An integration of the hopelessness theory of depression and the stress generation model was offered and tested in a longitudinal study of 169 undergraduates. It was expected that hopelessness would lead to increases in both depressive symptoms and interpersonal stress. Furthermore, it was predicted that hopelessness would produce increases in depressive symptoms partly as a function of generating interpersonal stress. Results conformed to prediction. Suggestions for future theoretical and empirical work on the hopelessness and stress generation views were offered.

According to the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989), hopelessness is a proximal and sufficient cause of a specific subtype of depression, hopelessness depression, mainly characterized by motivational- and emotional-related symptoms. Hopelessness is viewed as the expectation that highly desirable outcomes will not occur or that highly aversive events will occur, and hopelessness mediates the relation between the negative cognitive style (e.g., the tendency to attribute negative events to stable, global causes) and increases in stress-related depressive symptoms. Several reports have supported the general association between hopelessness and depression (e.g., Whisman, Miller, Norman, & Keitner, 1995), as well as the prediction that the attributional style – depression relation is mediated, at least in part, by hopelessness (Joiner, Metalsky, & Wonderlich, 1995; Metalsky & Joiner, 1992; Needles & Abramson, 1990).

Working from a different conceptual basis, Hammen, Davila, and colleagues have proposed an account of depression that is distinct
from, but not incompatible with, the hopelessness theory (Adrian & Hammen, 1993; Daley, Hammen, Burge, & Davila, 1997; Davila, Bradbury, Cohan, & Tochluk, 1997; Davila, Hammen, Burge, & Paley, 1995; Hammen, 1991; Hammen, Davila, Brown, Ellicott, & Gitlin, 1992; Potthoff, Holahan, & Joiner, 1995; Simons, Angell, Monroe, & Thase, 1993; Wagner, Berenson, Harding, & Joiner, 1998). Stated briefly, these researchers have theorized that depressed people, at least in part, generate their own negative life stress. For example, in a one–year study of women with depression, bipolar disorder, medical illness, or no disorder, Hammen (1991) showed that depressed women experienced more stress to which the women themselves contributed, even compared to the women with bipolar disorder and medical illness. This finding has been replicated in samples of men and women (Hammen et al., 1992), marital couples (Davila et al., 1997), adolescent women (Daley et al., 1997; Davila et al., 1995), children (Adrian & Hammen, 1993), as well as by other research groups (Potthoff et al., 1995; Simons et al., 1993; Wagner et al., 1998).

In a series of studies on the mechanism by which stress generation occurs, Joiner, Wingate, Gencoz, & Gencoz (in press) reported that increases in hopelessness fully accounted for (i.e., mediated) the relation between baseline depression levels and prospective increases in self–reported stress (i.e., stress generation). This result raised the possibility that depression does not generate stress per se, but rather, generates the belief, through hopelessness, that stress has worsened—a belief that may inflate scores on a self–report stress inventory. However, a follow–up study refuted this possibility—hopelessness predicted actual changes in negative interpersonal events, as reported by the target participant’s roommate, so hopelessness accounted not only for the self–reported stress generation but also for the stress–generation reported by a second party. Moreover, when both depression and hopelessness were simultaneously entered as predictors of interpersonal stress changes, hopelessness, not depression, was the significant predictor. Joiner et al. (in press) thus concluded that hopelessness may be the key aspect of depression in explaining the stress generation effect.

