

# Transactional Associations Between Adolescents' Emotion Dysregulation and Symptoms of Social Anxiety and Depression: A Longitudinal Study

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## Abstract

Emotion dysregulation has been associated with increased risks for psychopathology. During adolescence when the onset of mental illnesses peak, emotion dysregulation may be particularly problematic. In this study, we examined transactional associations between young adolescents' self-report of their emotion dysregulation and symptoms of social anxiety and depression at three assessments over 3 years. Participants were 391 Australian students (56% female; 79% White/Caucasian) in Grades 6 to 8 followed until Grades 9 to 11. Using structural equation modeling (SEM), emotion dysregulation was a latent variable indicated by lack of emotional clarity, nonacceptance of emotional responses, impulse control difficulties, limited access to emotion regulation strategies, and difficulties engaging in goal-directed behavior. A sixth aspect of emotion dysregulation, lack of emotional awareness, which was not correlated with the other five subscales, was examined separately

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in the SEM. Transactional associations over time were identified between emotion dysregulation and symptoms of social anxiety and depression, with adolescents higher in emotion dysregulation at Time 1 (T1) reporting higher social anxiety and depressive symptoms between T1 and Time 2 (T2), and T2 emotion dysregulation predicting increases in adolescents' depression, but not social anxiety, symptoms from T2 to Time 3 (T3), after controlling for baseline symptoms. In addition, earlier social anxiety, but not depressive, symptom level at T2 was significantly associated with later emotion dysregulation between T2 and T3, after baseline emotion dysregulation was controlled. Furthermore, girls were higher in emotion dysregulation, social anxiety, and depression than boys, but sex did not moderate temporal links between emotion dysregulation and symptoms.

### **Keywords**

emotion regulation, emotion dysregulation, social anxiety, depression, adolescence

An estimated 20% to 25% of Australian youth between 12 and 25 years suffer from mental health problems (Lester, Dooley, Cross, & Shaw, 2012; Waters et al., 2016), and similar prevalence estimates have also been reported in other western countries, such as the United States (Kessler, Chiu, Demler, & Walters, 2005) and the United Kingdom (Patel, Flisher, Hetrick, & McGorry, 2007). Research also suggests that the most common mental health concerns among this age group are the highly comorbid conditions of social anxiety and depression, with both disorders showing significantly higher prevalence among females than males (Farmer & Kashdan, 2012; Stein & Stein, 2008; Zimmer-Gembeck, Hunter, Waters, & Pronk, 2009; Zimmer-Gembeck & Skinner, 2008).

Various theoretical models have been proposed to explain the emergence and chronicity of social anxiety and mood disturbance in young people. Some theories point to the role of emotion dysregulation, and there is accumulating evidence to suggest that emotion regulation or dysregulation may be important processes associated with the pathogenesis of both social anxiety and depression (e.g., Eastabrook, Flynn, & Hollenstein, 2014; Klemanski, Curtiss, McLaughlin, & Nolen-Hoeksema, 2017; Loughheed & Hollenstein, 2012; Mathews, Kerns, & Ciesla, 2014). Emotion regulation has been defined as the "ability to respond to the ongoing demands of experience with a range of emotions, and in ways that are socially tolerable and sufficiently flexible to permit, delay or modify spontaneous reactions as needed" (Cole, Michel, & O'Donnell,

1994, p. 76). The development of emotion regulation is believed to follow a trajectory from extrinsic assistance with regulation during infancy (e.g., parents or caregivers guiding the efforts of infant regulation), to more intrinsic forms of regulation that increase in complexity across the life span (Gross, 2013; J. Ö. Schäfer, Naumann, Holmes, Tuschen-Caffier, & Samson, 2017; Zimmer-Gembeck & Skinner, 2011; Zimmer-Gembeck et al., 2017). There is also increasing evidence within the literature to suggest that adequate and adaptive emotion regulation is associated with greater psychosocial functioning and overall well-being, and that dysfunctional patterns of emotion regulation (or “dysregulation”) are etiologically related with symptoms of a number of psychopathologies, including social anxiety and depression (Compas et al., 2017; Schäfer et al., 2017).

Adolescence is one developmental period that is marked by significant challenges to and changes in emotion regulation and symptoms (Gross, 2013; Skinner & Zimmer-Gembeck, 2016), which are often linked to the physical and cognitive changes associated with the onset of puberty, as well as to changes in social relationships with the transition from primary to secondary school (Gross, 2013; Gullone & Taffe, 2012; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). These changes present new challenges for adolescents’ emotion regulation, at a period in development where the prefrontal cortex (often described as having a critical role in regulating emotions and attention) is still developing, and where intrinsic rather than extrinsic (from parents or caregivers) forms of regulation become more dominant, although not yet mature (Gross, 2013; Zimmer-Gembeck & Skinner, 2011). It has been argued that when an overreliance on juvenile forms of self-regulation occurs during this time of significant change and transition, it may result in increased emotion dysregulation (Cole et al., 1994), thus placing some adolescents at considerably greater risk for developing internalizing disorders, such as social anxiety and depression.

Emotion dysregulation has been described as a complex and dynamic process involving multiple facets (J. Ö. Schäfer et al., 2017). Not surprisingly, the complexity of this construct has resulted in multiple approaches to conceptualizing and measuring emotion dysregulation. In one widely used approach, Gratz and Roemer (2004) defined emotion dysregulation to include six emotion regulation difficulties, which were expected to contribute to the development and maintenance of psychopathology. These factors were described as (a) lack of emotional awareness, (b) lack of emotional clarity, (c) nonacceptance of emotional responses, (d) impulse control difficulties, (e) limited access to regulation strategies, and (f) difficulties engaging in goal-directed behavior when aroused.

