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## What Happens When Youth Talk About Their Problems? Co-Rumination as a Mechanism of Stress Generation

Jaclyn T. Aldrich

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What Happens When Youth Talk About Their Problems?

Co-Rumination as a Mechanism of Stress Generation

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A dissertation submitted in partial fulfillment of the requirements for the degree of

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In

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## Table of Contents

<b>List of Tables .....</b>	<b>v</b>
<b>List of Figures.....</b>	<b>vi</b>
<b>Abstract.....</b>	<b>viii</b>
<b>Chapter I: Introduction and Literature Review .....</b>	<b>1</b>
Adolescent Depression .....	3
Stress Generation Theory of Depression .....	4
Stress Generation and Depression .....	6
Response Styles Theory .....	7
Rumination and Depression .....	8
Rumination and Stress Generation .....	8
Co-Rumination as a Stress Generation Mechanism .....	9
Defining Co-Rumination.....	9
Co-Rumination and Depressive Symptoms.....	10
Co-Rumination and Stress Generation .....	10
Current Study.....	12
<b>Chapter II: Method.....</b>	<b>13</b>
Participants .....	13
Procedure.....	13
Baseline laboratory visit.....	14
Follow-up laboratory visits .....	14
Measures.....	14
Demographic variables.....	14
Episodic life stress.....	14
Co-rumination .....	15
Depressive symptoms.....	16
Data Analytic Plan.....	16

<b>CHAPTER III: Results</b> .....	<b>21</b>
Data Preparation and Descriptive Analyses .....	21
Path Analysis Models .....	25
Post-Hoc Analyses.....	36
<b>CHAPTER IV: DISCUSSION</b> .....	<b>44</b>
Limitations.....	47
<b>References</b> .....	<b>49</b>

## List of Tables

Table 1. Descriptive statistics for primary variables

Table 2. Correlations between primary variables

Table 3. Fit statistics of path analysis models

Table 4. Indirect effects of all path analysis models

Table 5. Descriptive statistics for post-hoc analyses

Table 6. Fit statistics for post-hoc models

Table 7. Indirect effects of post-hoc models.

## List of Figures

Figure 1. Proposed bidirectional associations between co-rumination, acute stress, and depressive symptoms.

Figure 2. Model 1: Proposed path diagram of the effect of co-rumination on depressive symptoms through interpersonal, dependent stress.

Figure 3. Model 2: Proposed path diagram of the effect of co-rumination on depressive symptoms through interpersonal, independent stress.

Figure 4. Model 3: Proposed path diagram of the effect of co-rumination on depressive symptoms through non-interpersonal stress.

Figure 5. Model 4: Proposed path diagram of the effect of depressive symptoms on co-rumination through interpersonal, dependent stress.

Figure 6. Model 5: Proposed path diagram of the effect of depressive symptoms on co-rumination through interpersonal, independent stress.

Figure 7. Model 6: Proposed path diagram of the effect of depressive symptoms on co-rumination through non-interpersonal stress.

Figure 8. Path analysis model 1 of associations between co-rumination, interpersonal dependent stress, and depressive symptoms

Figure 9. Path analysis model 2 of associations between co-rumination, interpersonal independent stress, and depressive symptoms

Figure 10. Path analysis model 3 of associations between co-rumination, non-interpersonal stress, and depressive symptoms

Figure 11. Path analysis model 4 of associations between depressive symptoms, interpersonal dependent stress, and co-rumination

Figure 12. Path analysis model 5 of associations between depressive symptoms, interpersonal independent stress, and co-rumination

Figure 13. Path analysis model 6 of associations between depressive symptoms, interpersonal independent stress, and co-rumination

Figure 14. Post-hoc path analysis model 7 of associations between depressive symptoms, interpersonal dependent stress, and co-rumination

Figure 15. Post-hoc path analysis model 8 of associations between depressive symptoms, interpersonal independent stress, and co-rumination

Figure 16. Post-hoc path analysis model 9 of associations between depressive symptoms, non-interpersonal stress, and co-rumination



## Abstract

During adolescence, rates of depression increase significantly, necessitating understanding of interpersonal and intrapersonal factors that contribute to the occurrence of depressive symptoms. Prominent theories of depression, such as stress generation theory, suggest that depressed individuals experience more interpersonal stress that is dependent on their own actions or behavior. The current study sought to examine the role of co-rumination in the generation of stress and development of depression over the course of a year. Participants were 150 adolescents (48.7% female, 77.5% Caucasian) ages 11 to 14 years old ( $M = 13.03$ ,  $SD = 0.93$ ). Three models assessed the directional relationship between co-rumination, three types of acute stress (interpersonal dependent, interpersonal independent, and non-interpersonal) and depressive symptoms; three models assessed the directional relationship between depressive symptoms, three types of acute stress, and co-rumination. Results were largely unresponsive of hypotheses; co-rumination did not consistently predict any type of acute stress, though T2 co-rumination predicted T3 interpersonal dependent stress in one model,  $B(SE) = -.15(.07)$ ,  $p = .02$ . Depressive symptoms did predict interpersonal dependent stress across more timepoints (e.g., T1 to T2,  $B[SE] = .23[.10]$ ,  $p = .02$ ) compared to interpersonal independent and non-interpersonal stress. Acute stress across did not mediate the relation between co-rumination and depressive symptoms or depressive symptoms and co-rumination. Post-hoc analyses simultaneously examined the previously separate directional relationships addressed the first six models. Results of these three models displayed a similar pattern of findings, with depressive symptoms predicting the occurrence of interpersonal dependent stress, ( $B[SE] = .29[.10]$ ,  $p = .005$ ) but not interpersonal independent ( $B[SE] = .06[.08]$ ,  $p = .45$ ) or non-interpersonal stress ( $B[SE] = .13[.10]$ ,  $p = .19$ ). Unexpectedly, interpersonal dependent stress negatively predicted co-rumination ( $B[SE] = -$

.20[.09],  $p = .02$ ). No stress variable mediated the relation between co-rumination and depressive symptoms or depressive symptoms and co-rumination. Overall, results suggest that co-rumination may not be a mechanism that generates interpersonal or non-interpersonal stress, supporting other prior research that has suggested the co-rumination may be a moderating factor in the relationship between stress and depressive symptoms. However, methodological concerns such as low sample size may have limited the current study.

## **Chapter I: Introduction and Literature Review**

Depression is a major public health concern, as prevalence rates of depression significantly increase during adolescence. This is troubling, as an episode of depression during adolescence increases the chance of both depressive symptoms and depressive episodes during adulthood (Kim-Cohen et al., 2003). The occurrence of depressive symptoms during adolescence can influence academic performance, psychosocial development, and interpersonal relationships (Essau & Chang, 2009) and increases risk for substance use (Marmorstein, 2010) and suicide attempts (Nock et al., 2013). Given that the prevalence of depression rises sharply during middle to late adolescence (Hankin et al., 1998; Kessler et al., 2001), it is necessary to understand vulnerabilities that contribute to the onset of depression during this time. The stress generation model of depression suggests that one pathway to depression is the bidirectional relationship between the occurrence of stressful events and depressive symptoms during adolescence (Harkness & Stewart, 2009).

Hammen (1991, 2006) proposed that the characteristics of depressive symptoms lead depressed individuals to experience an increased number of stressful life events. Specifically, the behavioral tendencies and cognitions associated with depression cause individuals to generate stress within in their lives, primarily within the interpersonal domain. The occurrence of stressful interpersonal events also seems to increase the likelihood of experiencing depressive symptoms, creating a bidirectional relationship. Prior research supports this effect in children and adolescents as well as adults (see Liu, 2013 for review). During adolescence, interpersonal conflict frequently occurs between youth and their friends and parents, whereas non-interpersonal stress includes failure to achieve goals and poor academic performance. Although there is empirical support for the stress generation model of depression in adolescence, research

is necessary to examine *how* adolescent's interpersonal behaviors contribute to the generation of interpersonal conflict and stress.

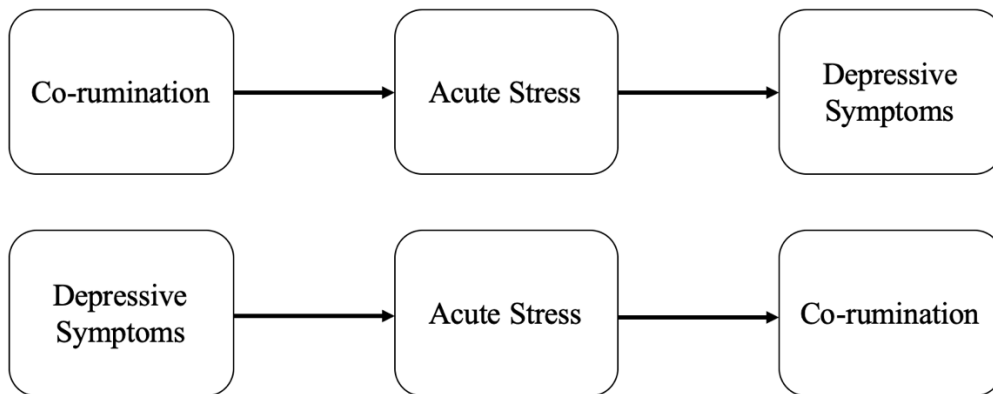
One interpersonal behavior that may contribute to stress generation is co-rumination. Co-rumination (Rose, 2002) is the tendency to engage in excessive problem talk with other individuals. Problem talk includes extensive discussion of problems, including the causes and consequences of problems, and focusing on negative emotions and distress. Rose (2002) described co-rumination as an interpersonal manifestation of rumination. Co-rumination predicts depressive symptoms concurrently (Calmes & Roberts, 2008; Starr & Davila, 2009) as well as the onset of depressive symptoms over time (Stone et al., 2011). Rose and colleagues (2017) found that co-rumination exacerbated stress generation among depressed adolescent girls, but not adolescent boys. This finding suggests that co-rumination moderates the relation between depressive symptoms and stress generation. However, Hankin and colleagues (2010) suggested that co-rumination may be an interpersonal behavior that contributes directly to stress generation and depressive symptoms among adolescents, indirectly influencing the course of both stress and depressive symptoms over time.

In this study, I propose to examine the relations between stress generation, co-rumination, and depressive symptoms among adolescents over the course of the year. Adolescents may co-ruminate with others about their distress, causing interpersonal tension or problems and possibly inhibiting more adaptive processes what might help adolescents manage such distress. This may lead to higher levels of interpersonal stress, thus resulting in depressed mood. Additionally, depressed adolescents likely experience more stress, increasing the likelihood that they would co-ruminate with peers about this interpersonal distress. Thus, I hypothesize that stress will act

as a mediator between co-rumination and depressive symptoms and depressive symptoms and co-rumination (see Figure 1).

Figure 1

*Proposed bidirectional associations between co-rumination, acute stress, and depressive symptoms.*



### **Adolescent Depression**

Depression during adolescence occurs at significantly higher rates compared to childhood and can have lasting effects across the lifespan. The Substance Abuse and Mental Health Administration (2017) estimates that within one year, 12.8% of adolescents aged 12 to 17 experience a major depressive episode. Adolescents who experience recurrent episodes of major depression before age 18 are likely to have more severe depressive episodes across the lifespan and poorer functioning in multiple domains (Hammen et al., 2008; Glied & Pine, 2002). Adolescent depression is also associated with higher rates of suicidal behaviors compared to depressive episodes during adulthood (Rohde et al., 2013). Thus, it is imperative to identify risk factors for adolescent depression in order to intervene appropriately.

Depression is characterized as a disorder of affect dysregulation (Forbes & Dahl, 2005). According to the *DSM-5* (American Psychiatric Association, 2013), a diagnosis of major

depressive disorder requires at least five of the following symptoms, occurring within the same two-week period: persistent depressed mood, loss of interest or pleasure in daily activities, significant appetite decrease or increase, insomnia or hypersomnia, fatigues/loss of energy, feelings of worthlessness or inappropriate guilt, difficulty concentrating, and recurrent thoughts of death or suicidal ideation. The occurrence of these symptoms must represent a change in functioning, be associated with clinically significant distress or impairment, and not due to a substance or medical illness. For children and adolescents, depressed mood may also be expressed as irritability. Just as it is necessary to study the occurrence of depressive disorders, it is also necessary to understand the occurrence of depressive symptoms.

Depression represents a continuum of symptoms differing in severity and duration. For example, subsyndromal depression occurs when an individual experiences at least one of the nine diagnostic symptoms for at least two weeks, without meeting criteria for a major depressive episode. Even individuals who present with subsyndromal depression experience a significant reduction in health status, above and beyond other significant predictors of health, compared to non-depressed individuals (Ayuso-Mateos et al., 2010). Subthreshold depression is associated with similar risk factors and risk for impairment as depressive episodes (Judd et al., 1994). This supports the study of depressive symptoms as opposed to only depressive episodes or disorders. Accordingly, depressive symptoms can be studied within the context of theories explaining the occurrence of depressive episodes.

### **Stress Generation Theory of Depression**

The stress generation theory of depression (Hammen, 1991, 2006) is a transactional model highlighting the bidirectional relationship between stress and depressive symptoms. According to this model, depressed and depression-prone individuals are not

passive recipients of stressful events, but rather active generators of these events. As such, these individuals not only experience more stressful life events but also generate stress within their lives (Hammen, 1991, 2006). This generation of stress is in part due to the person's characteristics as well as the characteristics of the depressive symptoms. Hammen (2006) explained that individuals who experience recurrent episodes of depression possess certain beliefs or characteristics, or engage in certain behaviors, which both make them vulnerable to depression and contribute to life stress. In turn, life stress may cause or intensify depressive symptoms. Hammen (2006) also noted that this does not mean that depressed individuals *cause* their own depression, but rather that there is an important bidirectional relationship between depressive symptoms and life stress, in which individuals are active participants. It is also important to note that within this theory, specific kinds of stress are more likely to both be generated by depressed individuals as well as contribute to depression.

