

RECIPROCAL RELATIONS BETWEEN PEER STRESS AND INTERNALIZING
AND EXTERNALIZING SYMPTOMS DURING ADOLESCENCE

By

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To my mother, Mary Heintz Richardson, and to my husband, Leslie James Carter.

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CHAPTER I

INTRODUCTION

Evidence of a significant relation between stress and psychopathology in children and adolescents has been found in both cross-sectional (Compas, 1987; Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001) and longitudinal studies (Hammen & Goodman-Brown, 1990; Hilsman & Garber, 1995; Rudolph, Lambert, Clark, & Kurlakowsky, 2001). Higher levels of stress have been found to be significantly associated with higher levels of both internalizing (Aseltine, Gore, & Gordon, 2000; Barrerra et al., 2000; Rudolph et al., 2000) and externalizing symptoms (Aseltine et al., 2000; Barrerra et al., 2000; Barrett & Heubeck, 2000; Jackson & Warren, 2000; von Weiss et al., 2000; Windle & Windle, 1996). Significant, positive relations between stress and depression, in particular, have been found in both community and outpatient samples of children and adolescents (Barrett & Heubeck, 2000; Cole & Turner, 1993; Daniels & Moos, 1990; Dumont & Provost, 1999; Larson, Moneta, Richards, & Wilson, 2002; Leadbeater, Kuperminc, Blatt, & Hertzog, 1999; Sandler, Reynolds, Kliwer, & Ramirez, 1992; Sandler, Tein, & West, 1994; Wadsworth & Compas, 2002; Windle & Windle, 1996) as well as in college freshmen and young adults (Compas, Slavin, Wagner, & Vannatta, 1986; Nezu, Nezu, Saraydarian, Kalmar, & Ronan, 1986; although for contrary results, see Benfield, Palmer, Pfefferbaum, & Stowe, 1988). The purpose of the present study was to compare three different models of the relation between peer stress, in particular, and both internalizing and externalizing symptoms in adolescents across four years.

At least three models have been proposed to explain the relation between stress and psychopathology: stress exposure, stress generation, and reciprocal. According to the stress exposure model, individuals who have experienced stressors will have more symptoms than those who have not (Hammen & Goodman-Brown, 1990; Hilsman & Garber, 1995; Rudolph, Lambert, Clark, & Kurlakowsky, 2001). Prospective studies showing that stress temporally precedes increases in symptoms have provided support for this model (e.g., Compas et al. 1989; Hilsman & Garber, 1995; Rudolph et al., 2001; Siegel & Brown, 1988). For example, Hilsman and Garber (1995) measured depressive symptoms one week before, the morning after, and five days after children received a lower grade than they would have liked, and found that, controlling for symptoms one week prior, the stressor predicted increases in depressive symptoms five days later. Other studies with children similarly have shown that controlling for initial levels of depression, stress significantly predicts increases in depressive symptoms six (Rudolph et al., 2001) and nine months later (Compas et al., 1989).

With regard to externalizing symptoms, Mathijssen, Koot, and Verhulst (1999) found in a sample of Dutch children and adolescents referred to outpatient mental health clinics that those whose life stressors had increased during the year between assessments had increases in both externalizing and total problem scores on the Child Behavior Checklist, although not in internalizing symptoms. Aguilar, Sroufe, Egeland, and Carlson (2000) showed that individuals who had onsets of externalizing behavior in childhood that continued across adolescence were more likely to have had life stress earlier in their childhood. Thus, some support for the stress exposure model has been found for both internalizing and externalizing problems.

The stress generation model (Hammen, 1991,1992) posits that individuals with psychopathology, particularly depression, tend to generate dependent stressors, the stressors in their lives that occur as a function of their own behavior. For example, controlling for baseline levels of depression, dependent interpersonal stressors predicted increased levels of depressive symptoms at follow-up (Davila, Hammen, Burge, Paley, & Daley, 1995). In a short-term longitudinal study of college freshman, self-reports of depressive symptoms were associated with stressors two weeks later (Potthoff, Holahan, & Joiner, 1995).

Leadbeater et al. (1999) examined whether externalizing and internalizing symptoms in adolescents predicted stressful life events one year later. In females, externalizing behaviors predicted subsequent stressful life events; no effects were found for males or with internalizing symptoms, however. Aseltine et al. (2000) found for both males and females, involvement in delinquent activities predicted higher levels of life stress and family conflict. These studies (Aseltine et al., 2000; Leadbeater et al., 1999; Potthoff et al., 1995), however, did not control for earlier levels of stress, making it difficult to determine whether symptoms actually predicted changes in levels of stress (Kim, Conger, Elder, & Lorenz, 2003).