Indeed, emotion dysregulation and internalizing symptoms during adolescence have been found to be interrelated (e.g., Eastabrook et al., 2014; Klemanski et al., 2017; Lanteigne, Flynn, Eastabrook, & Hollenstein, 2014). However, almost all studies to date have relied on a cross-sectional research design. In fact, there have been only few studies that have investigated the transactional associations of emotion dysregulation and adolescents' internalizing disorders, such as social anxiety and depression, and even fewer studies that have examined the common and unique contributions of each of the six emotion regulation difficulties identified by Gratz and Roemer (2004) on the escalation of adolescents' social anxiety and depressive symptoms over time.

Nevertheless, from the limited longitudinal research that has been conducted with adolescents, there has been preliminary, albeit mixed, evidence in support of a transactional relationship between emotion dysregulation and symptoms of anxiety and depression. For instance, in one study, Feng and colleagues (2009) found that earlier emotion dysregulation, when limited to a measure of dysregulated sadness, predicted increases in depressive symptoms in a sample of preadolescent girls ( $N = 225$ ;  $\bar{X}_{\text{age}}$ : Wave 1 = 9.10 years) over a 1-year period. Similarly, Folk, Zeman, Poon, and Dallaire (2014) found that earlier emotion dysregulation (defined as dysregulated anger, sadness, worry, and inhibition) predicted increases in later anxiety symptoms in children ( $N = 102$ ;  $\bar{X}_{\text{age}}$ : Wave 1 = 9.65 years) over a 2-year period. These same authors also found that earlier emotion dysregulation (defined as dysregulated anger and worry coping) predicted increases in later depressive symptoms.

In a series of studies (McLaughlin, Hatzenbuehler, & Hilt, 2009; McLaughlin et al., 2011) with a community sample of slightly older, early adolescents (aged 11-14 years), the results were not completely consistent. For instance, McLaughlin et al. (2009) found that when examining the transactional relationships between adolescents' ( $N = 1,065$ ) internalizing symptoms (e.g., anxiety and depression) and emotion dysregulation (defined as poor emotional understanding, dysregulated expression of sadness and anger, and rumination), no significant association was identified between anxiety or depressive symptoms at Time 1 (T1) and emotion dysregulation at Time 2 (T2; 4 months later) after controlling for baseline emotion dysregulation. In contrast, McLaughlin et al. (2011) found that emotion dysregulation (again defined as poor emotional awareness, dysregulated expression of sadness and anger, and rumination) at T1 predicted relative increases in adolescents' anxiety, aggressive behavior, and eating pathology, but not depressive symptoms by T2. In addition, McLaughlin et al. (2011) also found that after controlling for baseline emotion dysregulation, symptoms at T1 were no longer associated with emotion dysregulation at T2 (relative to dysregulation at T1). Thus,

while preliminary evidence exists in support of a transactional association between emotion dysregulation and internalizing symptoms, further research is needed to extend on, and ideally help to clarify, previous findings.

There is also evidence within the literature to suggest that subforms of emotion dysregulation may have common and unique associations with internalizing symptoms during adolescence. For instance, Neumann, van Lier, Gratz, and Koot (2010) found that symptoms of anxiety and depression were higher among adolescents ( $N = 870$ ; aged 11-17 years) who reported greater deficits in emotional clarity, greater nonacceptance of emotional responses, and more limited access to emotion regulation strategies. Whereas, no association was found between symptoms of anxiety and depression with lack of emotional awareness, impulse control difficulties, and difficulties engaging in goal-directed behavior. Similarly, Bender, Reinholdt-Dunne, Esbjørn, and Pons (2012) found that symptoms of anxiety were higher in adolescents ( $N = 544$ ; aged 9-16 years) who reported greater deficits in emotional clarity, greater nonacceptance of emotional responses, and more limited access to emotion regulation strategies. However, these authors also found that greater difficulties engaging in goal-directed behavior were also significantly associated with higher anxiety symptoms in this slightly younger adolescent sample.

Furthermore, in another study of anxiety during early adolescence ( $N = 90$ ; aged 11-14 years), Mathews et al. (2014) found that, when controlling for generalized anxiety, social anxiety symptoms were higher when adolescents reported more deficits in emotional clarity, greater nonacceptance of emotional responses, more negative self-evaluations of their ability to manage their emotions, and a greater negative reactivity to emotions. Whereas, in a more recent study of adolescents ( $N = 336$ ; aged 12-15 years; Hambour, Zimmer-Gembeck, Clear, Rowe, & Avdagic, 2018), lack of emotional clarity, nonacceptance of emotional responses, impulse control difficulties, limited access to regulation strategies, and difficulties engaging in goal-directed behavior when aroused were positively associated with social anxiety symptoms, but lack of emotional awareness had no significant association with social anxiety symptoms. Thus, the common and unique associations between individual difficulties in emotion regulation and symptoms of social anxiety and depression were also investigated in the current study. This will also be the first study to our knowledge to investigate these associations across 3 years spanning the transition from early to middle adolescence.

In research on emotion dysregulation and internalizing symptoms, the role of gender should not be overlooked. Gender differences in the levels of emotion dysregulation and internalizing symptoms have been reported in multiple cross-sectional studies of adolescents, with early adolescent females almost

always reporting greater difficulties regulating their negative emotions and higher levels of internalizing symptoms, such as anxiety and depression, in comparison with similarly aged male peers (e.g., Bender et al., 2012; Neumann et al., 2010). However, we also expand on these findings in the current study by investigating gender invariance in the pathways linking emotion dysregulation and symptoms over time. We could not locate any studies that have investigated whether gender may moderate the transactional associations between adolescents' emotion dysregulation and symptoms of social anxiety and depression over time.