Stressful life events can be characterized based on the life domain of the event and the role of the individual within the event. Relevant life domains include interpersonal versus non-interpersonal events. Interpersonal events are those that involve at least two individuals (Rudolph & Hammen, 1999), such as a child and parent, and that directly affect the relationship between the two persons. Examples include two children completing an activity together, an argument between a child and parent, and conflict between two parents. Non-interpersonal events are those that do not involve an interaction between two individuals, such as failing a test or performing in a recital. Next, events can be categorized based on the extent to which an individual's behavior or personal characteristics contribute to the event's occurrence (Rudolph & Hammen, 1999). Independent events, or fateful events, are events in which occurrence of the event is separate from the particular individual. Conversely, dependent events occur partially or

completely due to an individual's behavior. The distinction between independent and dependent and between interpersonal and non-interpersonal is important when considering what types of stressful events contribute to depressive symptoms.

### **Stress Generation and Depression**

Starting with Hammen's seminal study (Hammen, 1991), researchers have consistently demonstrated that depressed individuals generate more dependent, interpersonal events compared to non-depressed individuals. This means that depressed individuals experience more interpersonal stressful events that occur, in part, to their own behavior or characteristics (Hammen, 2006). This suggests that depressed individuals effect their environment in such a way that they generate interpersonal stress within their lives. Depressed individuals also generate non-interpersonal stress as well (Flynn et al., 2010; Rudolph et al., 2009); however it is particularly the occurrence of dependent, interpersonal events that predict the occurrence of depressive symptoms (Auerbach et al., 2014; Rudolph et al., 2009).

The stress generation theory is supported by a significant body of research in various populations (see Liu, 2013 for review). Among adolescent populations, generated interpersonal stressors have predicted depressive symptoms over time (Davila et al., 1995; Hankin et al., 2007; Little & Garber, 2005; Rudolph et al., 2009). Rudolph and colleagues (2009) found that depressed adolescent girls generated interpersonal stress, which prospectively predicted depressive symptoms and partially explained the continuity of depressive symptoms over time.

Depressive symptoms also likely contribute to increased stressful experiences, such as those discussed previously (Hammen, 1991; Rudolph, 2008). Among youth, symptoms of depression inhibit or interfere with social activities; sadness, irritability, or affect dysregulation may create tension within a youth's interpersonal relationships (Rudolph et al., 2009).



Interactions with a depressed youth may be unpleasant or unrewarding, discouraging peers from engaging with the adolescent. Additionally, the cognitive characteristics of depression, such as guilt or hopelessness, or cognitive styles associated with depression may affect how a youth interacts with family, friends, or teachers. Other interpersonal behaviors or individual characteristics likely contribute to the generation of interpersonal stress.

Though the theory of stress generation has garnered significant support in adult and adolescent populations, it does not take into consideration other specific personal characteristics or behaviors that may contribute to stress generation. For example, research has demonstrated that daily reassurance seeking predicts interpersonal stress generation over time (Eberhart & Hammen, 2009). How an individual responds to personal distress and interpersonal conflict will affect not only the individual's mood, but likely the duration and intensity of the interpersonal stress as well. One theory that may shed light on individual characteristics or processes that contribute to stress generation is response styles theory.

### **Response Styles Theory**

Response styles theory suggests that the way in which individuals respond to their distress affects the occurrence and severity of depressive symptoms (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 1993). Nolen-Hoeksema (1991) proposed two styles of responding: distraction and rumination. Rumination is defined as “repetitively and passively focusing on the symptoms of distress and on the possible causes and consequences of these symptoms” (Nolen-Hoeksema et al., 2008, p. 400). The process of rumination—intense, perseverative thoughts about problems, feelings, and distress—causes or intensifies depressed mood. Distraction, however, is defined as the use of pleasant or neutral activities to remove attention from distress or symptoms of distress (Nolen-Hoeksema, 1991). Distraction does not necessarily mean that an

individual will engage in more adaptive responses, but distracting responses do not predict depressive symptoms to the same degree that rumination does (Nolen-Hoeksema et al., 1993; Nolen-Hoeksema et al., 2008). Given the effect of rumination on depressive symptoms, the remainder of this section will focus on that relationship.

### ***Rumination and Depression***

Among adult and adolescent populations, rumination consistently predicts or is associated with an individual's depressive symptoms over time. Specifically, this relationship has been supported in samples of children (Abela et al., 2007; Abela et al., 2002), young adolescents (Abela et al., 2007; Burwell & Shirk, 2007; Hankin, 2008; Jose & Brown, 2007) and older adolescents (Abela et al., 2009; Nolen-Hoeksema et al., 2007). There are several reasons why rumination has such an effect on an individual's depressive symptoms. Rumination maintains an individual's focus on symptoms and distress, which may prevent that individual from trying to engage in other behaviors that may be constructive or distracting (Nolen-Hoeksema et al., 2008). Ruminators also have difficulty engaging in effective problem solving due to interference of rumination; rumination rarely results in effective solutions even though ruminators believe it will (Papageorgiou & Wells, 2001). Furthermore, research has demonstrated a relationship between rumination and stress.

### ***Rumination and Stress Generation***

Rumination likely initiates or maintains processes that contribute to the generation of stress. For example, rumination may interfere with instrumental social processes that promote supportive interpersonal relationships (Nolen-Hoeksema & Davis, 1999). After a stressful event occurs, rumination may also prolong an individual's distress concerning the event (McLaughlin & Nolen-Hoeksema, 2012). Research suggests that rumination exacerbates the relationship

between distress and depressive symptoms, prospectively, among adolescents (Abela et al, 2010) and moderates the relation between negative events and future depressive symptoms (Abela & Hankin, 2011). Addressing the role of rumination as an intrapersonal process that contributes to stress generation does not fully capture related interpersonal behaviors that also likely influence stress generation.

Among adolescents, the tendency to ruminate is associated with the tendency to co-ruminate (Rose, 2002). Although co-rumination has been described as the interpersonal manifestation of rumination, prior research has demonstrated that rumination and co-rumination are distinct processes (Calmes & Roberts, 2008), such that co-rumination significantly contributes to the occurrence of depression during adolescence above and beyond rumination (Stone et al., 2011). Co-rumination also reinforces the individual tendency to ruminate (Aldrich et al., 2019, Stone & Gibb, 2015), supporting the idea that co-rumination is a separate process and not an expression of internal rumination. Given that co-rumination appears to emerge during adolescence (Hankin et al., 2010) in conjunction with depressive symptoms, co-rumination may be one mechanism through which adolescents generate interpersonal stress.

### **Co-Rumination as a Stress Generation Mechanism**

#### ***Defining Co-Rumination***

Whereas rumination is an internal focus on distress, co-rumination is defined as excessive discussion of problems between two or more individuals, including rehashing the problem, conjecturing about causes and consequences of the problems, and focusing on negative emotions (Rose, 2002). The process of co-rumination involves discussing the same problem repeatedly, mutual encouragement of problem discussions, and a noticeable lack of problem solving. Characteristics of co-rumination like responding supportively to a friend or asking questions

about a problem reinforce the tendency to co-ruminate within a dyad (Rose et al., 2014). Among children and adolescents, co-rumination is associated with increased friendship quality (Rose, 2002; Rose et al., 2007) and internalizing symptoms, such as depression and anxiety (see Spendelov et al., 2017 for review).

### ***Co-Rumination and Depressive Symptoms***

Like rumination, co-rumination has been consistently associated with depressive symptoms. In a recent meta-analysis, Spendelov and colleagues (2017) found that co-rumination has a moderate, significant effect on depressive symptoms across all ages. Specifically, among children and adolescents, studies have found that co-rumination predicts depressive symptoms concurrently (Rose, 2002; Rose et al., 2014; Starr & Davila, 2009) and prospectively (Hankin et al., 2010; Rose et al., 2007; Stone et al., 2011). Co-rumination has also been associated with a lifetime history of depressive episodes (Stone et al., 2010). Additionally, youth with major depressive disorder tend to co-ruminate more and problem-solve less daily compared to healthy peers (Waller et al., 2014). Rose (2002) also believed that co-rumination within friendships would reinforce the individual tendency to rumination. Stone and Gibb (2015) confirmed this hypothesis in a sample of undergraduate students; co-rumination predicted increases in individual rumination over time, indirectly increasing depressive symptoms. Thus, co-rumination is an interpersonal behavior that contributes to the onset and maintenance of depressive symptoms.

### ***Co-Rumination and Stress Generation***

Co-rumination appears to have a relationship with the process of stress generation. Several studies have assessed the moderating role of co-rumination; White and Shih (2012) found that co-rumination moderated the relationship between daily stressful events and

depressed mood, such that higher levels of co-rumination were related to higher depressed mood. Rose and colleagues (2017) found that, among adolescents, co-rumination interacted with depressive symptoms to predict interpersonal stressors, but not non-interpersonal stressors, suggesting that co-rumination exacerbates stress generation. Neither of these studies addressed the role of co-rumination in the bidirectional relationship between depression and stress. However, Hankin and colleagues (2010) found that co-rumination predicted interpersonal stressors; more specifically, co-rumination predicted dependent interpersonal stressors but not dependent non-interpersonal or independent stressors. Additionally, Hankin and colleagues (2010) found support for a transactional model of stress generation. Internalized symptoms and interpersonal stress predicted co-rumination over time, while co-rumination predicted internalizing symptoms through generated interpersonal stressors. This suggests that co-rumination is one interpersonal behavior that generates stress within the lives of adolescents, as opposed to exacerbating stress generation or depressive symptoms.

Co-rumination clearly plays a role in the development of depressive symptoms and likely plays a role in stress generation. Adolescents who engage in co-rumination with peers fail to use adaptive problem solving skills, which would otherwise allow the adolescent to resolve interpersonal problems effectively. Youth who respond to everyday social stressors ineffectively tend to generate more interpersonal stress over time compared to those who utilize effective stress management skills (Flynn & Rudolph, 2011). Co-rumination also reinforces the individual tendency to ruminate (Stone & Gibb, 2015), increasing the likelihood that the youth will experience and dwell on negative affect and distress. This perpetuating cycle between stress, co-rumination, and depressive symptoms brings together two leading theories of depression, but requires more research to understand.

## **Current Study**

In this dissertation, I aimed to address gaps within the stress generation and response styles theories of depression by examining the relations between co-rumination, stress generation, and depressive symptoms in an adolescent population. Further, I aimed to find support for a transactional model of stress generation, in which stress mediates the relationship between co-rumination and depressive symptoms and depressive symptoms and co-rumination over time. Given previous research, I hypothesize the following:

**Hypothesis 1.** Co-rumination will predict interpersonal, dependent stressors more strongly compared to interpersonal, independent and non-interpersonal stressors prospectively.

**Hypothesis 2.** Interpersonal, dependent stress will mediate the relationship between co-rumination and depressive symptoms prospectively more strongly compared to interpersonal, independent and non-interpersonal stressors.

**Hypothesis 3.** Depressive symptoms will predict interpersonal, dependent stressors more strongly compared to interpersonal, independent and non-interpersonal stressors prospectively.

**Hypothesis 4.** Interpersonal, dependent stress will mediate the relationship between depressive symptoms and co-rumination prospectively more strongly compared to interpersonal, independent and non-interpersonal stressors.

## Chapter II: Method

### Participants

The current study utilized a participant pool from an ongoing longitudinal study investigating cognitive, affective, and physiological vulnerabilities of adolescent depression. Path values from Hankin and colleagues (2010) provided estimates to conduct a power analysis, in which their results indicated low magnitude of regression pathways (e.g., co-rumination to stress,  $B = .15$ ; stress to depressive symptoms,  $B = .18$ ). Utilizing this information, and assuming a direct power of approximately .45, and indirect power of approximately .06, guidelines from Wolf and colleagues (2013) on sample sizes for structural equation models suggested that a sample size of 130 would be adequate.

Participants were 150 adolescents (51.3% female) recruited from middle schools in the Pacific Northwest. Participants ranged in age from 11 to 14 years old ( $M = 13.03$ ,  $SD = 0.93$ ). Approximately 77.5 % of adolescents were Caucasian; 9.3% identified as biracial or other; 7.8% were Asian-American; 1.6% were African-American; 1.6% were Hispanic/Latin, and .8% were Native American/Pacific Islander.

### Procedure

Recruited participants completed an eligibility phone screening. Parents were interviewed via phone to determine if the youth met criteria for the study. Eligible youth had to be able to read English and not have significant learning or attention problems that may interfere with the youth's ability to remain seated and relatively still for 30 minutes at a time. Youth taking stimulant medications had to be able to abstain from the medication for 36 hours prior to the laboratory visit. Parents and study staff jointly determined the participant's eligibility based

on the criteria above, as well as the parents' ability to read and answer questions in English. Eligible youth were invited to participate in a baseline laboratory visit.

### ***Baseline laboratory visit***

Eligible youth and their parents completed a university-based laboratory visit, which took approximately four hours. Parents provided consent and adolescents provided assent prior to the start of the visit. During the visit, adolescents and their parent were interviewed separately to gather information on acute stressful events that may have occurred for the youth and their family over the past six months. Adolescents reported on their depressive symptoms and tendency to co-ruminate via online survey. Youth were paid \$35 and parents \$50 for their participation in the first laboratory visit.