A third perspective regarding the relation between stress and symptoms is the reciprocal model. Symptoms at one time are hypothesized to produce stressors at a later time, and similarly, stressors at one time are presumed to lead to symptoms at a later time. Studies testing this model treat both stress and symptoms as predictor and outcome measures across multiple periods. This allows one to control for earlier levels of stress or

symptoms when predicting outcomes and to examine cross-sectional and longitudinal relations among these variables.

Cohen, Burt, and Bjorck (1987) reported reciprocal relations between life events and psychological outcomes. Controlling for symptom levels five months earlier, they found that negative life events positively predicted anxiety and depression in young adolescents. The reverse relation also was found; that is, symptoms predicted change in level of stress. The direction of effects varied between anxiety and depression however, with high levels of depression predicting increases in stress whereas high levels of anxiety predicted decreases. Cohen et al. suggested that anxiety may prevent youth from engaging in risk-taking behaviors and thus contribute to lower levels of stress. Thus, the study by Cohen et al. appears to support the reciprocal model, although the effect differed for anxious versus or depressive symptoms.

Kim et al. (2003) tested separate reciprocal models for stress and internalizing and externalizing symptoms across adolescence. The results for internalizing and externalizing symptoms were similar in that stress predicted increased levels of symptoms at the following time-point while symptoms predicted increased levels of stress. This study adds to earlier work by showing that reciprocal relationships are present throughout multiple measurement periods in adolescence.

Building on the study by Kim et al. (2003), the current investigation used an autoregressive model to examine the direction of the relations between stress and internalizing and externalizing symptoms over four time points during early- to mid-adolescence. The present study differs from that of Kim et al., however, in several ways. First, we included both internalizing and externalizing symptoms in the same model

because they tend to be correlated and have trajectories that may influence each other (Hinshaw, Lahey, & Hart, 1993; Keiley, Bates, Dodge, & Pettit, 2000; Kiesner, 2002; Loeber, Russon, & Stouthamer-Loeber, 1994; Mesman, Bongers, & Koot, 2001, Panak & Garber, 1992; Seiffge-Krenke, 2000). Including both internalizing and externalizing symptoms in the same model allowed us to explore reciprocal relations between stress and one type of symptom while controlling for levels of the other type of symptom.

Second, Kim et al. (2003) as well as many other studies have used measures of general levels of stress rather than specific types of stressful events. The current study examined the relation between peer stress, in particular, and adolescent symptoms. Peer stress has been found to be the most frequently occurring stressor during early and middle adolescence (Isakson & Jarvis, 1998). Moreover, interpersonal stressors have been found to be associated with both internalizing (Compas, 1986; Davila et al., 1995; Sim, 2000) and externalizing symptoms (Barrett & Heubeck, 2000; Sim, 2000). For example, Rudolph et al. (2000) measured both interpersonal and non-interpersonal stressors in a sample of outpatient clinic youth and found that interpersonal stressors were significantly related to depressive and marginally related to externalizing symptoms; non-interpersonal stressors were significantly related to externalizing symptoms. Further, children with comorbid depressive and externalizing symptoms had a greater number of interpersonal stressors than did children who had high symptom levels in only one problem area. Thus, interpersonal stressors may be particularly linked with depression, but externalizing symptoms might exacerbate that relation further. Compas et al. (1986) also have shown that interpersonal stressors (i.e., problems with family and parents) were positively related to internalizing symptoms in adolescents, and social stressors such as “having

problems with roommates,” or “not having as many friends as you would like” were positively correlated with depressed and anxious symptoms in college students (Connor-Smith & Compas, 2002).

The association between social stressors and symptoms also has been found with culturally diverse samples. In Australian children, Barrett and Heubeck (2000) reported that hassles with peers specifically predicted anxiety symptoms, whereas hassles with teachers predicted conduct problems. Among Korean adolescents, hassles with friends and with parents were significantly related to both depressive symptoms and antisocial behaviors (Sim, 2000). Similar results were found in a sample of American inner-city adolescents in which stress with family members and peers was significantly associated with depression (Deardorff, Gonzales, & Sandler, 2003). Thus, the social domain is a particularly important context for the experience of negative life events that are linked with psychopathology in youth.

The present study also examined possible moderators of the relation between stress and symptoms. In particular, we tested whether the hypothesized models fit equally well for boys and girls and for offspring of depressed and non-depressed mothers. There is some evidence that girls experience more interpersonal stressors than boys (Rudolph & Hammen, 1999; Santa-Lucia, Gesten, Rendina-Gobioff, Epstein, Kaufmann, & Salcedo, 2000). This effect may depend on the specific type of interpersonal stress that is studied, however. Seiffge-Krenke & Stemmler (2002) reported a non-significant trend for boys to have more stress with peers than girls across adolescence, whereas girls tended to report more stress with mothers at age 15 than did boys.

