synchronic network variables of the HS group were able to account for a somewhat greater percentage of the variance in psychological symptoms scores at Time 2 than was the case among the LS group, although these differences were not statistically significant (Table II). The amount of variance explained by each of the data sets were comparable between HS and LS groups except for a somewhat higher percentage of variance accounted for by the Member Attribute Data Set among the HS Group.

In looking at the network change variables, we found minimal differences between the groups in the overall variance explained by networks (Table III). However, there were marked differences between groups in terms of which network data sets accounted for the variance which was explained. For the LS groups, interactional set was the important set, whereas for the HS group the structural set was determinant.

Once again, the variable sets as a whole yielded more fruitful data than did the individual variables. For the HS group, in examining the partial correlations only one change variable, number of contacts lost in the past year, attained statistical significance. Thus, increased symptoms was associated with a diminution in the number of linkages, regardless of levels of subjective importance.

Because of the low number of respondents ($n = 33$) in the HS group and the relatively large number of variables (20) entered into the regression equation involving the synchronic network variables, we performed a second regression analysis using the previously described scale comprising seven network variables together with the remaining network variables and the psychological symptoms at Time 1. Hence the number of variables entered into the equation was reduced to 14. The variance contributed by the synchronic network variables to explaining Psychological Symptoms at Time 2 was 22 and 18% for the HS and LS groups, respectively. Although there was some diminution in the percentages of explained variance from the original analysis, the data still confirmed the findings of the initial analyses shown in Tables II and III.

Analysis II

For both Method 2 and Method 3 analyses, Time 1 synchronic network variables and the change variables were entered in the same order as Analyses I; however, only those network variables which would contribute substantially to the explanation of variance (F values of 1.3 or greater) were included. Interaction terms were created by multiplying the network variables by the stress variable. The regression equation was:

Psychological Symptoms Time 2 = Stress Psychological Symptoms Time 1 + (each) network variable + (each)Network Variable × Stress

In order to test for a Method 2 type of buffering effect seven synchronic network variables were entered into the regression analysis (Table IV). None of the four partial correlations of the Network Variable × Stress interactional variables were significant; moreover, the tolerances of three of the interactional terms were too low to permit entry into the analysis. As a group, the four interactional variables accounted for only 1% of the variance of psychological symptoms.

Four network change variables were entered into the regression analysis (Table V). While the network change variables explained 16% of the variance after entering stress and psychological symptoms at Time 1, the stress interactional term contributed almost nothing to the explanation of

Table IV. Hierarchical Regression Analysis of Psychological Symptoms at Time 2 on Stress, Psychological Symptoms Time 1, Selected Synchronic Network Variables, and Stress × Network Variables[a]

Variables	Contribution to R^2	Unstandardized regression coefficient	Standardized regression coefficient
Stress	.01	.032	.162
Psychological Symptoms Time 1	.20	.417[b]	.437[b]
Frequency of contact	.02	.017	.086
Directionality	.02	−.013	−.031
Network size	.01	.000	−.146
Degree	.00	.000	.061
Number of clusters	.00	.020	.073
Number of large clusters	.00	−.011	−.020
Age homogeneity	.01	−.001	−.101
Stress × Frequency of contact	.00	.003	.052
Stress × Directionality	.01	−.015	−.221
Stress × Network size	.00	.002	.119
Stress × Degree	.00	.000	−.156

[a]Network variables were selected if their initial F values for analysis were 1.3 or greater. Total R^2 = .28.
[b]$p > .01$.

Table V. Hierarchical Regression Analysis of Psychological Symptoms at Time 2 on Stress, Psychological Symptoms Time 1, Selected Network Change Variables, and Stress × Change Variables[a]

Variables	Contribution to R^2	Unstandardized regression coefficient	Standardized regression coefficient
Stress	.01	−.003	−.014
Psychological Symptoms Time 1	.20	.378[b]	.396[b]
Numplex change	.05	−.067	−.203
Very important change	.05	−.027	−.214
Frequency of contact change	.02	−.041	−.189
Compensatory Contacts	.04	.084	.207
Numplex Change × Stress	.00	.000	.004

[a]Network change variables were selected if their initial F values for analysis were 1.3 or greater. Total $R^2 = .36$.
[b]$p < .01$.

variance. Thus, we must conclude that utilizing this method that the stress/network variable interaction did not indicate any buffering effect.

Analysis III

We next tested for a Method 3 type buffering effect by looking at changes in the standard regression coefficients (β) of the stress variable after the network variable were entered into the equation. A diminution in the β would suggest that a Method 3 buffering effect existed. When psychological symptoms at Time 2 was regressed on the stress variable the initial β was +.093. The addition of psychological symptoms at Time 1 reduced the β of stress to −.013, indicating that much of effect of stressful life events on subsequent psychological symptoms could be accounted for by the previous level of psychological symptoms. When the seven individual synchronic network variables in Analysis II were entered into the equation there was virtually no change in the β of stress (it increased by .001). Similarly, in a separate analysis, when the four individual network change variables in Analysis II were entered into the equation, there was no change in the β of stress (it decreased by .002). Hence, the data did not support a Method 3 type buffering effect.

DISCUSSION

Prefatory to the discussion, it is important to underscore that although the coping strategies used by hotel residents described here may be similar to those used by other at risk inner-city elderly, one must exercise

appropriate caution in generalizing the findings to other populations. Accordingly, we have concentrated our discussion on the data sets rather than on individual variables, since these larger sets may be more stable and thus have broader applicability. A second caveat concerns the relatively small size of the HS group. Although we corroborated our findings by performing a second analysis with a reduced number of variables in the analysis, it is likely that the results would have been attenuated had a larger sample been used. Finally, our method of calculating the change scores is problematic in that change measures are intrinsically unreliable and therefore less powerful in predicting outcomes.

