Social Support and Treatment Response in Older Depressed Primary Care Patients

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A previously tested theoretical model that specifies relationships among depression, activities of daily living (ADL) impairment, and social support components was validated with 307 patients aged 60 and older from a multisite effectiveness trial of 3 treatments (antidepressant, placebo, Problem-Solving Treatment) for dysthymia or minor depression in primary care. Participants completed interviews and self-reports at baseline and at 6 and 12 weeks. The short-term, longitudinal data were analyzed with covariance structure modeling techniques. Consistent with the previous model, impairment in ADLs was associated with subsequent increases in depression, a larger emotionally close network that made frequent visits was associated with subsequent increases in perceived support, and perceived support was associated with subsequent decreases in depression. This last effect was significant only among participants randomly assigned to receive placebo with clinical management. The similar results in 2 different studies are a substantial validation of the theoretical model. The effect of perceived support primarily in the placebo group suggests that those with greater perceived social support and subsyndromal depression may be more likely to have a positive response to nonspecific clinical treatment components.

ANY researchers have identified an association between social support and depression in older persons (Bowling & Browne, 1991; Henderson, Byrne, & Duncan-Jones, 1981; Krause, Liang, & Yatomi, 1989; Matt & Dean, 1993; Oxman, Berkman, Kasl, Freeman, & Barrett, 1992). In addition, social support is frequently associated with less impairment in activities of daily living (ADLs) in elderly persons (Cummings et al., 1988; King, Reis, Porter, & Norsen, 1993; Wilcox, Kasl, & Berkman, 1994). The interrelation of social support, ADLs, and depression is particularly relevant in older medical patients because of the greater likelihood of functional impairment (difficulty in performing ADLs; Oxman et al., 1992; Oxman, Barrett, Freeman, & Manheimer, 1994; Schulz & Decker, 1985; Siegal, Calsyn, & Cuddihee, 1987; Turner & Noh, 1988).

Notwithstanding these important lines of research, few studies have simultaneously examined the relationship of social support, depression, and ADLs. ADL impairment is consistently a more powerful predictor of depression in elderly persons than is social support (Bowling & Farquhar, 1991; Oxman et al., 1992). In addition, ADL impairment as a consequence of illness is often associated with changes in social support (Bloom & Kessler, 1994; Stoller & Pugliesi, 1991; Wilcox et al., 1994). Unless depression, social support, and ADL impairment are analyzed simultaneously and longitudinally, researchers cannot determine the extent to which their interrelations are independent and which domain warrants priority for targeting interventions. This is particularly relevant for the primary care physician faced with a depressed patient. The primary care physician is constantly facing competing demands that limit his or her time and the health care system's resources to simultaneously address all three domains (Klinkman, 1997; Rost et al., 2000).

If treated for depression, the majority of older patients with depression and functional impairment are likely to be treated by primary care physicians (Regier et al., 1993). There is substantial evidence that patients with major depression will respond to pharmacological or psychotherapeutic treatment (Gloaguen, Cottraux, Cucherat, & Blackburn, 1998; Reynolds, 1997; Schneider & Olin, 1995; Schulberg, Katon, Simon, & Rush, 1998). However, in older primary care patients, subsyndromal depressions, such as minor depression or dysthymia, are the most common types (Barrett, Barrett, Oxman, & Gerber, 1988; Blazer, Hughes, & George, 1987; Johnson, Weissman, & Klerman, 1992; Olfson et al., 1996), and the effectiveness of treatments for these other depressive types is less certain (Coyne, Klinkman, Gallo, & Schwenk, 1997; Katon et al., 1995; Kendrik, 1996; Oxman, 1997; Public Health Service Agency for Health Care Policy and Research, 1993). Spontaneous remission without pharmacotherapy or psychotherapy (Malt, Robak, Madsbu, Bakke, & Loeb, 1999; Rapp, Parisi, & Wallace, 1991) and response to clinical management alone (Miller, Frank, & Reynolds, 1999) may be higher in minor depression than in major depression. Social support may play a significant role in promoting such responses.

Because of the uncertainty of effective treatments for these common conditions in primary care, we have previously evaluated two primary-care-based-treatments in an 11-week multicenter randomized trial (Barrett, Williams, Oxman, Katon, Frank, Hegel, et al., 1999) comparing placebo, the antidepressant paroxetine, and Problem-Solving Treatment for Primary Care (PST-PC; Mynors-Wallis, 1996), a cognitive behavioral therapy for primary care. All three treatment groups received clinical management (Miller et al., 1999). In the overall results for the study (Barrett, Williams, Oxman, Katon, Frank, Cornell, et al., 1999), paroxetine showed a small but significant treatment effect compared to placebo but not to PST-PC. PST-PC showed a faster response than placebo but not a significantly better response at the end of the trial. The relatively small differences suggest that clinical management, regardless of additional depression-specific treatment, appears to be a reasonably effective treatment for many patients. Primary care physicians have long been using "watchful waiting" as a treatment approach for mild depression. Clinical management in this study was more intense than the watchful waiting of usual care, but it is less costly than a commitment to antidepressants or psychotherapy. An enhanced watchful waiting by primary care physicians, along with predictors of patients who are not likely to respond to clinical management alone, seems indicated. Social support may be one such important predictor.