Gender differences have been found with regard to specific types of peer stress, with girls reporting more stressful events in their close friendships whereas boys reporting more stressful events in their larger peer group (Rudolph, 2002). Interestingly, although girls reported fewer peer group stressors, they were more likely to experience anxious and depressive symptoms in response to such peer stress than were boys. Girls also were more likely to experience symptoms in response to stress within close friendships (Rudolph, 2002). Similarly, although both girls and boys who had difficulties in their close friendships and with peers had more symptoms of social anxiety, the relation was stronger for girls than boys (LaGreca & Lopez, 1998). In contrast, interpersonal conflict stress has been found to correlate significantly with depressive symptoms for boys, but not for girls (Rudolph & Hammen, 1991). Compas et al. (1986) also found the relation between negative events and internalizing symptoms to be significantly stronger for boys than for girls. In a sample of German adolescents, major stressful life events were related to depressive symptoms in boys only when they were 14 years old and to girls only when they were 17 (Seiffge-Krenke & Stemmler, 2002), whereas stress with peers at age 17 was significantly related to girls' depressive symptoms and marginally related to boys' symptoms. With respect to externalizing symptoms, prior research has shown that externalizing symptoms are related to stress for boys, but not girls (Santa Lucia et al., 2000). Thus, the strength of the relation between social stress and symptoms may differ for girls and boys. Therefore, the present study explored the extent to which the relation between peer stress and internalizing and externalizing symptoms differed as a function of gender.

Finally, studies have shown that offspring of depressed parents experience higher levels of stress (Adrian & Hammen, 1993; Anderson & Hammen, 1993; Billings & Moos, 1983; Conrad & Hammen, 1993; Hammen, Burge, & Adrian, 1991; Hammen & Goodman-Brown, 1990; Hammen, Shih, Altman, & Brennan, 2003), more impaired peer relations and poorer social skills (Goodman, Brogan, Lynch, & Fielding, 1993; Murray, Woolgar, Cooper, & Hipwell, 2001; Zahn-Waxler, Denham, Iannotti, & Cummings, 1992), and more symptoms (e.g., Downey & Coyne, 1990; Goodman & Gotlib, 1999; Zahn-Wexler, Denham, Iannotti, & Cummings, 1992) compared to offspring of nondepressed parents. Hammen et al. (2003) found that mothers' current depressive symptoms interacted with both children's stressful life events and social functioning to predict children's depressive symptoms. The nature of the interaction was that children with greater stress and lower social functioning whose mothers were currently depressed had more depressive symptoms than did children with the same level of stress and functioning whose mothers were not currently depressed. In addition, Hammen and Goodman-Brown (1990) reported that among children who had stressors that matched their specific vulnerabilities (i.e., affiliative- or achievement-oriented), those who became depressed were more likely to have mothers with depression than were those whose mothers had not been depressed. Therefore, we examined whether the relations between stress and symptoms differed for offspring of depressed versus non-depressed mothers.

In summary, the goal of the present study was to compare three different models of the relation between stress and adolescents' internalizing and externalizing symptoms across four years. In addition, we examined the extent to which these relations varied as a function of gender and maternal depression history.

CHAPTER II

METHOD

Participants

Participants were 240 adolescents and their mothers. The first assessment occurred when the children were in 6th grade (mean age = 11.86, SD = .57). The sample was 54.2% female, 82% Caucasian, 14.7% African-American, and 3.3% other (Hispanic, Asian, Native American, or mixed ethnic background). Participants were predominantly lower-middle to middle class; the mean socioeconomic status (Hollingshead, 1975) was 41.84 (SD = 13.25).

Parents of 5th grade children from metropolitan public schools were invited to participate in a study about child development. A brief health history questionnaire comprised of 24 medical conditions (e.g., heart disease, cancer, depression) and 34 medications (e.g., Prozac, Elavil) was sent with a letter describing the project. Of 1495 parents who returned these questionnaires, telephone screening interviews were conducted with the 587 who had endorsed either a history of depression, use of antidepressants, or no history of psychopathology. The Structured Clinical Interview for DSM diagnoses (SCID; Spitzer, Williams, Gibbon, & First, 1990) was then conducted with 349 mothers who indicated during the screening calls that they had had a history of some depression or had had no psychiatric problems. Families were excluded if the mother indicated a history of a psychiatric diagnosis that did not also include a mood disorder, reported a serious medical condition, or if the child had a serious and/or chronic

medical illness, was not primarily in a regular classroom, or had a pervasive developmental disorder. The final original sample of 240 families consisted of 185 mothers who had histories of depressive disorders (147 mothers had had diagnoses of Major Depressive Disorder; the remaining 38 mothers had diagnoses of Dysthymia, Depression NOS, or Adjustment Disorder with Depressed Mood); 55 mothers were lifetime free of psychopathology. Of the 240 families who participated in the initial data collection, 177 were followed through the fourth data collection. Families who did not complete the study did not differ from those who did on any demographic or study variables. In the current sample, 185 mothers had a history of a mood disorder and 55 did not.