Furthermore, Joiner et al. (2005) suggested an integration of the hopelessness theory and the stress generation literatures. Specifically, the finding that hopelessness may generate stress, taken together with past research showing that hopelessness may generate depression, indicates that hopelessness may generate depression as a function, in part or in full, of generating stress; or, somewhat similarly, hopelessness may simultaneously generate depression and stress, with depression and stress mutually reinforcing each other’s persistence over time. These possibilities are depicted in Figure 1.
Several features of these models deserve emphasis. The models overlap considerably but are not incompatible, and are not presented as alternatives to one another. But it is useful to differentiate between them, to highlight two points. First, the model in the top panel of Figure 1 depicts a mediational sequence, in which the effect of hopelessness on depressive symptoms is mediated by interpersonal stress. This model represents, then, a temporal sequence in which hopelessness (perhaps previously developed as a function of negative cognitive style activated by stress, as the hopelessness theory of depression states) has to generate interpersonal stress in order to then cause the depressive symptoms. In other words, depressive symptoms emerge (or increase) because of the occurrence of interpersonal stress generated previously by hopelessness. The curved arrow from hopelessness to depressive symptoms acknowledges the possibility that hopelessness may directly affect depressive symptoms, as well as indirectly affect depressive symptoms through interpersonal stress (i.e., partial mediation). From a data-analytic standpoint, as is described in detail later, the key strategy in testing this model is to assess the impact of partialling the mediator (i.e., interpersonal stress) from the relation between the predictor (i.e., hopelessness) and the outcome variable (i.e., depressive symptoms).
The model in the bottom panel of Figure 1 is intended to depict the possibility of simultaneous and independent effects of hopelessness on both interpersonal stress and depressive symptoms, separate from the issue of mediation. That is, hopelessness can generate, at the same time, interpersonal stress and depression without the necessity of one (either stress or depressive symptoms) having to affect the other previously, in other words (and unlike the model in the top panel figure), hopelessness can generate interpersonal stress or depression at the same time without any mediation effect. Support for this model would exclude, for example, the epiphenomenal explanation that hopelessness predicts interpersonal stress only because the latter is related to depressive symptoms (also predicted by hopelessness). From a data–analytic standpoint, the exclusion of this latter possibility would entail partialling depression from the relation between hopelessness and interpersonal stress, to demonstrate that the relationship nonetheless remains. If so, the view would be supported that hopelessness has genuine effects on the development of both stress and depressive symptoms.

Empirical support for these models would broaden the role of hopelessness in the hopelessness theory. Currently, hopelessness is framed as the mediator between negative cognitive style and stress–induced depressive reactions. Hopelessness may perform this function by generating interpersonal stress, which in turn, may heighten depression, and, importantly, encourage the persistence of depression by producing stress that may further aggravate the negative cognitive style, leading to more hopelessness, more symptoms, more stress, and so on.

The present longitudinal study of undergraduates was designed to test these issues. First, consistent with hopelessness theory (and with both models depicted in Figure 1), it was expected that hopelessness would predict increases in depressive symptoms. Second, consistent with research on stress generation (e.g., Davila et al., 1997; Hammen, 1991), and similar to Joiner et al. (in press), it was hypothesized that hopelessness would also be predictive of prospective increases in negative interpersonal events (again, consistent with both models depicted in Figure 1). Third, the possibility was tested that hopelessness predicts increases in depressive symptoms as a function of predicting increases in interpersonal stress (cf. the top panel of Figure 1). Analyses were also conducted to assess the possibility that hopelessness has simultaneous and unique effects on both increased depression and increased interpersonal life stress (cf. the bottom panel of Figure 1).
METHOD

PARTICIPANTS AND PROCEDURE

One hundred and sixty-nine participants (78 men; 91 women) were drawn from Introductory Psychology classes at a large state university, and received class credit in exchange for participation under conditions of full informed consent. Mean age was 19 (range = 17 to 29); the majority of subjects were Caucasian (68% Caucasian; 19% Asian–American; 8% Hispanic; 5% African–American). Participants were tested in groups towards the beginning of the school semester. Upon arrival at Session 1, participants were informed that they would be filling out questionnaires about their personal feelings and attitudes. They also were asked to return for a second session in five weeks, at which time they completed similar questionnaire packets.