### ***Follow-up laboratory visits***

Youth completed three follow-up laboratory visits every three months following the baseline visit. At each visit, youth and parents were interviewed separately to assess the presence of acute stressful events that occurred during the three-month time period between visits. Adolescents reported on depressive symptoms and co-rumination via questionnaires at each visit. Parents were compensated \$25 for each follow up visit; youth were paid \$15.

## **Measures**

### ***Demographic variables***

Demographic variables including age, gender, race, and ethnicity were collected at the first laboratory visit. Age was also collected at every follow-up visit.

### ***Episodic life stress***

Episodic life stress was assessed using the Children's Life Stress Interview (LSI; Rudolph & Hammen, 1999). The LSI is a semi-structured interview and was administered to



youth and parents separately by trained research assistants. The LSI uses the contextual threat method (Brown & Harris, 1978) to determine the occurrence and impact of episodic or acute stressful events within a designated time frame. Probes within the LSI allowed researchers to gather information on specific life events, including the nature and date of each event, whether the event was expected or not, how long the event lasted, and consequences of the event. Information about each event from parent and child was pooled and presented to a coding team; events only reported by parent or child were also presented. The trained coding team rated each event based on negative impact, positive impact, dependence/independence, and goal attainment, and categorize the event as interpersonal or non-interpersonal. Based on the ratings, indices were created based on the three types of acute stressors: interpersonal-dependent, interpersonal-nondependent, and non-interpersonal. Each index was computed through a count of the respective type of stressor where the negative impact rating was higher, thus representing the number of each type of negative stressor the child experienced in the designated time period. As the LSI was administered at each time point, the index scores at the baseline laboratory visit represented stressors experienced in the six months prior to the first visit, whereas index scores at each follow-up represented stressors experienced in the time between each visit, typically four months.

### ***Co-rumination***

Co-rumination was assessed using the Co-Rumination Questionnaire (CRQ; Rose, 2002). The original CRQ is a 27-item measure that assess the extent to which youth typically co-ruminate with same-sex friends. In the current study, a modified 16-item CRQ provided by Calmes and Roberts (2008) was used due to concerns of time burden on participants. Instructions for the modified measure ask participants to consider how they usually are in all of

their close relationships, not just same-sex friendships. Participants responded to each item on a 5 point Likert scale from 1 (*Not at all true*) to 5 (*Really true*). Both the original CRQ and the modified CRQ have demonstrated adequate internal consistency ( $\alpha = .94$ ; Calmes & Roberts, 2008; Rose, 2002), adequate test-retest reliability and validity (Hankin et al., 2010; Rose et al., 2007). The CRQ was administered at each laboratory visit; the modified CRQ demonstrated adequate internal consistency at each timepoint ( $\alpha = .92 - .94$ ).

### ***Depressive symptoms***

Depressive symptoms were measured using the Children's Depression Inventory-2 (CDI-2, Kovacs, 2010). The CDI-2 is a 28-item self-report inventory that assesses the presence of depressive symptoms across the previous two weeks. Each item contains three statements and asks the participant to select the statement that best represents their mood and behavior. For example, one of the items assessing anhedonia contains the statements "I have fun in many things," "I have fun in some things," and "Nothing is fun at all." Each statement corresponds to a 0-, 1-, or 2-point rating. Scores on the CDI-2 range from 0 to 54, with higher scores indicating more depressive symptoms; scores above 14 indicate the presence of clinically significant symptoms. The CDI-2 has demonstrated adequate internal consistency ( $\alpha = .89$ ), as well as satisfactory construct validity (Kovacs, 1985). The CDI-2 was administered to participants at all laboratory visits and demonstrated adequate internal consistency at each timepoint ( $\alpha = .81 - .84$ ).

### **Data Analytic Plan**

Data were analyzed using path analysis with Mplus Version 8.1.6 (Muthén & Muthén, 2017). To address Hypotheses 1 and 2, a total of three models were run (see Figures 2, 3, and 4) in which co-rumination, acute stressors, and depressive symptoms were entered as observed

variables in order to assess the structural relationships between variables. The three types of acute stress, interpersonal independent, interpersonal dependent, and non-interpersonal, were entered separately, resulting in the total of three models. Similarly, to answer Hypotheses 3 and 4, three models were run, assessing the role of the different type of acute stressor in each (see Figures 5, 6, and 7). To assess model fit, several indices were used, including the  $\chi^2$  test of model fit, the comparative-fit-index (CFI), and the standardized root mean square residual (RMSEA). Good model fit was represented by a non-significant  $\chi^2$  test, CFI value greater than 0.90, and RMSEA value less than 0.05 (Hu & Bentler, 1999). The significance and strength of the path coefficients was used to assess the direct effect of co-rumination on the type of acute stress (Models 1-3) and type of acute stress on co-rumination (Models 4-6). Meaning, the direct effects were first assessed based on whether the pathways were significant or not, followed by the magnitude of the standardized beta coefficient. The MODEL INDIRECT command was used to assess the indirect effect of co-rumination on depressive symptoms through the type of acute stress (Models 1-3) and the indirect effect of depressive symptoms on co-rumination through type of acute stress (Models 4-6). Comparison of indirect effects across models was first accomplished through assessing the significance of the indirect effects, followed by the magnitude of indirect effect estimates. The MODEL CONSTRAINT and MODEL TEST commands in MPlus would be utilized to further explore whether indirect effects significantly differed across models, given the criteria of significance previously listed.

Figure 2

*Model 1: Proposed path diagram of the effect of co-rumination on depressive symptoms through interpersonal, dependent stress.*

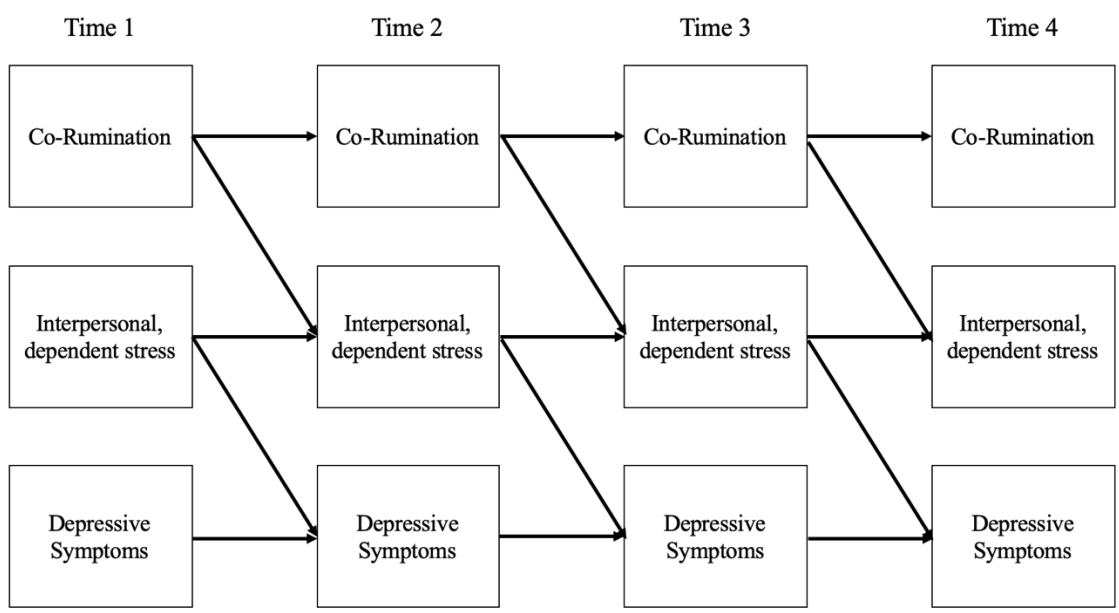


Figure 3

*Model 2: Proposed path diagram of the effect of co-rumination on depressive symptoms through interpersonal, independent stress.*

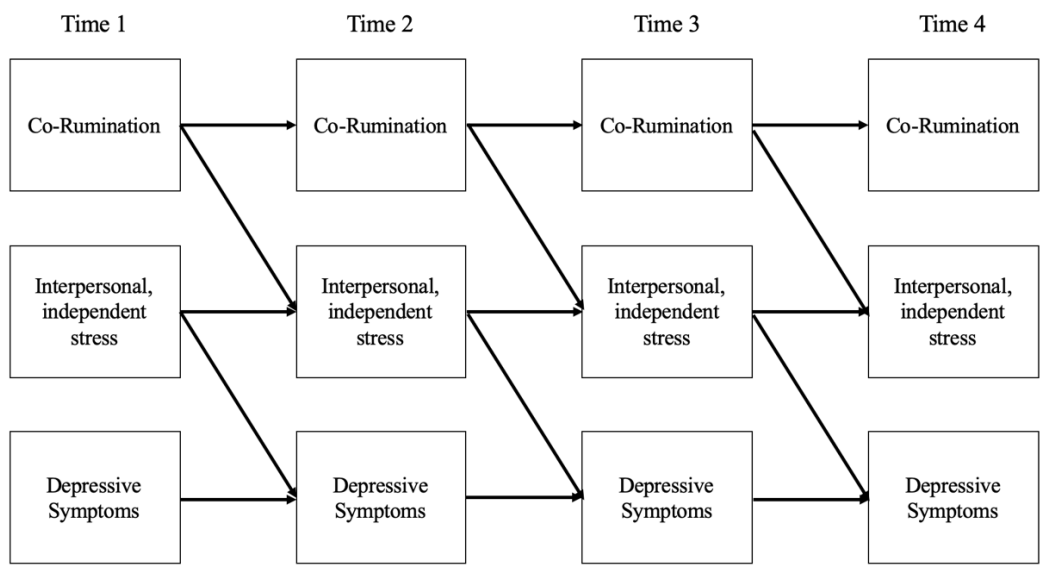


Figure 4

*Model 3: Proposed path diagram of the effect of co-rumination on depressive symptoms through non-interpersonal stress.*

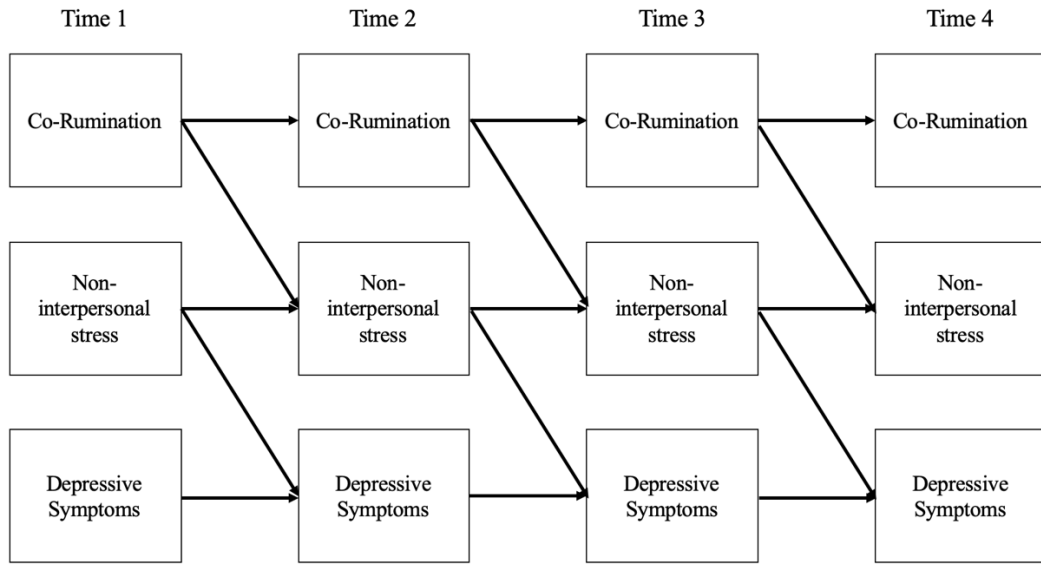


Figure 5

*Model 4: Proposed path diagram of the effect of depressive symptoms on co-rumination through interpersonal, dependent stress.*

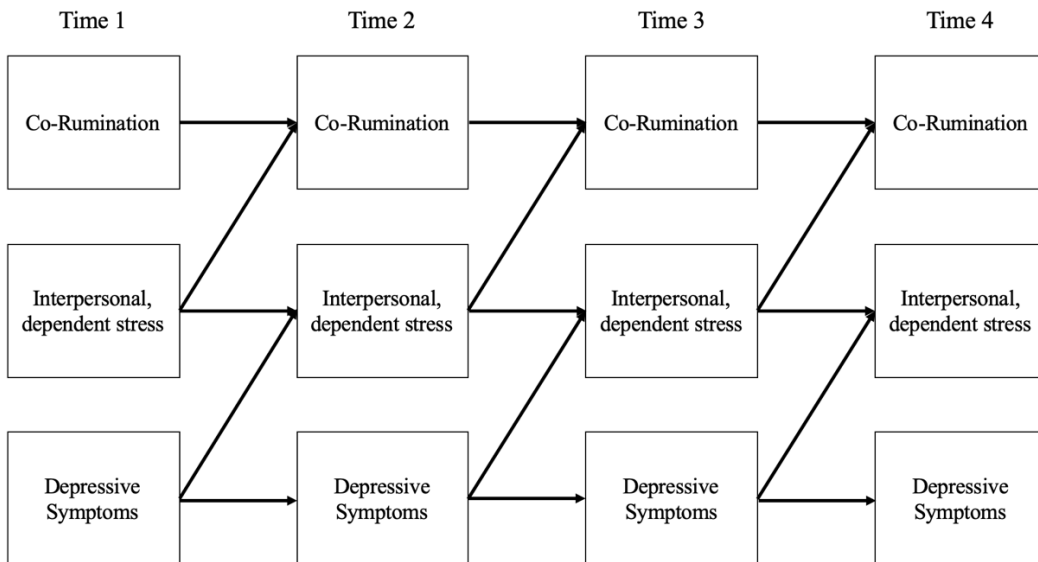


Figure 6

Model 5: Proposed path diagram of the effect of depressive symptoms on co-rumination through interpersonal, independent stress.