A research assistant who was unaware of the mothers' psychiatric history individually administered a battery of questionnaires separately to the mother and adolescent. The present study reports the results of the assessments of the adolescents in grades 6, 7, 8, and 9. Only those measures relevant to the current study are described here.

Measures

Maternal Depression. Maternal depression was assessed with the Structured Clinical Interview (SCID; Spitzer et al., 1990), a widely used, semi-structured clinical interview from which DSM diagnoses (American Psychiatric Association, 1987; 1994) can be made. Inter-rater reliability was calculated on a random 25% of these interviews and yielded a Kappa of .88 for diagnoses of depressive disorders.

Peer stress. Peer relationship stressors were assessed by a questionnaire about school-related stressors (Robinson, Garber, & Hilsman, 1995). Although this measure

included general and academic types of school stress, the current investigation used only those items related to peer relationships. These items were selected based on agreement between two independent raters of the nature of the stress contained in each item. The raters agreed on the classification 80% of the time (Kappa = .71) (Little & Garber, 2000); only items on which they agreed were included in the present investigation

The peer relationship measure used in the current analyses consisted of eleven items such as: “Other kids have picked on you, teased you, made fun of you, or spread rumors about you,” “You’ve had arguments with other kids at school,” and “Kids who were your friends last year are not your friends anymore.” Participants rated the degree to which the stressor had occurred on a five-point scale ranging from 1 (*not at all*) to 5 (*very much*). Items were recoded into a dichotomous scale indicating a count of whether or not the item had occurred (i.e., rated 2 or above). The eleven items were summed to create a total peer stress score ranging from 0 to 11. Because we wanted the index of peer stress to be consistent with the YSR *t*-scores that are normed separately for each gender, we subtracted the mean of each gender’s peer stress score at Time 1 from the stress score at all four time points. Means and standard deviations of all variables are shown in Table 1.

Internalizing and externalizing symptoms. Both internalizing and externalizing symptoms were assessed using the Youth Self Report (YSR; Achenbach, 1991). This measure includes 112 items of problem behaviors in multiple domains. For each item, respondents rate the extent to which each item applies to them on a 3-point scale ranging from 0 (*not true*) to 2 (*very true or often true*). Anxious-depressed, withdrawn, and somatic items comprise the internalizing broadband scale, whereas delinquent and

Table 1

Intercorrelations, means, and standard deviations of study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. T1 Peer Stress												
2. T1 Internalizing	.35											
3. T1 Externalizing	.35	.63										
4. T2 Peer Stress	.35	.24	.23									
5. T2 Internalizing	.30	.56	.40	.40								
6. T2 Externalizing	.18	.34	.61	.33	.63							
7. T3 Peer Stress	.20	.26	.23	.43	.31	.29						
8. T3 Internalizing	.21	.47	.39	.28	.49	.36	.36					
9. T3 Externalizing	.24	.34	.57	.33	.38	.64	.43	.56				
10. T4 Peer Stress	.20	.15	.12	.37	.27	.25	.49	.21	.25			
11. T4 Internalizing	.20	.34	.19	.20	.48	.32	.34	.56	.36	.37		
12. T4 Externalizing	.21	.24	.40	.16	.33	.50	.33	.32	.61	.33	.51	
Mean	.00	46.81	47.51	-0.92	44.08	46.9	-1.08	43.4	46.8	-1.00	43.99	47.01
Standard deviation	2.46	10.49	10.46	1.8	9.08	9.62	1.76	9.3	10.1	1.74	9.33	9.88

Note. All correlations > .15 are significant at the .05 level; all correlations > .18 significant at the .01 level.

aggressive behaviors comprise the externalizing scale. Across the four data collections, Cronbach's alphas for the internalizing scale ranged from .85 to .86 and from .86 to .89 for the externalizing scale.

CHAPTER III

RESULTS

Descriptive Statistics

Table 1 compares the means, standard deviations, and correlations of all variables at each time point. The measures of internalizing and externalizing symptoms and peer stressors were significant and positively related at each time-point and across time for the majority of variables. The exception to this pattern was that Externalizing at Time 1 and Peer Stress at Time 4 were not significantly correlated. The auto-correlations for measures at different time points were strong and internalizing and externalizing symptoms were highly correlated with each other contemporaneously (.51 - .63) and longitudinally (.19 - .63) though the correlations decreased as time points were further apart.

Independent samples t-tests were run to test for group differences according to gender and maternal depression history. Girls reported significantly higher levels of internalizing symptoms at Time 3 ($t = -3.19$, $df = 167$, $p < .01$) and Time 4 ($t = -2.43$, $df = 156$, $p < .05$). The groups did not significantly differ on symptoms levels as a function of maternal depression history.