Given the number of studies examining the relationships between social support and depression in elderly persons or social support and ADLs, we have previously described a theoretical framework that emerges from this literature and propose that it applies to older medical patients. Research supporting this model in a short-term (7-month) longitudinal study of older adults undergoing heart surgery is reported in Oxman and Hull (1997). In brief, our framework was organized around three sets of relationships.

- 1. Components of social support. Social support is multidimensional (Barrera, 1986; Heller, Swindle, & Dunsenbury, et al., 1986; Oxman & Berkman, 1990; Schaefer, Coyne, & Lazarus, 1981; Seeman & Berkman, 1988; Tardy, 1988). Three major components of social support are (a) the network of support providers, (b) the type and amount of support provided through that network, and (c) the adequacy of that support (George, 1989; Oxman & Berkman, 1990). The components of social support are interrelated. With respect to depression, the most important interrelationship is that types and frequency of network contacts (Kessler, Kendler, Heath, Neale, & Eaves, 1994; Oxman et al., 1992) are strongly associated with the perceived adequacy of support. Persons who have more frequent contact with network members to whom they feel emotionally close perceive their support as being more adequate (Oxman & Hull, 1997).
- 2. Social support and depression. One of the consequences of a severe mood disorder can be social withdrawal and a more negative perception of social support. The support may be more adequate than severely depressed persons perceive it to be. However, severely depressed persons may be difficult to be around and may indeed drive their support away. In other words, their perception of inadequate support may be accurate. In contrast, participants with less severe levels of depression are more likely to have diminished social support precede the onset of depressive symptoms (Alloway & Bebbington, 1987; Brown, Bifulco, & Harris, 1987; Brugha et al., 1982). Because we have been examining participants with the less severe diagnoses of minor depression or dysthymia, our framework included the hypothesis of a unidirectional relationship from social support components to depression. The greater the number of emotionally close network members making frequent contact or the greater the perceived adequacy of support, the less severe will be the level of depressive symptoms.

3. Social support and ADL impairment. The association of social support with ADL impairment may diminish or change direction unless new events occur (Fontana, Kerns, Rosenberg, & Colonese, 1989; Glass & Maddox, 1992; Marottoli, Berkman, & Cooney, 1992). Because we have been examining older patients with a mixture of chronic and acute conditions, we hypothesized overall, direct, unidirectional relationships from perceived support and network size to ADLs. The greater the perceived adequacy of support, the lower will be the level of impairment in ADLs. In turn, the lower the level of ADL impairment, the lower will be the level of depression. Social support thus can have both direct and indirect effects to reduce depression severity.

In this study, we tested the generalizability of these relationships by evaluating the relationship of social support to depression in older patients undergoing treatment for dysthymia or minor depression.

METHODS

Participants

The details of the study design have been described in detail elsewhere and are summarized here (Barrett, Williams, Oxman, Katon, Frank, Hegel, et al., 1999). Primary care patients aged more than 60 were recruited for a comparative treatment trial from patients currently enrolled in primary care practices (family medicine or general internal medicine) in four communities (Lebanon, NH, Pittsburgh, PA, San Antonio, TX, and Seattle, WA). The four participating centers were chosen for geographic diversity and diversity of clinical populations. Patients aged 60 and older were recruited through referral and screening at community, Veterans Affairs, and academic-affiliated primary care clinics. To be included, a participant needed to have had three or four of the DSM-IV symptoms of depression for at least 1 month, one of which was depressed mood or anhedonia as assessed by clinical interview, and to have a 17-item Hamilton Rating Scale for Depression HAM-D; (Hamilton, 1960) score of 10 or greater. Patients with dysthymia were also required to have had symptoms for at least 2 years. Depression diagnoses were made by a research psychiatrist or psychologist using the PRIME-MD, a diagnostic instrument designed for use in primary care (Spitzer et al., 1994). Patients were excluded if, within the past 6 months, they had had major depression, active substance abuse, uncomplicated bereavement, parasuicidal behavior, or antisocial personality. Patients currently taking psychotropic drugs or seeing a psychotherapist were also excluded because the study was a treatment trial. Patients with cognitive impairment (a Mini-Mental State Examination score of less than 23; Folstein, Folstein, & McHugh, 1975) or a terminal illness (less than 6 months to live) were also excluded.

Design

We randomly assigned patients giving consent to placebo, paroxetine, or PST-PC using a computer-generated random allocation table. Randomization was blocked and stratified by site and diagnosis. Treatment assignments were held by a pharmacist and were available to study personnel only in the event of emergent medical need.

For the overall study, 415 patients met eligibility criteria and were randomized to placebo (n = 140), paroxetine (n = 137), or PST-PC (n = 138). Patient characteristics were distributed equally among groups (mean age 71, 59% male, 78% White). Diagnoses were dysthymia (n = 211) or minor depression (n = 204). Participants were included in the analyses for this study only if they completed the 11-week trial and had complete data for baseline, mid-treatment (6-week), and end of treatment (11-week) interviews. The method of analysis we used—structural equation modeling—was designed to deal with complete data only (Bentler & Chou, 1988, p. 180).

These restrictions resulted in a sample of 307 patients randomized to placebo (n = 110), paroxetine (n = 93), or PST-PC (n = 104). The mean age was 71; 61% of participants were male and 76% were White. Diagnosis sample sizes were 161 persons with dysthymia and 146 persons with minor depression.

Procedures

At each site, we used a variety of methods to educate participating primary care providers about referral. We also used brief depression screening instruments at some sites to bring potential patients to the attention of their provider for referral. Participants thus were identified in the primary care practice setting as potentially having either dysthymia or minor depression and were referred for a research evaluation. A two-phase evaluation took place within 1 week of identification. The initial phase was a semi-structured clinical interview to determine eligibility. For patients meeting criteria, a complete description of the study was provided and written informed consent obtained. Those who agreed to participate were then administered additional measures, including social support, and randomized to one of the three treatment arms. Participants in all three arms were offered six subsequent treatment visits at 1, 2, 4, 6, 8, and 10 (for PST-PC) or 11 (for paroxetine and placebo) weeks.

Measures

The semi-structured interview included mood, anxiety, and alcohol modules from the Prime-MD (Spitzer et al., 1994), the 17-item HAM-D (Hamilton, 1960), and portions of the Structured Clinical Interview for Diagnosis (SCID; Spitzer, Williams, Gibbon, & First, 1992).

To assess depression severity, we used the interviewbased 17-item HAM-D (Hamilton, 1960), ranging from 0 to 53 for the single-rater version used in this study. The HAM-D has shown reliability and validity in measuring depression in elderly patients (McDowell & Newell, 1987; Oxman, Barrett, et al., 1994).

We used the interview-administered Sickness Impact Profile (SIP) (Bergner, 1978) to measure ADLs. The SIP measures functional impairments from illness and consists of 136 statements in 12 categories. All items concentrate on changes in performance. Participants are instructed to respond yes to a statement only if they are sure it describes them at the time of questioning and is related to health status. Category scores are calculated by adding predetermined relative scale values for each item responded yes within the category, dividing by the maximum possible score for that category, and then multiplying by 100. The potential range for each category is 0 to 100. Three categories—ambulation, mobility, and body care and movement—are summed to form the physical dimension, a basic ADL measure. In this study we used only the physical dimension score because the full SIP score contains items that overlap with social support and depression. Although the majority of the work on reliability and validity has been done with the total score, in other studies (e.g., Ott et al., 1983; Oxman, Freeman, Manheimer, & Stukel, 1994) reliability of the physical dimension score of the SIP has been sufficiently high.

To understand better how social support relates to improved outcomes, several investigators have described multidimensional models of social support (Barrera, 1986; Heller et al., 1986; Oxman & Berkman, 1990; Schaefer et al., 1981; Seeman & Berkman, 1988; Tardy, 1988). Three major components of social support are (a) the network of support providers, (b) the type and amount of support provided through that network, and (c) the adequacy of that support (George, 1989; Oxman & Berkman, 1990). In previous work we found that the dimension of the type and amount of support was not relevant to depressive symptoms. Accordingly, because this was an effectiveness trial with a limited number of measures, in this project we included only the dimensions of the network and the adequacy of support. To assess social networks we used the interview-based Social Network Questionnaire from the New Haven site of the Established Populations for the Epidemiologic Study of the Elderly (Seeman & Berkman, 1988). As a summary measure we used the total number of emotionally close network members (children, relatives, friends) who were seen regularly, that is, at least once per month. To assess perceived adequacy of support we used the Multidimensional Scale of Perceived Social Support (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988). The MSPSS is a 12-item self-report measure with a 7-point scale from "very strongly disagree" to "very strongly agree." Item scores are averaged for a total ranging from 1 to 7. The MSPSS provides a summary score as well as three subtype scores for perceived adequacy of support from a significant other, family, and friends. Internal and test-retest reliability are high, and validity has been demonstrated in older medical patients (Hann, Oxman, Ahles, Furstenberg, & Stukel 1995; Oxman, Freeman, et al., 1994).

Analysis

Hypotheses.—The following groups of hypotheses were incorporated into a single structural equation model that we tested using data obtained from older patients in the treatment trial of dysthymia or minor depression. The perceived adequacy of social support at 6 weeks, after three treatment visits, is a positive function of the number of emotionally close network members seen regularly before the start of treatment. A similar relationship exists between the 6-week assessment and the end of treatment assessment at 11 weeks. The theoretical framework we are testing includes a unidirectional path from social support to depression. We

hypothesized that depressive symptoms at the 6-week assessment are a negative function of the perceived adequacy of support pretreatment and the number of network members seen regularly pretreatment, and a positive function of the amount of impairment in pretreatment ADLs. Similar relationships exist between predictors assessed at the 6-week assessment and depression at the 11-week assessment. Impairment in ADLs at the 6-week assessment is a negative function of the perceived adequacy of support and the number of network members seen regularly pretreatment. Similar relationships exist between the 6-week assessment and the 11-week assessment.