MATERIALS

Hopelessness Scale (HS; Beck, Weissman, Lester, & Trexler, 1974). We used the original HS, which includes 20 true–false items which tap the general construct of hopelessness (e.g., “I look forward to the future with hope and enthusiasm” [reversed]). The scale’s reliability and validity have been well supported (for recent work with an extended version of this scale, see Metalsky & Joiner, 1992; Needles & Abramson, 1990). The scale was administered at Time 1.

Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979; Beck & Steer, 1987). Level of depressive symptoms was assessed by the BDI, a 21–item self–report inventory. Although the BDI is not indicative of the full clinical syndrome of depression, it is a reliable and well–validated measure of depressive symptomatology (see Beck, Steer, & Garbin, 1988, for a review). BDI was administered at Times 1 and 2.

Negative Life Events Questionnaire—Interpersonal Events (NLEQ; Saxe & Abramson, 1987). The NLEQ was developed specifically for use with college students and includes several categories of events to ensure broad coverage (e.g., family, friends, etc.). Similar to Joiner and Rudd (1995), as well as to studies in the stress generation literature (e.g., Hammen, 1991), the present study focused only on interpersonal negative life stressors (e.g., “Fight or disagreement with romantic partner”). Although the focus was only on interpersonal negative life stressors, the whole scale was administered, and interpersonal– and achievement–related stressors were also assessed. Items were rated on a 0 to 4 scale (0 = “Never present”; 4 = “always present”) on how frequently they had occurred during the past five weeks. Scores for the 28 items can thus range from 0 to 112. Scores were also computed using a dichotomous criterion.
(0 = event absent; 1 = event present). Results were similar to those using the 0 to 4 scale.

The scale is reliable (e.g., Saxe & Abramson, 1987) and valid (e.g., Alloy & Clements, 1992; Metalsky & Joiner, 1992). The NLEQ has been used in a previous study of stress generation (Joiner et al., in press). The measure was administered at Times 1 and 2, covering the five-week interval prior to each assessment session.

RESULTS

Descriptive statistics and intercorrelations are summarized in Table 1. Gender did not moderate any findings, and thus is not included. As can be seen there, a high test–retest correlation was obtained for the life events measure \( r = .68, p < .001 \), similar to Joiner et al. (in press), implying person–centered involvement in stress maintenance, a consistent reporting style bias, and/or chronic stressors. Notably, the data–analytic approach accounts for this by partialling earlier stress from later stress, and focusing on the residual term, which will correspond to the onset of new stress, or the exacerbation (or diminution) of existing stress. Because of the high association between earlier and later stress, the covariance of the former from the latter is stringent, and the ability of hopelessness to nonetheless predict the residual stress term would provide compelling support for its role as a stress generator. Other correlations in Table 1, as well as descriptive statistics, are in line with

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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<tr>
<td>1. T1 HOPELESSNESS</td>
<td>6.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>(10.87)</td>
<td></td>
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<td>2. T1 BDI</td>
<td></td>
<td>.60</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td>(7.85)</td>
<td></td>
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<td></td>
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<tr>
<td>3. T2 BDI</td>
<td></td>
<td>.59</td>
<td>.69</td>
<td></td>
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<td></td>
<td></td>
<td>(7.41)</td>
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<tr>
<td>4. T1 IP STRESS</td>
<td></td>
<td>.52</td>
<td>.42</td>
<td>16.91</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(7.41)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. T2 IP STRESS</td>
<td></td>
<td>.50</td>
<td>.39</td>
<td>.44</td>
<td>16.51</td>
</tr>
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<td></td>
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<td>(16.00)</td>
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Note: Means and standard deviations (in parentheses) on diagonal. HOPE = Hopelessness Scale. BDI = Beck Depression Inventory. IP STRESS = Interpersonal Negative Events from the Negative Life Events Questionnaire. All correlations significant to the .001 level.
expectation and with past work. For example, depressive symptoms were correlated with hopelessness and with negative interpersonal events (similar, e.g., to Metalsky & Joiner, 1992).