Data Analysis

All models were estimated using AMOS 4.0 (Arbuckle, 1997). This program is able to make use of all data points without deleting missing cases in either pair-wise or case-wise fashion. Model fit for the following analyses was evaluated using three

indices: χ^2 , the chi-square statistic, the Tucker-Lewis Index (TLI, Tucker & Lewis, 1973) and the root-mean-square-error of approximation (RMSEA; Browne & Cudeck, 1993). The TLI functions as a measure of the improvement in model fit that is compared to a null model in which all variables are uncorrelated. TLI scores greater than .95 represent good model fit (Hu & Bentler, 1999). As opposed to the TLI that measures relative fit compared to a baseline model, the RMSEA measures absolute fit between the observed and implied covariance matrices; values below .06 represent good fit (Hu & Bentler, 1999). Significant worsening in model fit among hierarchically-related models was tested using the standard chi-square difference test where a significant change in chi-square between two models indicates significantly worsened (or improved if model trimming) fit.

Model Comparisons

The base model used in the present study is presented in Figure 1. Consistent with the goal of examining reciprocal relations among peer stress and internalizing symptoms and externalizing symptoms over time, we included paths from each of these variables at one point in time to each of the other variables at the subsequent time point. Thus, there are three paths from each upstream variable to the downstream variables at the subsequent time point. This procedure allowed us to estimate both stability coefficients for each variable (i.e., the path from Time 1 peer stress to Time 2 peer stress) as well as cross-lagged paths between each variable (e.g., the path from Time 1 peer stress to Time 2 internalizing symptoms).

Given the moderately high correlation between the exogenous variables at the first measurement point, correlations were allowed between each of those three variables

for a total of three correlations to estimate. Because each of the variables was obtained using adolescent self-report, correlated disturbances were included between each of the three variables at each time point for a total of nine correlated disturbances. None of these correlations are shown in the figure for ease of presentation.

An inspection of the bi-variate correlations did not suggest that there were developmental differences in the relations among study variables so we placed time invariance constraints on each of the paths in the model to improve model parsimony. For example, as shown in Figure 1, the three paths leading from peer stress to internalizing symptoms are all labeled with the same variable e meaning that they were constrained to be equal across the measurement periods. The same constraints were placed on all other sets of paths in the model. None of the covariances were constrained to be equal over time.

Beginning with the fully reciprocal model, we performed two sets of multi-group comparisons. The first set examined whether gender moderated the fit of the reciprocal model. To perform this test, we compared two hierarchical models and tested the change in chi-square between them for significance. The first model allowed the paths for boys and girls to be estimated freely without cross-group constraints, whereas the second model constrained the paths for boys and girls to equal each other. The full model with the lack of constraints between groups fit the data well: $\chi^2(90, N = 240) = 130.95, p = .01$; RMSEA = .04; TLI = .99, whereas the model with equality constraints between the groups also fit the data well: $\chi^2(99, N = 240) = 134.81, p = .01$; RMSEA = .04; TLI = .99. The change in chi-square between the two models [$\Delta \chi^2(9, N = 240) = 3.86, p = ns$] reveals that the model with equality constraints between boys and girls did not fit the data