Covariance structure models.—We conducted all covariance structure models using the statistical program EOS (Bentler, 1989) applied to the covariance matrix of the observed variables for participants without missing data. Whereas usual test statistics are appropriate when listwise deletion is used for missing data, pairwise deletion or substituting estimates for missing values raises questions regarding the appropriateness of standard error estimates and goodness of fit statistics (Bollen, 1989). To test the adequacy of our theorized model, we used the recommended (e.g., Anderson & Gerbing, 1988) procedure of specifying nested models: (a) a rudimentary base model and (b) a theorized model that includes all of the paths in the base model plus a set of additional paths that were theorized a priori. To demonstrate the utility of one's theorized model, one must show that (a) it fits significantly better than the base model and (b) the theorized paths that distinguish it from the base model are statistically significant on an individual basis.

RESULTS

The mean HAM-D scores pretreatment and after 6 weeks of treatment were 13.5 (SD 2.65), range 10-22, and 8.96 (SD 4.95), range 0-25, respectively. The mean HAM-D score at 11 weeks was 7.25 (SD 4.92), range 0-27. Mean SIP physical dimension pretreatment was 12.1 (SD 12.7), range 0-68.0. The most common medical conditions were hyperlipidemia (29.3%), arthritis (25.4%), diabetes mellitus (19.2%), benign prostatic hypertrophy (18.9%), and hypertension (17.9%). Ninety-eight percent of participants had at least one comorbid medical diagnosis, and 46.3% had five or more diagnoses. Mean SIP physical dimension score after 6 weeks of clinical management was 11.5 (SD 12.7) and at 11 weeks was 10.4 (SD 12.3), range 0-80.1. Tables 1-3 display the means, standard deviations, and correlation matrix for each treatment group (paroxetine, PST-PC, placebo) across the three time points.

An initial model was specified in which all exogenous variables were allowed to intercorrelate and only autoregressive structural effects were included (Figure 1). A multigroup analysis was conducted in which this model was simultaneously applied to all three treatment groups (placebo, paroxetine, PST-PC) with the depicted paths, the variances of the exogenous variables, and the errors for the endogenous variables constrained to be equal across groups. This model provided a reasonable fit to the data, χ^2 (204, n = 307) = 386.75, CFI = .89, NFI = .80, NNFI = .90.

We developed an initial theoretical model by adding the

paths depicted in Figure 2 to the paths included in the base model. Once again, a multigroup analysis was conducted in which this model was simultaneously applied to all three treatment groups. In addition to the constraints applied to the base model, all paths depicted in Figure 2 were constrained to be equal across groups. This model also provided a reasonable fit to the data, χ^2 (192, n = 307) = 365.18, CFI = .91, NFI = .82, NNFI = .90, and a significantly better fit than the base model, difference χ^2 (12, n = 307) = 21.57, p < .05. Conceptually, this improvement in the fit of the model is equivalent to an omnibus demonstration that the theorized short-term, longitudinal relationships (depicted in Figure 2) have predictive validity over and above the cross-sectional correlation of the variables at Time 0 and their longitudinal stability over Time 1 and 2 (depicted in Figure 1).

As an explicit test of the hypothesis that the theorized associations varied as a function of treatment, a set of models was specified in which the equality constraints specified in the initial theoretical model were systematically lifted. The fit of each resultant model was then compared against the fully constrained, initial theoretical model. In all, six additional models were specified. These models differed in terms of the paths they allowed to vary across groups. Conceptually, each of these models represents a test of a specific interaction or moderation in the strength of a relationship between variables as a consequence of treatment group. If a particular model fits better than the initial theoretical model, then the paths being tested vary in strength across groups.

Among the six models, Model A was of greatest interest insofar as it allowed the perceived support to depression paths to vary across treatment groups. Model B allowed the ADL impairment to depression paths to vary across groups. Model C allowed the perceived support to ADL impairment paths to vary across groups. Model D allowed the network size to perceived support paths to vary across groups. Model E allowed the network size to depression paths to vary across groups. Finally, Model F allowed the network size to ADL impairment paths to vary across groups. In all cases, constraints were released for both the Time 0 to Time 1 and Time 1 to Time 2 paths. Of these six models, only Model A fit better than the fully constrained initial theoretical model, difference χ^2 (4, n = 307) = 9.85, p < .05. This finding supports the hypothesis that the association of perceived social support with subsequent depression varies as a function of treatment group. Inspection of the effects within groups revealed that perceived support at Time 0 is predictive of decreased depression at Time 1 and perceived support at Time 1 is predictive of decreased depression at Time 2 in the placebo group (standardized path coefficients = -.18, z = -2.03, p < .05, and -.22, z = -2.79, p < .01, respectively), but not in the antidepressant group (standardized path coefficients = -.02, z = -.22, ns, and .11, z = 1.23, ns, respectively) or the PST-PC group (standardized path coefficients = -.02, z = -.25, *ns*, and -.09, z = -1.09, ns, respectively).