HOPELESSNESS AS A “GENERATOR” OF DEPRESSIVE SYMPTOMS

To determine whether Time 1 hopelessness scores were related to changes in depressive symptoms from Time 1 to Time 2, a setwise hierarchical multiple regression/correlation procedure was used (Cohen & Cohen, 1983). T2 BDI scores served as the dependent variable. T1 BDI scores were entered first into the regression equation, thereby creating residual change scores in BDI depressive symptoms from T1 to T2. Next, T1 hopelessness scores were entered into the equation.

As can be seen in Table 2, T1 hopelessness scores were significantly related to increases in BDI depressive symptoms from T1 to T2 ($r = .30, t_{166} = 4.01, p < .001$). This result is consistent with a wealth of past research documenting the relation of hopelessness and depressive symptoms (e.g., Metalsky & Joiner, 1992).

HOPELESSNESS AS A “GENERATOR” OF INTERPERSONAL STRESS

A similar approach was taken to determine whether Time 1 hopelessness scores were related to changes in interpersonal stress from Time 1 to Time 2. As can be seen in Table 3, in a regression equation with T2 interpersonal stress scores as the dependent variable, T1 interpersonal stress scores as the covariate, and T1 hopelessness scores as the predictor, T1 hopelessness scores emerged as a significant predictor of interpersonal stress changes from T1 to T2 ($r = .21, t_{166} = 2.83, p < .01$). This result is consistent with past work on stress generation and depression (e.g., Hammen, 1991), and with the view that hopelessness may be a stress generator (Joiner, 1998).

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1. As can be seen in Table 1, each of the variables was positively skewed. In accord with the recommendations of Cohen and Cohen (1983), a square–root transformation was applied to all variables before regression analyses, described next, were conducted. Results using the transformed variables were virtually identical to those using untransformed versions, with respect to direction, magnitude, and significance. Untransformed results are reported hereafter.
### TABLE 2. Hopelessness Scores Predicting Increases in BDI Depressive Symptoms

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>$R^2$ for set</th>
<th>$F$ Change</th>
<th>$t$ for individual within-set predictors</th>
<th>df</th>
<th>Partial correlation (pr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T1 BDI</td>
<td>155.08*</td>
<td>12.45*</td>
<td>.69</td>
<td>1,167</td>
<td>.48</td>
</tr>
<tr>
<td>2</td>
<td>T1 HOPELESSNESS</td>
<td>16.06*</td>
<td>4.01*</td>
<td>.30</td>
<td>1,166</td>
<td>.53</td>
</tr>
</tbody>
</table>

Note. pr = partial correlation for within-set predictors. BDI = Beck Depression Inventory. HOPELESSNESS = Hopelessness Scale. *p < .01.

### TABLE 3. Hopelessness Scores Predicting Increases in Interpersonal Stress

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>$R^2$ for set</th>
<th>$F$ Change</th>
<th>$t$ for individual within-set predictors</th>
<th>df</th>
<th>Partial correlation (pr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T1 IP STRESS</td>
<td>151.59*</td>
<td>12.31*</td>
<td>.68</td>
<td>1,167</td>
<td>.46</td>
</tr>
<tr>
<td>2</td>
<td>T1 HOPELESSNESS</td>
<td>8.01*</td>
<td>2.83*</td>
<td>.21</td>
<td>1,166</td>
<td>.48</td>
</tr>
</tbody>
</table>

Note. pr = partial correlation for within-set predictors. IP STRESS = Interpersonal Negative Events from the Negative Life Events Questionnaire. HOPELESSNESS = Hopelessness Scale. *p < .01.
Baron and Kenny (1986) articulated several conditions that should characterize the interrelations between predictor, mediator, and dependent variables. In order to serve as a mediator of a predictor’s effect on a dependent variable, a potential mediator variable must itself be predictive of the dependent variable. In the present case, then, the condition requires that T1 interpersonal stress (the mediator) be associated with changes in depressive symptoms (the outcome).