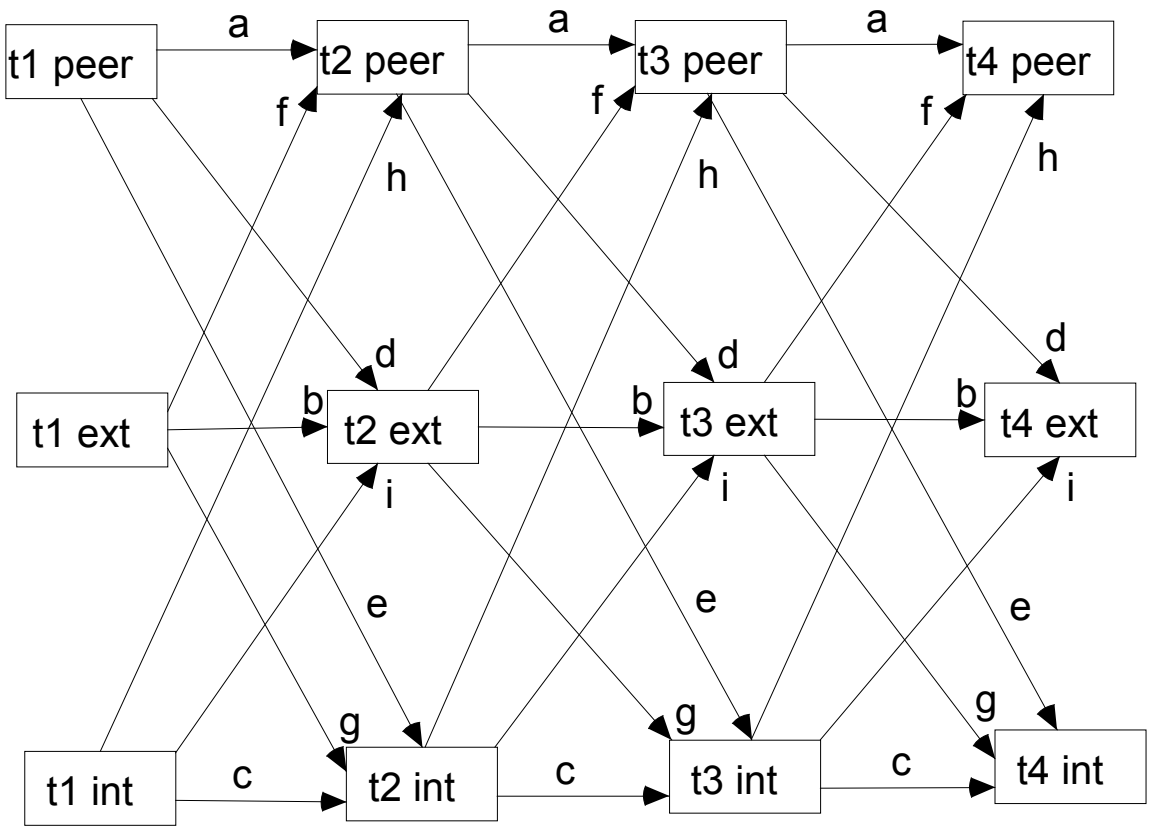


Figure 1

Reciprocal model for peer stress, internalizing symptoms, and externalizing symptoms

significantly worse than the model with equality constraints. For the sake of parsimony, we interpreted the model with cross-group constraints as well as used it as the base model for testing the alternative models: stress generation and stress exposure.

Once a model has been determined to fit adequately, the next step is to examine its lower-order parameters such as path coefficients. The path from peer stress to externalizing symptoms, from peer stress to internalizing symptoms, from internalizing symptoms to peer stress, and from externalizing symptoms to peer stress were all significant at the .05 level. All the paths were in the expected positive direction such that high levels of peer stress at one time-point predicted higher levels of internalizing and externalizing symptoms at the subsequent time-point and higher levels of internalizing and externalizing symptoms at one time-point predicted increases in peer stress at the subsequent time-points (see Table 2 for beta coefficients, standard errors, and *p*-values). Thus, this model provided preliminary support for the reciprocal model though some paths were stronger than others.

Although this model provides an acceptable fit to the data, there are several competing models that might fit the data equally well. Consistent with the model comparison approach, we wanted to compare the full reciprocal model with both the stress generation and stress exposure models. We obtained both of these models by constraining paths in the full reciprocal model to equal zero and used the chi-square statistic to compare relative model fit.

The stress generation model involved constraining the paths from peer hassles to internalizing and externalizing symptoms to equal zero (eliminating the *c* and *d* paths from the model in Figure 1). This model fit the data significantly worse than did the full

Table 2

Path coefficients, standard errors, and p-values for the two group comparison models.

Path	Gender		Risk status	
	Girls and boys	Low	High	
Peer Stress to Internalizing	.67 (.17)**	-.08 (.32)	.86 (.20)**	
Peer Stress to Externalizing	.40 (.18)*	-.37 (.36)	.63 (.21)**	
Internalizing to Peer Stress	.02 (.01)*	.06 (.02)**	.01 (.01)	
Externalizing to Peer Stress	.02 (.01)*	-.01 (.02)	.03 (.01)**	

Note. Because there was not a significant difference between the model that constrained the paths between boys and girls to be equal and the model that estimated both parameters separately, path coefficients, standard errors and p-values are presented for the total sample.

* $p < .05$, ** $p < .01$

reciprocal model [$\Delta\chi^2(2, N = 240) = 15.34, p < .001$]. The significant decrement in χ^2 suggests that the paths omitted from this model helped to recreate the sample covariance matrix and that the paths from peer stress to internalizing and externalizing symptoms were necessary to represent the data.

Next we tested the stress exposure model. Again, working from the reciprocal model, we constrained the paths from internalizing and externalizing symptoms to peer stress to equal zero by removing the *f* and *h* paths from the full model in Figure 1.

This reduced model also fit the data significantly worse than the full reciprocal model [$\Delta\chi^2(2, N = 240) = 22.23, p = .001$] suggesting that the omitted paths were necessary to minimize discrepancies between the sample and implied covariance matrices.

The second set of multi-group comparisons tested whether there were differences in the reciprocal model due to risk status. High-risk adolescents were those whose mothers had a history of mood disorders, whereas low risk were those whose mothers had no psychiatric history. Because the sample sizes of the two groups were quite different (185 high risk; 55 low risk), these results are considered exploratory.