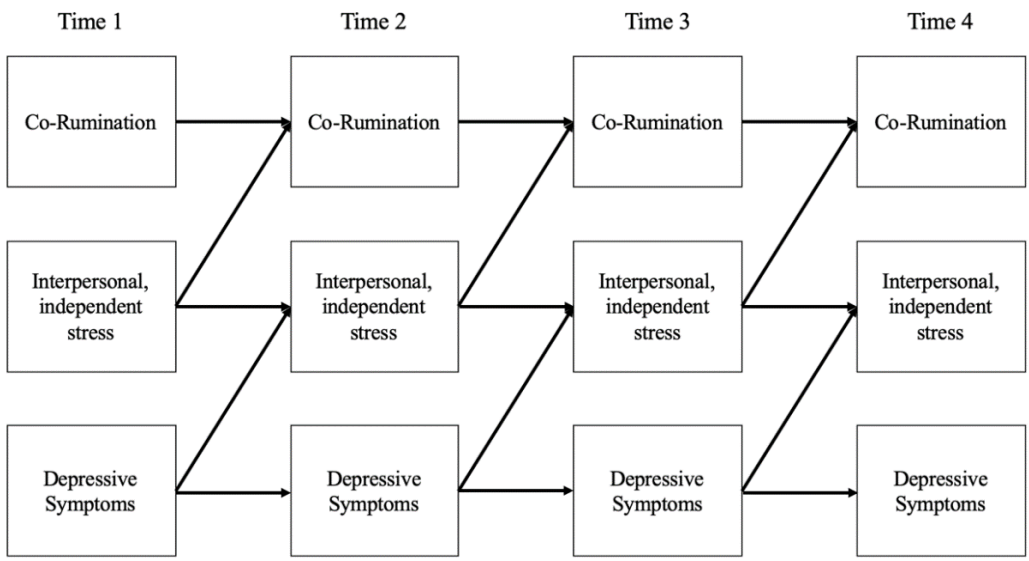
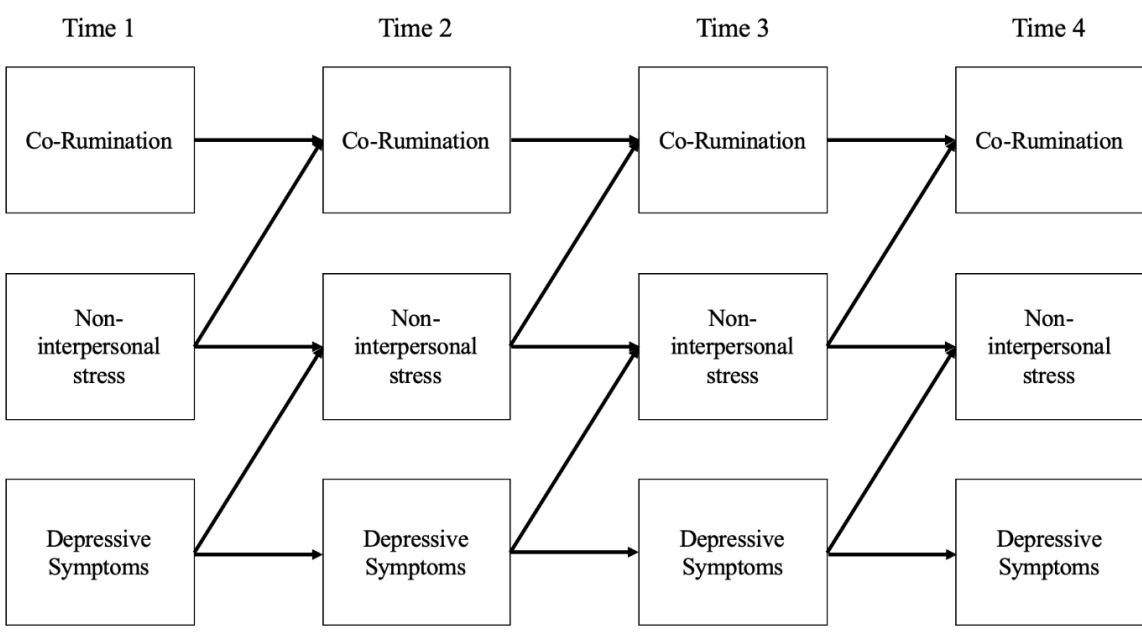


Figure 7

Model 6: Proposed path diagram of the effect of depressive symptoms on co-rumination through non-interpersonal stress.



## CHAPTER III: Results

### Data Preparation and Descriptive Analyses

Missing data for the CRQ and CDI were handled through multiple imputation in the Statistical Package for the Social Sciences (SPSS) 26.0. Data were available for 150 participants at T1, 138 at T2, 127 at T3, and 127 at T4 based on loss of participants due to attrition. Missing data at the item level ranged from .98% at T3 to 4.17% at T1. For participants with less than 24% of item level data CDI-2 and CRQ at each time point (Olinsky et al., 2003), data were imputed using multiple imputation by timepoint, with placeholder scores for the CDI-2 and CRQ at the time points not being imputed as predictor variables. Additionally, age and gender were included in the imputation as predictors. No participants were excluded from the imputation due to missing greater than 24% of items at each time point. Due to method of data collection for stress variables, this data could not be imputed.

Following imputation, data were examined to ensure that all parametric assumptions were met. To assess data normality, variable skewness and kurtosis were examined (see Table 1) and Shapiro-Wilk tests of normality were conducted. As expected, the kurtosis for several count variables (i.e., acute stress variables) and CDI-2 scores were above acceptable ranges. Additionally, the Shapiro-Wilk test of normality indicated that all variables (i.e., depressive symptoms, acute stress) except the four co-rumination variables were non-normal, based on the significant test statistics. Given this, I decided to utilize robust maximum likelihood (MLR) as the estimation method in Mplus, as MLR does not assume normality (Kline, 2016). Additionally, as non-normal data was expected for these variables, transformation of the variables would potentially fundamentally alter the variable (Bentler, 1987; Kline, 2016). However, this method

of estimation does not allow for the use of bootstrapping and utilizes listwise deletion, meaning that all path analysis models were based on the 108 participants with complete data.

Correlations between all variables can be found in Table 2. Of note, T2 interpersonal dependent stress was significantly, positively, correlated with T1 ( $r = .30, p < .001$ ), T2 ( $r = .29, p < .001$ ), T3 ( $r = .25, p = .01$ ), and T4 ( $r = .23, p = .01$ ) depressive symptoms. Additionally, T3 interpersonal dependent stress was significantly positively correlated with T1 depressive symptoms ( $r = .23, p = .01$ ) but not T2 ( $r = .14, p = .12$ ), T3 ( $r = .15, p = .11$ ), or T4 depressive symptoms ( $r = .14, p = .14$ ). Co-rumination at T1 was significantly positively correlated with T3 interpersonal dependent stress ( $r = .21, p = .02$ ). Finally, T4 interpersonal dependent stress was significantly positively correlated with depressive symptoms at T1 ( $r = .21, p = .02$ ), T2 ( $r = .22, p = .02$ ), T3 ( $r = .23, p = .01$ ), and T4 ( $r = .20, p = .03$ ).



Table 1

*Descriptive statistics for primary variables*

	<i>Min</i>	<i>Max</i>	<i>M</i>	<i>SD</i>	<i>Skewness</i>	<i>Kurtosis</i>
T1 Age	11.47	14.87	13.03	0.90	0.26	-0.93
T1 CDI	0.00	29.00	6.90	5.92	1.75	3.55
T2 CDI	0.00	30.01	5.99	5.46	1.59	3.31
T3 CDI	0.00	39.12	6.50	6.31	2.08	6.53
T4 CDI	0.00	33.12	6.13	6.17	1.99	5.06
T1 CRQ	1.00	4.88	2.86	0.86	0.12	-0.49
T2 CRQ	1.00	4.88	2.93	0.79	-0.01	-0.51
T3 CRQ	1.00	4.94	3.03	0.83	-0.13	-0.04
T4 CRQ	1.00	4.95	3.01	0.90	-0.16	-0.22
T1 Count Interpersonal Independent	0.00	3.00	0.33	0.62	2.07	4.43
T2 Count Interpersonal Independent	0.00	4.00	0.39	0.70	2.17	5.80
T3 Count Interpersonal Independent	0.00	3.00	0.25	0.54	2.38	6.40
T4 Count Interpersonal Independent	0.00	3.00	0.31	0.61	2.08	4.11
T1 Count Interpersonal Dependent	0.00	3.00	0.27	0.58	2.70	8.76
T2 Count Interpersonal Dependent	0.00	3.00	0.35	0.60	1.76	2.98
T3 Count Interpersonal Dependent	0.00	4.00	0.44	0.76	2.37	7.24
T4 Count Interpersonal Dependent	0.00	4.00	0.40	0.79	2.06	4.08
T1 Count Non-Interpersonal	0.00	4.00	0.82	1.01	1.32	1.33
T2 Count Non-Interpersonal	0.00	4.00	0.82	0.97	1.29	1.43
T3 Count Non-Interpersonal	0.00	5.00	1.07	1.10	1.02	0.84
T4 Count Non-Interpersonal	0.00	6.00	1.01	1.07	1.54	4.03

*Note.* CDI = Children's Depression Inventory-2, CRQ = Co-Rumination Questionnaire.

Table 2

*Correlations between primary variables.*

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	
1. Gender																						
2. T1 Age	.02																					
3. T1 CDI	.09	.14																				
4. T1 CRQ	.20 <sup>a</sup>	-.02	.03																			
5. T1 Count Non-Int	-.04	-.05	.09	-.03																		
6. T1 Count Int Ind	-.09	-.04	.03	-.10	-.14																	
7. T1 Count Int Dep	-.11	.03	.10	.11	.01	.09																
8. T2 CDI	.08	.16	.81 <sup>c</sup>	.08	.07	.12	.06															
9. T2 CRQ	.14	-.01	-.06	.56 <sup>c</sup>	-.02	-.05	.09	.00														
10. T2 Count Non-Int	-.02	-.07	.00	-.05	.08	-.05	-.03	.01	-.05													
11. T2 Count Int Ind	-.26 <sup>b</sup>	-.02	.07	-.11	.17 <sup>a</sup>	-.01	.12	-.04	-.02	.13												
12. T2 Count Int Dep	.05	.14	.30 <sup>c</sup>	.07	.11	.09	.26 <sup>b</sup>	.29 <sup>b</sup>	.08	.17 <sup>a</sup>	.03											
13. T3 CDI	.10	.09	.73 <sup>c</sup>	-.02	.04	.15	.08	.88 <sup>b</sup>	-.07	.00	-.06	.25 <sup>b</sup>										
14. T3 CRQ	.11	-.01	-.15	.43 <sup>c</sup>	.05	.03	.08	-.07	.65 <sup>c</sup>	-.06	.05	-.04	-.16									
15. T3 Count Non-Int	.18 <sup>a</sup>	-.18	.16	.02	.13	.08	-.08	.13	-.09	.06	-.03	-.02	.12	.02								
16. T3 Count Int Ind	.00	-.02	.05	.16	.03	.00	.10	.10	.11	.04	.01	.09	.08	.06	-.10							
17. T3 Count Int Dep	.20 <sup>a</sup>	.21 <sup>a</sup>	.23 <sup>a</sup>	.21 <sup>a</sup>	.02	.21 <sup>a</sup>	.12	.14	-.11	-.05	-.01	.23 <sup>a</sup>	.15	-.11	.15	.11						
18. T4 CDI	.19 <sup>a</sup>	.04	.68 <sup>c</sup>	.02	.03	.07	.07	.81 <sup>c</sup>	-.04	.04	-.17	.23 <sup>a</sup>	.86 <sup>c</sup>	-.11	.10	.13	.14					
19. T4 CRQ	.18 <sup>a</sup>	.01	-.08	.49 <sup>c</sup>	.05	.00	.09	-.09	.59 <sup>c</sup>	-.02	.09	-.04	-.12	.79 <sup>c</sup>	.03	.11	.05	-.11				
20. T4 Count Non-Int	-.15	-.01	.02	.12	-.13	.12	.14	.00	-.07	.02	-.12	.25 <sup>a</sup>	-.05	.04	.04	.00	.05	-.04	-.07			
21. T4 Count Int Ind	.11	.08	.01	.02	-.10	.01	-.02	.04	-.01	.02	-.05	.08	.02	-.05	.05	-.03	-.02	.09	-.01	-.02		
22. T4 Count Int Dep	-.03	.07	.21 <sup>a</sup>	.04	-.10	.30 <sup>b</sup>	.15	.22 <sup>a</sup>	.09	-.08	.21 <sup>a</sup>	.22 <sup>a</sup>	.23 <sup>a</sup>	.03	.00	.17	.24 <sup>a</sup>	.20 <sup>a</sup>	.06	.00	.14	

*Note.* CDI = Children's Depression Inventory-2, CRQ = Co-Rumination Questionnaire, Non-Int = Non-Interpersonal Stress, Int Ind = Interpersonal Independent Stress, Int Dep = Interpersonal Dependent Stress.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

## Path Analysis Models

Model fit was first assessed by examining each model with age and gender entered as correlates of the CRQ, CDI, and corresponding acute stress variable at T1. All initial models demonstrated acceptable fit (see Table 3). If the correlation between age or gender and the CRQ, CDI, or acute stress variable were non-significant, the pathway was trimmed from the model unless the trimmed model demonstrated worse fit than the initial model. Fit statistics for final, trimmed models can be found in Table 3.