## **The Current Study**

The primary aim of the current 3-year longitudinal study was to examine the temporal, transactional associations between adolescents' emotion dysregulation and symptoms of social anxiety and depression. Thus, the tested models allowed for associations between earlier internalizing symptoms (i.e., social anxiety and depression) and later emotion dysregulation, and between earlier emotion dysregulation and later internalizing symptoms. We not only modeled a latent construct of emotion dysregulation but also tested separate models of each of the Emotion Dysregulation subscales. Furthermore, given the higher incidence rates of emotion dysregulation and internalizing symptoms in girls versus boys, gender differences and gender invariance in directional model paths were also investigated. The following hypotheses were tested:

**Hypothesis 1:** There will be significant transactional associations between a latent construct of emotion dysregulation (indicated by multiple subscales) and symptoms of social anxiety and depression over time.

**Hypothesis 2:** Given evidence that some subscales of emotion dysregulation may be a stronger correlate of symptoms than others (e.g., Hambour et al., 2018), when each subscale of emotion dysregulation is tested, significant transactional associations will be found for each of the more active aspects of emotion dysregulation (lack of emotional clarity, nonacceptance of emotional responses, impulse control difficulties, limited access to regulation strategies, and difficulties engaging in goal-directed behavior when aroused), and less support will be found for the influence of any passive aspect of emotion dysregulation (lack of emotional awareness) on symptoms.

**Hypothesis 3:** Girls will report more emotion dysregulation and symptoms than boys. However, given the current lack of evidence regarding gender invariance (or moderation) of the transactional associations

between emotion dysregulation and symptoms over time, no specific hypotheses were made.

## Method

### *Participants and Procedure*

At T1 of this longitudinal study, 391 students participated (56% female). Students were attending one of three urban Australian schools. At T1, students were aged 10 to 14 years ( $\bar{X}_{\text{age}} = 12.0, SD = 0.90$ ) and were in Grades 6 (27%), 7 (30%), or 8 (43%). Participants reported their ethnicity as White/Caucasian (79%), Asian (15%), Aboriginal, Torres Strait Islander, or Pacific Islander (1%), or from a variety of other ethnic/racial backgrounds (5%).

Study approval was obtained from the university Human Research Ethics Committee prior to seeking principals' approvals to participate in this study. All contacted schools agreed to participate and students were provided with a detailed parental consent package to take home and return to school, which included a short survey for parents to report their demographic information. To encourage the return of parental consent forms (irrespective of parental consent to participate), a party was awarded to the class within each grade, at each school, that returned the most consent forms. A minority of parents (16%) actively declined participation, and a further 42% of students failed to return consent forms. Participating students received a small gift (e.g., novelty pen, sticker) after each survey was completed. Students were also able to decline participation on the day of the survey, although this was without incidence.

Data were collected 4 times from students, but data from the first two assessments were collapsed creating three time points for analyses in this study, with one assessment in each of 3 different years. To be more specific, students completed the first two assessments about 6 months apart in the same year. In these assessment sessions, a planned missingness design (Little & Rhemtulla, 2013) was used so that 50% of students completed the social anxiety measure first and completed the depression measure 6 months later. This pattern was reversed for the other 50% of students. Students completed the full emotion dysregulation measure both times. Planned missingness was used to reduce the time burden on schools and students, given the number of other measures also completed by students and the evidence that social anxiety and depression scores would be highly correlated and could be accurately estimated using new methods of estimating missing data (Graham, 2009; J. L. Schafer & Graham, 2002). For the current study, however, rather than estimating missing data, we have collapsed the data across these two

assessments by drawing on all measures and calculating the average of the emotion dysregulation scores so that all students have only one set of data. We did this to create one assessment per student, per year. We refer to this merged data as T1 data in the present study. Because this meant that we needed to account for the different timing of symptom measures for students, we also created a dichotomous variable indicating whether students first completed social anxiety items or depressive symptom items and included this indicator as a control variable in all SEMs. This variable was not associated with any measures and did not change the study results in any way. Thus, it was not included in the results reported here. T2 data were collected about 9 months after the last assessment for T1. Time 3 (T3) data were collected about 18 months after T2. All measures were completed by all students at T2 and T3.

The first, second, and third assessments were conducted in students' regular classrooms, whereas in the final assessment, students were contacted individually and completed an online survey or completed the survey via mail.

## Measures

**Social anxiety symptoms.** The Social Anxiety Scale for Adolescents (SAS-A; La Greca & Lopez, 1998) was used to assess social anxiety symptoms. The SAS-A consists of 18 self-report items, adapted from the Social Anxiety Scale for Children–Revised (La Greca & Stone, 1993) for an adolescent age group. Each item (e.g., “I worry what others say about me”) has five response options, ranging from 1 (*not true*) to 5 (*very true*). The SAS-A contains three subscales: Fear of Negative Evaluation (FNE; eight items, Cronbach's  $\alpha$  ranged from .88-.91 across the four waves), Social Avoidance and Distress in New Situations (SAD-New; six items, Cronbach's  $\alpha$  ranged from .83-.91), and General Social Avoidance and Distress (SAD-General; four items, Cronbach's  $\alpha$  ranged from .75-.81). Items on each subscale were averaged for use as measured indicators of a latent variable of social anxiety. At T1, there were 81 students (21%) identified to be within the clinical range for symptoms of social anxiety (La Greca, 1999).

**Depressive symptoms.** The Short Mood and Feelings Questionnaire (SMFQ; Angold et al., 1995) was used to assess depressive symptoms. The SMFQ is a 13-item self-report questionnaire. All items are negatively worded (e.g., “I felt lonely”) and had response options ranging from 1 (*not true*) to 5 (*very true*). Calculating the mean of the 13-items formed the total score, with higher scores indicating greater depressive symptoms. In previous studies, the child version of the SMFQ has been found to have good internal



reliability ( $\alpha = .85$ ; Angold et al., 1995) and reasonable criterion validity (area under the curve [AUC] = .73; Rhew et al., 2010). In the present study, Cronbach's  $\alpha$  at each wave ranged from .92 to .96. At T1, there were 112 (29%) students identified to be within the clinical range for symptoms of depression (Angold et al., 1995).

*Emotion dysregulation.* The Difficulty in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) was originally developed to assess emotion dysregulation in adults and has since been validated for use with adolescents (Neumann et al., 2010). The DERS consists of 36 items (e.g., "I am attentive to my feelings") that were rated on a 5-point scale ranging from 1 (*not true*) to 5 (*very true*). The DERS contains six subscales: Lack of Emotional Awareness (six items, Cronbach's  $\alpha$  ranged from .78-.83), Lack of Emotional Clarity (five items, Cronbach's  $\alpha$  ranged from .74-.88), Nonacceptance of Emotional Responses (six items, Cronbach's  $\alpha$  ranged from .84-.91), Impulse Control Difficulties (six items, Cronbach's  $\alpha$  ranged from .82-.87), Limited Access to Emotion Regulation Strategies (eight items, Cronbach's  $\alpha$  ranged from .88-.91), and Difficulties Engaging in Goal-Directed Behavior (five items, Cronbach's  $\alpha$  ranged from .82-.87). Several items were reversed coded before items were averaged to form subscale scores for use as measured indicators of a latent variable of emotion dysregulation.

### Overview of Analyses

Other than the planned missingness of measures (see "Procedures" section above), all 391 students completed a questionnaire at T1, T2, or T3. Overall, 370 of the 391 students (95%) completed the questionnaires at T1, 328 (84%) completed questionnaires at T2, and 263 (67%) completed questionnaires at T3. To maintain all 391 participants in the preliminary analyses (e.g., correlations), we used multiple imputation (15 imputed datasets) and the pooled results are reported below.

The primary analyses included fitting structural equation models (SEMs) with AMOS using maximum likelihood estimation. We used full information maximum likelihood (FIML) in the SEM to estimate missing data and maintain all 391 participants in all analyses. Model fit was examined with regularly used fit indices, including the  $\chi^2$  test statistic and the comparative fit index (CFI; Bentler & Bonett, 1980). The CFI is believed to be more acceptable as it approaches values of 1, and values over .90 are considered to be representative of good model fit. An estimate of error due to the approximate fit of the model was also assessed using the root mean square

error approximation (RMSEA; Browne & Cudeck, 1992). RMSEA is considered a good fit if values are below .05, a fair fit if values are between .05 and .08, and a mediocre fit if values are between .08 and .10 (Kaplan, 2000; Zimmer-Gembeck, Hunter, & Pronk, 2007).

A sequential process was used to conduct SEM (Kaplan, 2000). The first step within this process was to determine the invariance of the measurement model across the three time points. In this model, the total SMFQ score was used as an indicator of depressive symptoms. For social anxiety symptoms, we used three indicators of FNE, SAD-New, and SAD-General. For emotion dysregulation, lack of emotional awareness, lack of emotional clarity, non-acceptance of emotional responses, impulse control difficulties, limited access to emotion regulation strategies, and difficulties engaging in goal-directed behavior were used as indicators. The covariances between these latent variables were all freed within each time point.

The second step in the sequential process was to fit the structural model to test Hypothesis 1, which incorporated all aspects of the measurement model, as well as the transactional associations between T1 emotion dysregulation with symptoms of T2 depression and T2 social anxiety, and, in turn, the associations between T2 depression and T2 social anxiety symptoms with T3 emotion dysregulation. Similarly, the transactional associations between T1 depression and T1 social anxiety symptoms with emotion dysregulation at T2, and, in turn, the associations of T2 emotion dysregulation with symptoms of T3 depression and T3 social anxiety were also included in this model. Additional SEMs were fitted to test Hypothesis 2, which involved fitting a SEM for each Emotion Dysregulation subscale separate from the others. To test Hypothesis 3, *t* tests were used to compare boys and girls on all measures, and two-group (boy/girl) models were fit to thoroughly test gender invariance. This involved first examining measurement invariance in boys compared with girls and gender invariance in within-time covariances, before testing gender invariance in directional pathways linking emotion dysregulation and symptoms over time.

## **Results**

### *Correlations and Sex Differences*

As can be seen in Table 1, social anxiety and depressive symptoms and emotion dysregulation at T1 were positively and significantly associated with symptoms and emotion dysregulation at T2 and T3. Pooled means, *SDs*, and independent-groups *t* tests comparing boys and girls are reported in Table 2. Supporting Hypothesis 3, sex differences were identified for all variables

**Table 1.** Correlation Between Social Anxiety, Depressive Symptoms, and Emotion Dysregulation (*N* = 391).

Measure	1	2	3	4	5	6	7	8
1 T1 Social anxiety								
2 T1 Depressive symptoms	.39							
3 T1 Emotion dysregulation	.57	.63						
4 T2 Social anxiety	.74	.47	.63					
5 T2 Depressive symptoms	.46	.65	.62	.65				
6 T2 Emotion dysregulation	.45	.53	.76	.60	.73			
7 T3 Social anxiety	.56	.23	.38	.62	.41	.43		
8 T3 Depressive symptoms	.38	.44	.46	.45	.50	.49	.62	
9 T3 Emotion dysregulation	.41	.40	.58	.48	.49	.62	.64	.76

Note. All *p* < .01.

**Table 2.** Means and Standard Deviations of All Measures and Comparison Between Boys and Girls (*N* = 391).

Measure	All participants $\bar{X}$ (SD)	Boys $\bar{X}$ (SD) <i>n</i> = 174	Girls $\bar{X}$ (SD) <i>n</i> = 217	Sex comparison <i>t</i> (1, 389)
T1 Social anxiety	2.20 (0.77)	2.01 (0.70)	2.35 (0.80)	-4.15***
T1 Depressive symptoms	1.70 (0.74)	1.56 (0.56)	1.82 (0.86)	-3.32**
T1 Emotion dysregulation	2.27 (0.52)	2.21 (0.46)	2.32 (0.56)	-2.01*
T2 Social anxiety	2.24 (0.84)	2.05 (0.75)	2.41 (0.87)	-4.05***
T2 Depressive symptoms	1.72 (0.87)	1.58 (0.75)	1.84 (0.94)	-2.82**
T2 Emotion dysregulation	2.36 (0.66)	2.24 (0.60)	2.45 (0.70)	-2.92**
T3 Social anxiety	2.47 (0.88)	2.33 (0.86)	2.59 (0.89)	-2.58**
T3 Depressive symptoms	2.00 (1.01)	1.79 (0.94)	2.18 (1.08)	-3.53***
T3 Emotion dysregulation	2.54 (0.66)	2.42 (0.59)	2.65 (0.70)	-2.93**

\**p* < .05. \*\**p* < .01. \*\*\**p* < .001.

with girls, relative to boys, reporting significantly more social anxiety, depressive symptoms, and emotion dysregulation.

### Measurement Invariance

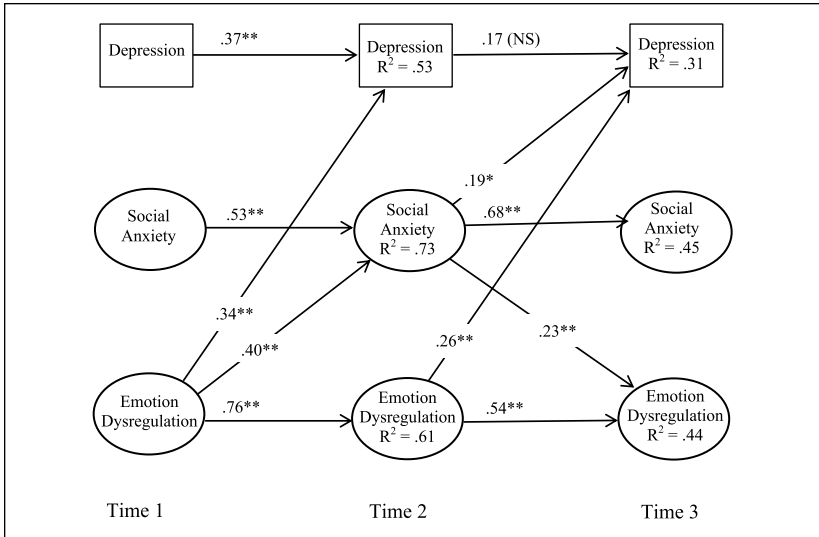
Prior to testing structural models, we fit preliminary models to test for measurement invariance over the 3 times of assessment. In the first model, all factor loadings were .65 or above with the exception of lack of emotional

awareness, which did not load significantly onto the latent variable of emotion dysregulation at any time point. Because of this, lack of emotional awareness was subsequently examined as a separate factor and the model was again fit. In this second model, there was evidence for measurement invariance of the latent variables of emotion dysregulation and social anxiety. More specifically, the fits of the model when we (a) fixed all loadings to equality over time and (b) freed the loadings to vary over time were not significantly different from each other,  $\chi^2$ -difference (12) = 17.5,  $p > .05$ .

### ***Structural Paths: Associations Between Symptoms and Emotion Dysregulation***

Given the low loadings of emotional awareness on the latent emotion dysregulation variables, we next fit two structural models, one specifying a model with lack of emotional awareness as a separate exogenous variable and one without lack of emotional awareness included. When lack of emotional awareness was included as a separate exogenous variable, the model fit was adequate,  $\chi^2(359, N = 391) = 981.66, p < .001, CFI = .91, RMSEA = .067$  (90% confidence interval [CI] = [.062, .072]),  $p < .001$ . However, lack of emotional awareness had no associations with symptoms either within waves or over time, with associations ranging from  $-.04$  to  $.14$  (all  $p > .05$ ). Thus, lack of emotional awareness was trimmed from the model and another model was fit. The final trimmed model had an adequate fit to the data on most indicators,  $\chi^2(298, N = 391) = 756.5, p < .001, CFI = .93, RMSEA = .063$  (90% CI = [.057, .068]),  $p < .001$ .

As shown in Figure 1, four of the cross-lag paths between symptoms and emotion dysregulation were significantly larger than 0, suggesting an escalating cycle of emotion dysregulation and symptoms of social anxiety and depression over time. First, emotion dysregulation predicted later symptoms. In particular, both symptoms of social anxiety and depression at T2 were predicted by T1 emotion dysregulation. Also, depressive symptom level at T3 was predicted by T2 emotion dysregulation. In turn, symptoms also predicted increasing emotion dysregulation between T2 and T3, with social anxiety symptom level predicting increased emotion dysregulation at T3 relative to T2. In addition, although freed in the analyses, only one of the prospective paths from symptoms of depression to social anxiety and vice versa was significant, with T2 social anxiety symptoms significantly associated with increased depressive symptoms at T3. This suggests a limited influence of one internalizing disorder on the other over time once emotion dysregulation was adjusted.



**Figure 1.** Results of testing the transactional associations between depression and social anxiety symptoms and emotion dysregulation over time.

Note. All transaction paths were freed in this model, but only significant associations are shown here. Covariance between measures within time points, and nonsignificant paths between measures across time are not shown here, but were freed in the model,  $\chi^2(298, N = 391) = 756.5, p < .001, CFI = .93, RMSEA = .063$  (90% CI = [.057, .068]),  $p < .001$ . NS = not significant; CFI = comparative fit index; RMSEA = root mean square error of approximation; CI = confidence interval.

\* $p < .05$ . \*\* $p < .01$ .

### Models for Each Emotion Dysregulation Subscale

To test Hypothesis 2, we fit six additional models. In each model, we replaced the latent variable of emotion dysregulation with a single subscale from the emotion dysregulation measure (i.e., either lack of emotional awareness, lack of emotional clarity, low goals, impulsivity, nonacceptance, or lack of strategies). Overall, there were different patterns identified between each of the individual subcomponents of emotion dysregulation and symptoms of social anxiety and depression at T1 and T2 compared with T2 and T3, with the weakest findings for lack of emotional awareness. Specifically, regarding emotion dysregulation as a predictor of later symptoms, emotion dysregulation had widespread influence on later symptoms, especially between T1 and T2, but was also important for predicting depressive symptoms at T3. In particular, the majority of subcomponents of emotion dysregulation at T1 were associated with later (T2) symptoms of depression and social anxiety,

with path estimates ranging from .11 to .28, all  $p < .05$ , with the exception of the association between T1 lack of emotional clarity and T2 social anxiety (path estimate = .07,  $p = .14$ ). There was also no significant associations between T1 lack of emotional awareness and T2 symptoms of depression and social anxiety, with path estimates of .02 and  $-.03$ ,  $p = .37$  and  $.68$ , respectively. Moreover, two subscales of emotion dysregulation at T2 (Nonacceptance and Lack of Strategies) were associated with symptoms of depressive symptoms at T3, with path estimates of .26 and .17,  $ps < .001$  and  $< .05$ , respectively. Thus, T2 lack of emotional awareness, lack of emotional clarity, low goals, and impulsivity were not significantly associated with T3 depressive symptoms, with path estimates ranging from .05 to .13, and  $p$  from .054 to .36. There were also no significant associations found between any of the subcomponents of emotion dysregulation at T2 with symptoms of social anxiety at T3, with path estimates ranging from .01 to .10, and  $p$  from .15 to .90.

Regarding symptoms as predictors of later emotion dysregulation, this pattern was also supported, showing evidence for the significant role of earlier depressive symptoms in the escalation of emotion dysregulation between T1 and T2 and the significant role of earlier social anxiety symptoms in the escalation of emotion dysregulation between T2 and T3. In particular, T1 depressive symptom level was associated with the majority of subcomponents of emotion dysregulation at T2 (path estimates ranging from .09-.17,  $p < .05$ ), with the exception of the associations between T1 depressive symptoms and T2 lack of emotional awareness and low goals (path estimates of .05 and .09,  $p = .31$  and  $.07$ , respectively). Whereas, there were no significant associations identified between T1 symptoms of social anxiety and any subcomponent of emotion dysregulation at T2, with path estimates ranging from .01 to .10, and  $p$  from .06 to .92. Between T2 and T3, T2 social anxiety symptom level was associated with the majority of subcomponents of emotion dysregulation at T3 (path estimates ranging from .17-.32,  $p < .05$ ), with the exception of the associations between T2 social anxiety symptoms and T3 lack of emotional awareness and low goals (path estimates =  $-.02$  and  $.16$ ,  $ps = .79$  and  $.06$ , respectively). Whereas, no significant associations were identified between T2 depressive symptoms and any subcomponent of emotion dysregulation at T3 (path estimates ranging from  $-.16$  to  $.05$ , and  $p$  from  $.06$ -.99), with the exception of the association between T2 depressive symptoms and T3 lack of emotional awareness, path estimate =  $.18$ ,  $p < .05$ .

### ***Gender Invariance: Sex Moderation of the Temporal Paths***

Prior to testing for gender invariance in model paths, we confirmed measurement invariance over time in both boys and girls. When we compared models

with loadings for emotion dysregulation and social anxiety fixed to measurement invariance over time to models with these loadings free to vary across time, the  $\chi^2$ -difference tests comparing model fits were not significant,  $\chi^2$ -difference (12) = 15.4,  $p > .05$  for boys and  $\chi^2$ -difference (12) = 7.9 for girls. Thus, measurement was invariant over time for both boys and girls.

Building on this measurement invariant structure, two-group models were fit to test for gender invariance (Hypothesis 3). We began by fitting a model with all paths fixed to gender equality, and the model had an adequate fit to the data,  $\chi^2(641, N = 391) = 1,226.8, p < .001$ ; CFI = .91, RMSEA = .048 (90% CI = [.044, .053]),  $p = .73$ . Next, to assess whether correlations within times of measurement differed for boys and girls, we fit a model where we allowed the within-wave correlations to differ for boys and girls. This model also had an adequate fit to the data,  $\chi^2(614, N = 391) = 1,164.0, p < .001$ ; CFI = .92, RMSEA = .048 (90% CI = [.044, .052]),  $p = .78$ . The difference between these two models was significant ( $p < .01$ ),  $\chi^2$ -difference (27) = 62.8, and further analyses fixing only one covariance at a time to gender equality indicated that the correlation between T1 emotion dysregulation and T1 depressive symptoms was stronger for girls (.77) in comparison with boys (.53), and that the correlation between T3 emotion dysregulation and T3 symptoms of social anxiety was stronger for boys (.77) in comparison with girls (.51).

In a final model, we allowed only the cross-lag paths to differ for boys and girls (i.e., keeping all other models paths fixed to gender equality). This model also had an adequate fit to the data,  $\chi^2(629, N = 387) = 1,205.9, p < .001$ ; CFI = .91, RMSEA = .049 (90% CI = [.044, .053]),  $p < .01$ . The difference in the fit of this model compared to the one with all paths fixed was not significant ( $p > .05$ ),  $\chi^2$ -difference (12) = 20.9,  $p > .05$ . Thus, overall, these results of all the models we tested suggest that there were two gender differences in correlations between measures *within* time points, but there was no evidence that structural paths between symptoms and emotion dysregulation over time differed between boys and girls. Thus, there was gender invariance in the transactional associations between emotion dysregulation and symptoms over time.

## Discussion

The primary purpose of the present longitudinal study was to investigate the transactional associations between emotion dysregulation and internalizing symptoms of social anxiety and depression in adolescents. Broadly, the findings from this study provide evidence to suggest that there is an unfolding of emotional problems over time among adolescents as they progress from the

early to middle adolescent years. Earlier emotion dysregulation was found to be associated with later symptoms of social anxiety and depression, and earlier symptoms of social anxiety and depression were, in turn, associated with increasing emotion dysregulation. More specifically, there were three primary findings from this study.

### ***Structural Paths: Associations Between Symptoms and Emotion Dysregulation***

The first primary finding was in support of Hypothesis 1. Higher levels of earlier emotion dysregulation were found to predict relative increases in social anxiety and depressive symptoms over time, even after controlling for adolescents' baseline internalizing symptoms. However, there were also differences in the identified associations depending on the time period under examination, with T1 emotion dysregulation predicting increases in both adolescents' social anxiety and depressive symptoms between T1 and T2, and T2 emotion dysregulation predicting increases in adolescents' depressive, but not social anxiety, symptoms from T2 to T3. These findings are consistent with previous research on children (e.g., Feng et al., 2009; Folk et al., 2014), and partially consistent with McLaughlin et al. (2011), who found that earlier emotion dysregulation predicted increases in early adolescents' anxiety, but not depressive symptoms. Furthermore, and consistent with other research on the trajectories of internalizing symptoms during adolescence (Beesdo et al., 2007; McLaughlin & King, 2015), earlier social anxiety symptoms (i.e., T2) were also found to predict relative increases in later depressive symptoms (i.e., T3). Thus, when taken together, these findings suggest that emotion dysregulation may be an important early predictor of adolescent internalizing symptoms. It may also be the case that once internalizing symptoms have developed, they may also lead to further difficulties with regulating ones' emotional experience, as well as an ongoing escalation of depressive symptoms into middle adolescence.

In further support of Hypothesis 1, higher levels of earlier social anxiety symptoms (i.e., T2) were also found to predict relative increases in emotion dysregulation (by T3), even after earlier emotion dysregulation was controlled. However, there were no significant associations identified between earlier depressive symptoms and later emotion dysregulation (using the latent construct of emotion dysregulation) at any time point. These findings are in contrast to previous work conducted by McLaughlin et al. (2009; McLaughlin et al., 2011), which found that after controlling for baseline emotion dysregulation, there were no significant associations between internalizing symptoms at T1 and emotion dysregulation at T2.



There are many methodological differences that may provide possible reasons for the disagreement in findings across studies. These include the components of dysregulation that were assessed in each study (and the measure used to assess emotion dysregulation), the age of participants, and the lag time between assessments. In the present study, a measure of emotion dysregulation was used that incorporated five emotion dysregulation factors (i.e., lack of emotional clarity, nonacceptance of emotional responses, impulse control difficulties, limited access to emotion regulation strategies, and difficulties engaging in goal-directed behavior). Whereas, McLaughlin et al. (2011) measured four different factors of emotion dysregulation (e.g., lack of emotional understanding, dysregulated expression of sadness and anger, and rumination) and used each of these emotion dysregulation factors as indicators of a latent emotion dysregulation construct. Furthermore, while McLaughlin et al. identified that all of their individual emotion dysregulation measures had significant cross-sectional relationships with internalizing symptoms (i.e., depression and anxiety), post hoc linear regression analysis identified that their measure of lack of emotional understanding did not significantly correlate with later depression symptoms. Thus, it is possible that this lack of significant association between earlier emotional understanding and later depressive symptoms may have contributed to this study's inability to demonstrate a significant relationship between the latent factor of emotion dysregulation and symptoms of depression over time.

It is also possible that the older age range and longer lag time between assessments in the current study may provide further possible explanations for the discrepancy between the results of these studies. For instance, it is well known that adolescents typically become better able to regulate their emotions and/or cope with stressors as they progress throughout the developmental period of adolescence (Gross, 2013; Skinner & Zimmer-Gembeck, 2016). Thus, given that the current study followed early adolescents (aged 9-14 years at T1) over a 3-year period (aged 12-17 years at T3), it is not surprising that the unique contributions of emotion dysregulation on internalizing symptoms, and vice versa, were more widespread between T1 and T2 but more limited between T2 and T3. Furthermore, it is also possible that the shorter lag times used by McLaughlin et al. (e.g., three assessments over a 7-month period) may have been less sensitive to detect change over time. Nevertheless, the findings from the present study are consistent with some other previous longitudinal studies on children and preadolescents (e.g., Feng et al., 2009; Folk et al., 2014), as well as other cross-sectional research on adolescence (e.g., Bender et al., 2012; Eastabrook et al., 2014; Lanteigne et al., 2014; Loughheed & Hollenstein, 2012; Mathews et al., 2014). Overall, the current research is the first study, to our knowledge, to demonstrate a

bidirectional relationship between emotion dysregulation and symptoms of social anxiety and depression during the developmental periods of early and middle adolescence.

### *Follow-Up Models for Each Emotion Dysregulation Subscale*

In the present study, the second key finding was that individual and *active* components of emotion dysregulation have unique effects on increases in both social anxiety and depressive symptoms over time. Moreover, the findings in total suggest that the influence of individual and *active* components of emotion dysregulation on internalizing symptoms may become more localized or specific as adolescents get older. For example, in partial support of Hypothesis 2, models of each subscale of emotion dysregulation suggested that earlier (i.e., T1) nonacceptance of emotional responses, impulse control difficulties, limited access to emotion regulation strategies, and difficulties engaging in goal-directed behavior, but not lack of emotional clarity, were significantly associated with increases in adolescents' social anxiety during early adolescence (i.e., T2), but T2 emotion dysregulation had no significant association with social anxiety symptoms at T3. Whereas, all five earlier emotion regulation difficulties (i.e., T1) were significantly associated with increases in later depressive symptoms during early adolescence (i.e., T2), but only an earlier (i.e., T2) nonacceptance of emotional responses and limited access to emotion regulation strategies were significantly associated with later depressive symptoms in middle adolescence (i.e., T3).

Converse of the above, social anxiety and depression symptoms were also found to have unique effects on individual and *active* components of emotion dysregulation over time. For instance, our models of each subscale of emotion dysregulation suggested that earlier depressive (i.e., T1), but not social anxiety, symptoms predicted relative increases in adolescents' lack of emotional clarity, impulse control difficulties, nonacceptance of emotional responses, and limited access to emotion regulation strategies, but not difficulties engaging in goal-directed behavior during early adolescence (i.e., T2). Thus, when examined at an individual component level, rather than as a latent factor, earlier symptoms of depression predicted increases in unique difficulties in early adolescents' emotion regulation. Whereas, this association appears to shift during middle adolescence, and it was earlier social anxiety (i.e., T2), not depressive, symptoms that predicted increases in these same emotion regulation difficulties at a later time point (i.e., T3). Hence, the current study provides preliminary evidence to suggest that there may be differential trajectories between earlier emotion dysregulation and symptoms of social anxiety and depression, as well as between earlier symptoms of social

anxiety and depression and emotion dysregulation. Future research in this area would therefore benefit from focusing on investigating and understanding the different patterns of these constructs at an individual person level, with a focus on answering the question of how patterns of symptoms and dysregulation might unfold differently for different youth, while also attending to potential differences by age and gender.

### *Gender Invariance: Sex Moderation of the Temporal Paths*

The third finding to highlight, concerns adolescents' gender. In support of Hypothesis 3, and also consistent with previous research (e.g., Bender et al., 2012; Neumann et al., 2010), girls and boys differed in their cross-sectional levels of emotion dysregulation and symptoms of social anxiety and depression (e.g., girls identifying more difficulties in emotion regulation and internalizing symptoms). However, the associations between emotion dysregulation and symptoms over time did not differ between boys and girls. Thus, while, on average, girls report higher levels of internalizing symptoms and greater difficulties regulating their emotions (to the extent that any adolescent experiences internalizing symptoms and emotion dysregulation), the pathways to internalizing disorders and emotion dysregulation (at least in the developmental transition from early to middle adolescence) are the same for boys and girls. Future research should attempt to further clarify the role of gender differences on the longitudinal associations between emotion dysregulation and symptoms of social anxiety and depression in adolescents at different ages and developmental transitions (e.g., adolescence to adulthood).

### *Study Limitations and Future Directions*

When interpreting the results of this study, it is important to note that this study had some limitations. First, there were differences in the lag time across assessments (e.g., 6-12 months between T1 and T2, and 24 months between T2 and T3), which have the potential to explain some of the differences in results between time points. For instance, it is possible that some of the differences identified between T1 and T2, and T2 and T3, may be due to age and/or other developmental factors (e.g., pubertal and/or cognitive maturation) that were not specifically measured in this study. Nevertheless, measurement invariance was established for each of the measures in this study, indicating that the same constructs were being measured across time points irrespective of assessment lag times.

Moreover, a planned missingness design for measures of adolescents' social anxiety and depressive symptoms was used to reduce the time commitment

from schools, given the known moderate (sometimes high) correlation between social anxiety and depressive symptoms. Although we expected that a shorter survey would result in better quality data from all participants, estimation of this missing data proved too unstable. Thus, we choose to reduce the data to 3 times of measurement and to control for timing of measurement in analyses presented here.

Self-report may also be a limitation of the present study, given that we relied on adolescent self-report to measure all constructs. This method of administration was used, as it is commonly accepted in the literature that adolescents are the best reporters of their internalizing symptoms and experiences (e.g., Costello & Angold, 1995). However, it is possible that the reliance on self-report data may have resulted in shared method variance, potentially inflating the identified relationships between measures. Follow-up studies that include data from multiple informants, such as teacher, parent, and/or peer reports, could be useful in determining the extent to which shared method variance influenced the current findings.

Finally, lack of emotional awareness did not load onto the latent variable of emotion dysregulation, which may suggest some revision to the scoring procedure for the DERS (Gratz & Roemer, 2004), at least when used with adolescents. This deserves further research attention. Indeed, there have been previous factor analytic studies that have highlighted the lack of relationships of other Dysregulation subscales with the Lack of Emotional Awareness subscale (Fowler et al., 2014; Hambour et al., 2018).

## **Conclusion**

The findings of the present study highlight the relevance of emotion dysregulation in escalating symptoms of social anxiety and depression during early adolescence. Furthermore, and to the best of our knowledge, the current longitudinal study also represents the first research to formally investigate and demonstrate a bidirectional, longitudinal relationship between earlier emotion dysregulation (including all of the individual emotion regulation difficulties as operationalized by Gratz & Roemer, 2004) and later symptoms of social anxiety and depression in adolescence, and, in turn, earlier symptoms of social anxiety and depression with later emotion dysregulation.

While it is important to continue this line of research to better understand how specific emotion regulation difficulties exacerbate adolescents' internalizing symptoms, the current study provides evidence to suggest that the early identification of difficulties in emotion regulation may be particularly important during early adolescence, as emotion dysregulation appears to predict internalizing symptoms. Whereas once these problems have developed,

internalizing symptoms further increase difficulties in emotion regulation, which in turn exacerbate depressive symptoms. Thus, the current findings provide support for interventions focused on fostering and enhancing early adolescents' emotion regulation abilities, as well as targeting specific emotion regulation difficulties in middle adolescence, to reduce social anxiety and depression within this demographic.

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