It should also be underscored that from the outset our desire to include as many independent variables as possible was tempered by a consideration of statistical power (i.e., the probability of finding a true difference to be statistically significant). In regression analysis, power is inversely related to the number of independent variables, all other things being equal: the greater the number of independent variables the lower the power or, conversely, the greater the number of subjects required to achieve or maintain a given power level. Deciding on .05 as an acceptable significance level for hypothesis testing, common usage suggests that a power level of .80 be adopted. We anticipated finding an effect (R^2) of approximately .40 (Cohen et al., 1985). Given a subject N of 133, power computations suggest that as many as 15 independent variables could be included in the analysis. It turned out that additional analyses, involving smaller subgroups, were of interest. Admittedly, the inclusion of as many as 15 variables violates power considerations, that is, given only 33 subjects (used in one analysis with 14 independent variables) the desired power level of .80 would require an effect of not less than .50. On the other hand, the concept of power addresses only Type II (false-negative) statistical error; if a significant effect is found, power restrictions are moot. This being the case, we persevered in some analyses where the effect/variable mix was not completely justified.

A major issue addressed here concerns whether social networks exert a direct independent influence on psychological symptomatology. Most studies have been cross-sectional and have therefore been unable to control for the antecedent effects of the dependent variable (i.e., the previous level of a respondent's psychological symptoms). In fact very few longitudinal studies have adequately controlled for the dependent variable at Time 1 (Monroe, 1983). As our study confirmed, one of the best predictors of current psychopathology is past psychopathology. As a consequence of failing to control for the antecedent levels of the dependent variable, it had been difficult to determine whether those positive relationships between social networks and psychopathology were valid in that there may have been a

large degree of shared variance between the network variables and the prior level of psychological symptoms. Cause-and-effect relationships become especially difficult to determine.

In the present study, by controlling for psychological symptoms at Time 1 and employing a longitudinal design, we were able to demonstrate that social networks do indeed exert a direct independent influence on subsequent psychopathology. The variance explained by network variables alone at Time 1 was 26% for the LS group and 31% for the HS group. Also to be noted with respect to causality was the independent effects of the change variables on psychopathology. The variance explained by changes in various network dimensions between Time 1 and Time 2 was 20% for the LS group and 24% for the HS group.

A second issue that has remained unresolved has been whether networks exert only a direct effect on psychological outcome or whether they provide predominantly a buffering effect against symptoms. The results of this study support those theorists who have contended that social networks have only a direct, independent effect on psychological well-being and that they do not provide any additional buffering effect. All three methods to assess buffering failed to reveal any significant effects. These results contrast with an earlier study of this sample in which we examined the relationship between social networks and the SRO residents' ability to meet a variety of needs (Cohen et al., 1985). The dependent variable (need fulfillment) comprised predominantly material items such as finance, transportation, physical self-maintenance, recreation, and the like. In this case, a buffering effect seemingly existed in that differences in the explained variance in need fulfillment between the HS and LS groups was 25% for the synchronic network variables and 14% for the change variables. This suggests that for the population studied here, under stressful circumstances, the social networks are especially effective in helping to meet the more material requirements of daily living. This is not to negate the networks' influence on emotional well-being but to highlight the fact that the additional benefits they provide during stressful conditions seems to be predominantly in the material sphere. Of course, it may also be true that network effects on emotional well-being do not manifest themselves within a 1-year period.

To propose that there are only minimal differences between the effects of networks during stressful and nonstressful periods is to fall victim to a linear conceptual framework of viewing social networks. Above, we suggested that the social support model views exchanges between individuals as beneficial to the recipient, that is, more is generally better. The social network concept employed here is a broader category which while encompassing social support, includes a variety of measures of social interaction that do not necessarily imply benefits to the recipient. Our findings dramatically

underscored the nonlinearity of the network concept. This was especially evident with respect to the change variables. In the LS group, the interactional variables accounted for virtually all of the explained variance in psychological symptoms, whereas among the HS group the structural variables were paramount. Thus, increases in stress are not accompanied by a simple accretion of additional support but by an alteration in the relative importance of different aspects of social relationships. The data lend support to a qualitative disjunction between high- and low-stress groups. For those undergoing low stress and who have generally lower levels of psychological symptoms, interactional features of their networks (e.g., subjective importance, number of transactions, frequency of interaction) enhance well-being. On the other hand, for those who already have somewhat higher levels of psychopathology and who then experience high levels of stress, structural network features, especially loss of network contacts, irrespective of subjective importance, can result in further diminution in psychological well-being.

It would be especially useful to know if the network patterns found among those with fewer symptoms in the HS group represented a network response to moving from a low-stress situation to a high-stress one, or whether it was merely serendipitous for the individual to have a health-promoting network pattern. Unfortunately, since we had no antecedent variable for life events, we could not answer this question adequately.

These data indicate that clinicians working with network systems face a more complex challenge than was supposed previously. Depending on their client's stress level, different network dimensions must be emphasized and strengthened. For the population examined here, structural dimensions assume greater importance for those undergoing greater stress. Although different dimensions may assume importance in other populations, it is likely that disparities in the relative values of particular network dimensions depending upon stress level will be generalizable across samples.

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