Table 3

*Fit statistics of path analysis models.*

	RMSEA	CFI	TLI	$\chi^2$	df
Model 1: Interpersonal Dependent CRQ → CDI	.05	.97	.95	88.33*	67
Model 1 Trimmed	.05	.97	.96	91.32	71
Model 2: Interpersonal Independent CRQ → CDI	.04	.98	.98	79.61	86
Model 2 Trimmed	.04	.98	.98	83.97	73
Model 3: Non-Interpersonal CRQ → CDI	.04	.98	.98	79.38	68
Model 3 Trimmed	.03	.99	.98	81.22	72
Model 4: Interpersonal Dependent CDI → CRQ	.05	.98	.97	82.57	68
Model 4 Trimmed	.04	.98	.97	86.09	72
Model 5: Interpersonal Independent CDI → CRQ	.04	.98	.97	80.35	68
Model 5 Trimmed	.04	.98	.98	82.98	72
Model 6: Non-Interpersonal CDI → CRQ	.04	.98	.98	78.93	68
Model 6 Trimmed	.03	.99	.98	80.81	72

*Note.* Chi-square statistics cannot be compared within nested models due to model estimator.

\* $p < .05$ .

Results of Models 1, 2, and 3 can be found in Figures 8, 9, and 10, respectively.

Inconsistent with Hypothesis 1, co-rumination did not more strongly predict interpersonal dependent stress compared to interpersonal independent or non-interpersonal stress. The only

significant path between co-rumination and type of acute stress was the pathway between T2 co-rumination and T3 interpersonal dependent stress, in which co-rumination negatively predicted interpersonal dependent stress,  $B(SE) = -.15(.07)$ ,  $p = .02$ . There were no occurrences in which co-rumination predicted either interpersonal independent or non-interpersonal stress; the magnitude of path coefficients was generally low ( $< .20$ ) and varied between positive and negative directions. Interpersonal dependent stress predicted the continuous occurrence of interpersonal dependent stress across the year, whereas interpersonal independent and non-interpersonal stress did not.

Furthermore, inconsistent with Hypothesis 2, interpersonal, independent stress did not mediate the relation between co-rumination and depressive symptoms prospectively. A summary of the indirect effects for all models can be found in Table 4. The indirect effects for Models 1, 2, and 3 were non-significant with the magnitude of the effects low ( $< .013$ ). Direction of effects varied based on model; the indirect effects of interpersonal dependent stress were more positive compared to interpersonal independent and non-interpersonal stress, which were generally negative.

Results of Models 4, 5, and 6 can be found in Figures 11, 12, and 13, respectively. Partially consistent with Hypothesis 3, depressive symptoms at T1 predicted interpersonal dependent stressors at T2 ( $B[SE] = .23[.10]$ ,  $p = .02$ ) and T4 ( $B[SE] = .17[.09]$ ,  $p = .04$ ), whereas it did not predict interpersonal independent (e.g., at T2,  $B[SE] = .05[.07]$ ,  $p = .46$ ) or non-interpersonal stressors (e.g., at T2,  $B[SE] = .006[.10]$ ,  $p = .95$ ). This indicates that higher depressive symptoms at T1 and T3 predicted more interpersonal dependent stressors at T2 and T4, respectively. Inconsistent with Hypothesis 3, T2 depressive symptoms predicted non-interpersonal stressors at T3 ( $B[SE] = .15[.07]$ ,  $p = .04$ ) compared to interpersonal dependent

( $B[SE] = .12[.07]$ ,  $p = .09$ ) and interpersonal independent stressors ( $B[SE] = .11[.10]$ ,  $p = .25$ ) at T3. Furthermore, no type of stress predicted co-rumination, with the magnitude of path coefficients low ( $< .16$ ) and direction fluctuating between positive and negative. Inconsistent with Hypothesis 4, interpersonal dependent stress did not mediate the relation between depressive symptoms and co-rumination more strongly than interpersonal independent and non-interpersonal stress (see Table 4). The indirect effects for Models 4, 5, and 6 were non-significant with the magnitude of the effects low ( $< .008$ ).

Table 4

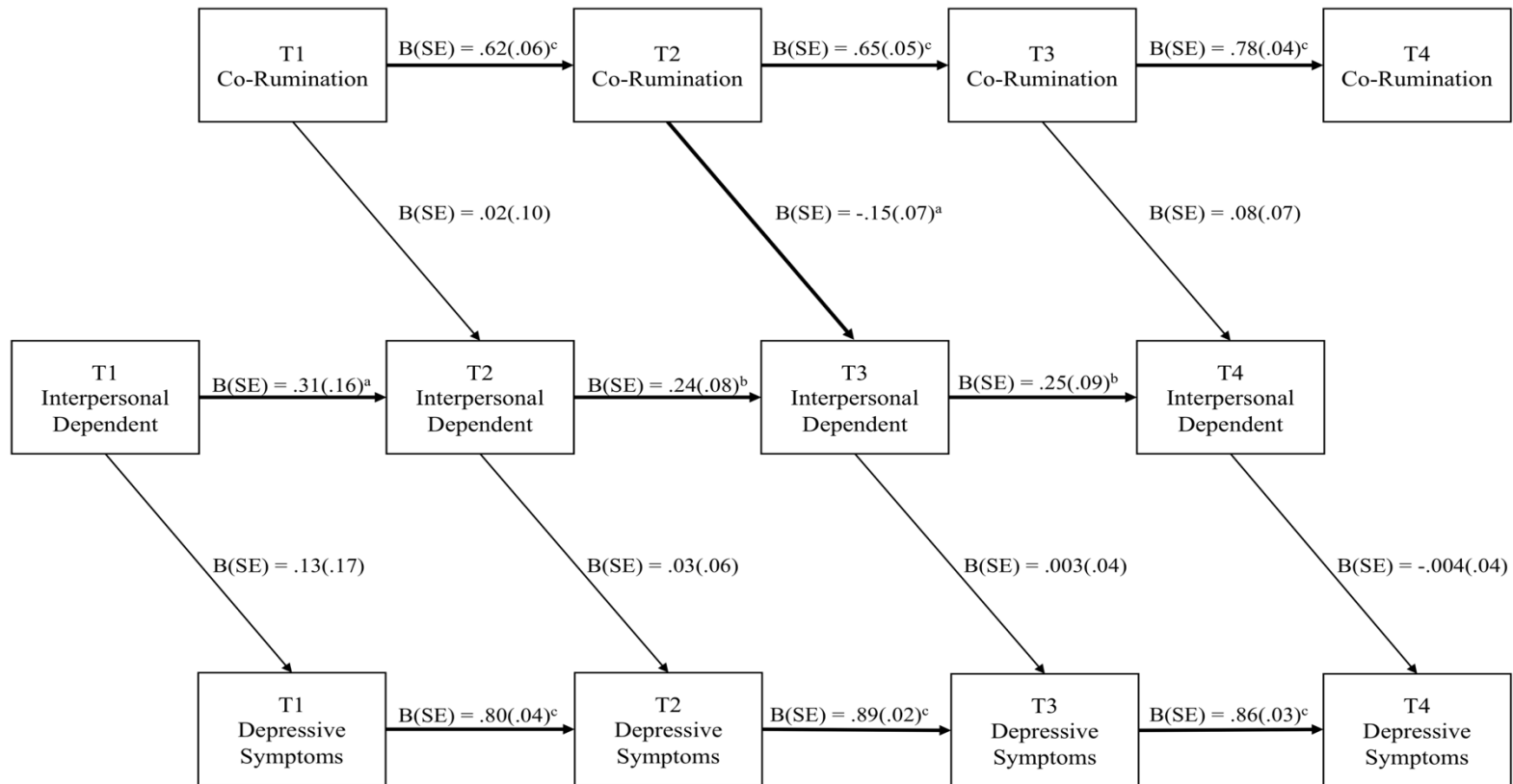
*Indirect effects of all path analysis models.*

	Estimate	SE	95% CI		<i>p</i>
			LL	UL	
Model 1. Interpersonal Dependent					
T1 CRQ → T2 CDI	.001	.003	-.008	.009	.859
T2 CRQ → T3 CDI	.000	.006	-.013	.016	.952
T3 CRQ → T4 CDI	.000	.003	-.007	.008	.932
Model 2. Interpersonal Independent					
T1 CRQ → T2 CDI	.013	.011	-.010	.042	.270
T2 CRQ → T3 CDI	-.003	.005	-.014	.011	.541
T3 CRQ → T4 CDI	-.003	.001	-.013	.011	.585
Model 2. Non-Interpersonal					
T1 CRQ → T2 CDI	-.002	.005	-.013	.008	.669
T2 CRQ → T3 CDI	-.002	.005	-.011	.008	.736
T3 CRQ → T4 CDI	-.001	.002	-.005	.004	.749
Model 4. Interpersonal Dependent					
T1 CDI → T2 CRQ	.008	.021	-.033	.061	.708
T2 CDI → T3 CRQ	-.005	.008	-.022	.016	.512
T3 CDI → T4 CRQ	.006	.011	-.015	.033	.586
Model 5. Interpersonal Independent					
T1 CDI → T2 CRQ	.007	.011	-.011	.036	.518
T2 CDI → T3 CRQ	-.006	.008	-.019	.014	.456
T3 CDI → T4 CRQ	-.001	.003	-.006	.008	.763
Model 6. Non-Interpersonal					
T1 CDI → T2 CRQ	.000	.001	-.003	.004	.949
T2 CDI → T3 CRQ	.006	.009	-.011	.030	.479
T3 CDI → T4 CRQ	.006	.009	-.011	.030	.472

*Note.* CDI = Children's Depression Inventory-2; CRQ = Co-Rumination Questionnaire.

Figure 8

*Path analysis model 1 of associations between co-rumination, interpersonal dependent stress, and depressive symptoms.*

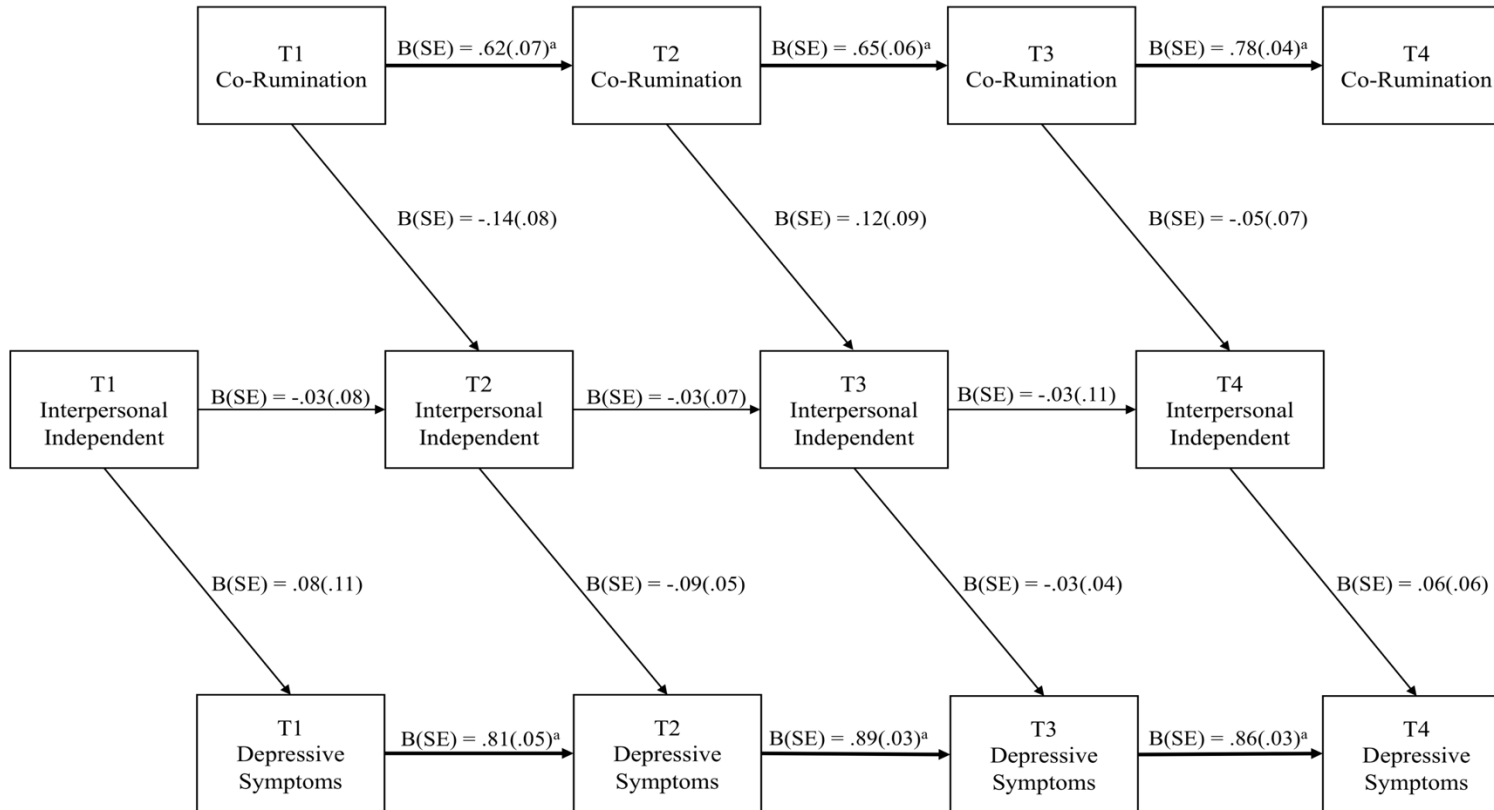


*Note.* This path analysis model shows the association between co-rumination, interpersonal dependent stress, and depressive symptoms across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 9