We compared these groups in the same manner as analyses of gender. Two hierarchical models were tested: one in which the path coefficients for the high- and low-risk were estimated independently and one in which the paths for both groups were constrained to equal each other. The full model without risk level constraints fit the data well: $\chi^2(90, N = 240) = 145.44, p = .01$; RMSEA = .05; TLI = .98; the model with equality constraints also fit the data well: $\chi^2(99, N = 240) = 166.80, p = .01$; RMSEA = .05; TLI = .98. The change in chi-square between the two models [$\Delta\chi^2(9, N = 180) =$

21.36, $p < .05$] was significant, however. This indicates that the model that freely estimated parameters for the different groups fit the data significantly better than did the constrained model. Therefore, we interpreted the coefficients for the full model without the group equality constraints and used this model as the basis for comparison to the stress generation and stress exposure models.

For the low-risk group, the path from internalizing symptoms to peer stress were significant and positive indicating that higher levels of internalizing symptoms predicted increases in peer stress at the subsequent assessment time. For the high risk group, the path from peer stress to internalizing symptoms, from peer stress to externalizing symptoms, and from externalizing symptoms to peer stress were significant and positive. The path from internalizing symptoms to peer stress was not significant for the high risk group, however. Table 2 shows the complete path coefficients, standard deviations, and p - values for the high and low risk groups.

The next step was to compare the full reciprocal model with paths separately estimated for high and low-risk adolescents to the stress generation and stress exposure models. We followed the same procedure for testing alternative, nested models as with gender. For the risk-status comparisons, both the stress-generation model [$\Delta\chi^2(11, N = 240) = 35.73, p < .05$] and the stress-exposure model [$\Delta\chi^2(11, N = 240) = 41.58, p < .05$] fit the data significantly worse than the reciprocal model. These model fit decrements suggest that all cross-lag paths were needed to represent the data, and that the reciprocal model provided the best representation of the inter-relations among peer stress and internalizing symptoms and externalizing symptoms.

CHAPTER IV

DISCUSSION

The current study investigated the relations between peer stress and internalizing and externalizing symptoms across four years of adolescence. Results indicated that the reciprocal model provided the best fit to the data compared to either the stress exposure or stress generation models. The reciprocal model fit equally well for girls and boys but varied as a function of risk status. Children whose mothers had histories of mood disorders (high risk) had more significant relations among stress and symptoms than did children whose mothers had no history of psychological disorder (low risk).

Both the stress exposure and stress generation models are uni-directional with one set of variables predicting another over time. The stress exposure model posits that stress at one time will predict increases in symptoms at the next time point, and the stress generation model predicts that symptoms at one time will predict increases in stress at subsequent time points. In contrast, the reciprocal model is bi-directional and predicts that both sets of variables serve as predictor and outcome variables across time. The reciprocal model provided a significantly better fit to the data than did either the stress exposure or stress generation models indicating that all the cross-lag paths between stress and symptoms were necessary to best represent the relations among peer stress and internalizing and externalizing symptoms. These results are consistent with other studies that have tested the reciprocal model (Cohen et al., 1987; Kim et al., 2003) and found that

stress predicted increases in symptoms after controlling for prior symptom levels, and symptoms predicted increases in stress after controlling for prior stress levels.

The differences between the current investigation and these other studies, however, highlight the contribution of this study to this literature. Cohen et al. (1987) found support for the reciprocal model using measures of only depressive and anxious symptoms, although depressive symptoms were positively related to stress whereas anxious symptoms correlated negatively with subsequent stress. The present study used a composite index that included anxious, depressive, and somatic symptoms and found positive relations between these internalizing symptoms and stress in both directions. Future studies should further explore possible differences in the relation between stress and these different types of internalizing symptoms as well as clinical diagnoses.

Kim et al. (2003) examined the relation of stress and both internalizing and externalizing symptoms, although they tested the models separately for each symptom type. Because of the often found co-occurrence of internalizing and externalizing symptoms, the present study included them in the same model. In addition, the measure of externalizing symptoms used by Kim et al. was based on delinquent activities only, whereas we examined delinquent and aggressive symptoms in a single index of externalizing problems. Thus, the findings of the present study replicate and extend the findings of Kim et al. by showing further support for the reciprocal model with regard to a measure of externalizing problems that included both delinquency and aggression.

In addition, the present study focused on a specific type of stress rather than a general index of stressful life events or daily hassles across multiple domains. Interpersonal stress and peer stress in particular have been shown to be especially

relevant for adolescents (Isakson & Jarvis, 1998). The findings of the current study provide further support for the notion of a vicious interpersonal cycle that can develop between social stressors and symptoms and vice versa (Coyne, 1976).

With regard to gender, no differences were found in the reciprocal model. The model fit equally well for boys and girls suggesting that the same patterns among variables held for both. These results are consistent with other studies that have not found gender differences in the relation between stress and symptoms (e.g., Barrerra et al., 2002; Kim et al., 2003; Santa-Lucia et al., 2000; Wadsworth & Compas, 2002). Gender differences in this relation that have been found have been inconsistent, with some studies showing a greater relation for girls than boys (LaGreca & Lopez, 1998; Rudolph, 2002) and others showing a greater relation for boys than girls (Compas, 1986; Rudolph & Hammen, 1991). Moreover, Seiffgre-Krenke and Stemmler (2002) found that gender differences may change with development such that major life events were significantly correlated with depressive symptoms in boys at age 14, and peer stress was related to depressive symptoms in girls at age 17. Finally, differences in results regarding gender do not appear to be explained solely by the type of outcome measured.