This condition was satisfied. A regression equation, similar in form to that depicted in Tables 2 and 3, was constructed, with T2 BDI as the dependent variable, T1 BDI as a covariate, and T1 interpersonal stress as a predictor. T1 interpersonal stress did in fact serve as a significant predictor of changes in BDI depressive symptoms \( (p = .22, t [166] = 2.87, p < .01) \), consistent with past research documenting the contribution of stress to depressive symptoms (e.g., Brown & Harris, 1978). It was also the case that changes in interpersonal stress from T1 to T2 (standardized residual term) were predictive of changes in depressive symptoms from T1 to T2 \( (p = .16, t [166] = 2.11, p < .05) \).

A second condition requires that the significant relationship between the predictor and the outcome be reduced when the effects of the mediator are controlled. With regard to this condition, Baron and Kenny (1986) distinguished between partial versus complete mediation. In the case of partial mediation, the predictor’s effect (see the curved arrow in the top panel of Figure 1) would be reduced but would continue to serve as a significant predictor of the outcome variable when the mediator is controlled. In contrast, for complete mediation, the predictor’s effect would be reduced to non-significance when the mediator is controlled.

Consistent with the criteria for partial mediation, the previously reported significant partial correlation between T1 hopelessness and changes in BDI depression \( (r = .30; \text{see Table 2}) \) was reduced, but continued to serve as a significant predictor of BDI increases, when T1 and T2 interpersonal stress scores were controlled \( (r = .19, t [164] = 2.54, p < .01) \). The Sobel (1982) test indicated significant mediation \( (Z = 1.99, p < .05) \). This finding suggests that hopelessness exerts both a direct effect on depression increases, as well as an indirect effect through increases in interpersonal stress. Interestingly, it appeared to be interpersonal stress specifically, and not stress generally, that (partially) mediated this effect. When an index of achievement-related negative life events was used in the analyses in place of interpersonal stress, mediation did not occur (i.e., the relation between T1 hopelessness and changes in depres-
sive symptoms was virtually unchanged; $pr = .29$, $t (164) = 3.93, p < .01$; cf. $pr = .30$ when mediators not controlled).

HOPELESSNESS AS A SIMULTANEOUS “GENERATOR” OF BOTH INTERPERSONAL STRESS AND DEPRESSIVE SYMPTOMS

It remains possible that hopelessness leads to stress changes only because it leads to depression changes. This appears not to be the case. Specifically, when T1 and T2 BDI depressive symptoms were partialled from the relation between T1 Hopelessness and changes from T1 to T2 in Interpersonal Stress, the significant relationship remained ($pr = .17$, $t [164] = 2.14, p < .05$; cf. $pr = .21$ in Table 3), at odds with the view that hopelessness’ role as a stress generator is epiphenomenal vis-à-vis its relation to depressive symptoms. Rather, it appears that hopelessness leads to both depressive symptoms and interpersonal stress (cf. bottom panel of Figure 1).

DISCUSSION

In this study, we hypothesized that hopelessness would explain interpersonal stress increases from T1 to T2, as well as increases in depressive symptoms, and that this relationship between hopelessness and depression would be mediated, at least in part, by the interpersonal stress. Results were supportive with our predictions showing that hopelessness explained interpersonal stress increases from T1 to T2, and also explained increases in depressive symptoms from T1 to T2. Moreover, the relationship between hopelessness and depressive symptoms was reduced after controlling for interpersonal stress at T1 and T2, thereby showing partial mediation. Additionally, we also demonstrated that the relationship between hopelessness and depression was not spurious, that is, merely due to a common relation of both variables to interpersonal stress.

The present findings, thus, suggest an interpersonal elaboration to Abramson et al.’s (1989) hopelessness theory of depression. According to the hopelessness theory, hopelessness produces depression, and current results are supportive of this prediction (see Table 2). In addition, present findings indicated that hopelessness may also generate stress (see Table 3; Joiner, 1998, reported similar results).