*Path analysis model 2 of associations between co-rumination, interpersonal independent stress, and depressive symptoms.*



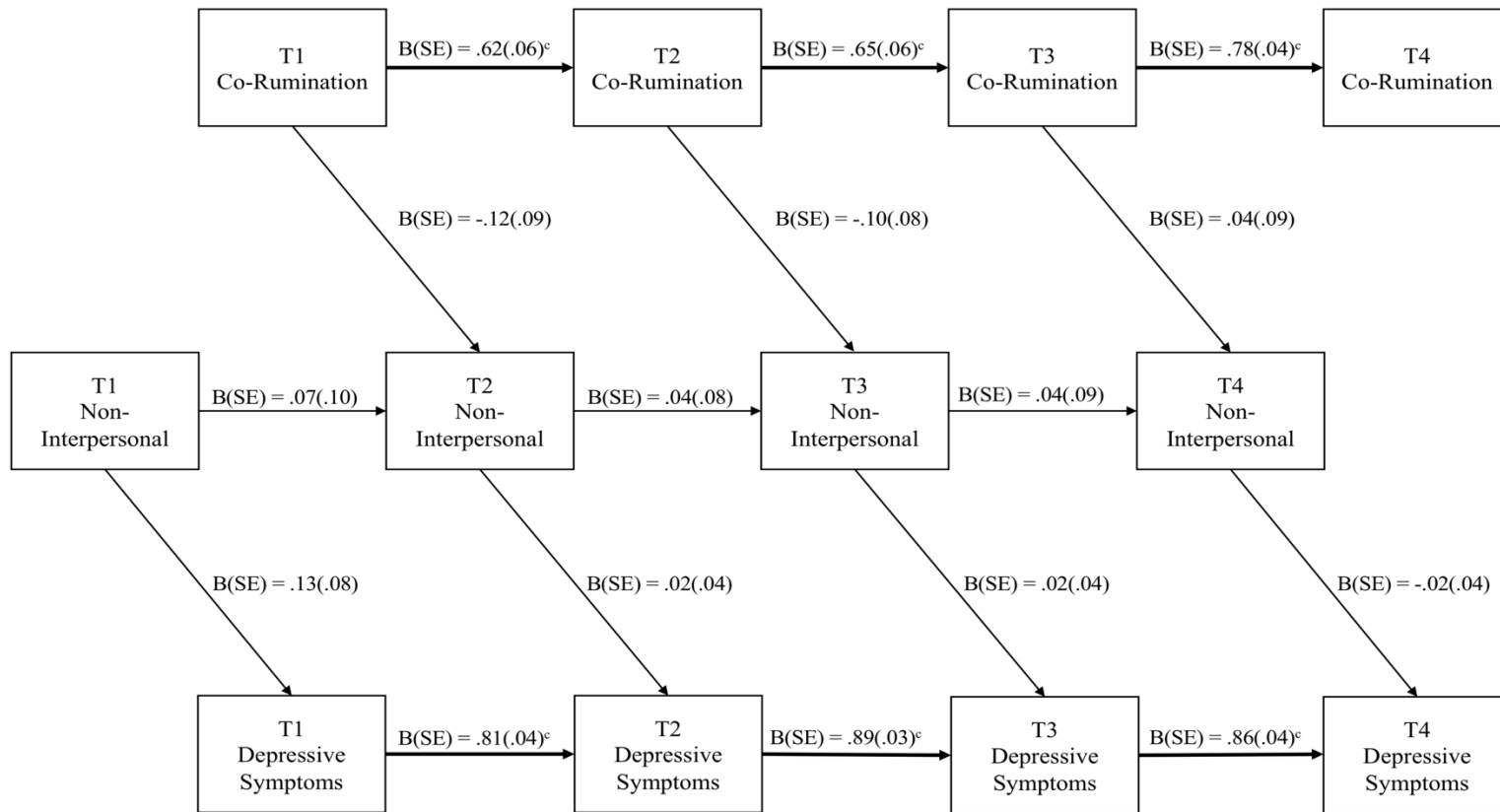
*Note.* This path analysis model shows the association between co-rumination, interpersonal independent stress, and depressive symptoms across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .



Figure 10

*Path analysis model 3 of associations between co-rumination, non-interpersonal stress, and depressive symptoms.*

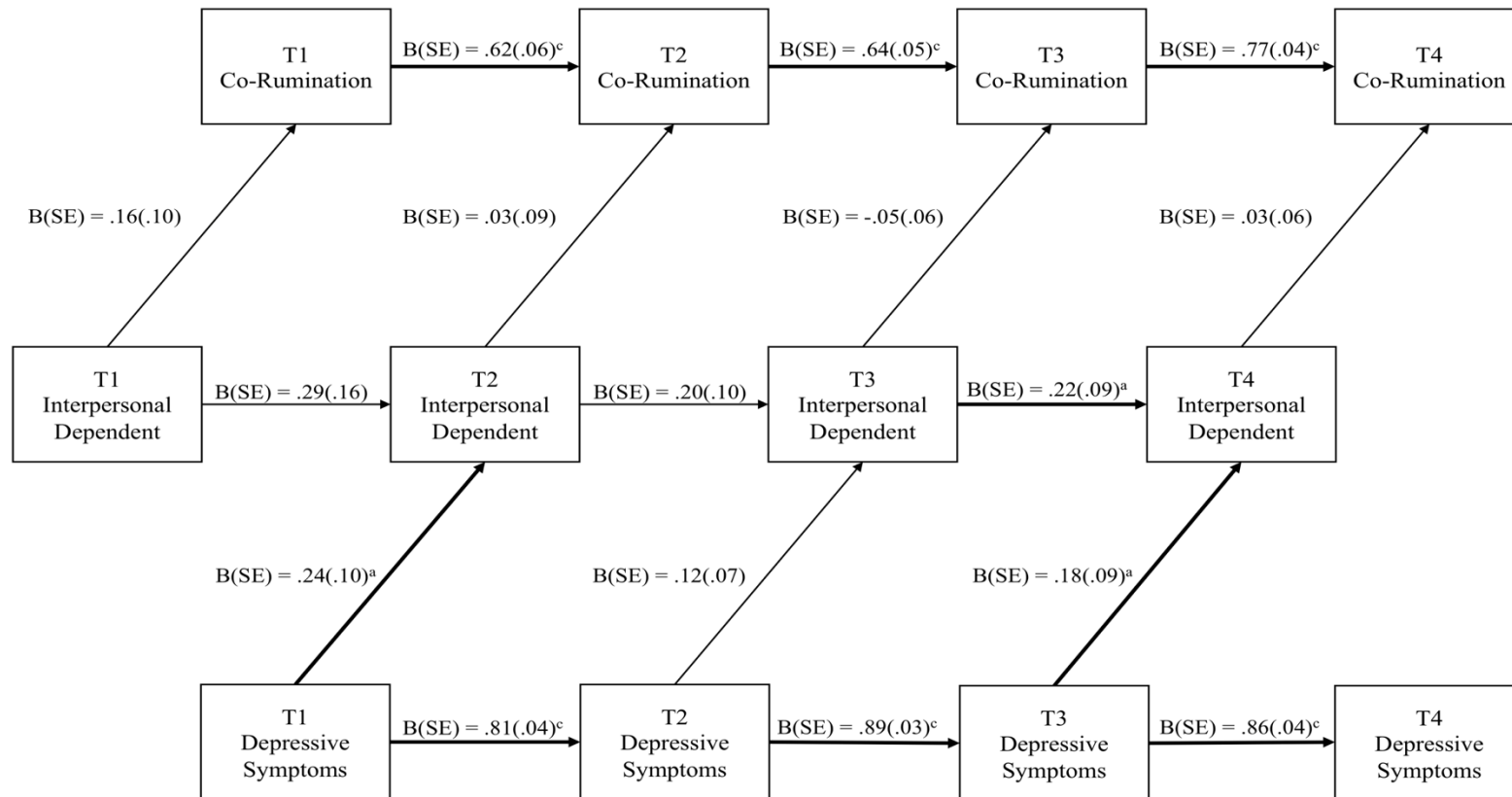


*Note.* This path analysis model shows the association between co-rumination, non-interpersonal stress, and depressive symptoms across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 11

*Path analysis model 4 of associations between depressive symptoms, interpersonal dependent stress, and co-rumination.*

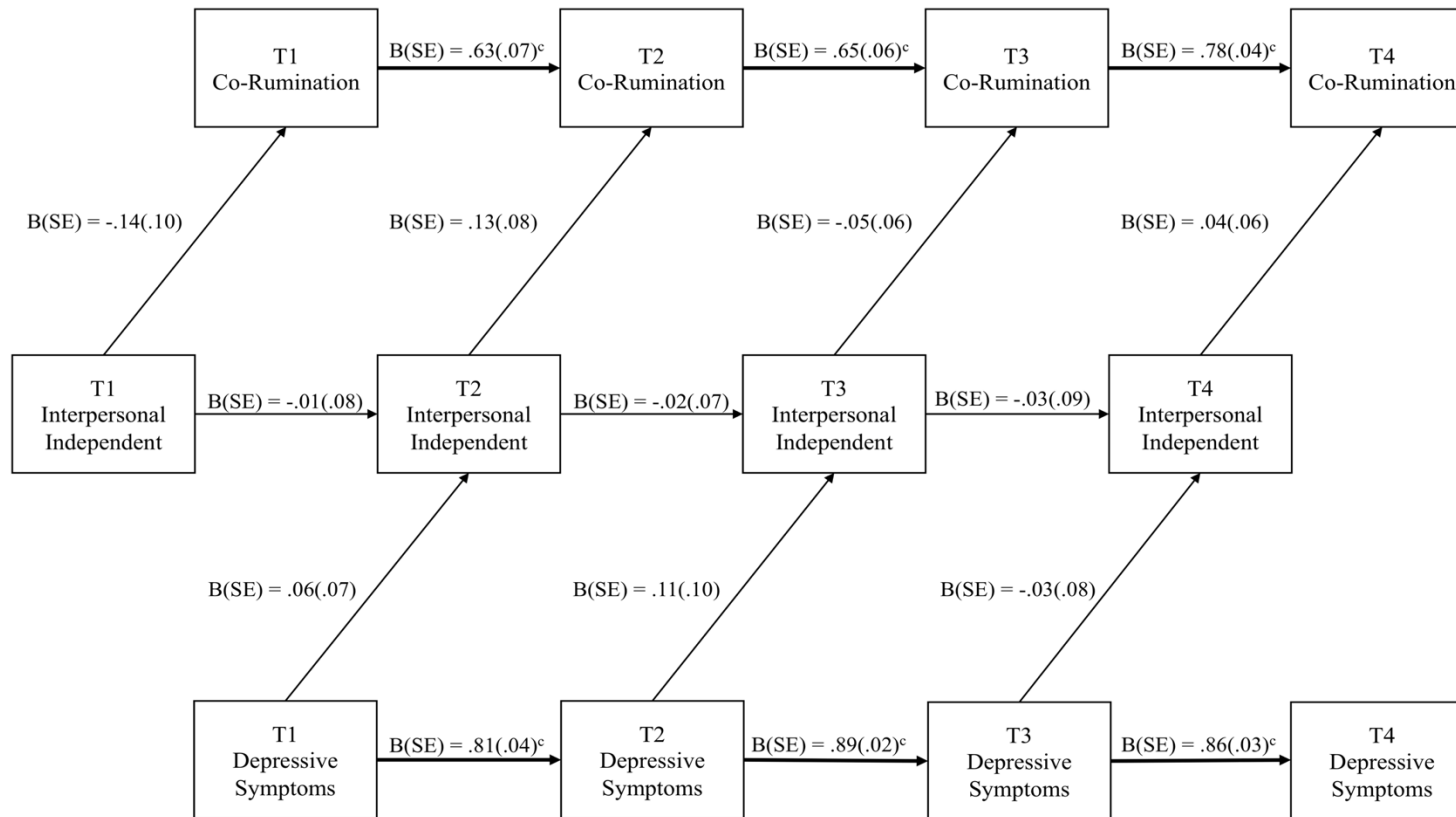


*Note.* This path analysis model shows the association between depressive symptoms, interpersonal dependent stress, and co-rumination across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 12

*Path analysis model 5 of associations between depressive symptoms, interpersonal independent stress, and co-rumination.*

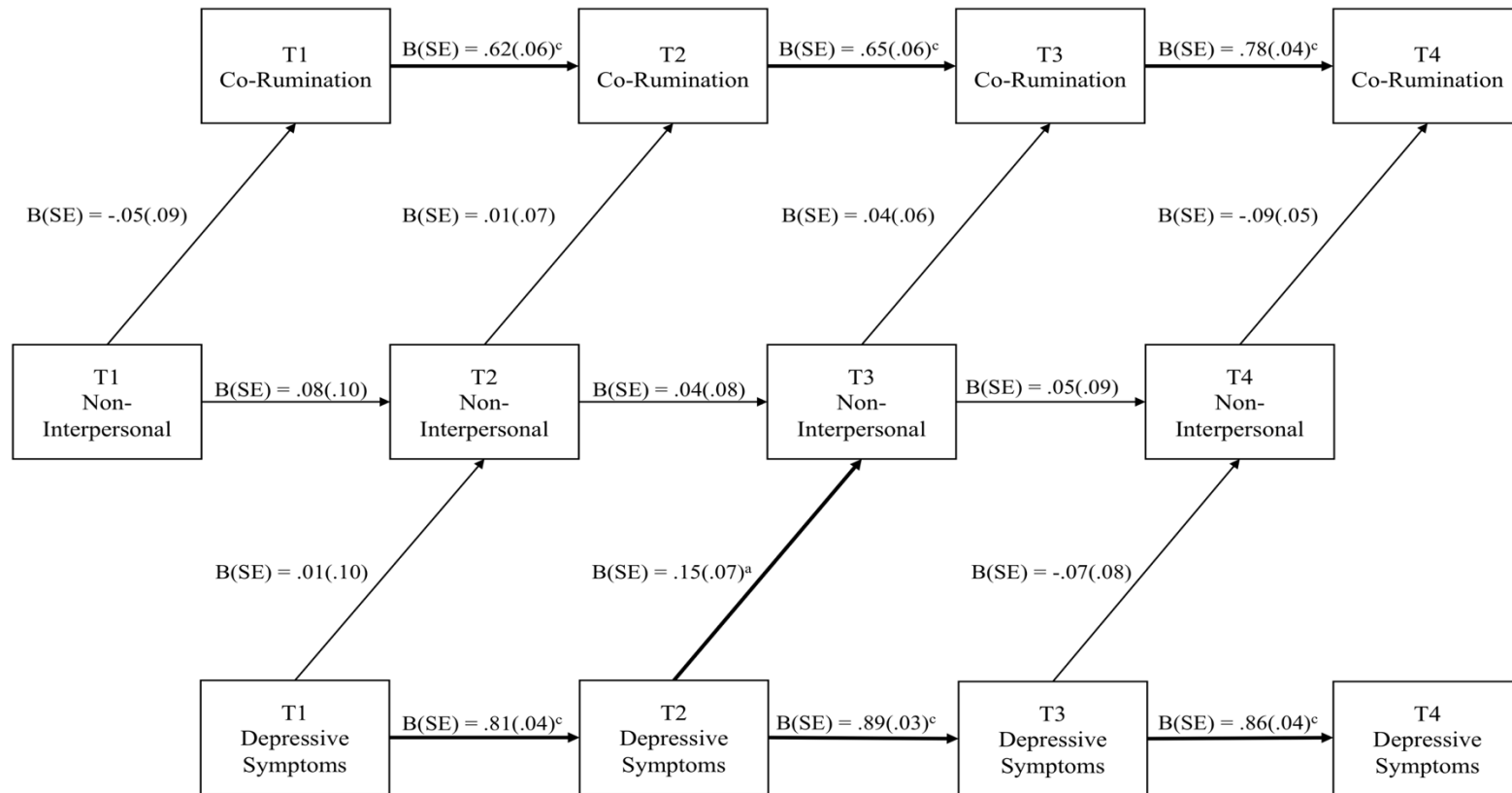


*Note.* This path analysis model shows the association between depressive symptoms, interpersonal independent stress, and co-rumination across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 13

*Path analysis model 6 of associations between depressive symptoms, interpersonal independent stress, and co-rumination.*



*Note.* This path analysis model shows the association between depressive symptoms, non-interpersonal stress, and co-rumination across one year period. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

## Post-Hoc Analyses

Given the findings of the initial six models, I decided to run post-hoc path analysis models that examined the bidirectional relationships not assessed in the first six models. Due to the restrictions of the MLR estimation method utilized (e.g., listwise deletion), I decided to limit the path models to examine the timepoints of T1 through T3 in order to increase the overall number of participants from 108 to 118. Additionally, I decided to collapse across timepoints, given the low magnitude of the effects found in the primary analyses. To do this, I added the number of stressors at T2 and T3 for each type of stress to create a composite stress score. However, this still allows for prospective analyses as the models assessed the effect of depressive symptoms and co-rumination on stress across the following 6 months. Each model assessed the effect of T1 stress on T1 co-rumination, depressive symptoms, and the composite T2/T3 stress score. Models also assessed the effect of both T1 co-rumination and depressive symptoms on T2/T3 stress, and T2/T3 stress on T3 co-rumination and depressive symptoms. Thus, I ran a total of three models based on each type of stress, examining the four initial hypotheses.

Descriptive statistics for post-hoc analyses variables can be found in Table 5. Of note, T1 depressive symptoms were significantly positively correlated with T2/T3 interpersonal dependent stress ( $r = .24, p = .01$ ). Additionally, T1 co-rumination was significantly positively correlated with T2/T3 interpersonal dependent stress ( $r = .20, p = .03$ ). Significant correlations were not noted between T1 co-rumination and depressive symptoms and the other stress variables. As the kurtosis for several variables was above acceptable levels, MLR was again utilized as the estimation methods for all models. Model fit was assessed using the same method

and parameters as the initial models. Results of initial and trimmed models can be found in Table 6.

Table 5

*Descriptive statistics for post-hoc analyses*

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Gender												
2. Age	0.02											
3. T1 CDI	0.09	0.14										
4. T1 CRQ	0.20 <sup>a</sup>	-0.02	0.03									
5. T1 Count Int Dep	-0.11	0.03	0.10	0.11								
6. T1 Count Int Ind	-0.09	-0.04	0.03	-0.10	0.09							
7. T1 Count Non-Int	-0.04	-0.05	0.09	-0.031	0.01	-0.14						
8. T3 CDI	0.10	0.09	0.73 <sup>c</sup>	-0.02	0.08	0.15	0.04					
9. T3 CRQ	0.11	-0.01	-0.15	0.43 <sup>b</sup>	0.08	0.03	0.05	-0.16				
10. T2/T3 Count Int Dep	0.19 <sup>a</sup>	0.24 <sup>b</sup>	0.32 <sup>c</sup>	0.20 <sup>a</sup>	0.24 <sup>b</sup>	0.20 <sup>a</sup>	0.03	0.24 <sup>b</sup>	-0.11			
11. T2/T3 Count Int Ind	-0.17	-0.03	0.06	0.00	0.17	0.01	0.16	0.00	0.12	0.09		
12. T2/T3 Count Non-Int	0.10	-0.20 <sup>a</sup>	0.14	-0.05	-0.08	0.06	0.16	0.09	-0.03	0.13	0.02	
<i>Min</i>		11.47	0.00	1.00	0.00	0.00	0.00	0.00	1.00	0.00	0.00	0.00
<i>Max</i>		14.87	29.00	4.88	3.00	3.00	4.00	39.12	4.94	5.00	4.00	6.00
<i>M</i>		13.03	6.90	2.86	0.27	0.33	0.82	6.50	3.03	0.74	0.63	1.88
<i>SD</i>		0.90	5.92	0.86	0.58	0.62	1.01	6.31	0.83	1.06	0.89	1.52
<i>Skewness</i>		0.26	1.75	0.12	2.70	2.07	1.32	2.08	-0.13	1.54	1.46	0.77
<i>Kurtosis</i>		-0.93	3.55	-0.49	8.76	4.43	1.33	6.53	-0.04	2.21	1.73	0.02

*Note.* CDI = Children's Depression Inventory-2, CRQ = Co-Rumination Questionnaire, Non-Int = non-interpersonal stress, Int Ind = interpersonal independent stress, Int Dep = interpersonal dependent stress.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Table 6

*Fit statistics for post-hoc models*

	RMSEA	CFI	TLI	$\chi^2$	df
Model 7. Interpersonal Dependent	.05	.97	.92	153.193*	28
Model 7 Trimmed	.04	.98	.95	153.196*	28
Model 8. Interpersonal Independent	.03	.99	.96	129.713*	28
Model 8 Trimmed	.01	.99	.99	129.713*	28
Model 9. Non-Interpersonal	.07	.94	.86	141.76*	28
Model 9 Trimmed	.04	.98	.96	128.016*	21

*Note.* Chi-square statistics cannot be compared within nested models due to model estimator.  
\* $p < .05$ .

Results of Model 7, 8, and 9 can be found in Figures 14, 15, and 16, respectively; a summary of the indirect effects of the models can be found in Table 7. Inconsistent with Hypothesis 1, co-rumination did not predict any type of stress, though it was noted that the regression coefficient between T1 co-rumination and T2/T3 interpersonal dependent stress ( $B[SE] = .15[.09]$ ,  $p = .08$ ) was stronger compared to that of co-rumination to T2/T3 interpersonal independent ( $B[SE] = -.007[.09]$ ,  $p = .94$ ) and non-interpersonal stress ( $B[SE] = -.06[.09]$ ,  $p = .49$ ). Consistent with Hypothesis 3, T1 depressive symptoms positively predicted T2/T3 interpersonal dependent stress,  $B(SE) = .29(.10)$ ,  $p = .005$ , but did not predict T2/T3 interpersonal independent ( $B[SE] = .06[.08]$ ,  $p = .45$ ) or non-interpersonal stress ( $B[SE] = .13[.10]$ ,  $p = .19$ ). Furthermore, inconsistent with Hypotheses 2 and 4, no indirect effects were significant. Indirect effects were noted to be low in magnitude ( $< .06$ ), and primarily positive in directionality, though the strongest indirect was negative in directionality, opposite of the hypothesized direction.



Table 7

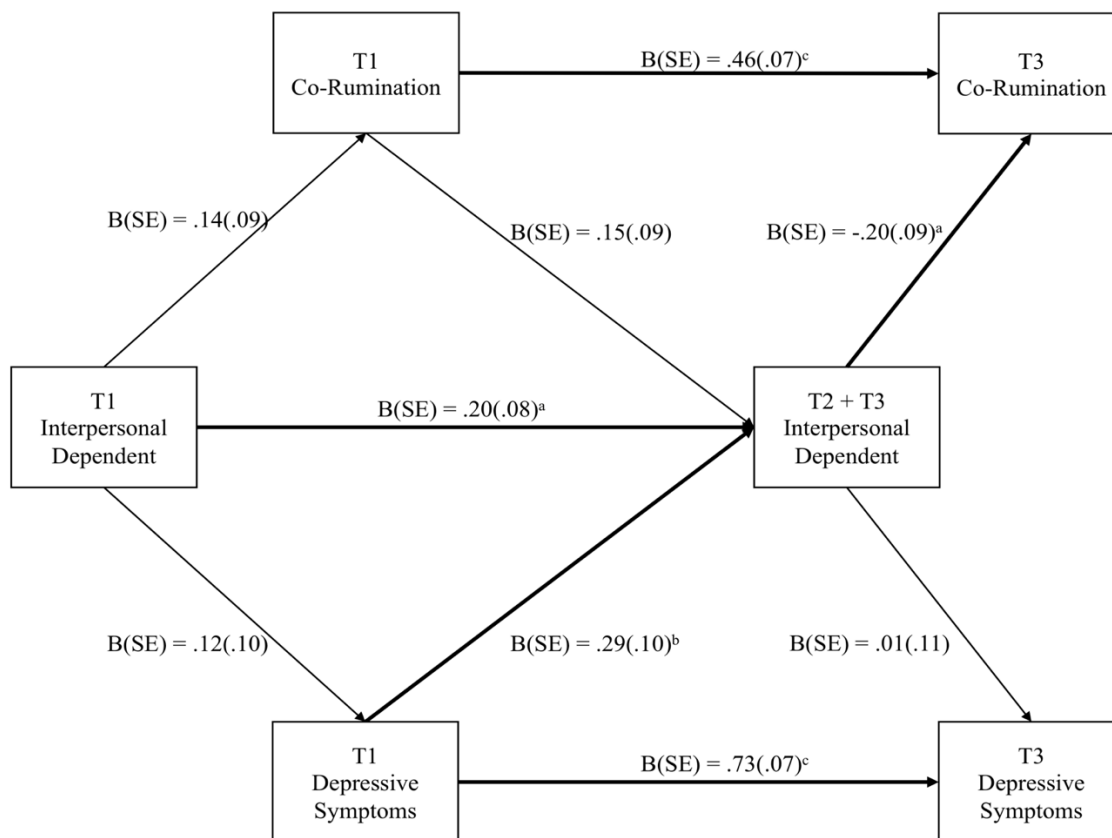
Indirect effects of post-hoc models.

	Estimate	SE	95% CI		<i>p</i>
			LL	UL	
Model 7. Interpersonal Dependent					
T1 CRQ → T3 CDI	.001	.016	-.030	.032	.955
T1 CDI → T3 CRQ	-.057	.040	-.140	.020	.146
Model 8. Interpersonal Independent					
T1 CRQ → T3 CDI	.000	.004	-.008	.008	.941
T1 CDI → T3 CRQ	.007	.011	-.014	.028	.510
Model 9. Non-Interpersonal					
T1 CRQ → T3 CDI	.001	.004	-.020	.017	.834
T1 CDI → T3 CRQ	-.001	.009	-.007	.009	.876

*Note.* CDI = Children's Depression Inventory-2; CRQ = Co-Rumination Questionnaire.

Figure 14

*Post-hoc path analysis model 7 of associations between depressive symptoms, interpersonal dependent stress, and co-rumination.*

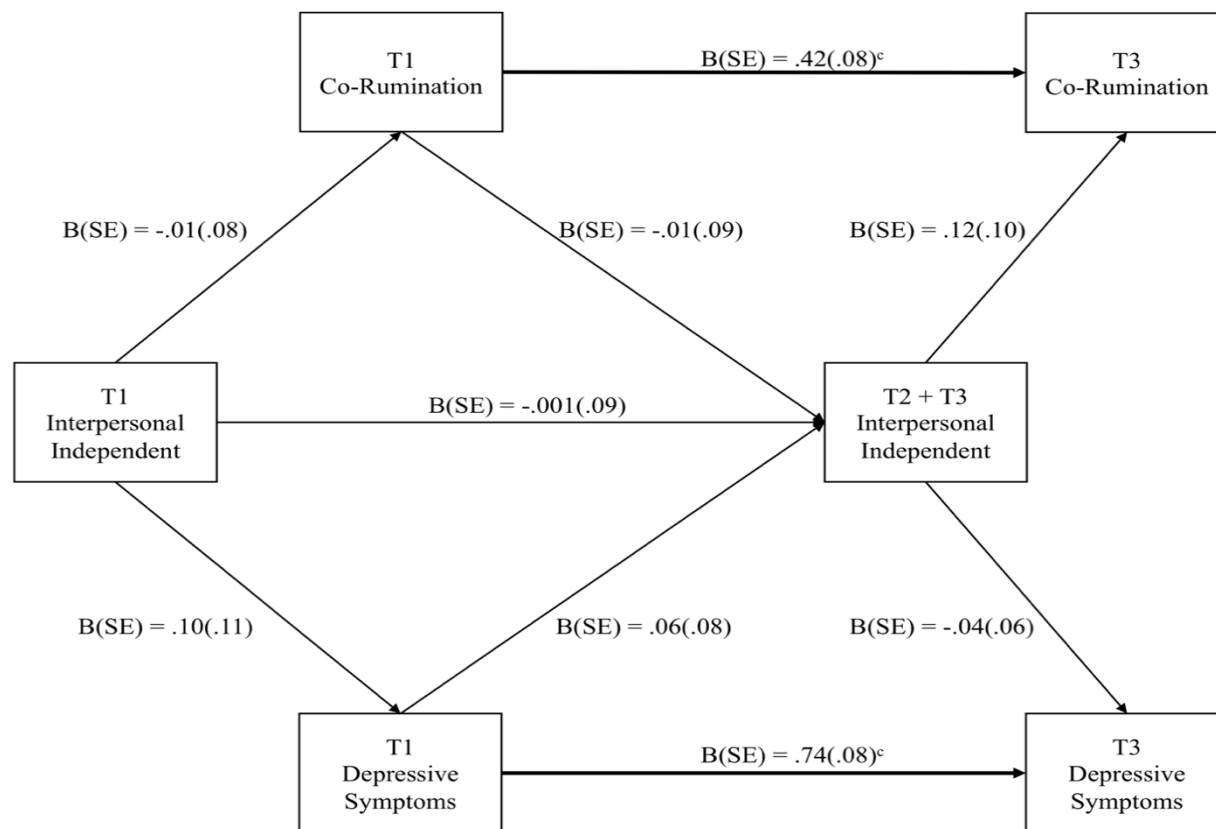


*Note.* This path analysis model shows the association between depressive symptoms, interpersonal dependent stress, and co-rumination based on post-hoc analyses. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 15

*Post-hoc path analysis model 8 of associations between depressive symptoms, interpersonal independent stress, and co-rumination.*

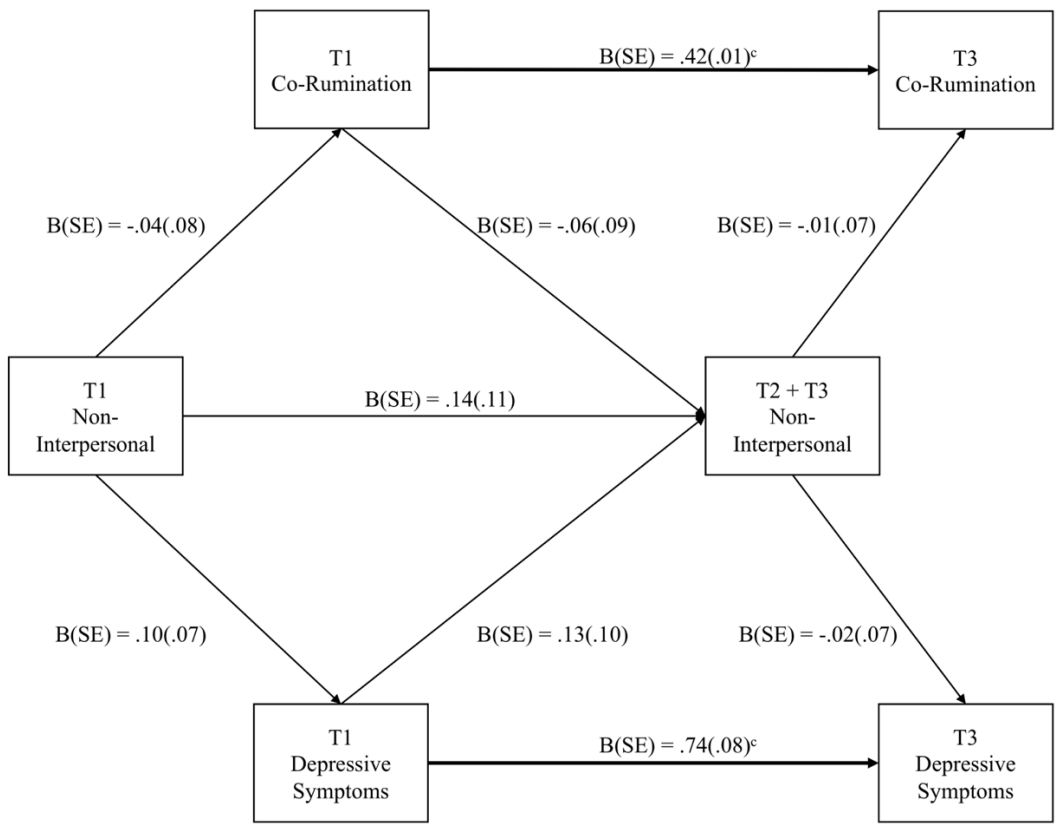


*Note.* This path analysis model shows the association between depressive symptoms, interpersonal independent stress, and co-rumination based on post-hoc analyses. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

Figure 16

*Post-hoc path analysis model 9 of associations between depressive symptoms, non-interpersonal stress, and co-rumination.*



*Note.* This path analysis model shows the association between depressive symptoms, non-interpersonal stress, and co-rumination based on post-hoc analyses. Coefficients presented are standardized linear regression coefficients.

<sup>a</sup> $p < .05$ , <sup>b</sup> $p < .01$ , <sup>c</sup> $p < .001$ .

## CHAPTER IV: DISCUSSION

Depression is a common mental health disorder that occurs with high prevalence rates during adolescence and is associated with significant impairment. Many theories have attempted to explain the occurrence of depressive symptoms. Stress generation theory (Hammen, 1991; 2006) posited that depressed and depression-prone individuals are active generators of stressful events in their life, rather than passive recipients, meaning individuals experience and generate more stressful events in their life. The generation of such stress is due to individual characteristics as well as characteristics of depressive symptoms themselves, leading to likely recurrence or intensification of depressive symptoms. Another prevalent theory, response styles theory (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 1993) posited that the way in which individuals respond to stressful events will affect the occurrence and severity of depressive symptoms. One type of response, rumination, is characterized by intense, perseverative thoughts about the distress and has been repeatedly shown in the literature to predict and intensify depressed mood (Nolen-Hoeksema et al., 2008). A more recent expansion of this theory suggested that rumination between individuals, or co-rumination (Rose, 2002), may contribute to ongoing interpersonal distress and depressive symptoms.

The current study had two purposes: first, to examine the intersection of stress generation and response styles theory by assessing the association between co-rumination, acute life stress, and depressive symptoms and two, to evaluate co-rumination as a mechanism of stress generation among adolescents. I hypothesized that co-rumination and depressive symptoms would more strongly predict the occurrence of interpersonal, dependent stress, meaning stress that involves at least two individuals and occurs at least partially due to the individual's behavior, compared to interpersonal, independent and non-interpersonal stress. I also

hypothesized that interpersonal, dependent stress would mediate the pathway between co-rumination and depressive symptoms and depressive symptoms and co-rumination compared to interpersonal, independent and non-interpersonal stress.

Results were largely unresponsive of proposed hypotheses. As indicated in Models 1, 2, and 3 and post-hoc models, co-rumination did not significantly predict the occurrence of any type of stress, with one exception. Co-rumination negatively predicted interpersonal dependent stressors between T2 and T3, contrary to the hypothesis that co-rumination would positively predict interpersonal dependent stress. Thus, this result suggests that higher levels of co-rumination predicted less interpersonal dependent stress over time. Additionally, results of Models 4, 5, and 6 and post-hoc models partially supported the hypothesis that depressive symptoms would more strongly predict the occurrence of interpersonal, dependent stress. Depressive symptoms did positively predict interpersonal, dependent stress in Model 7, indicating that higher levels of depressive symptoms predicted more interpersonal dependent stressors over time, whereas depressive symptoms did not predict the other types of stress. This is consistent with stress generation theory, in that depressed youth may actively contribute to stress within their environment.

There could be several explanations for this pattern of findings. First, the negative association between co-rumination and interpersonal, dependent stress and positive association between depressive symptoms and interpersonal, dependent stress may be somewhat consistent with stress generation theory. If a youth exhibits higher levels of depressive symptoms, he or she may be withdrawing from peers or displaying negative mood symptoms (i.e., sadness, irritability) that may make peers less likely to interact with that youth. Without the presence of peers, it would be difficult for youth to co-ruminate. These interpersonal difficulties would

likely increase a depressed youth's interpersonal stress, consistent with stress generation theory (Rudolph, 2008; Rudolph et al., 2009), as was illustrated in the relationship between depressive symptoms and interpersonal dependent distress in the current study. It may be interesting to explore whether peer stress occurs more in response to depressive symptoms as opposed to family stress, both of which occur at higher rates for depressed youth (Beavers et al., 2007; Ge et al., 1994).

Another consideration comes from response styles theory and more recent research on the relationship between co-rumination and rumination. First, research has consistently demonstrated a strong relationship between rumination and depressive symptoms in various ages of youth (Abela et al., 2002; Abela et al., 2007; Abela et al., 2009; Hankin, 2008; Jose & Brown, 2007). In turn, rumination has been shown to moderate the relation between negative events and distress and depressive symptoms (Abela & Hankin, 2011; Abela et al., 2010). More recent research has suggested that co-rumination fosters the tendency to ruminate (Aldrich et al., 2019; Stone & Gibb, 2015), meaning that engagement in co-rumination increases an individual's tendency to utilize rumination alone. Thus, rumination may be an important component missing from the current models that may help to explain the relationships between co-rumination, acute stress, and depressive symptoms.

Furthermore, prior research on co-rumination and stress has resulted in mixed findings regarding the role of co-rumination in the occurrence of depressive symptoms. Several studies have found that co-rumination acts as a moderator within the stress and depressive symptoms paradigm. Rose and colleagues (2017) found that co-rumination exacerbated the occurrence of stress generation among depressed girls, meaning depressive symptoms predicted the occurrence of interpersonal stress when girls co-ruminated with friends. Additionally, Bastin and colleagues

(2015) examined the reciprocal relationship and found that co-rumination interacted with interpersonal stress to predict depressive symptoms over time; however, this relationship was true for adolescent girls only, not boys. These studies are contrasted against research conducted by Hankin and colleagues (2010), who found that co-rumination predicted the occurrence of interpersonal dependent stress but not interpersonal independent or non-interpersonal stress. Additionally, dependent interpersonal stress predicted increases in co-rumination over time. Thus, it appears that co-rumination may act differently based on the relationship under examination. In the case of this study, I was not able to replicate findings along the same line as Hankin and colleagues (2010). As discussed below, there are methodological limitations that may have contributed to lack of significant results. Future research should continue to examine the differing role of co-rumination and replicate previous findings in order to determine co-rumination's strongest influence on depressive symptoms.

### **Limitations**

Results of the current study should be considered in light of the study's limitations. First, the a priori power analysis indicated that a sample size of 130 would be necessary, based on the magnitude of effects from previous studies. The current study utilized a sample of 108 participants in primary analyses and 118 participants in post-hoc analyses. Due to the non-normality of several variables (e.g., depressive symptoms), it was necessary to utilize an estimation method that could account for the non-normal distribution of the variables. Unfortunately, due to the limitations of the statistical software utilized, the estimation method used listwise deletion, which lowered the total number of participants available for analyses. Additionally, due to the method of data collection for stress variables, it was not possible to impute data for participants missing data at the follow-up visits. This further influenced the data



available for analyses. As previously mentioned, the current models did not examine other factors that have strong connections to co-rumination, stress, and depressive symptoms, such as rumination. It would be difficult to address the role of rumination within these models, given the methodological limitations listed above; a larger sample size would be necessary in order to examine other factors that may be pertinent.

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