Values for the standardized coefficients in Model A are depicted in Figure 3. In order to reduce clutter, we do not depict values for the base paths shown in Figure 1, values

	Time 0					Time	: 1		Time 2			
Variables	1	2	3	4	5	6	7	8	9	10	11	12
Time 0, baseline												
1. Perceived adequacy of support	1.00	0.28*	-0.03	-0.04	0.66****	0.36***	-0.05	-0.01	0.71****	0.33***	-0.09	-0.01
 No. of close network members seen ≥1/month 		1.00	-0.09	0.07	0.30**	0.60****	-0.17	0.05	0.18	0.47****	-0.01	0.13
3. HAM-D			1.00	0.19	-0.01	-0.01	0.21*	0.09	-0.05	0.05	0.02	0.07
4. SIP physical dimension				1.00	0.01	0.09	0.31*	0.82****	0.08	0.35***	0.43****	0.86****
Time 1, 6 weeks of treatment												
5. Perceived adequacy of support					1.00	0.41****	-0.07	-0.01	0.72****	0.34***	0.10	0.05
 No. of close network members seen ≥1/month 						1.00	-0.07	0.09	0.45****	0.70****	0.02	0.18
7. HAM-D							1.00	0.38****	-0.06	0.05	0.46****	0.31**
8. SIP physical dimension								1.00	0.04	0.37***	0.40****	0.88****
Time 2, 11 weeks of treatment												
9. Perceived adequacy of support									1.00	0.40****	0.09	0.07
10. No. of close network members seen ≥1/month										1.00	0.16	0.42****
11. HAM-D											1.00	0.48****
12. SIP physical dimension												1.00
Μ	4.69	4.24	14.0	13.2	5.02	4.68	8.27	13.18	5.00	4.60	6.16	11.18
SD	1.14	3.36	2.88	13.9	1.21	3.13	4.52	14.86	1.20	3.57	4.23	13.21

Table 1. Means, Standard Deviations, and Correlations of Social Support, Depression, and ADL Variables for Paroxetine Group (n = 93)

Notes: ADL = activities of daily living; HAM-D = Hamilton Rating Scale for Depression; SIP = Sickness Impact Profile.

 ${}^{*}p \leq .05; \, {}^{**}p \leq .01; \, {}^{***}p \leq .001; \, {}^{****}p \leq .0001.$

for the network size to ADL impairment paths (Time 0 to Time 1 standardized path coefficient = -.00, z = -.01, ns; Time 1 to Time 2 standardized path coefficient = .04, z = 1.31, ns), and values for the perceived support to ADL impairment paths (Time 0 to Time 1 standardized path coefficient = -.01, z = -.44, ns; Time 1 to Time 2 standardized path coefficient = 2.00, z = -.10, ns), although these values were included in the specification of the model. Finally, the

three values for the perceived support to depression paths varied depending on treatment group and reflected those observed in the placebo, antidepressant, and PST-PC groups, respectively.

The basic conclusions to be drawn from the effects depicted in Figure 3 are (a) ADL impairment was associated with subsequent increases in depression, (b) larger network size was associated with subsequent increases in perceived

Table 2. Means, Standard Deviations, and Correlations of Social Support,	Depression, and ADL Variables for PST	-PC Group $(n = 104)$
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	Time 0				Time 1				Time 2			
Variables	1	2	3	4	5	6	7	8	9	10	11	12
Time 0, baseline												
 Perceived adequacy of support 	1.00	0.36****	-0.02	-0.11	0.69****	0.31***	-0.07	-0.10	0.66****	0.26**	-0.12	-0.08
2. No. of close network members seen ≥1/month		1.00	-0.16	-0.05	0.36***	0.48****	-0.09	-0.06	0.36***	0.55****	-0.18	-0.05
3. HAM-D			1.00	0.34***	-0.01	0.03	0.42****	0.32***	-0.02	-0.04	0.28**	0.36***
4. SIP physical dimension				1.00	-0.22*	-0.03	0.25**	0.80****	0.19	-0.13	0.21*	0.81****
Time 1, 6 weeks of treatment												
 Perceived adequacy of support 					1.00	0.38****	-0.12	-0.18	0.72****	0.33***	-0.16	-0.09
6. No. of close network members seen ≥1/month						1.00	-0.20*	0.04	0.39****	0.54****	-0.12	0.04
7. HAM-D							1.00	0.34***	-0.21*	-0.26^{**}	0.57****	0.39****
8. SIP physical dimension								1.00	-0.14	-0.05	0.23*	0.85****
Time 2, 11 weeks of treatment												
 Perceived adequacy of support 									1.00	0.46****	-0.24**	-0.09
10. No. of close network members seen ≥1/month										1.00	-0.13	-0.03
11. HAM-D											1.00	0.40****
12. SIP physical dimension												1.00
Μ	4.51	4.66	13.4	11.5	4.62	4.64	9.56	11.1	4.68	4.93	7.60	10.1
SD	1.16	4.79	2.54	12.0	1.18	4.13	5.28	11.8	1.26	4.02	5.36	12.0

Notes: ADL = activities of daily living; PST-PC = Problem-Solving Treatment for Primary Care; HAM-D = Hamilton Rating Scale for Depression; SIP = Sickness Impact Profile.

 $p \le .05; **p \le .01; ***p \le .001; ****p \le .0001.$

Table 3. Means, Standard Deviations, and Correlations of Social Support, Depression, and ADL Variables for Placebo Group (n = 110)

			Time 0			Tir	ne 1					
Variables	1	2	3	4	5	6	7	8	9	10	11	12
Time 0, baseline												
 Perceived adequacy of support 	1.00	0.18*	-0.01	-0.02	0.70****	0.34***	-0.20*	-0.01	0.67****	0.34***	-0.23**	-0.06
2. No. of close network members seen ≥1/month		1.00	0.18*	-0.12	0.18	0.42****	-0.11	-0.10	0.13	0.40****	-0.14	-0.09
3. HAM-D			1.00	0.22*	-0.19*	0.08	0.45****	0.28**	-0.08	0.03	0.11	0.20*
4. SIP physical dimension				1.00	0.01	-0.11	0.07	0.85****	-0.07	-0.02	0.18*	0.79****
Time 1, 6 weeks of treatment												
 Perceived adequacy of support 					1.00	0.34****	-0.30**	-0.07	0.74****	0.30**	-0.35***	-0.12
 No. of close network members seen ≥1/month 						1.00	-0.17	-0.18	0.27**	0.73****	-0.14	-0.12
7. HAM-D							1.00	0.19*	-0.23*	-0.23*	0.50****	0.10
8. SIP physical dimension								1.00	-0.07	0.05	0.22*	0.83****
Time 2, 11 weeks of treatment												
 Perceived adequacy of support 									1.00	0.29**	-0.35****	-0.17
10. No. of close network members seen ≥1/month										1.00	-0.10	0.03
11. HAM-D											1.00	0.23*
12. SIP physical dimension												1.00
М	4.75	5.05	13.3	11.6	4.80	5.16	8.99	10.5	4.88	5.06	7.85	9.96
SD	1.09	4.61	2.51	12.3	1.15	4.19	4.94	11.5	1.21	4.39	4.91	11.8

Notes: ADL = activities of daily living; HAM-D = Hamilton Rating Scale for Depression; SIP = Sickness Impact Profile. $p \le .05; p \le .01; p \le .01; p \le .001; p \le .0001.$

social support, and (c) perceived support was associated with subsequent decreases in depression, although this last effect varied as a function of treatment group in that it was only the case among participants randomly assigned to receive a placebo drug.

a function of diagnosis. A multigroup analysis was con-

Subsidiary Analyses

Qualification of model by diagnosis.—We performed a set of analyses to determine if the fit of the model varied as





Figure 1. Base structural model for relationships among depression, ADL impairment, and social support components.







Figure 3. Trimmed structural model for relationships among depression, ADL impairment, and social support components. Numbers are standardized coefficients. The three values for perceived support are those observed in the placebo, antidepressant, and PST-PC groups, respectively. All parameters associated with the base model were estimated but for clarity are not depicted. Similarly, the nonsignificant coefficients for network size to ADL impairment and for perceived support to ADL impairment are not shown. *p < .10; **p < .05.

models to test the hypothesis that the paths of principal theoretical interest (i.e., those that appear in Figure 2) varied as a function of diagnosis. These six models parallel in form Models A-F described previously. None of these models resulted in an improvement in fit over the initial theoretical model. Inspection of the univariate modification statistics (Lagrange Multiplier Tests) revealed that we could improve the model by relaxing several constraints on equivalent stabilities and exogenous correlations across diagnosis (i.e., the paths that appear in Figure 1) and equivalent exogenous variances and endogenous errors. However, univariate modification statistics revealed that none of the equality constraints of theoretical interest (i.e., those related to paths that appear in Figure 2) significantly degraded the model. In terms of the substantive interests of the present research, it can be concluded that the fit of the model did not vary as a function of diagnosis group.

Qualification of model by site.—We performed a set of analyses to determine if the fit of the model varied as a function of data collection site. A multigroup analysis was conducted in which the initial theoretical model was simultaneously applied to data from each of the four sites (Lebanon, Pittsburgh, San Antonio, Seattle) with all paths, exogenous variances, and endogenous errors constrained to be equal across sites. This model provided a poor fit to the data, χ^2 (270, n = 307) = 731.05, CFI = .73, NFI = .63, NNFI = .73. As in the treatment group analyses, we specified six additional models to test the hypothesis that the paths of principal theoretical interest (i.e., those that appear in Figure 2) varied as a function of location. These six models parallel in form Models A-F described previously. None of these models resulted in an improvement in fit over the initial theoretical model. Inspection of the univariate modification statistics (Lagrange Multiplier Tests) revealed that we could improve the model by relaxing several constraints on equivalent stabilities and correlations across sites (i.e., the paths that appear in Figure 1) and equivalent exogenous variances and endogenous errors. Indeed, simply relaxing the constraints on equivalent variances and errors (while maintaining the constraint that all paths that appear in Figures 1 and 2 be equal across groups) yielded a model with a reasonable fit to the data, χ^2 (234, n = 307) = 430.24, CFI = .88, NFI = .78, NNFI = .87. However, for both this model and the previous model, univariate modification statistics revealed that none of the equality constraints of theoretical interest (i.e., those related to paths that appear in Figure 2) significantly degraded the model. In terms of the substantive interests of the present research, it can be concluded that the fit of the model did not vary as a function of data collection site.

DISCUSSION

From the broadest perspective, the results of this study replicate a short-term, longitudinal model relating social support, ADL impairment, and depression, suggesting that the proposed theoretical model is quite robust. In this model, ADL impairment is associated with subsequent increases in depressive symptoms, but depressive symptoms are not associated with increases in ADL impairment. A larger, emotionally close network that makes frequent visits is subsequently associated with higher perceived adequacy of social support. In turn, perceived social support is associated with subsequent decreases in depression, but only among participants randomly assigned to treatment with placebo plus clinical management.

The participants in the present study were different in numerous ways from the sample of heart surgery patients on which the original model was tested (Oxman & Hull, 1997). Participants in the earlier study were seen because they were undergoing the same intensive surgical procedure at the same hospital. They were not all depressed. Participants in the present study were all depressed, were all primary care outpatients suffering sufficiently to enter a randomized controlled trial for depression, had more impairment in ADLs than did the heart surgery patients, and came from four geographically diverse regions. Despite these differences, there was substantial replication of the theoretical model across these samples. Particularly encouraging for the stability of the model is that in the present study there was internal replication of findings from Time 0 to Time 1 in the findings from Time 1 to Time 2.

The unidirectional nature of the relationships is helpful in better understanding the significance and mechanisms of social support. The perception of adequate support appears to be related to having contact with persons with whom one feels emotionally close. The confirmation of a unidirectional path from adequate support to depression suggests that the perception of adequate support is not a secondary effect of mood on perception. In addition, the present results replicate the earlier finding of a unidirectional relationship between impairment in ADLs and depression. Impairment in ADLs leads to more depressive symptoms, but depressed mood does not contribute significantly to impairment in ADLs.

In the present study, conducted in participants with more severe and chronic depression and ADL impairment, the relative replication of the model, especially in the context of a mental health intervention rather than a surgical intervention, suggests that key aspects of the model are firm and meaningful. Despite replication of multiple associations, some aspects of the model were not confirmed in this sample. There was no direct effect of the social network size on lower depression or less ADL impairment as there was in the heart surgery sample. In addition, there was no effect of adequate perceived support on less ADL impairment as there was for the heart surgery patients. Such differences raise a question as to whether there were different events in the surgical sample (presurgical anticipation, acute postsurgical recovery, longer term improvement in function) with different psychological impacts. These events suggest that part of the earlier model may have been an effect of those particular stimuli and events.

Social Support

Two key issues arise in understanding the validity of these results: (a) the specificity of the effect of social support to the placebo group and (b) the mechanisms by which social support has these effects.

Specificity.—Depression has different components such as mood, cognition, and somatic symptoms. Antidepressants such as paroxetine have direct effects on these features and, at least in milder depression, do not seem to be augmented by social support. In cognitive-behavioral approaches such as PST-PC, patients develop strategies to cope and successfully implement these strategies. Implementation has been explicitly theorized to be linked to changes in cognition and mood (Mynors-Wallis, 1996). Use of active coping strategies has also been related to improvements in mental health (Sherbourne, Hays, & Wells, 1995) and is different than the specific focus on symptoms, side effects, and adherence that occurs in clinical management. Thus, for active, specific depression treatments like paroxetine or PST-PC, naturalistic, social support appears to be redundant and without additive benefit. In contrast, in the placebo group social support appears to be additive to the benefits of attention from clinical management and the expectations of positive benefit from placebo.

Mechanisms.—The experience of adequate (or inadequate, Coyne & Downey, 1991) social support can have effects on depression through biological, psychological, or social mechanisms. It is likely that indirect, if not direct, effects of social support are mediated through the central nervous system, even if these mechanisms are poorly understood. For example, just as negative conditioning can result in anticipatory vomiting during chemotherapy for cancer, positive conditioning can evoke better mood in the presence of emotionally supportive contacts. Having close confidants to turn to during stressful times can reduce the surge of negative emotions that occurs with depression. There are also subtle but potentially powerful nonverbal interactions that occur between persons that influence mood both positively and negatively (Coyne & Downey, 1991; Geerts, Bouhuys, & Van den Hoofdakker, 1996; Gerin, Milner, Chawla, & Pickering, 1995; Levenson, Carstensen, Friesen, & Ekman, 1991). Put simply, feeling cared for leads persons to feel good about themselves.

ADL Impairment

ADL impairment is a powerful predictor of depression in elderly persons (Beekman et al., 1997; Bowling & Farquhar, 1991; Koenig, 1998; Oxman et al., 1992; Sherbourne et al., 1995). This relationship may be stronger for minor depressions or for the chronicity of major depression than for acute major depressions (Beekman et al., 1997; Hays, Wells, Sherbourne, Rogers, & Spritzer, 1995). In our analyses, a comparison of the standardized coefficients for ADL impairment and depression to those of perceived adequacy of support and depression suggests that for subsyndromal depressions the magnitude of effects are equivalent.