The addendum to hopelessness theory is depicted in Figure 2. The highlighted aspects represent the suggested elaboration to the theory. Specifically, the Figure shows that hopelessness, once developed (perhaps as a function of negative cognitive style activated by stress): a)
leads directly to depressive symptoms; b) leads indirectly to depressive symptoms by generating interpersonal stress; and c) in generating stress, provides more “grist” for negative cognitive style’s “mill,” thus propagating the sequence and perpetuating depressive symptoms.

It should also be noted that the present study was consistent with the stress generation phenomenon, as reported by Hammen, Davila and colleagues (e.g., Hammen, 1991; Davila et al., 1997). The Joiner et al. (in press) finding that hopelessness is the key aspect of depression that drives the stress generation effect was also replicated.

Joiner et al. (2005) provided several explanations of how hopelessness may operate as a stress generator. Hopelessness appears not to lead to increased stress via impaired interpersonal behaviors and attitudes. The results of Davila et al. (1995) and Joiner et al. (in press) converge on this conclusion.

Rather, hopelessness may be involved in stress generation through its association with depression chronicity. As depression persists, sufferers may become more and more hopeless, and significant others may become more and more burdened and disaffected. Consistent with this view, Sacco, Milana, and Dunn (1989) provided participants with transcripts of depressed people whose episodes were long–lasting, and transcripts of depressed people who experienced short episodes. Compared to the latter, long–duration “depressives” elicited more anger and more rejecting attitudes. Notably, Hammen’s (1991) study on stress generation in depression focused on women with chronic forms of depression.

A second possibility is that hopelessness embitters and stultifies the sufferer, potentially producing negative and persistent mental repre-
sentations of the sufferer in the minds of significant others. Sacco (1999) argued that, once developed, such representations become autonomous, in that they selectively guide attention toward and confirm the representation of the depressed person by others (cf. Fiske, 1993). These social–cognitive processes may occur spontaneously and outside the awareness of the perceiver (Lewicki, Hill, & Czyzewska, 1992), and may be particularly salient with regard to negative as compared to positive features of the represented person. There is evidence that, as compared to positive behaviors, negative behaviors are more likely to draw attention (Pratto & John, 1991), and when attributed to the person (as opposed to the situation), more likely to be remembered (Ybarra & Stephan, 1996). Once crystallized, representations of negative behaviors are more difficult to alter than representations of positive behaviors (Rothbart & Park, 1986).

Hopelessness, then, may generate stress by instilling in others a person–schema that propagates itself and that negatively biases subsequent perceptions of the hopeless person. Others’ negative views may, in turn, subserve critical communications from others (i.e., negative interpersonal stress) toward the hopeless person, and such communications have been shown to be strong predictors of depression and its recurrence (Hooley & Teasdale, 1989).

The sequence depicted in Figure 2 is recurrent (i.e., feeds back on itself), and without a “braking mechanism,” spirals infinitely downward. Of course, “infinite downward spirals” exist, in the form of depression–related suicide as well as chronic forms of depression (e.g., dysthymia). But most depressions remit (even if most recur; Belsher & Costello, 1988; Bothwell & Scott, 1997)—what is the “breaking mechanism?” Some speculations are offered, none of which have been thoroughly investigated. They thus represent a potentially fruitful avenue for future research. First, once hopelessness and depression develop, the sufferer may socially withdraw. Although social withdrawal certainly has costs (cf. Joiner, 1997), it also confers the benefits of distance from interpersonal criticism. During a phase of social withdrawal, the sufferer may initially become more symptomatic, due to the lingering effects of previous interpersonal strife and the current experience of loneliness. But with time, the effects of previous relationship strife may fade, and the ongoing experience of loneliness may compel the sufferer to access alternative, under–utilized, and (perhaps) healthier sources of social support, which in turn, may instill hope (cf. Needles & Abramson, 1990) and encourage recovery.