Externalizing symptoms have been found to be related to stress in girls (Jackson & Warren, 2000) and to internalizing symptoms in boys (Rudolph & Hammen, 1991).

In the present study, the measure of peer stress included items concerning rejection, loneliness, withdrawal, transition, and victimization. Rudolph (2002) found gender differences in the relation between specific types of peer stress and symptoms with girls showing more anxious and depressive symptoms in response to peer group and friendship stress. It is possible that had we had measures of each of the different forms of

peer stress rather than a single measure that combined all of them, gender differences might have emerged.

Regarding risk status, more significant relations between stress and symptoms were found for offspring of mothers with histories of mood disorders than for children of mothers with no psychiatric history. These results should be considered preliminary, however, because of the relatively smaller number of low risk participants. For the low-risk group, the only significant path was from internalizing symptoms to peer stress in grades 8 and 9. Thus, the low-risk youth may have been less adversely affected by stress than the high-risk group. This is consistent with the study by Malcarne, Hamilton, Ingram, and Taylor (2000) who showed that stress was not associated with symptoms in children of non-depressed mothers. The one significant path for the low-risk group was consistent with the stress-generation model and studies that have shown that higher levels of depressive symptoms predicted increases in stressful events in community samples of mothers who were less likely to have experienced psychological disorder (Davila et al., 1995; Pothoff et al., 1995).

For the high-risk sub-group, there were significant paths from externalizing symptoms to peer stress and from peer stress to internalizing and externalizing symptoms. These results are similar to others showing that children of depressed mothers are more likely to experience distress in relation to stressors (Hammen & Goodman-Brown, 1990; Hammen et al., 2003, Malcarne et al., 2000). Hammen and Goodman-Brown (1990), for example, found that children whose depressive symptoms increased over 6 months were more likely to have experienced stressful interpersonal events and to have mothers with histories of mood disorders

Another difference between the current study and those by Cohen et al. (1987) and Kim et al. (2003) was the samples. Participants in the Kim et al. (2003) study were selected because of the economic strain in their community; Cohen et al. (1987) also used a community sample to examine relations between stress and symptoms. In contrast, the present study used a high risk design involving offspring of depressed and non-depressed mothers. An advantage of this sample is that it included children who were likely to show greater variability on the variables of interest (e.g., peer stress, symptoms). A limitation, however, is that the findings might not generalize to a more general normative sample. In addition, the smaller size of the low risk sample may have reduced the power to detect effects for this group. The overall finding of support for the reciprocal model in the current investigation, however, was consistent with the results of the two other studies that used community samples.

The relatively small sample size also prevented us from being able to test developmental hypotheses regarding the relations between peer stress and symptoms over time. Kim et al. (2003) found shifts in these relations throughout adolescence. The current study did not have enough power to estimate these effects separately for each time-point. The correlational analyses, however, did not reveal any developmental differences in the relations between stress and symptoms over time.

Another limitation of this study was that information about peer stress and symptoms was provided by the same informant. Thus it is possible that some of the observed relations among the constructs were due to method variance. Nevertheless, even with data from a single informant it was possible to make meaningful comparisons of the three different models of the relations between peer stress and adolescent symptoms. Future

studies should explore whether these findings generalize to data obtained from multiple informants.

In addition, the measure of peer stress used in the current study was an 11-item subscale from a general measure of stress. Other research (e.g., Rudolph, 2002) has shown that the relation between peer stress and outcomes differ according to the specific type of peer stress studied., Therefore, future studies should use a measure that includes several dimensions of peer stress such as peer rejection, peer conflict, and problems within friendships.

Finally, future studies should explore possible moderators and mediators of the relations between peer stress and symptoms. Variables such as coping, social support, and cognitive appraisals have been shown to affect these relations (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001; Compas, Malcarne, & Fondacaro, 1988; Goodman, Gravitt, & Kaslow, 1995; Panak & Garber, 1992). For example, Goodman et al. (1995), found that children experiencing high levels of stress who were better problem solvers had lower levels of depressive symptoms than children with the same level of stress who were worse problem solvers.

In summary, the present study provided further support of a reciprocal model of the relation between peer stress and adolescent internalizing and externalizing symptoms during early adolescence. This study adds to the literature by focusing on a specific type of stress that is particularly relevant for adolescents, peer stress, and the use of a high-risk sample of individuals who are particularly likely to have vulnerabilities with regard to peer stress and symptoms.

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