Our finding of no effect of social support on ADL impairment is consistent with a longer term temporal model (Stoller & Pugliesi, 1991; Wilcox et al, 1994). Acute events affecting ADLs such as those surrounding heart surgery are more likely to show a relationship with social support. The most frequent illnesses in our participants were chronic conditions (e.g., diabetes mellitus, arthritis) or findings that lead to illness (e.g. hyperlipidemia, hypertension). In the present study primary care physicians followed participants who were outpatients rather than hospitalized for an acute event. With chronicity social support is associated with weaker and conflicting effects on the incidence (Seeman et al., 1995) and course (Glass & Maddox, 1992; Wilcox et al., 1994) of ADL impairment. Theories to explain this include unbalanced exchange theory (Dowd, 1975) and network attrition (Lewis & Meredith, 1988). Thus, for longer periods the associations of social support with ADL impairment appear to diminish or even change direction unless new events occur (Fontana et al., 1989; Glass & Maddox, 1992; Marottoli et al., 1992).

Limitations

There are several limitations of this study that qualify interpretation or generalizability. First, we had to rely on a subset of participants with no missing data. This can result in outcomes that are less conservative than analyses in which the last value obtained is carried forward. Second, because of the relatively small sample size we did not include latent variable representations. Third, these findings cannot necessarily be extrapolated to older persons with major depressive disorder. The associations between social support and depression may be weaker in persons with more severe depression (Andrew, Hawton, Fagg, & Westbrook, 1993; Brugha et al., 1987; Ezquiaga, Garcia, Bravo, & Pallares, 1998).

Implications

These longitudinal results from a randomized trial strongly confirm the importance of both social support and ADL impairment to depression in elderly persons. These results also help to put into perspective the value of striving to change social network contacts or perceived adequacy in milder depressions. These results, along with others (e.g., Beekman et al., 1997; Kendrik, 1996), support a cost-effective watchful waiting approach coupled with more selective use of depression-specific treatments. For older primary care patients with minor depression or dysthymia, it would appear reasonable for physicians to pursue an initial strategy of "watchful waiting" as long as the person is embedded in an emotionally close network that makes regular contact, particularly if ADL impairment is low. Watchful waiting is an approach with which primary care physicians are familiar for conditions such as depression (Williams et al., 1999). Although watchful waiting is usually not as intense as clinical management (three to six visits over 3 months), it would appear to deserve increased use in milder depressions.

In contrast, for patients without emotionally close network contact, particularly when coupled with more severe ADL impairment, beginning depression-specific treatments sooner would seem to be indicated. Treatment for such individuals will also be improved if physicians incorporate a diathesisstress model of depression (Coyne & Whiffen, 1995). Treatment and future research for such individuals need simultaneously to address the possibility of enduring personality traits that lead to smaller supportive networks as well as modification of the current social and life cycle context that might result in less support (Coyne & Downey, 1991).

From the broadest perspective, characterizing patients along dimensions of social support and ADL impairment could lead to a more cost-effective use of resources, including learning how to improve inadequate social support.

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Braceland Center For Mental Health and Aging The Institute of Living: Hartford Hospital's Mental Health Network

The Braceland Center for Mental Health and Aging at the Institute of Living/Hartford Hospital conducts research and education aimed at improving mental health care and other types of services for older persons.

Research, evaluation and education projects address the following major areas: clinical mental health services and policy (primarily focusing on depression and dementia), organization, financing and quality of long term care, and ethical/legal issues in aging. Projects are funded through a variety of federal and state sources and private foundations. The Center also has a substantial and growing endowment to support its activities. The Center is formally affiliated with the Center on Aging at the University of Connecticut Health Center.

Director—The Director will ensure achievement of the Center's mission by:

- providing direction and oversight for all aspects of the aging health policy and clinical mental health research agendas;
- identifying potential funding and directing grant and contract proposal preparation in order to secure adequate support for Braceland Center programs;
- and promoting the Braceland Center as a scholarly research and education institute through applied research in the Hartford Hospital system, national dissemination of original research and faculty appointments.

Required credentials include a Ph.D. or M.D. or its equivalent; a history of extramurally funded scholarly research relevant to mental health and aging; qualifications appropriate for rank as Associate Professor including publications; and experience in research administration.

Senior Scientist—Will enhance the Center's research program through:

- developing a program of scholarly research;
- competing successfully for external grants;
- collaborating with colleagues in a multidisciplinary environment

Required credentials include a Ph.D. or equivalent degree in a health related/behavioral science area with specialization in gerontology. Demonstrated ability to secure independent funding in the field of aging, skills in research design and data analysis and a record of peer-reviewed publications are essential. Experience with mental health services and/or policy research is preferred.

Salary commensurate with experience. The Institute of Living is an equal opportunity employer. Review of applications for both jobs will begin January 1, 2001. Applicants should submit 2 copies of a letter of application and vita. The letter of application should specify the following: 1) a summary of research and teaching experience/interests, and 2) recent samples of scholarly work. Please send application materials to:

> Harold I. Schwartz, MD Psychiatrist-in-Chief The Institute of Living/Hartford Hospital 400 Washington Street Hartford, CT 06106