A related possibility is that severe hopelessness and depressive symptoms serve as a powerful distress signal, with both negative and adaptive qualities. Much has been made of the negative stimulus value of this
signal, and with reason, insofar as one main point of this paper is to argue that hopelessness generates interpersonal stress, and one main point of the literature on depression and the response of others is to explain depressed people’s negative interpersonal impact (e.g., Coyne, 1976; Joiner, Alfano, & Metalsky, 1992). Nonetheless, the communication value of hopelessness and depression may also be adaptive, in that it recruits interpersonal solace (cf. the sociophysiological model of Price, Sloman, Gardner, Gilbert, & Rohde, 1994; Price & Gardner, 1999). Indeed, anecdotally, many people who have recovered from depression remark that, through the experience, they “found out who their real friends were.” Joiner et al. (1992) obtained some empirical support for the view that people with tolerant, supportive, or emotionally empathic interpersonal styles were less rejecting than others, even toward people most vulnerable to rejection (i.e., high reassurance–seeking, depressed people). The heightened support of tolerant others—including mental health professionals—may thus serve to break the cycle depicted in Figure 2.

It must also be remembered that positive life events occur, to some degree or another, in everyone’s lives. Should a hopeless or depressed person fortuitously experience a positive event (e.g., supportive phone call from an old friend), the experience may foster improvement, especially if the event is attributed to stable and global causes (e.g., “my old friend really cares about me”; cf. Needles & Abramson, 1990).

In conclusion, a few cautions and considerations are noted. First, with regard to stress generation, the present study examined self-reported stress, the assessment of which has the possible disadvantage of bias from such sources as current hopelessness and depression. This point should be noted, but in addition, it should be emphasized that in a previous study Joiner et al. (in press) demonstrated that the stress generating effect of hopelessness was genuine, and not due merely to the generation of negative perceptions of events. Also, the index of stress used in this study did not differentiate between stress specifically related to a participant’s actions, versus stress from random, non-contingent sources (cf. Hammen, 1991). It should be emphasized, however, that this imprecision would likely have worked against, not for, the detection of the stress generation effect.

2. One could legitimately argue that cognitively vulnerable people, especially those who are currently depressed, are unlikely to make adaptive attributions. However, this point, while reasonable, begs the questions of the cross-situational stability of attributional style, as well as possible contextual effects on attributional style. Both of these questions deserve heightened attention within the literature on cognitive style and depression.
Second, the present study did not address the important issue of specificity of effects to depressive symptoms versus other psychopathological symptoms. Here again, past work mollifies this concern; Hammen (1991) demonstrated the relative specificity of stress generation to depression versus bipolar disorder, and Joiner et al. (in press) provided a similar finding regarding depressive versus anxious symptoms. Third, the present study focused on depressive symptoms as they occurred in a general sample of undergraduate students, not on clinical forms of depression. Hammen (1991) and Daley et al. (1997) found evidence for the stress generation effect in participants with a depression diagnosis. The current study, like that of Davila et al. (1995), assessed the generality of stress generation to depressive symptoms vs. depressive diagnoses. Depressive symptoms in unselected populations may (Vredenburg, Flett, & Krames, 1993) or may not (Coyne, 1994; Tennen, Hall, & Affleck, 1995) represent a legitimate analog of clinical depression. Future research will benefit from considering these limitations in testing and elaborating upon the hopelessness theory, stress generation, and the recursive and multi-directional inter-relationships of hopelessness, depressive symptoms, and interpersonal stress.

REFERENCES


3. A focus on symptoms (as opposed to diagnoses) is legitimate in the context of past work (e.g., Davila et al., 1995), the “continuity-category” debate (see Vredenburg, Flett, & Krames, 1993), and the pernicious qualities of sub-clinical depressive symptoms (Judd, 1997). This focus is also legitimate given the features of the data set (from which symptoms counts are readily available, but diagnostic status is more difficult to establish).


Joiner, T., Wingate, L.R., Gencoz, T., & Gencoz, F. (2005). Stress generation in depression:


